To: Osterhoudt, Darrell[dosterhoudt@asdwa.org]
Cc: Burneson, Eric[Burneson.Eric@epa.gov]; Lopez-Carbo, Maria[Lopez-Carbo.Maria@epa.gov]; Carroll, Gregory[Carroll.Gregory@epa.gov]; Christ, Lisa[Christ.Lisa@epa.gov]; Hautman, Dan[Hautman.Dan@epa.gov]; Huff, Lisa[Huff.Lisa@epa.gov]; Rodgers-Jenkins, Crystal[Rodgers-Jenkins.Crystal@epa.gov]; Albert, Ryan[Albert.Ryan@epa.gov]
From: Oshida, Phil
Sent: Wed 2/10/2016 2:27:16 PM
Subject: SRMD Comments on the Draft ASDWA Member Meeting Agenda

February 10, 2016

Good morning Darrell.

Thanks for the opportunity to review and comment on the draft ASDWA Member Meeting Agenda.

Our comments:

•□□□□□□□ The "Six Year Review issues" will be covered in the "SRMD projects/Rule Development Update". It does not need a separate heading on the agenda.

•□□□□□□□ We will address the PFOA/PFOS activities in the "SRMD Projects/Rule Development Update".

•□□□□□□□ Please revise the Cyanotoxin title to read "Cyanotoxin and Legionella Activities".

•□□□□□□□ We are good with the remainder of the "Regulatory Tool & Rule Development" agenda.

•□□□□□□□ Per Maria's suggestion, we support a topic on Flint, MI. It should have a DWPD (Maria Lopez-Carbo) lead and SRMD will provide assistance, as necessary. This is likely to have an implementation focus.

Thanks.

-Phil-

Phil Oshida S Associate Director S Standards and Risk Management Division S Office of Ground Water and Drinking Water S Office of Water S U.S. EPA S 202.564.6594

To: Carroll, Gregory[Carroll.Gregory@epa.gov]
From: BNA Highlights
Sent: Tue 3/29/2016 3:20:04 AM
Subject: Mar. 29 -- BNA, Inc. Daily Environment Report

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Tuesday, March 29, 2016 · Number 60

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NEWS

Air Pollution
California Adopts Diisocyanate Airborne Exposure Levels
California's Office of Environmental Health Hazard Assessment adopted airborne concentrations of toluene diisocyanate and methylene diphenyl diisocyanate state regulators will use in assessing exposure risks for the two toxic...

Air Pollution
Cincinnati Air Standards Litigation Denied Review
The U.S. Supreme Court will not review a lower court decision that vacated the Environment Protection Agency's redesignation in 2011 of Greater Cincinnati, Ohio, as meeting national air standards for particulate matter (Ohio v. Sierra...

Air Pollution
Litigation Tracker: Ozone Briefing to Begin, Refinery Litigation Halted
Briefing on the legality of the Environmental Protection Agency's national ozone standards issued in 2015 will begin in April and continue through the summer of 2016....

Air Pollution
OMB Reviewing Ozone, Particulate Analysis Guidance
The White House Office of Management and Budget is reviewing a guidance document on permitting issues under ozone and fine particulate matter air quality standards, according to the office's website. The guidance document (RIN 2060-ZA24),...

Climate Regulation
Allowance Allocations Key for Clean Power Plan: Paper
How and to whom states allocate carbon dioxide emissions allowances as part of any mass-based compliance strategy for the Environmental Protection Agency's Clean Power Plan will depend on

the goals state regulators hope to achieve,...

Climate Regulation
EPA Defends Generation Shifting in Clean Power Plan
A Clean Air Act requirement that the Environmental Protection Agency determine the "best system" for cutting carbon dioxide emissions is broad enough to encompass the reductions anticipated in the Clean Power Plan, the agency argued...

Drinking Water
EPA to Release Nonbinding Heath Advisory for PFOA
The Environmental Protection Agency is poised to release a nonbinding health advisory for perfluorooctanoic acid this spring, EPA Region III Administrator Shawn M. Garvin said in a March 25 letter in response to pressure to act on West Virginia...

Drinking Water
FEMA Extends Emergency Status in Flint Until August
The Federal Emergency Management Agency has extended its emergency declaration for Flint, Mich., through Aug. 14 in response to a March 14 request by the state. The agency said in a March 25 letter released by Michigan Gov. Rick Snyder (R) that...

Drinking Water
Halliburton Energy Consent Order Bars Suit Over Well Water
A consent order between Oklahoma and Halliburton Energy Services authorizing the state to address a groundwater plume bars a lawsuit to expedite the cleanup of well-water contamination, the U.S. District Court for the Western District of...

Endangered Species
Federal Protections Defended for Sage Grouse
The Idaho governor and state Legislature lack legal standing to sue over federal protections for the greater sage grouse because the plaintiffs cannot claim any injury until federal agencies get down to the details of blocking projects or...

EPA
House Science Panel Presses McCabe Again on Air Travel
Republicans on the House Science, Space and Technology Committee are reiterating demands for information on the travel habits of the Environmental Protection Agency's top air official, warning they may subpoena the records....

Forests
High Court Refuses Tongass Roadless Case Review
The U.S. Supreme Court refused March 28 to review an appeals court decision affirming tough restrictions on road construction in the Tongass National Forest in Alaska (Alaska v. Organized Vill. of Kake, U.S., No. 15-467, 3/28/16). The Roadless...

Hazardous Waste
Connecticut Companies Settle on Alleged PCB Violations
Two Connecticut companies have settled claims by the Environmental Protection Agency that they violated federal laws on toxic substances in their handling of waste contaminated by polychlorinated biphenyls, the agency announced March...

Hydraulic Fracturing
Quake Outlook Includes Human-Induced Temblors for First Time
The U.S. agency responsible for monitoring earthquakes has for the first time issued a short-term seismic forecast that includes both natural and human-induced risks. ...

International Issues

Canada Builders Get Environmental Details for Cement

The Cement Association of Canada voluntarily registered an environmental product declaration for general-use and portland-limestone cement, Canada's independent industrial standards organization, the CSA Group, said March 23....

Pesticides

EPA Inspector General to Look Into 'Superweeds'

The Environmental Protection Agency's inspector general is launching an evaluation of how effective the agency is at delaying or preventing the spreading of herbicide-resistant weeds....

Pipeline Safety

Industry Seeks Comment Extension for Pipeline Rule

The Transportation Department should extend the 60-day comment period for its recently proposed rule for new pipeline safety requirements, the American Petroleum Institute, American Gas Association and other trade groups wrote in a letter...

Water Pollution

South Dakota Senator Seeks Override of Buffer Bill Veto

South Dakota state Sen. Jim Peterson (D) is leading an effort to override a veto of legislation that would have allowed farmers to reclassify some of their property if they created buffer strips along the state's public waters....

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Comment Deadlines

MARCH 28 FEDERAL REGISTER

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NEWS

Air Pollution
Briefing in Ozone Litigation to Begin in April
Briefing on the legality of the Environmental Protection Agency's 2015 national ozone standards of 70 parts per billion will begin in April and conclude in September, according to court documents (Murray Energy Corp. v. EPA, D.C. Cir.,...

Air Pollution
Dallas Officials Want Portion of Volkswagen Settlement
Dallas-area transportation officials want to ensure that any settlement with Volkswagen AG over the company's use of illegal technology in its diesel vehicles provides funding to air quality projects in nonattainment areas that saw...

Air Pollution
Eighth Circuit Agrees to Rehear Minnesota Regional Haze Dispute
The Eighth U.S. Circuit Court of Appeals March 10 agreed to rehear a case challenging Minnesota's plan to reduce regional haze (Nat'l Parks Conservation Ass'n v. EPA, 8th Cir., No. 12-2910, 3/10/16)....

Air Pollution
House Rules to Consider Coal Refuse Emissions Bill
The House Rules Committee is set to consider a bill (H.R. 3797) that would alter the way coal refuse power plants are regulated under the Cross-State Air Pollution Rule and the Mercury and Air Toxics Standards. The legislation, introduced by...

Air Pollution
Los Angeles Area Emissions Trading Plan Challenged

Southern California air quality officials violated state law in approving industry-backed changes to a regional emissions trading program, environmental advocates said in a lawsuit filed late March 9 (Cmtys. for a Better Env't v. S....

Air Pollution
Ontario Reports Significant Smog Improvements
Ontario's air quality is significantly better than it was a decade ago, with notable decreases in smog-causing pollutants, the province's Ministry of the Environment and Climate Change said March 9. There were no smog advisories...

Asbestos
Tennessee Lawmakers Approve Asbestos Claim Limits
Plaintiffs filing lawsuits in Tennessee over harm from asbestos exposure may face new hurdles in seeking monetary relief, under a bill approved by the state Legislature March 10....

Biotechnology
India Cuts Monsanto Modified Cotton-Seed Royalty Fees by 70%
India cut royalties for genetically-modified cotton seeds, defying Monsanto Co., which has said such a move would cause it to reevaluate its business in the country, the largest grower of the fiber....

Brownfields
Wisconsin Expands Brownfield Liability Protections
Environmental remediation projects in Wisconsin targeting sediment underlying state waterways will be less costly, complicated and risky under a new law enacted by state lawmakers, an environmental attorney told Bloomberg BNA March 10....

Budget
House Appropriators Discuss Use of Equity Formula
The House Appropriations Committee is considering the use of an equity formula in upcoming appropriations bill language that would direct a portion of funds toward impoverished communities. ...

Chemical Testing
New Software Expected to Help Chemical Safety Reviews
An updated chemical data access and management system the European Chemical Industry Council expects to release within days will make it easier for companies to comply with regulations governing commercial chemicals, cosmetics and other...

Chesapeake Bay
Lawsuit: Problems Seen With Baywide Pollution Limits
The adoption of a cleanup plan for the Chesapeake Bay as a whole shouldn't relieve Maryland of its obligation to develop localized pollution-reduction strategies for other bay tributaries listed as impaired under the Clean Water Act,...

Climate Change
California Updates Plan for Extreme Weather, Sea Level Rise
The California Natural Resources Agency (CNRA) has updated its plans for addressing sea level rise and extreme weather events caused by climate change across a variety of sectors. ...

Climate Change
Canada to Reduce Oil, Gas Methane Emissions Up to 45 Percent
Canada will introduce regulations to reduce methane emissions from the oil and gas sector by 40 percent to 45 percent from 2012 levels by 2025 and harmonize the nation's regime with that of the

U.S., Environment and Climate Change Canada...

Climate Change
Seattle to Electrify Transport, Add Electric Vehicles
Seattle Mayor Ed Murray (D) announced a sweeping series of measures March 10 to reduce the city's carbon footprint by electrifying the city's transportation sector using the Northwest's abundant hydropower....

Climate Regulation
Clean Power Plan Opponents Turn Ginsburg's Words on EPA
Opponents of the Clean Power Plan are turning the words of a U.S. Supreme Court justice widely viewed as sympathetic to the Environmental Protection Agency into another weapon to attack the carbon dioxide standards for power plants....

Climate Regulation
EPA Moving With 'Urgency' on Methane From Existing Wells
The Environmental Protection Agency will solicit better methane emissions data from existing oil and gas wells as it pursues regulations for the industry long sought by environmental advocates, the White House announced March 10....

Climate Regulation
Inhofe: EPA Pressuring States on Power Plan Compliance
The Environmental Protection Agency is using the prospect of truncated deadlines to force states to continue compliance efforts for the Clean Power Plan even though the rule has been stayed by the U.S. Supreme Court, Sen. James Inhofe (R-Okla.)...

Coal Mining
Arch Coal to Abandon Montana Mine
Citing financial difficulty, Arch Coal said March 10 that it will no longer seek a mining permit for the Otter Creek coal reserves in southeastern Montana....

Coal Mining
Cantwell Urges Interior on Banning Self-Bonding in Mining
Intensifying her efforts to probe self-bonding in the mining industry, Sen. Maria Cantwell (D-Wash.) asked the Interior Department March 10 to consider prohibiting the practice....

Congress
Republicans See Window to Nullify Late-Term Obama Rules
A confluence of political factors could open the door for Republicans to successfully nullify Obama administration environmental regulations issued in the final months of his presidency, Senate lawmakers told Bloomberg BNA. ...

Drinking Water
EPA's McCarthy: Broader Justice, Water Debate Needed
While the Environmental Protection Agency is placing much of its efforts on helping Flint, Mich., residents deal with the ramifications of the city's lead-contaminated drinking water "disaster," the incident also has raised...

Drinking Water
Governors Call for Federal PFOA Contamination Guidance
The Environmental Protection Agency should establish national guidance addressing perfluorooctanoic acid (PFOA) contamination to help states assess the safety of their drinking water, three Democratic governors said in a March 10 letter....

Drinking Water
Ohio Seeks Faster Notification of Lead-in-Water Issues
Public utilities in Ohio should notify their customers within two days, not 30, if sampling shows lead contamination in the water supply, the state's environmental regulator is recommending. ...

Drinking Water
Unsafe Lead Levels in Water at 30 Newark Schools
Investigators are still searching for the source of high levels of lead found in the water at 30 schools in Newark, N.J., the New Jersey Department of Environmental Protection told Bloomberg BNA March 10....

Energy
Cornyn: McConnell May File For Cloture on Flint Bill
Senate Majority Leader Mitch McConnell (R-Ky.) may move to bring an energy bill and legislation that would provide aid to Flint, Mich., using procedural moves to sidestep senators who have been blocking the measure by using unanimous consent,...

Energy Efficiency
Government Seeks Companies' Help on Energy Goal
The nation's chief sustainability officer has called on companies such as Johnson Controls and AECOM to help meet the federal government's energy savings goal before the Obama administration ends. ...

Energy
U.S. Nuclear Industry Largely Safer After Fukushima Accident
The U.S. nuclear industry is safer after making fleet-wide improvements over the past five years since the meltdown of three nuclear reactors at the Fukushima Dai-Ichi nuclear plant in Japan, industry, regulators and safety advocates say....

Enforcement
EPA Limited in Next Steps on Flint Water Crisis,
After a slow start, the Environmental Protection Agency is doing what it can to make sure municipal and state officials resolve the Flint, Mich., lead-tainted drinking water crisis, and further actions such as penalties or a federal takeover...

General Policy
Green Policies Boost Industry, Not Pollution Havens: OECD
Stringent environmental policies can boost industrial innovation without diverting trade to less regulated markets, the Organization for Economic Cooperation and Development said....

Hazardous Waste
China Soil Pollution Cleanup Plans Face New Delays
Deliberation on China's long-sought Soil Pollution Prevention and Treatment Law to address severe contamination of the country's farmlands and areas around industrial sites will likely run into 2017, a top government official...

Hazmat Transport
Senate FAA Bill Bans Lithium Battery Shipments on Planes
The bulk transport of lithium ion batteries on passenger planes would be banned under Senate legislation reauthorizing the Federal Aviation Administration. The bill, unveiled March 9 by the Senate Commerce Committee, would also prohibit...

Hydraulic Fracturing
Fracking Court Issue: What Does FLPMA Authorize?

In the legal fight over regulation of hydraulic fracturing on federal lands, states have grounded their argument in a basic position: federal land management and leasing laws are about apportioning various uses of the surface and mineral...

International Climate
Obama, Trudeau Pledge Quick Paris Deal Implementation
The U.S. and Canada agreed to speed up implementation of the Paris climate agreement and lead by example in completing in 2016 plans that will outline how they will put themselves on a low-emissions development trajectory between now and 2050....

Oil & Gas
New York Gas Line Delayed Pending Environmental Permits
The opening of Williams Partners LP's proposed $925-million Constitution natural gas pipeline will be delayed to the second half of 2017 as the developer awaits a permit to clear trees in New York and other environmental approvals....

Pesticides
Canada Moves to End Pesticide Testing on Dogs
Canada will soon end a requirement that pesticides used on crops be tested on dogs for toxicity and safety, bringing rules in line with those in the U.S., Canada's health department said March 9....

Renewable Energy
Florida Voters to Decide Tax Break for Solar Panels
A proposal to exempt renewable energy equipment installed on commercial and industrial buildings from property tax assessments will go before Florida voters in August. ...

Risk Assessment
EPA: Lower Doses of Explosive May Harm Health
A military explosive found in the soil and groundwater of some hazardous waste sites can harm the nervous system at lower doses than previously estimated, according to draft assessment the Environmental Protection Agency released March...

Storage Tanks
EPA Inspector General to Vet Tribal Storage Tank Programs
The Environmental Protection Agency's Office of Inspector General is launching a review into the agency's underground storage tank programs in Indian country, according to an OIG memo released March 9....

Superfund
Superfund Claim Tossed in Hazmat Personal Injury Case
The Superfund law can't be used to establish a negligence claim over trichloroethylene that leached from a ball bearing factory into the groundwater of a nearby residential community, a federal court has ruled (Kirk v. Schaeffler Grp....

Toxic Substances
Canada Cites Impact of Foreign Sources on Mercury
Mercury remains a risk to Canadian ecosystems and human health, Environment and Climate Change Canada said March 8, in releasing the country's first comprehensive evaluation of scientific research on mercury. ...

Worker Safety
Federal Workers Given Zika Travel Warning
The top personnel management official in the U.S. issued a warning to all federal employees at risk

of contracting the Zika virus currently spreading in the Americas....

INTERVIEW

Risk Assessment
EPA Science Adviser Discusses Public Health Role
Bloomberg BNA's Steven Gibb interviewed EPA Science Adviser and Deputy Assistant Administrator for the Office of Research and Development Thomas Burke* March 4 on the role he envisions for public health in environmental decision-making....

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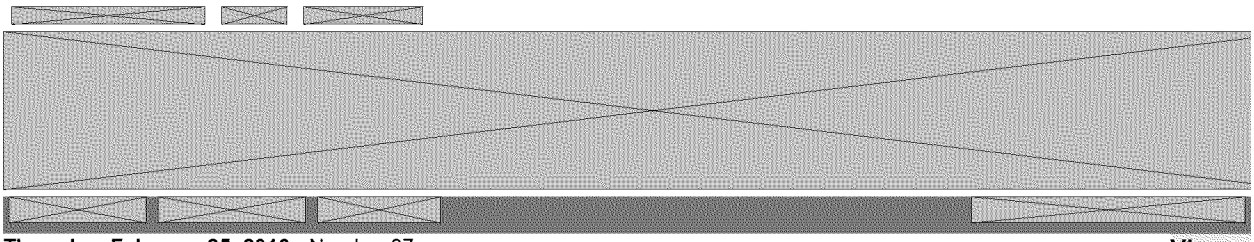

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NEWS

Air Pollution
20 States Ask Supreme Court to Stay EPA Mercury Rule
A coalition of 20 states led by Michigan asked the U.S. Supreme Court to stay implementation of a multibillion-dollar rule to limit mercury emissions from power plants, a development that attorneys said will test the high court's willingness...

Air Pollution
Court Upholds Federal Plan for Haze in Arizona
A federal appeals court upheld the Environmental Protection Agency's decision to impose a federal plan setting emissions limits on a coal-fired power plant in Arizona after partially rejecting the state's plan as inadequate (Arizona...

Air Pollution
EPA Ends Recordkeeping Requirements for Pollutant
Businesses will no longer be subject to recordkeeping and reporting requirements under the Clean Air Act related to the use of t-butyl acetate, which is used as a solvent in paints, inks and adhesives. The agency, in a final rule scheduled for...

Air Pollution
EPA Keeps Testing Component in Sewage Sludge Plan
A final federal plan to implement sewage sludge incinerator emissions guidelines in states that choose not to develop their own plans will not include a revised testing procedure sought by sewage system operators....

Air Pollution
Haze Lawsuit to Continue After Cross-State Ruling
Lawsuits challenging a 2012 Environmental Protection Agency rule that allowed states to use

emissions trading under the Cross-State Air Pollution Rule to fulfill obligations under the regional haze program will finally be allowed to continue,...

Air Pollution
India's Air Pollution Levels Said to Surpass China's
The average Indian faced more particulate pollution than the average Chinese person in 2015, the first time on record, Greenpeace India said in a study. Air pollution levels in India, especially North India, have risen over the past decade...

Biotechnology
Senate Moving to Invalidate State GMO Laws
Congress may be headed for an election-year decision on whether to help big food companies that don't want to comply with a new Vermont state law designed to give consumers more information about what is on their plates....

Chemicals
California Study Finds PBDEs Levels Lower in Breast Milk
Levels of polybrominated diphenly eithers (PBDEs) found in breast milk of California women have dropped dramatically since the state's 2008 ban on the manufacture and sale of products with the flame retardant, according to a study by...

Chemicals
Seven Chemicals May Cause Human Cancer, WHO Agency Says
The International Agency for Research on Cancer has classified seven widely used chemicals as either probably or possibly carcinogenic to people, the agency announced Feb. 24....

Climate Change
Bill Gates Q&A on Climate Change: 'We Need a Miracle'
All we need is an energy miracle. So came the call from Bill Gates on Feb. 22 with the release of his annual letter. ...

Climate Change
Free-Market Groups Urge Congress to Oppose Carbon Tax
Twenty free-market organizations released a letter Feb. 24 urging Congress against pursuing any sort of carbon tax because they said allowing one would harm the nation's economy....

Climate Change
Investors Seek Vote on Climate Proposal at Exxon Mobil
Investors representing more than $1 billion in Exxon Mobil Corp. shares are asking the Securities and Exchange Commission to let a shareholder resolution on climate change onto the company's proxy....

Climate Change
McKenna: Canada Will Review 'Climate Test' Call
Canada will review a proposal by environmental groups for a "climate test" that would call on policy makers to consider international climate change goals before approving major new energy projects....

Climate Regulation
McCarthy: EPA Underestimated Methane From Oil and Gas
The Environmental Protection Agency has been "substantially" underestimating emissions of methane from the oil and natural gas sector, and that could drive new agency regulations going forward, Administrator Gina McCarthy said...

Corporate Responsibility
Vermont Pension Board Softens Stance Against Coal Divestment
Vermont Gov. Peter Shumlin's effort to divest the state of coal, gas and oil stocks inched forward Feb. 23 when a pension committee voted to vet the issue....

Drinking Water
Class Action Filed Over PFOA Contamination in N.Y.
Residents of Hoosick Falls, N.Y., filed a class action lawsuitFeb. 24 in federal court against Saint-Gobain Performance Plastics Corp. and Honeywell International Inc., seeking damages for contamination of their water supply with perfluorooctanoic...

Drinking Water
EPA Recommends Bottled Water in PFOA Contaminated Areas
The Environmental Protection Agency is urging people to use bottled water for cooking and drinking in areas such as Hoosick Falls in upstate New York where tap water has been found to contain concentrations well over the EPA's provisional...

Drinking Water
Flint Deal Paves Way for Senate Action on Energy Bill
A deal announced Feb. 24 that would steer federal funding to help Flint, Mich., fix its lead-tainted drinking water system paves the way for the U.S. Senate to resume debate on broad energy legislation....

Elections
Climate Activists Use Cameras to Put Pressure on Clinton
Griffin Sinclair-Wingate was pressed against the edge of the stage after a debate in New Hampshire when he got his crack at Hillary Clinton....

Emissions Trading
Guangdong to Include New Industries in Carbon Exchange
China's southern province of Guangdong is planning to expand the number of industries to be included in its carbon-trading exchange amid steps to integrate the regional trading program into a planned national emissions exchange....

Energy
Energy Department Promotes More Research
The Energy Department announced Feb. 24 the launch of its Energy Materials Network to enhance collaboration on research and development of advanced materials. The initiative will use funding channeled through the department's Office...

Energy
Justices Appear Convinced State Subsidy Enters FERC Turf
The majority of the U.S. Supreme Court justices appeared convinced during oral arguments that a Maryland subsidy program to encourage new electricity generation in the state crossed into the Federal Energy Regulatory Commission's...

Energy
Moniz: Aliso Canyon Takeaway Is Aging Infrastructure
The recent gas leak in Southern California highlights the importance of addressing the aging energy infrastructure, Energy Secretary Ernest Moniz said Feb. 24 in Houston....

Energy
Sixth Circuit Says Gas Storage Case Should Be Stayed
A suit brought by 40 landowners against a natural gas company should be stayed, not dismissed,

pending a class certification decision in an earlier filed case, the U.S. Court of Appeals for the Sixth Circuit ruled Feb. 24 (Baatz v. Columbia Gas...

Environmental Justice
'Conservative' for States to Develop Power Plans: White House
Preparing to comply with the Clean Power Plan would be the "conservative" option for states and communities seeking the environmental benefits despite the U.S. Supreme Court's decision to halt the rule's implementation,...

Hazardous Waste
HHS Agency Seeks Public Review on Bromopropane, Jet Fuel
The Agency for Toxic Substances and Disease Registry (ATSDR) floated two new toxicological profiles for public review in a Federal Register notice Feb. 24 as part of a long-standing effort to identify hazards at defense facilities....

Hydraulic Fracturing
Colorado Lawmakers Kill Bill to Stop Fracking Bans
A Colorado legislative committee Feb. 24 defeated a bill (H.B. 1181) that would have required local governments to compensate mineral royalty owners when a local ordinance banning hydraulic fracturing prevents them from recovering oil...

Insurance
Insurer Owes No Coverage to Condo for Mold Cleanup
An insurer owes no coverage for a condominium management company's mold abatement because it had no legal obligation to conduct the cleanup, the Eighth Circuit ruled Feb. 24 (Busch Prop. Inc. v. Nat'l Union Fire Ins. Co. of Pittsburgh,...

International Climate
Decline of Power Plant Emissions Likely Without EPA Limits
Carbon pollution from the U.S. power sector will likely continue to fall sharply through 2025 with or without Obama administration carbon pollution limits recently put on hold by the U.S. Supreme Court, an analyst said Feb. 24....

International Climate
No Reason to Deepen EU Carbon Goal Now, Group Says
Europe should not toughen its pollution-reduction goals in immediate reaction to a global climate agreement reached in Paris in December, according to the region's biggest association of employers....

Mining
Murray Energy: Mining Rule Would Eliminate Coal Industry
A pending mining waste rule would drive between 40,000 and 77,000 coal miners out of work and slash as much as $58.7 billion from the gross domestic product, a mining company official told a House subcommittee Feb. 24....

Mining
Peabody Energy No Longer Able to Self-Bond, Group Alleges
WildEarth Guardians has given Peabody Energy notice of intent to sue, saying the financial condition of the large mining company means it no longer qualifies to "self-bond" its mines in Colorado, New Mexico and Wyoming. ...

Pesticides
European Commission to Propose Glyphosate Reapproval
The European Commission will propose to renew until 2031 the authorization in the European

Union of the widely used pesticide glyphosate, according to a draft regulation obtained by Bloomberg BNA....

REACH
REACH Enforcement Project Finds High Level of Compliance in EU
A coordinated enforcement project involving 18 countries turned up only three breaches of decisions taken under the European Union's REACH regulation to phase out two hazardous substances, the European Chemicals Agency (ECHA) said...

Regulatory Policy
No Consensus on Fixing Unfunded Mandates Act
Although there is broad agreement that the Unfunded Mandates Reform Act of 1995 has holes and needs to be fixed, no consensus about how to do that emerged from a Senate hearing on the issue....

Regulatory Policy
Zombie Working Group May Come Back to Life: Lankford
A bipartisan working group in the Senate tasked with crafting a package of changes to the regulatory process has fallen apart for the moment, although Republicans are hopeful that Democrats will come back to the table, said Sen. James Lankford...

Renewable Energy
Brooklyn Site Seen as Base to Build Offshore Wind Farm
Deepwater Wind LLC, the company building the only U.S. offshore wind farm, is looking at a Brooklyn waterfront site as a staging ground as it pursues a potential project off the south shore of Long Island, according to a person familiar with the...

Sustainability
Gore Sees Sustainable Investments Eclipsing 'Tens of Trillions'
Al Gore started an investment business a dozen years ago, aiming to put money in the sort of companies that aligned with his vision of a world in transition....

Trade
U.S. Prevails in WTO Dispute Against India's Solar Program
A World Trade Organization dispute panel backed U.S. allegations that India's national solar program violated international trade rules and unfairly discriminated against imported solar cells and modules. ...

Vehicle Fuels
Inhofe: Congress Must Act on Fuel Standard by 2022
Congress must decide what to do with the renewable fuel standard by 2022 to prevent the Environmental Protection Agency from having full control over it, Sen. James Inhofe (R-Okla.) said Feb. 24 at an oversight hearing on the requirements for...

Water Pollution
NPDES Permit for Beef Plant Can Transfer to New Owner
The city of Brawley, Calif., has approved the transfer of a Clean Water Act discharge permit held by the prior owner of a beef plant to the prospective proprietor....

Water Pollution
Research: Microplastics in Rivers Despite Treatment
Thousands and perhaps millions of tiny shards of plastic that are less than 5 millimeters wide are entering urban rivers in Illinois every day after eluding capture at wastewater treatment plants, according to research by Chicago-based Loyola...

Water Resources
Lawmakers Soften Criticism of Water Project Work
The U.S. Army Corps of Engineers is getting better at reviewing potential water infrastructure projects and reporting them to Congress but still needs improvement, lawmakers told corps leaders Feb. 24....

Water Resources
Texas Supreme Court Lets Stand Drought Rules Invalidation
A ruling that invalidated emergency drought rules allowing Texas's water regulator to suspend some junior water rights, but not those held by power generators and cities for public health, safety and welfare concerns, will stand after...

REGULATORY AGENDA

Comment Deadlines

FEBRUARY 24 FEDERAL REGISTER

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Afternoon Briefing - Your Preview of Today's News

The following news provides a snapshot of what Bloomberg BNA is working on today. Read the full version of all the stories in the final issue, published each night.

Methane From Oil, Gas Sector Higher Than Thought: EPA Chief

Posted February 24, 2016, 4:21 P.M. ET

By Nushin Huq

Methane emissions from the oil and gas sector are much higher than the Environmental Protection Agency previously thought, Administrator Gina McCarthy said today in Houston.

The most recent greenhouse gas inventory revealed higher levels from all parts of the energy sector, from storage to pipelines, McCarthy said. While emissions have not necessarily gone up, she said, the new report better captures data.

Voluntary agreements on emission reductions have not been as productive with the oil and gas industry compared to other industries, such as utilities and mining, McCarthy said at the IHS CERAWeek conference.

EPA will finalize its methane rules to regulate emissions from new oil and gas wells this spring, McCarthy said. When asked if this means the EPA will regulate natural gas more, she said, “We already are.”

20 States Ask U.S. Supreme Court to Stay EPA Mercury Rule

Posted February 24, 2016, 12:45 P.M. ET

By Patrick Ambrosio

A coalition of 20 states, led by Michigan, asked the U.S. Supreme Court to stay implementation of a multibillion-dollar rule limiting mercury emissions from power plants.

The states, in a petition filed today, said a stay is appropriate because the Supreme Court already decided in June 2015 that the Environmental Protection Agency violated the Clean Air Act when it failed to consider cost in making a finding that it was “appropriate and necessary” to regulate those emissions. That EPA finding triggered a requirement that the agency move ahead with a rulemaking process that resulted in promulgation of the Mercury and Air Toxics Standards.

The Supreme Court, in its 2015 opinion authored by Justice Antonin Scalia, remanded the rule back to the D.C. Circuit, which opted in December to leave the regulation in place while the EPA works to address the Supreme Court's holding.

“This is an unmistakable example of agency overreach,” the states said today. “An executive agency strayed far beyond the limited authority the legislative branch gave it, and then, when this court corrected the agency's error, the EPA requested on remand that the unauthorized, unlawful regulation should be left in place to have the force of law.”

The states argued that a stay is even more warranted for the MATS rule than it was for the Clean Power Plan, the Obama administration's carbon dioxide standards for power plants. The Supreme Court, in a 5-4 order issued prior to Scalia's death, granted a stay of the Clean Power Plan pending review by the D.C. Circuit and the Supreme Court.

Flint Legislation ‘Hotlined,' Inhofe Says

Posted February 24, 2016, 2:53 P.M. ET

By Ari Natter

Legislation that would provide funding to help Flint, Mich., deal with its lead-tainted drinking water and in the process allow stalled Senate energy legislation (S. 2012) to proceed has been “hotlined,” Sen. Jim Inhofe (R-Okla.) told Bloomberg BNA today.

The term refers to when the Senate majority leader and minority leader agree to bring a measure to the floor by unanimous consent, and use a special phone line to lawmakers' offices to see if there are any objections.

Inhofe said he expects to know if there are any objections later this afternoon. “I feel pretty optimistic that we’ll go ahead and do it,” Inhofe said. But he added that once the measure gets to the Senate floor: “It might not pass. It will take 60 votes.”

‘About 30’ Energy Bill Amendments Possible as Part of Flint Deal: Cornyn

Posted February 24, 2016, 1:30 P.M. ET

By Ari Natter

A deal in the works to move back to stalled Senate energy legislation (S. 2012) includes a request to hold a voice vote on “about 30 amendments,” Sen. John Cornyn (R-Texas) told reporters today.

In addition, “a handful of other items” would receive roll call votes, he said without elaborating.

Sen. John Thune (R-S.D.) said: “It sounds like they are close to reaching an agreement.”

Power Emissions to Decline Even Without Clean Power Plan: Analyst

Posted February 24, 2016, 4:13 P.M. ET

By Dean Scott

Carbon pollution from the U.S. power sector is likely to continue to fall sharply through 2025 with or without Obama administration carbon pollution limits recently put on hold by the U.S. Supreme Court, an analyst said today.

U.S. carbon dioxide emissions from the sector would total nearly 2.08 billion metric tons in 2025 if the high court ultimately decides to overturn the power sector limits, according to John Larsen, director of the Rhodium Group and a former official with the Energy Department.

But Larsen characterized that as a relatively modest impact compared to if the carbon limits were untouched by the court and fully implemented by the Environmental Protection Agency. That scenario would bring the sector's total down to about 1.76 billion tons in 2025.

The impact of leaving the EPA limits in place is so small—just over 300 million metric tons—largely because of other positive trends that have little to do with the regulations, Larsen said, speaking at a forum held by the New America

think tank and the Center for Climate and Energy Solutions.

Two big factors, Larsen said, are declining costs of solar and wind energy, which he said will only continue to fall, and the five year-extension of solar investment and wind production tax credits included in the omnibus funding bill enacted in December.

Companies Face Class Action Over PFOA Contamination in N.Y.

Posted February 24, 2016, 2:35 P.M. ET

By Amena H. Saiyid

Residents of Hoosick Falls, N.Y., filed a class action lawsuit against Saint-Gobain Performance Plastics Corp. and Honeywell International Inc., holding the two companies responsible for contaminating the aquifer underlying the village with the perfluorooctanoic acid (PFOA).

The lawsuit also accuses the two companies of being responsible for the presence of PFOA in the municipal water supply, private wells and local aquifer, “creating a public health crisis for residents and hurting property values.”

The Environmental Protection Agency on Jan. 28 recommended that people in the Town of Hoosick and Village of Hoosick Falls who have private wells with PFOA concentration of 100 parts per trillion or more use bottled water for cooking and drinking. The state of New York also in January declared the village to be a Superfund site, thereby activating emergency funds to address the problem.

The New York-based Weitz & Luxenberg P.C. filed the lawsuit on behalf of the village residents in U.S. District Court for the Northern District of New York.

Insurer Owes No Coverage to Condo Manager for Mold Cleanup

Posted February 24, 2016, 3:45 P.M. ET

By Peter Hayes

An insurer owes no coverage for a condominium management company's mold abatement because it had no legal obligation to conduct the cleanup, the Eighth Circuit ruled today.

Without a settled claim or a settlement or judgment arising from a suit, the management company wasn't “legally obligated to pay by reason of liability imposed by law,” the court said. As a result, there is no coverage under Missouri law.

The management company voluntarily conducted abatement of mold caused by vinyl wallpaper that trapped moisture. Consent forms signed by each owner didn't include an admission of liability, but didn't include a release or settlement of potential claims the property owner might have against the management company.

Judge Lavenski R. Smith wrote the opinion. Judges Diana E. Murphy and Michael Melloy were also on the panel.

Dowd & Bennett in St. Louis represented Busch Properties.

Foland & Wickens in Kansas City represented National Union.

Seven Chemicals May Cause Human Cancer, WHO Agency Says

Posted February 24, 2016, 11:48 A.M. ET

By Pat Rizzuto

Hydrazine, a rocket propellent; 2-mercaptobenzothiazole, a chemical used in rubber; and dimethylformamide, an acrylic-fiber chemical have all been classified as probably carcinogenic to humans, a World Health Organization agency announced today.

A majority of the scientists that WHO's International Agency for Research on Cancer (IARC) asked to review seven

chemicals also classified tetrabromobisphenol A, a widely used flame retardant, as probably carcinogenic to humans.

The panel classified 1-bromopropane, a vapor degreaser; 3-chloro-2- methylpropene, an industrial chemical and seed fumigant; and dimethyl-p- toluidine, which is used to make dental materials, bone cements, industrial glues and artificial fingernail preparations, as possibly carcinogenic to humans.

Albemarle Corp., the Dow Chemical Corp. and the Chevron Corp. are among the companies that make some of these chemicals, some or all of which are produced in volumes of one million pounds or more per year.

IARC's conclusions about the hazards chemicals pose can affect how chemicals are labeled and the safety information that must be provided to workers. Its conclusions on these seven chemicals, reached during a Feb. 2-9 meeting of scientists, were published in the Lancet.

Justices Debate Whether State Subsidy Enters FERC Turf

Posted February 24, 2016, 1:58 P.M. ET

By Rebecca Kern

U.S. Supreme Court justices today questioned whether a Maryland subsidy encouraging new electricity generation in the state crossed into the Federal Energy Regulatory Commission's authority over the wholesale energy markets.

Justice Sonia Sotomayor appeared to side with the federal government in saying that the case represented conflict preemption, with Maryland's subsidy program intruding into FERC's mechanism for setting wholesale electricity rates.

Maryland ordered utilities to enter into 20-year contracts with CPV Maryland LLC to build a new generation facility, and bid into PJM Interconnection LLC capacity markets, which FERC oversees.

Scott Strauss argued on behalf of the Maryland Public Service Commission that the contracts that bid and cleared into PJM's markets did not distort market pricing. Strauss also said states have the authority to regulate and build new power plants and facilities as they see fit, which Maryland did in this case.

Hughes v. Talen Energy Marketing LLC was heard before eight justices, and was among the first cases argued at the Supreme Court since the death of Justice Antonin Scalia. A decision in the case is expected as early as April, analysts say.

Inhofe Urges Congress to Block EPA on Fuel Standard

Posted February 24, 2016, 12:38 P.M. ET

By Renee Schoof

Congress should reconsider the renewable fuel standard by 2022 to prevent the Environmental Protection Agency from having full control over it, Sen. James Inhofe (R-Okla.) said today.

Targeted amounts of total renewable fuels that must be blended into the fuel supply, including advanced and conventional, are set by law through 2022. After that, under current law, the EPA would decide how to administer the program.

The EPA has mismanaged the renewable fuel standard and should not be allowed to run the program on its own, Inhofe, chairman of the Senate Environment and Public Works Committee, said during an oversight hearing.

Inhofe said he wants the renewable fuel standard repealed, but did not propose any legislation to eliminate or change it during the hearing. Republicans and Democrats from farm states defend the renewable fuel standard.

Lawmakers Soften Criticism of Water Project Work

Posted February 24, 2016, 3:05 P.M. ET

By Alan Kovski

The U.S. Army Corps of Engineers is getting better at reviewing potential water infrastructure projects and reporting them to Congress but still needs improvement, lawmakers told corps leaders today.

The corps also needs to do a better job of explaining to local officials the complexities of how to propose a project that can make it through daunting bureaucratic complexities, members of the House Transportation and Infrastructure Committee said.

Neither point was disputed by the two witnesses, Jo-Ellen Darcy, assistant secretary of the Army for civil works, and Lt. Gen. Thomas Bostick, chief of engineers.

The 2016 annual project report, delivered by the corps to Congress in the first week of February, listed enough new or revised projects ready for congressional approval that both Republicans and Democrats appear mollified, in contrast to the bipartisan anger they expressed after the first such report was delivered in 2015 under authority of a 2014 law.

A Water Resources Development Act of 2016, if written and passed as promised by lawmakers, is expected to approve many of the projects in the latest annual report, although funding will not necessarily follow in all cases.

Study: Microplastics in Water Despite Wastewater Treatment

Posted February 24, 2016, 12:12 P.M. ET

By Amena H. Saiyid

Thousands and perhaps millions of tiny shards of plastic that are less than 5 millimeters wide are entering urban rivers in Illinois every day by eluding capture at wastewater treatment plants, according to a new Loyola University study.

Released today by the American Geophysical Union, the findings by assistant professor Timothy Hoellein and his team show that wastewater treatment plants were a source of microplastics in 80 percent of the 10 urban rivers examined in Illinois, “regardless of the size of the river or the size and type of wastewater treatment plant.”

Hoellein estimates that between 15,000 and 4.5 million pieces of microplastics enter those rivers every day.

He told the AGU that the findings are significant because rivers are a source of drinking water for many communities and also a habitat for wildlife. Fish and other aquatic creatures eat the tiny pieces of plastic in rivers, which then make their way up the food chain–possibly ending up in people's dinner, according to Hoellein.

Some of those microplastics—microbeads in personal care products—are to be phased out starting July 1, 2017, under a new federal law.

U.S. Prevails in WTO Dispute Against India's Solar Program

Posted February 24, 2016, 12:35 P.M. ET

By Bryce Baschuk

A World Trade Organization dispute panel today agreed with U.S. trade officials that India's national solar program violated international trade rules and unfairly discriminated against imported solar cells and modules.

Specifically, the WTO ruled that the domestic content requirements of India's Jawaharlal Nehru National Solar Mission program are discriminatory because they require solar power developers to use Indian-manufactured cells and modules, according to its decision.

The WTO urged India to bring its measures into compliance with the national treatment obligations in Article 2.1 of the WTO Agreement on Trade-Related Investment Measures and Article III:4 of the General Agreement on Tariffs and Trade 1994.

“The United States strongly supports the rapid deployment of solar energy around the world—including in India,” said U.S. Trade Representative Michael Froman in a news release. “But discriminatory policies in the clean energy space in fact undermine our efforts to promote clean energy by requiring the use of more expensive and less efficient equipment, raising the cost of generating clean energy and making it more difficult for clean energy sources to be

competitive."

Investors Seek Vote on Climate Proposal at Exxon Mobil

Posted February 24, 2016, 11:23 A.M. ET

By Andrea Vittorio

Investors representing more than $1 billion in Exxon Mobil shares are urging the Securities and Exchange Commission to let a shareholder resolution on climate change onto the company's proxy.

The shareholder proposal, filed by New York state's comptroller and four other investors, reflects their ongoing concern over how a changing climate could impact Exxon Mobil's business, especially if policy makers live up to the goals of a recent international deal to fight it.

"As investors, we need to know how ExxonMobil's bottom line will be impacted by the global effort to reduce emissions and what the company plans to do about it," New York State Comptroller Thomas DiNapoli said in a statement today.

Exxon Mobil, which is simultaneously facing an inquiry from the state's attorney general into whether it misled the public and investors on climate change, has asked the SEC to block the proposal from making it to a vote at its annual meeting in the spring. The SEC declined to comment.

Conservative Groups Urge Congress to Oppose Carbon Tax

Posted February 24, 2016, 11:14 A.M. ET

By Anthony Adragna

Twenty conservative organizations, including FreedomWorks, the American Energy Alliance and Competitive Enterprise Institute, released a letter today urging Congress against pursuing any sort of carbon tax.

"Our nation's citizens expect – and deserve – their duly elected lawmakers to institute policies that move our economy forward and allow all Americans an equal opportunity to succeed," the groups wrote to House Majority Whip Steve Scalise (R-La.). "A carbon tax would fail resoundingly on both of these fronts."

Scalise introduced a resolution (H. Con. Res. 89) last year expressing the sense of Congress that a carbon tax would harm the U.S. economy. There has been little momentum for any sort of carbon tax among lawmakers, though Democratic Sens. Sheldon Whitehouse (R.I.) and Brian Schatz (Hawaii) have introduced legislation (S. 1548) in hopes of building support for one.

SAFE PIPES Act Would Cost $50 Million from 2017-2021: CBO

Posted February 23, 2016, 5:36 P.M. ET

By Rachel Leven

The Senate pipeline safety reauthorization bill would cost $50 million over the course of five years, the Congressional Budget Office said in its cost estimate released today.

The SAFE PIPES Act (S. 2276) would require $525 million in appropriations from 2017 to 2021 for the Pipeline and Hazardous Materials Safety Administration's pipeline safety functions, it said. The bill would generate $462 million in fees from pipeline owners and $17 million in revenue from new underground natural gas storage facility assessments.

The Senate Commerce, Science and Transportation Committee passed the SAFE PIPES Act on Dec. 9. The bill would lower maximum funding levels for pipeline safety programs and aim to allow PHMSA time to address leftover mandates from its 2012 pipeline safety authorization law (Pub. L. No. 112–90), which lapsed Sept. 30.

European Commission to Propose Glyphosate Reapproval

Posted February 24, 2016, 2:10 P.M. ET

By Stephen Gardner

The European Commission will propose to renew until 2031 the authorization in the European Union of the widely used pesticide glyphosate, according to a draft regulation obtained by Bloomberg BNA.

The proposed renewal is controversial because glyphosate has been labeled “probably carcinogenic” by the World Health Organization, though the European Food Safety Authority (EFSA) in November judged that the substance was “unlikely to pose a carcinogenic hazard to humans.”

According to the draft regulation to reauthorize glyphosate, the current authorization for the substance will expire June 30, and EFSA's assessment that it should not be classified as carcinogenic provides the basis for its reapproval as safe to use in the EU.

A committee of EU member state representatives will discuss and possibly vote on the commission's draft regulation on glyphosate at a March 7-8 meeting.

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NEWS

Air Pollution
Air Standards Set for Los Angeles Area Near Gas Leak
California state and local agencies released criteria late Feb. 16 for determining when emissions from Southern California Gas Co.'s underground natural gas storage field near Los Angeles no longer affect air quality in nearby neighborhoods....

Air Pollution
Canada Proposes Tighter Ambient Sulfur Dioxide Standard
Canada's health agency has proposed tightening the country's national ambient air quality objectives for sulfur dioxide, last updated more than two decades ago and more than four times higher than allowable levels across the border...

Air Pollution
EPA Proposes 12 Nonattainment Areas for Sulfur Standards
The Environmental Protection Agency will propose designating 12 areas in eight states as not achieving 2010 federal air quality standards for sulfur dioxide....

Chemicals
EPA to Release RDX Draft Analysis at May Meeting
The Environmental Protection Agency's Integrated Risk Information System Program will release a draft assessment for a chemical used in explosives at an upcoming May meeting, it announced Feb. 17. The program will release the draft...

Climate Change
High Temperatures on Pace to Break 2015 Record
Here we go again. For the surface of planet Earth, 2015 was the hottest year on record by a stunning margin. But already, 2016 is on track to beat it....

Corporate Responsibility
Apple's $1.5B Green Bond Seen Spurring Corporate Issuers
Apple Inc.'s new green bond may trigger other companies to follow suit, with more corporate issuance this year for investments in renewable energy and climate change projects, analysts examining the niche market said....

Drinking Water
EPA Didn't Enforce Flint Drinking Water Violations: CRS
The Environmental Protection Agency did not use its authority under the Safe Drinking Water Act to enforce violations against the public water utility in Flint, Mich., despite being aware of the elevated lead levels in the city's tap water,...

Energy
Ostendorff to Leave Nuclear Regulatory Commission
William Ostendorff, a Republican commissioner at the Nuclear Regulatory Commission, will leave the agency after his five-year term ends June 30....

Energy
South Carolina Offers Renewable Energy Tax Credits
South Carolina is providing income tax credits for certain solar energy and geothermal installations as part of legislation (H.3874) signed into law by Gov. Nikki Haley (R) Feb. 16....

Enforcement
Trial Begins for Former BP Manager Linked to 2010 Oil Spill
Federal prosecutors have what they expect to be their last chance to send someone to jail over the deadly 2010 BP Plc Gulf of Mexico well blowout and the worst offshore oil spill in U.S. history (United States v. Kaluza, E.D. La., No. 12-cr-00265,...

Environmental Justice
EPA Solicits Nominations for Justice Council
The Environmental Protection Agency is requesting nominations be submitted for its National Environmental Justice Advisory Council, according to a notice to be published in the Federal Register Feb. 18. The council offers recommendations...

EPA
Acting EPA Human Resources Chief to Depart Agency
The Environmental Protection Agency's acting leader of human resources, contract and grant issues will leave the agency for a teaching position at the University of Texas–Austin, Administrator Gina McCarthy said in an e-mail...

Hazardous Waste
Oil Pipeline Cases Stay in State Court After Plaintiffs Changed
The addition of substituted plaintiffs in a mass action suit in Louisiana over oil pipeline waste doesn't create a new "window" for transfer of the cases from state to federal court under the Class Action Fairness Act, a federal...

Oil & Gas
Oklahoma Announces Plan for Wastewater Injection Cutbacks
In a continuing effort to address increased earthquake activity, Oklahoma's energy and gas regulators unveiled what they called the "largest volume reduction plan yet" for underground wastewater disposal from oil and gas...

Renewable Energy

Ontario Investing $72M in Efficiency, Clean-Tech Programs
The Province of Ontario's Green Investment Fund will invest C$99 million ($72 million) in energy efficiency and clean-tech innovation programs. The government has committed C$74 million in the clean-tech initiative, designed to reduce...

Renewable Energy
Solar Group Pushes for Broad Eligibility for Tax Credits
The IRS should take a broad approach as it considers what types of renewable energy equipment are eligible for tax credits, a solar energy trade group said....

Risk Assessment
More Time Given to Provide Chlorinated Paraffin Use Data
Companies that process or use any of seven chlorinated paraffins as lubricants or flame retardants and for other purposes will have until March 23 to provide the Environmental Protection Agency information about how they use these chemicals,...

Stormwater
Trading a Boon When Using Green Infrastructure: NRDC
Cities using green infrastructure to manage stormwater will glean greater benefits by engaging in stormwater credit trading, according to a new issue brief by the nonprofit advocacy group the Natural Resources Defense Council....

Sustainability
Ford Drops Membership in ALEC Lobbying Group
Ford Motor Co. dropped its membership in the conservative lobbying group the American Legislative Exchange Council (ALEC), the company confirmed in an e-mail Feb. 17 to Bloomberg BNA. ...

Water Pollution
EPA: DC Water Waived Potomac River E. Coli TMDL Claims
The D.C. Water and Sewer Authority failed to state a claim when it filed a complaint challenging the total maximum daily load for E. coli that the Environmental Protection Agency approved for the Potomac River and its tributaries, the agency...

Water Pollution
New York Classifies PFOA as Hazardous
The chemical perfluorooctanoic acid (PFOA) was classified by New York state as a hazardous substance Feb. 17 in an emergency regulation published in the New York State Register....

Water Pollution
Toxins From Algae Found in Southeast U.S. Streams
Toxins from algae were found in 39 percent of small streams sampled across the southeastern U.S., the U.S. Geological Survey said Feb. 17....

SPECIAL REPORT

Water Resources
Groundwater Reserved Right Appeal Raises Doctrine Questions
Although water rights are generally products of state law—adjudicated in state courts and administered by state agencies—a federal common law doctrine creates a water right for federal lands, such as national forests, monuments...

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Afternoon Briefing - Your Preview of Today's News

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EPA to Propose 12 Areas as Not Meeting SO2 Standard

Posted February 17, 2016, 12:43 P.M. ET

By Andrew Childers

The Environmental Protection Agency today announced it will propose to designate 12 areas in eight states as not achieving federal air quality standards for sulfur dioxide issued in 2010.

The EPA's proposal would designate to the list of areas not meeting the national ambient air quality standards for sulfur dioxide: Alton township and Williamson County, Ill.; Jefferson and Posey counties, Ind.; parts of Desoto Parish, La.; Anne Arundel and Baltimore counties, Md.; St. Clair County, Mich.; Franklin County, Mo.; Muskogee County, Okla.; and Freestone-Anderson, Rusk-Greg-Panola and Titus counties, Texas.

The EPA said it intends to complete the proposed designations by July 2.

The EPA in 2013 designated 29 areas in 16 states as being in nonattainment with the 2010 primary, one-hour national ambient air quality standard for sulfur dioxide of 75 parts per billion. The agency delayed action on the rest of the country due to inadequate air monitoring. The agency, under a court-approved consent decree with the Sierra Club and the Natural Resources Defense Council, agreed to take a phased approach to making attainment designations for the rest of the country.

EPA Didn't Enforce Drinking Water Violations in Flint: CRS

Posted February 17, 2016, 11:19 A.M. ET

By Amena H. Saiyid

The Environmental Protection Agency did not use its authority under the Safe Drinking Water Act to enforce violations against the public water utility in Flint, Mich., despite being aware of the elevated lead levels in the city's tap water, according to a Congressional Research Service report.

Released yesterday, the report said Section 1414 of the Safe Drinking Water Act authorizes the EPA to notify state and the public water systems when it learns of a drinking water violation, and to allow the systems to come into

compliance.

But if after 30 days the violation continues and the jurisdictions haven't taken action then the EPA “must” take enforcement action against the public utility, the report notes. “EPA has not used this authority in Flint,” wrote Mary Tiemann, author of the CRS report.

Air Standards Set for Leak in Gas Field Near Los Angeles

Posted February 17, 2016, 3:41 P.M. ET

By Carolyn Whetzel

California state and local agencies late yesterday released criteria for determining when emissions from Southern California Gas Co.'s underground natural gas storage field near Los Angeles no longer affect air quality in nearby neighborhoods.

The criteria, which sets thresholds for methane and other contaminants, are based on typical levels that predate the Oct. 23 discovery of the gas leak at the facility now in the final stages of being permanently capped.

At a Feb. 16 news conference at the SoCalGas Co. office in Chatsworth, U.S. Energy Secretary Ernest Moniz said lessons learned from the leak will inform a multi-agency effort by the federal government to address the nation's aging energy infrastructure.

Issues that have come to light in Aliso Canyon are obviously of huge local concern, but underscore the problems with the nation's aging energy storage infrastructure, Moniz said following a tour of the facility and meeting with local, state and federal officials. This incident identifies a problem we have to study more generally across the country, he said. “Frankly, gas storage fields need a fresh look in terms of the regulatory requirements,” Moniz said.

New York Classifies PFOA as Hazardous

Posted February 17, 2016, 11:22 A.M. ET

By Gerald B. Silverman

The chemical perfluorooctanoic acid (PFOA) was classified by New York as a hazardous substance today in an emergency regulation published in the New York State Register.

The regulation is a response to water contamination in the upstate town of Hoosick Falls, N.Y. The state can now expend funds from its Superfund program to remediate the water problems.

The New York State Department of Environmental Conservation said the classification will also allow the state to respond if elevated levels of the chemical are found elsewhere in the state.

PFOA is a chemical used in the manufacture of nonstick cookware and other consumer products that resist heat and repel oil, grease and water.

Health Canada Proposes Lower Ambient SO2 Standard

Posted February 17, 2016, 3:53 P.M. ET

By Peter Menyasz

Canada's health agency has proposed lowering the country's national ambient air quality objectives for sulfur dioxide, last updated more than two decades ago and more than four times higher than allowable levels across the border in the U.S.

While exposure to sulfur dioxide emissions has decreased significantly since the national objectives were adopted in 1989, research on the health impacts of short-term exposure to sulfur dioxide at current exposure levels shows a risk that should be addressed, Health Canada said.

“The human health assessment has identified potential health risks to the Canadian population from exposure to

ambient concentrations of SO2 that is below the current National Ambient Air Quality Objectives," it said. "It is therefore recommended that the current [standards] be revised or new ambient air quality objectives or standards be introduced."

Ford Drops Membership in ALEC Lobbying Group

Posted February 17, 2016, 11:51 A.M. ET

By Ari Natter

Ford Motor Co. has dropped its membership in the conservative lobbying group the American Legislative Exchange Council (ALEC), the company confirmed in an e-mail today to Bloomberg BNA.

"As part of our annual budget review in 2015, we adjusted our participation in several groups. We will not be participating in ALEC in 2016," spokeswoman Christin Baker said in an e-mail.

In leaving ALEC, Ford joins companies such as Google, Microsoft and others that have raised concerns about the group's position denying climate change and other stances deemed to be anti-environmental, according to the watchdog group the Center for Media and Democracy.

Duke Energy Loses $126M Tax Refund Bid in South Carolina

Posted February 17, 2016, 3:52 P.M. ET

By Erin McManus

Duke Energy Corp. can't use principal recovered from the sale of short-term securities to reduce the percentage of its income attributable to South Carolina.

The South Carolina Supreme Court ruled today that inclusion of principal in the calculation of the company's total sales—when determining how much of its income is subject to South Carolina tax—would result in distortion leading "to absurd results that could not have been intended by the General Assembly."

Duke sought a $126 million refund by claiming that the total proceeds from the sale by its treasury department of short-term securities, which had no connection to South Carolina, should be included in the denominator of the fraction of its income taxable by South Carolina, thereby reducing that tax.

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Daily Environment Report's 2016 Outlook articles are now available in one convenient PDF. To see the outlook, click here.

To see our outlook video with a review of key issues to watch in 2016, click here.

NEWS

Air Pollution
China Proposes Car Interior Air Quality Testing
China's Ministry of Environmental Protection proposed mandatory testing of air quality inside new motor vehicles, according to a draft of the policy released for public comment Jan. 28. The amendment would replace a voluntary testing...

Air Pollution
Measure Would Halt New Injections at L.A. Gas Field
The California Senate approved a bill Jan. 28 to impose an immediate moratorium on natural gas injections at Southern California Gas Co.'s underground storage field near Los Angeles. The unanimous vote, 40-0, sends S.B. 380 to the Assembly...

Air Pollution
Ozone Rule Not Protective, Environmental Groups Argue
The Environmental Protection Agency erred when it rejected the advice of its own science advisers and set revised ozone standards that are insufficient to protect health or vegetation, environmental and public health groups told a federal...

Air Pollution
Utah Governor Pushing State's Refineries to Make Tier 3 Fuel
Utah Gov. Gary R. Herbert (R) said he continues to push the state's refineries to produce "as soon as possible" fuel under the Tier 3 rule, which cuts sulfur in gasoline and strengthens vehicle

emissions standards....

Climate Change
Amendment Eyes Fossil Fuel Disclosure on Climate
The push for better fossil fuel-company disclosure on climate change could get an added boost from Congress....

Climate Change
Maryland Bill Would Boost Greenhouse Gas Cuts
A Maryland Senate committee scheduled a Feb. 9 hearing on legislation (S.B. 323) that would boost the state's target for greenhouse gas emissions reductions to 40 percent below 2006 levels by 2030. The measure would codify the recommendation...

Climate Change
Northwest Territories Premier Talks Climate Change
Canada's Northwest Territories will use challenges presented by climate change and the current oil-price slump to build infrastructure and craft legislation on environmental assessments and hydraulic fracturing so the province...

Climate Regulation
21 Senate Democrats Urge New Methane Standards
The Environmental Protection Agency should expand its regulation of methane emissions to include existing sources from the oil and natural gas sectors, 21 members of the Senate Democratic caucus wrote Jan. 28. ...

Climate Regulation
Feb. 4 Deadline Set for EPA Power Plan Stay Response
The Environmental Protection Agency has a Feb. 4 deadline to respond to petitions to the U.S. Supreme Court seeking to stay implementation of carbon dioxide standards for power plants while the standards are being litigated, according to...

Climate Regulation
Proposal Would Alter Oil, Gas GHG Emissions Reporting
Oil and gas operations that are large emitters of greenhouse gases would be allowed to use new monitoring methods to detect leaks from their equipment under a proposed Environmental Protection Agency rule that would alter how those facilities...

Drinking Water
Bill to Authorize $400M for Flint Water Infrastructure
Legislation that would provide $400 million in federal emergency funding to help Flint, Mich., respond to its drinking water crisis was announced Jan. 28 by three Democratic members of the Michigan congressional delegation. ...

Drinking Water
Flint Emergency Extended, $28M Assistance OK'd
On the heels of a $28 million supplemental appropriation measure (H.B. 5220) for Flint passing the Michigan Senate, Gov. Rick Snyder (R) extended the city's state of emergency until April 14. ...

Energy Efficiency
California Adopts New Lighting Standards
California has set the nation's first energy efficiency standards for small lamps used in track lighting and general purpose light-emitting diodes, or LEDs....

Energy

Senate Adopts Energy Amendments on Nuclear, Research
Measures that would promote advanced nuclear energy projects and increase funding for energy research were among the first amendments adopted to a broad energy bill (S. 2012) on the Senate floor Jan. 28. ...

Environmental Reviews
Canada's Energy Project Review Process Lauded
Canada's new environmental assessment process for pipelines and other major energy projects is being widely lauded, despite questions about whether it is enough to help the country meet its international climate change commitments....

Hazmat Transport
UN Body Urges Ban on Lithium Batteries on Passenger Planes
The International Civil Aviation Organization's top technical body has recommended a total ban on shipments of lithium batteries as cargo on passenger aircraft....

Oil & Gas
Oklahoma Company Agrees to Disposal Well Cuts
A recent agreement between Oklahoma's oil and gas regulator and one of the state's largest energy companies to reduce wastewater disposal in response to increased seismicity heads off potential legal action over the company's...

Renewable Energy
California Extends Payments for Rooftop Solar Customers
Rooftop solar customers of California's three investor-owned utilities will continue to get paid full retail rates for the excess energy their systems generate, under a net metering program the California Public Utilities Commission...

Toxic Substances
EU Food Authority Claims Breakthrough in Pesticide Exposure Studies
The European Food Safety Authority (EFSA) said it will use a new risk assessment tool to prepare "breakthrough" exposure studies on the risks to the human thyroid and nervous systems of the combined effects of pesticide residues in...

Vehicle Fuels
Plug Power Expects Higher Sales from Hydrogen Fuel Cells
Plug Power Inc., a maker of fuel-cell systems for forklifts, expects revenue to climb 50 percent in 2016, matching the growth rate set last year....

Water Pollution
GAO Report on WOTUS Lobbying Not Needed in Record: DOJ
A Government Accountability Office report criticizing alleged illegal lobbying by the Environmental Protection Agency to promote its water jurisdiction rule does not belong in the administrative record for litigation on the regulation,...

Water Pollution
Most Coastal Waters in Fair to Poor Health, EPA Says
Water quality in most U.S. coastal waters ranged from fair to poor in 2010, owing mostly to phosphorus pollution, the Environmental Protection Agency said in its most recent ecological assessment....

Water Pollution
N.Y. Takes Steps to Address Water Problem
Gov. Andrew M. Cuomo (D) took the unusual steps Jan. 27 of ordering emergency regulations to

classify the chemical perfluorooctanoic acid (PFOA) as a hazardous substance and to have a plastics plant operated by Saint-Gobain Corp. immediately...

Water Resources
Colorado Lawmakers Debate 2016 Water Policy Goals
Legislative proposals to conserve water, streamline permits and increase storage were the chief items of debate between two state lawmakers at a presentation of the Colorado Water Congress. ...

BNA INSIGHTS

Is Nuisance Litigation Undermining the Clean Air Act's Preemptive Economic Purpose?
The battle over federal Clean Air Act preemption continues to rage in the nation's courts, and until the U.S. Supreme Court decides to review the issue, the answer will probably remain unresolved. A group of slightly older cases held that...

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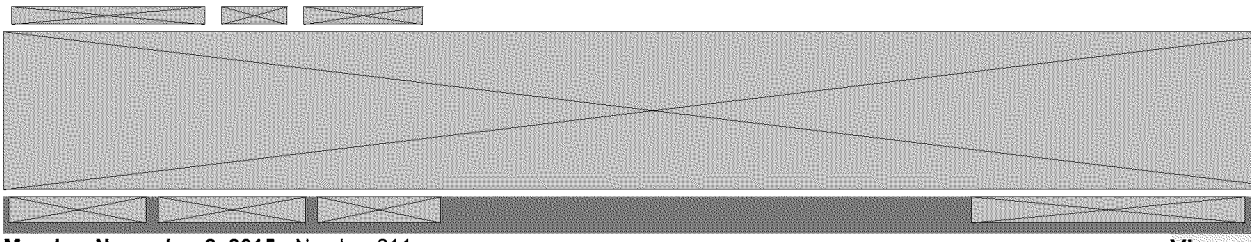

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NEWS

Air Pollution
Colorado Attorney General Says She Can Sue EPA
Colorado Attorney General Cynthia Coffman (R) said the state constitution clearly grants her the ability to sue the Environmental Protection Agency over the Clean Power Plan despite the objection of Gov. John Hickenlooper (D). ...

Air Pollution
EPA Seeks Delay in McCarthy's Job Lawsuit Deposition
The Environmental Protection Agency is asking a federal judge to delay deposition of Administrator Gina McCarthy in a lawsuit over a required Clean Air Act jobs review if it denies the agency's bid to shield its top official from the inquiry...

Biotechnology
New Authority Not Expected for Biotech Framework Update
Federal agencies that regulate products made with biotechnologies will clarify their roles and responsibilities as they update the Coordinated Framework for the Regulation of Biotechnology, said a senior White House scientist Oct. 30....

Budget
Budget Deal Clears Congress; Omnibus Bill Next Up
With a Senate vote that occurred just after 3 a.m. on the morning of Oct. 30, a budget deal allowing appropriators to exceed the sequester-mandated budget caps cleared Congress....

Chemicals
California to List Preservative Mixture as Carcinogen
California's Office of Environmental Health Hazard Assessment announced Oct. 30 plans to add the complex mixture of pentachlorophenol and byproducts of its synthesis to the state's list of

chemicals linked to cancer....

Chemicals
Science Advisers to Weigh in on Proposition 65 Proposal
Scientific advisers for California's Office of Environmental Health Hazard Assessment will weigh in Nov. 9 on whether there is sufficient scientific evidence to consider adding nickel, pentachlorophenol, perfluorooctanoic acid...

Climate Change
Maryland Panel Backs 2030 Greenhouse Gas Emissions Cut
The Maryland Commission on Climate Change has urged lawmakers to adopt a more ambitious goal of reducing greenhouse gas emissions 40 percent below 2006 levels by 2030 as they revisit an existing climate change law during the 2016 legislative...

Climate Change
Texas Unlikely to Submit Carbon Plan
Confident that the U.S. Supreme Court will eventually strike down the Obama administration's Clean Power Plan, a senior attorney in the Office of the Texas Attorney General said Oct. 30 his state probably wouldn't submit a compliance...

Climate Regulation
Suits Filed Over EPA Carbon Standards for New Power Plants
Murray Energy Corp. and a free market advocacy group filed lawsuits Oct. 30 challenging the Environmental Protection Agency's carbon dioxide standards for new and modified power plants (Murray Energy Corp. v. EPA, D.C. Cir., No. 15-1396,...

Courts
Standing Case Could Affect Environmental Plaintiffs
The U.S. Supreme Court could make it more difficult for environmental plaintiffs to allege standing in certain cases if it decides that a statutory violation isn't a sufficient injury-in-fact without there also being evidence of a...

Drilling
Shell Wins Nova Scotia Offshore Exploration Approval
The Canadian regulator for energy development in Nova Scotia's offshore areas has approved the launch of Shell Canada Ltd.'s proposed Shelburne Basin deepwater exploration project. The Canada-Nova Scotia Offshore Petroleum...

Drinking Water
EPA Drinking Water Council to Meet on Lead, Copper
The Environmental Protection Agency's National Drinking Water Advisory Council will meet Nov. 17-19 to consider long-term revisions to the agency's lead and copper rule, according to a notice scheduled for publication in the Federal...

Energy
Agencies to Prepare for Solar Storms, White House Says
The White House directed federal agencies, including the Energy Department, in a recent action plan to prepare for the rare occurrence of solar storms, which can harm the nation's electric grid and telecommunications equipment....

Hazmat Transport
Montana Auditor Calls for Improved Rail Safety
Anticipating an increase in oil shipments by rail, the Montana Legislative Audit Division called on the state to improve its railroad safety efforts....

International Climate
Forest Fires Push Indonesia Greenhouse Gas Emissions
The intense fire season in Indonesia means that country has temporarily become among the world's largest emitters of greenhouse gases, according to the World Resources Institute....

International Climate
Nations' Pledges Show Progress But Short of 2-Degree Goal
Roughly one-quarter of the greenhouse gas emissions cuts nations have pledged toward a global climate agreement hinge on whether developing nations get adequate funding from richer nations, the United Nations said Oct. 30....

Natural Resources
White House Memo: Consider Ecosystem Services
Federal agencies will develop policies to assess ecosystem services—benefits people gain from nature—as part of federal planning and decisions, under a memorandum issued by the White House....

Ozone Depletion
World's Worst Greenhouse Gas Spurs Global Smuggling Rings
Every year, diplomats gather to review progress in implementing the Montreal Protocol, a 1987 treaty that limited global use of chlorofluorocarbons and other ozone-eating chemicals. ...

Pesticides
After Delay, Farmworker Rule Set for Publication
After a delay of more than a month, the Environmental Protection Agency's update to its worker protection standards is set to be officially published in the Federal Register on Nov. 2, according to a pre-publication notice....

Pesticides
Under Court Order, EPA Proposes Ban on Dow Pesticide
Under the threat of a court order, the Environmental Protection Agency is proposing a ban on all agricultural uses of the Dow AgroSciences insecticide chlorpyrifos....

Pipeline Safety
NTSB Endorses API Oil, Gas Pipeline Safety Approach
The National Transportation Safety Board said Oct. 30 that the American Petroleum Institute's recently released oil and gas pipeline safety management system recommended practices exceeds expectations. The board recommended the...

Renewable Energy
China Said to Mull Wind, Solar Power Tariff Cuts
China is considering cutting the preferential rate it offers wind and solar power developers because the surcharges tacked onto electricity bills to pay for clean-energy subsidies aren't high enough....

Water Pollution
Additional Challenges to Water Jurisdiction Rule Filed
Farm and stormwater groups have joined various states, industry groups and companies in their challenges to the waters of the U.S. rule issued by the Environmental Protection Agency and U.S. Army Corps of Engineers, which delineates the federal...

Water Pollution
Comments on Washington Water Rule Due Dec. 28
The Environmental Protection Agency announced Oct. 30 it is extending the public comment

period until Dec. 28 for a proposed rule to revise federal water quality criteria applicable to Washington. The proposal (RIN 2040-AF56) would revise...

Water Pollution
Commission Seeks Input on Water Testing Methods
The Delaware River Basin Commission is seeking comment on the methodology it plans to use when testing the water quality in the Delaware River and Bay, the commission said Oct. 29. The proposed methodology will be used to compile the "2016...

Water Pollution
EPA Lawyer Points to Sixth Circuit on Clean Water Rule Issue
The threshold jurisdictional question relating to judicial review of challenges to the Obama Administration's clean water rule may be decided relatively quickly by the U.S. Court of Appeals for the Sixth Circuit, a senior attorney...

Water Pollution
Senate to Vote on Measures to Block Waters of U.S. Rule
The Senate will vote Nov. 3 on whether to proceed to legislation (S. 1140) that would force the Environmental Protection Agency and U.S. Army Corps of Engineers to rewrite their waters of the U.S. rule, Don Stewart, a spokesman for Senate Majority...

Water Pollution
Supreme Court Expected to Grant Review in Water Cases
The U.S. Supreme Court likely will resolve a split among the federal circuit courts of appeals about whether a jurisdictional determination under the Clean Water Act constitutes a judicially reviewable final agency action, legal panelists...

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Afternoon Briefing - Your Preview of Today's News

The following news provides a snapshot of what Bloomberg BNA is working on today. Read the full version of all the stories in the final issue, published each night.

EPA Strengthens Bush-Era Ozone Standards to 70 ppb

Posted October 01, 2015, 2:14 P.M. ET

By Patrick Ambrosio

The Environmental Protection Agency tightened national standards for ground-level ozone to a level of 70 parts per billion, a move that upset both industry opponents and public health organizations, the agency announced today.

The final rule, posted to EPA's website, is projected to cost the U.S. $1.4 billion in compliance costs in 2025, not including California, which will get longer to meet the standards due to its air quality control issues. The agency projected the standards will yield nationwide public health benefits of up to $5.9 billion in 2025 from avoided premature deaths and asthma attacks.

The existing 75 ppb ozone standards, set in 2008 under President George W. Bush, were judged by the agency to be inadequately protective of public health.

Public health and environmental groups, including the American Lung Association, the Natural Resources Defense Council and Earthjustice, issued statements describing the ozone rule as a small step to improve air quality that is not sufficient enough. Those groups had urged the agency to set the ozone standards at a level of 60 ppb.

The American Petroleum Institute, one of the leading opponents of the ozone rule, issued a statement urging Congress to take action to block implementation of the “unnecessary and costly” ozone rule.

Senate Will Push to Overturn EPA Ozone Rule: Sessions

Posted October 01, 2015, 4:10 P.M. ET

By Anthony Adragna

Sen. Jeff Sessions (R-Ala.) told Bloomberg BNA today the Senate plans to try to overturn the Environmental Protection Agency's newly finalized ozone standards using the Congressional Review Act.

“I believe it’s going to be clear the regulations exceed what is justifiable and it’ll be important for Congress to fight

back," Sessions said.

Sen. John Thune (R-S.D.) told reporters the EPA's decision to tighten the national ambient ozone standard to 70 parts per billion was "better than where we heard they were talking about going, but still it just doesn't make a lot of sense." Thune has introduced legislation that would effectively block the EPA from revising the ozone standard until 85 percent of the counties not meeting the current standard become compliant.

Manchin, Capito Say TSCA Vote Possible Next Week

Posted October 01, 2015, 4:13 P.M. ET

By Anthony Adragna

West Virginia Sens. Joe Manchin (D) and Shelley Moore Capito (R) say legislation (S. 697) that would reform the nation's chemical law may get a floor vote next week after the addition of language to address the concerns of Sen. Barbara Boxer (D-Calif.).

"It's time has come," Manchin said. "It's time for us to move forward on that bill."

The legislation, from Sens. Tom Udall (D-N.M.) and David Vitter (R-La.), has the backing of 56 senators from 36 states. Senate Majority Leader Mitch McConnell (R-Ky.) has not announced when the bill will hit the floor.

New Use Rules for 30 Chemicals Issued by EPA

Posted October 01, 2015, 2:18 P.M. ET

By Pat Rizzuto

Thirty chemicals, including five the Environmental Protection Agency concluded were analogous to perfluorooctanoic acid (PFOA), would be regulated under direct final new use rules the Environmental Protection Agency will publish in tomorrow's Federal Register.

The significant new use rules (SNURs) will be effective Dec. 1, unless the EPA receives by Nov. 2 adverse comments or a notice alerting it to a party's intention to file adverse comments.

The 30 chemicals include nine that already are subject to consent orders the EPA negotiated with chemical manufacturers using the authority provided under section 5(e) of the Toxic Substances Control Act.

The SNURs for the nine chemicals subject to these section 5(e) orders would require any future manufacturer or importer of the chemical to comply with the same requirements to which the original manufacturer already is bound.

Similarly, the SNURs for the remaining 21 chemicals would oblige any future manufacturer or importer to the manufacturing and use conditions described in the original manufacturers premanufacture notice (PMN). EPA reviewed those PMNs and did not object to those chemicals entering commerce.

FERC's Clark: Supreme Court Case Could Give Agency More Power

Posted October 01, 2015, 2:17 P.M. ET

By Rebecca Kern

Tony Clark, a Republican commissioner with the Federal Energy Regulatory Commission, said that an upcoming U.S. Supreme Court case on whether FERC has authority to compensate demand response programs in the wholesale electricity markets could in the end give the federal government more power than it currently has.

"The real headline coming out of that, in my mind, is something that if I was a state commissioner I would be very, very concerned about," Clark said at a Politico event in Washington today. He said that in its oral arguments scheduled for Oct. 14, the federal government is claiming that anything that affects wholesale rates is a federal jurisdiction activity, which in turn could affect states' rights.

"If you take that to its furthest logical conclusion, basically that light switch on the wall in some way affects wholesale

rates, which could be used as jurisdiction to really run right over states' governments because it strengthens the hand of FERC and the federal government with regards to the Federal Power Act," he said.

Clark said there a number of outcomes for the case. In one scenario, the Supreme Court could uphold the lower district court ruling, which vacated the FERC's Order 745 demand response program. Or it could narrowly overturn the ruling, essentially keeping the Federal Power Act as it has been. Or, they rule that "anything that affects wholesale rates is federal jurisdiction. That could be a blow for state rights in terms of the Federal Power Act," he said.

PHMSA Releases Hazardous Liquid Pipelines Proposal

Posted October 01, 2015, 4:16 P.M. ET

By Rachel Leven

The nation's pipeline safety regulator under a proposed rule released today would expand a requirement to periodically conduct in-line integrity assessments of hazardous liquid pipelines to apply to pipes located outside of high consequence areas, a requirement expected to cost operators $16.7 million annually.

The Pipeline and Hazardous Materials Safety Administration rule would also establish a timeline for inspections of pipelines affected by an extreme weather incident or natural disaster following the incident, require installation of leak-detection systems on all hazardous liquid pipelines, and require annual evaluations and improvements of pipelines with the highest environmental or human damage potential, among other steps, the proposal said.

The rule, which has been highly anticipated by groups such as the Association of Oil Pipe Lines and the American Petroleum Institute, would cost hazardous liquid operators an estimated $22.4 million per year with benefits between $3.5 million and $17.7 million annually. That is in addition to unquantifiable safety benefits such as prevented fatalities. PHMSA estimates 421 hazardous liquid pipeline operators may incur costs.

The rule will be available for public comment through Jan. 8, 2016.

Mosaic Fertilizer to Spend $630 Million on Cleanup, Trust Fund

Posted October 01, 2015, 3:03 P.M. ET

By Chris Marr

Mosaic Fertilizer will start a trust fund to pay for eventual closure and environmental management of four fertilizer plants, while also investing in near-term projects, worth a total commitment of almost $2 billion, the EPA said today in announcing a proposed settlement with the company.

Under the agreement, which is pending public comment and approval in two federal courts, Mosaic will deposit $630 million into a trust fund, which is to be invested and grown to $1.8 billion, according to the Environmental Protection Agency. The settlement resolves alleged violations of the federal Resource Conservation and Recovery Act as well as state laws in Florida and Louisiana, related to Mosaic's handling of hazardous waste and wastewater at its facilities.

John C. Cruden, an official with Department of Justice, which helped pursue the case on behalf of the EPA, called the settlement the government's "most significant enforcement action in the mining and mineral processing arena. … This sets the standard for our continuing enforcement of RCRA in the entire phosphoric acid industry."

Supreme Court Grants Certiorari in Alaska Case

Posted October 01, 2015, 1:24 P.M. ET

By Rebecca Wilhelm

The U.S. Supreme Court said today it will review whether Section 103(c) of the Alaska National Interest Lands Conservation Act prohibits the National Park Service from exercising regulatory control over state, native corporation and privately owned land that is physically located within the boundaries of the National Park System.

The Supreme Court granted certiorari in a lawsuit challenging the application of a park service regulation banning hovercraft use in all national parks to Alaska land and waters. The petitioner, John Sturgeon, argued that the regulation should not be extended to state navigable waters within Alaska's Yukon-Charley Preserve. Specifically, he argued that the plain statutory language removes nonfederal land from federal regulations promulgated to manage public land.

The U.S. Court of Appeals for the Ninth Circuit held that the plain text of Section 103(c) only excludes nonfederal land from regulations that are solely applicable to public land. The hovercraft ban regulation, the Ninth Circuit ruled, is not limited to public land but is generally applicable to all land and waters within the park system, including the Alaska preserve.

Oral argument has not yet been scheduled in the case.

Heitkamp Oil Bill Unlikely to Go Beyond Committee: Corker

Posted October 01, 2015, 12:53 P.M. ET

By Ari Natter

A bill to lift the ban on most crude oil exports approved today by the Senate Banking Committee likely won't be the legislative vehicle ultimately brought forward to end the trade restriction, Sen. Bob Corker (R-Tenn.) said during the markup.

The adoption of an amendment outside of the committee's jurisdiction by Sen. Pat Toomey (R-Pa.) related to Iran sanctions means "what we do today will not be the vehicle to move this effort ahead," Corker said.

The bill (S. 1372) by Sen. Heidi Heitkamp (D-N.D.) was approved on a 13-9 vote.

Oil Exports Could Be Included in Budget Deal, Corker Says

Posted October 01, 2015, 12:51 P.M. ET

By Ari Natter

A policy rider to lift the ban on most crude oil exports may be added to a year-end spending package to be negotiated with the White House to keep the government funded after Dec. 11, Sen. Bob Corker (R-Tenn.) said today.

"It's possible this ends up being one of the issues that is a part of that," Corker told Bloomberg BNA. "There are a lot of things being talked about, but I think this is something that could be a consideration as a part of the spending bill."

Senate Majority Leader Mitch McConnell (R-Ky.) said Sept. 29 he is preparing to begin talks with President Barack Obama and other congressional leaders soon on a possible two-year budget deal.

Major Corporations Urge Strong Climate Deal in Paris

Posted October 01, 2015, 10:42 A.M. ET

By Anthony Adragna

Mars Inc., General Mills, Inc., Nestle USA, Unilever, Ben & Jerry's, Kellogg Co. and other companies today urge U.S. and world leaders in a letter to reach a "sound agreement, properly financed" to combat climate change when nations from around the world negotiate later this year in Paris.

"Now is the time to meaningfully address the reality of climate change," the letter states. "We are asking you to embrace the opportunity presented to you in Paris, and to come back with a sound agreement, properly financed, that can affect real change."

The companies also pledged to redouble their efforts to make their supply chains more sustainable, speak more transparently about their efforts to address climate change and advocate for governments to set "clear, achievable, measurable and enforceable science-based targets for carbon emissions reductions."

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Subject: Seminar by Dr. Andrew Lindstrom (NERL-RTP): "Current research priorities for per and polyfluoroalkyl substances (PFAS): implications of UCMR3 results and industry reformulations" (presentation attached)
Start Date/Time: Tue 9/22/2015 6:00:00 PM
End Date/Time: Tue 9/22/2015 7:00:00 PM
Lindstrom Cincinnati 9-22-15.pptx

NERL VTC Locations:
Las Vegas - Exc Conference Room 212
RTP - E230A
Athens - no available rooms

Call-In Info:
US/Canada Dial-In Number:
Int'l/Local Dial-in #:
Conference Code: Conference Code/ Ex. 6

Conference Code/ Ex. 6

Biography
Andrew B. Lindstrom has a Ph.D. from the University of North Carolina's School of Public Health and has been working for the United States Environmental Protection Agency's (USEPA) National Exposure Research Laboratory (NERL) for the past 25 years. He is currently working in the Exposure Measurements and Analysis Branch where his areas of expertise include measurement

of trace level contaminants in environmental and biological matrices and human exposure assessment.

Abstract

Current research priorities for per and polyfluoroalkyl substances (PFAS): implications of UCMR3 results and industry reformulations
With more than a decade of intensive scientific research and increasing regulatory pressure worldwide, the sources of per- and polyfluoroalkyl substances (PFAS) in the environment and routes of human exposure still need to be fully characterized. Research has documented that PFAS contamination in the environment is often associated with direct emissions from chemical production facilities, effluents from municipal wastewater treatment plants, and the application of sewage sludge in agricultural areas. International regulatory efforts have succeeded in reducing the traditional PFAS such as PFOS and PFOA in many areas, but they have also served to relocate the production of these legacy materials to regions with developing economies and less stringent regulation. Moreover, a whole new generation of PFAS has replaced the regulated materials previously produced in the Western world. Interim results from the USEPA's Unregulated Contaminant Monitoring Rule 3 (UCMR3) program now provide detailed information on the occurrence of six PFAS in finished drinking water across the US. These data serve as an important source of information to: 1) identify and classify the sources of current PFAS contamination, 2) assess, test, and improve mitigation and control technologies used by water providers, 3) assess the importance of drinking water as a route of human exposure in specific populations, and 4) identify communities to conduct epidemiological studies to investigate the origins of PFAS-related disease.

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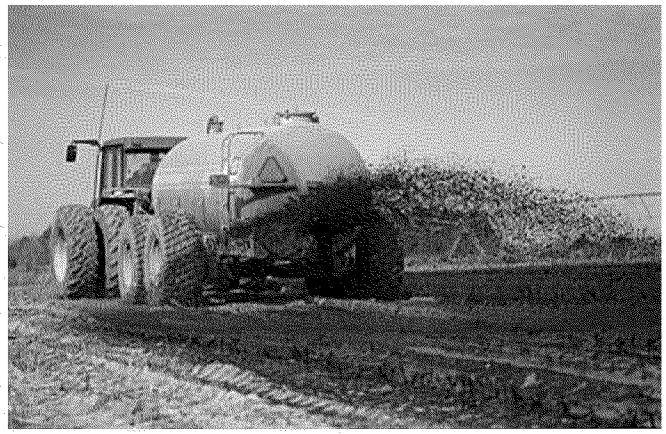
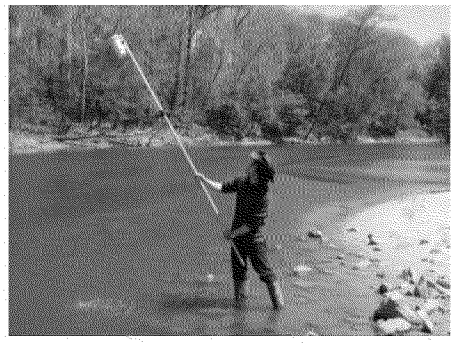
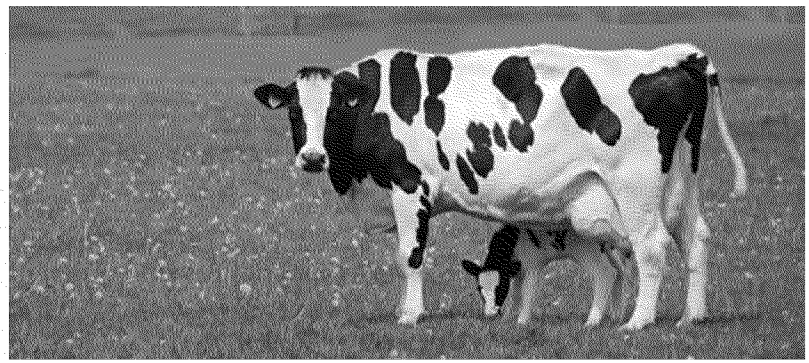
Current research priorities for per- and polyfluoroalkyl substances (PFAS): implications of UCMR3 results and industry reformulations

Andrew B. Lindstrom

U.S. Environmental Protection Agency, National Exposure Research Laboratory, Research Triangle Park, NC



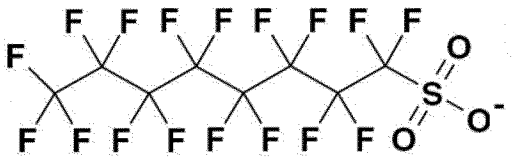




USEPA
AWBERC Auditorium
Cincinnati, OH
September 22, 2015

Presentation Outline

Discuss per- and polyfluoroalkyl substance (PFAS), their properties, toxicity, regulations



Discuss “replacement” PFAS

Summary of interim results from the UCMR3 program, implications, and opportunities

Long-term outlook regarding PFAS

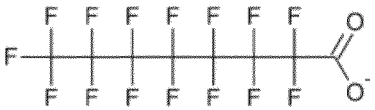
Three major considerations

Principle of Precaution…"where there are threats of serious or irreversible damage, lack of full scientific certainty shall not be used as a reason for postponing cost effective measures to prevent environmental degradation". *Commission of the European Communities: (2000)* *http://ec.europa.eu/dgs/health_consumer/library/pub/pub07_en.pdf*

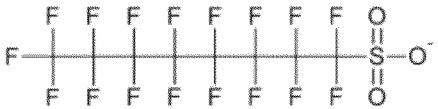
Green Chemistry…always strive to use the least toxic alternative available, with a preference for compounds that quickly and harmlessly degrade to their original starting materials
Anastas PT, Warner JC. 1998. Green Chemistry: Theory and Practice. New York, NY Oxford University Press

Environmental Justice…"the avoidance of hazards and acquisition of benefits through relationships that negatively impact the environment of others" *Steve Wing, (2015) University of North Carolina*

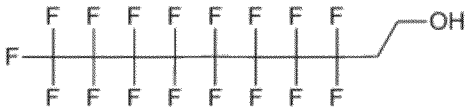
Some per- and polyfluoroalkyl substance (PFAS)



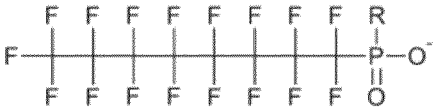
Perfluorocarboxylic acids
(ex. PFOA)



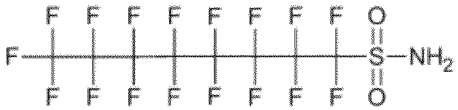
Perfluorosulfonic acids
(ex. PFOS)



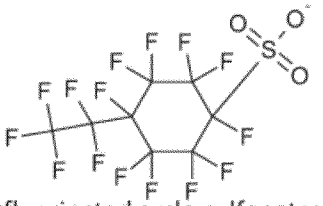
Fluorotelomer alcohol
(ex. 8:2 FTOH)



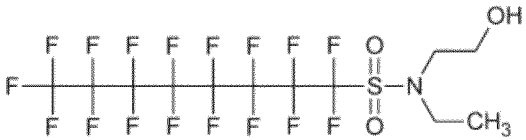
Perfluorophosphonic/phosphinic acids
(ex. If R=OH then PFOPA
If R=C8 perfluoroalkane then 8:8 PFPi)



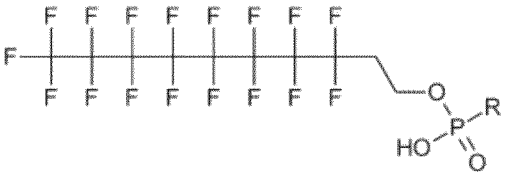
Perfluorosulfonamide
(ex. FOSA)



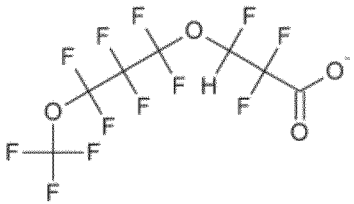
Perfluorinated cyclo sulfonates
(ex. PFECHS)



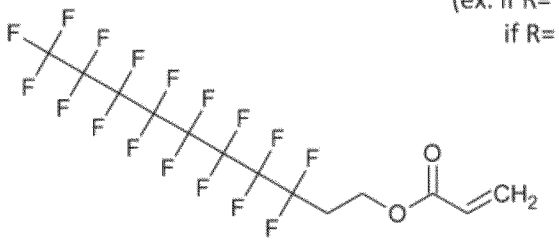
Perfluorosulfonamidoethanol
(ex. N-EtFOSE)



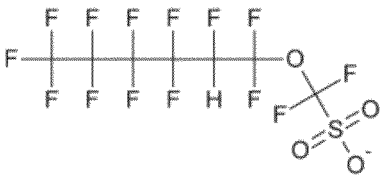
Fluorotelomer phosphate esters
(ex. if R= OH then 8:2 monoPAP
if R= 8:2 FTO ester then 8:2 diPAP)



Polyfluorinated ether carboxylates
(ex. 4,8-dioxa-3H-perfluorononanoate)



Polyfluorinated polymeric unit
(ex. 1H,1H,2H,2H-perfluorodecyl acrylate)

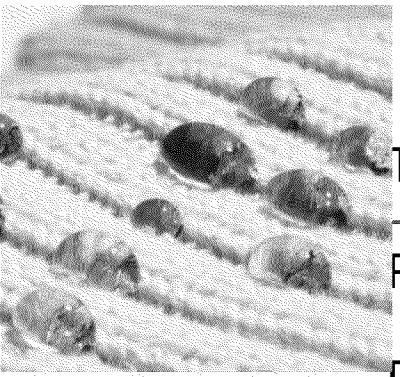


Polyfluorinated ether sulfonates
(ex. Perfluoro [hexyl ethyl ether sulfonate])

Figure 1. Generic structures for polyfluorinated compounds. The $n = 8$ linear carbon structures are shown for many of these examples, but $n = 4-14$ linear and/or branched carbon units are generally possible.

PFAS used in many products

Many PFAS are toxic, extremely persistent, and bioaccumulative



Textile treatments

Paints

Pesticides

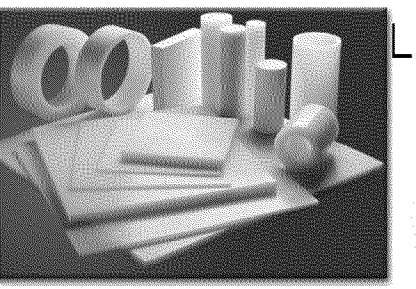
Floor polish

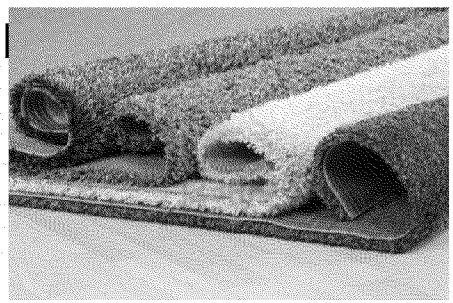
Denture cleaner

Polymers

Adhesives

Lul





Paper coatings

Surfactants

Fire-fighting foam

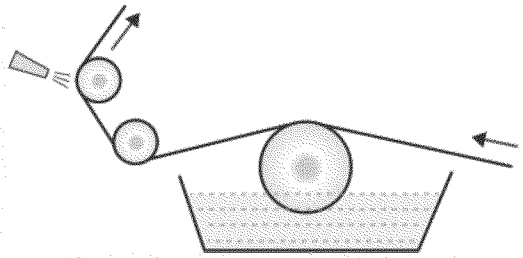
Photographic film

Shampoo

Non-stick cookware

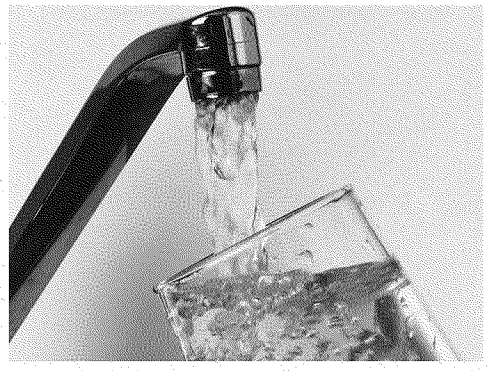
Caulks

Carpets



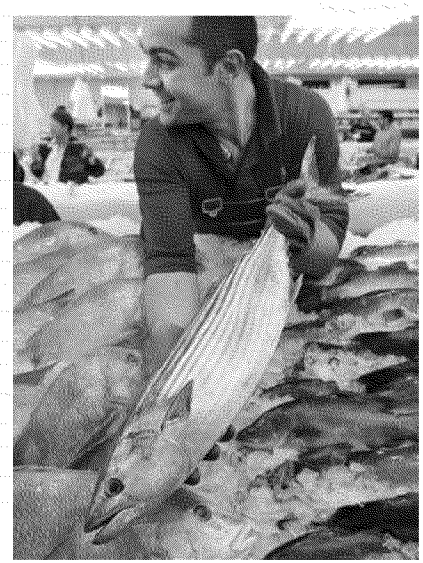
Sources of PFAS exposure for humans

Best documented source includes contaminated drinking water around industrial operations e.g., Cottage Grove, Minnesota; Parkersburg, West Virginia; Dalton, Georgia; Decatur, Alabama; Arnsberg, Germany; Osaka, Japan

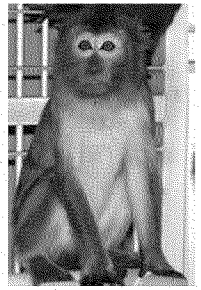


Food is also implicated in many studies (mostly modeling), but there are few good data on food items (complex matrices). Exception is fish, which is a well documented source.

Fromme et al. 2009, Inter. J. Hyg. & Envr. Heath (212) 239-270



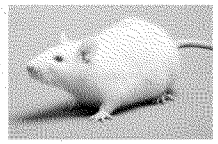
PFAS Health Effects Summary



Animal toxicity

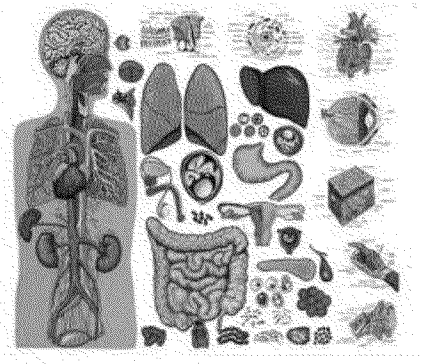
- Causes liver, immune system, developmental, endocrine, metabolic, and neurobehavioral toxicity.
- PFOA and PFOS caused tumors in chronic rat studies.

Post et al., (2012) Perfluorooctanoic acid (PFOA) , an emerging drinking water contaminant: A critical review of recent literature, *Environmental Research* (116) 93-117



Human health effects associated with PFAS in the general population and/or communities with contaminated drinking water include:

- ↑ cholesterol
- ↑ uric acid
- ↑ liver enzymes
- ↓ birth weight
- ↓ vaccine response
- Thyroid disease
- Osteoarthritis
- Diabetes
- Testicular and kidney cancer
- Pregnancy-induced hypertension
- Ulcerative colitis
- Effects in young adulthood from prenatal exposures
  - *Obesity in young women.*
  - *↓ sperm count in young men.*



Steenland et al., (2014), *Epidemiology* (25) 167-169; http://www.c8sciencepanel.org/

2009 US Environmental Protection Agency Short-term Provisional Health Advisories

Provisional Health Advisory levels for PFOS and PFOA in drinking water

PFOS = 200 ng/L PFOA = 400 ng/L

Short term exposure only (months) – no long-term (chronic) standard set

State of New Jersey long-term chronic health-based drinking water guidance for PFOA of 40 ng/L issued in 2007

* Some experts calling for reduction in EPA's Provisional standards by a factor of 100 – 1000 to be truly protective for chronic exposures

PFOS = 2 ng/L PFOA = 4 ng/L

* Immunotoxicity of perfluorinated alkylates: calculation of benchmark doses based on serum concentrations in children Grandjean, P ; Budtz-Jorgensen, E ;Environmental Health (12:35) DOI: 10.1186/1476-069X-12-35, APR 19 2013

US Environmental Protection Agency
PFOA Stewardship Program

In January 2006, USEPA started this program to help minimize impact of PFOA in the environment

Eight major international companies have agreed to participate (including 3M, DuPont, Asahi Glass, Daikin)

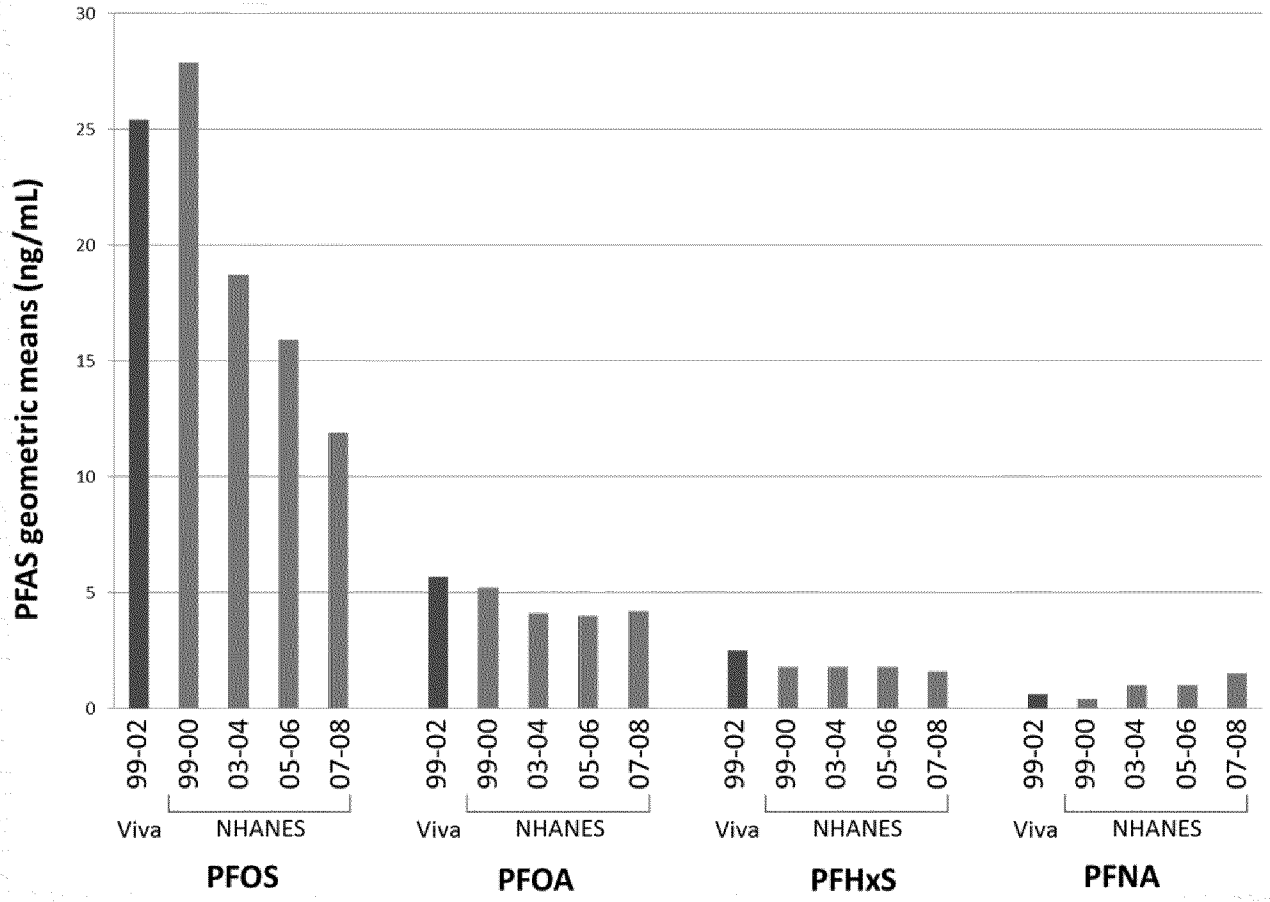
Agreement to voluntarily reduce factory emissions and product content of PFOA and related compounds* on a global basis by 95% no later than 2010

Agreement to work toward total elimination of emissions and product content of these compounds by 2015

Based on emissions and content determinations made for 2006

* Includes PFOA, precursor chemicals that can break down to PFOA, higher homologues (C9 and larger)

Trends in PFAS Serum Levels in US



Sagiv et al. (2015) *Environ. Sci. Technol.* DOI: 10.1021/acs.est.5b02489

Table 2. Geometric mean and 95% confidence interval and selected percentiles of PFOS, PFOA, PFHxS, and PFNA serum concentrations (ng/mL) for the U.S. population 12 years of age and older: Data from NHANES 2011-2012 [a]

	Geometric Mean (95% Confidence Interval)		Selected Percentiles			
			50th	75th	90th	95th
PFHxS	1.28	1.15-1.43	1.27	2.26	3.81	5.43
PFOS	6.31	5.83-6.82	6.51	10.48	15.62	21.68
PFOA	2.08	1.95-2.22	2.08	3.02	4.35	5.67
PFNA	0.88	0.80-0.97	0.86	1.30	1.95	2.54

Perspectives | Brief Communication

The Madrid Statement on Poly- and Perfluoroalkyl Substances (PFASs)

http://dx.doi.org/10.1289/ehp.1509934

As scientists and other professionals from a variety of disciplines, we are concerned about the production and release into the environment of an increasing number of poly- and perfluoroalkyl substances (PFASs) for the following reasons:

1. PFASs are man-made and found everywhere. PFASs are highly

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Environmental Health Perspectives • VOLUME 123 | NUMBER 5 | May 2015

Legacy PFAS continue to be a problem – persistent;

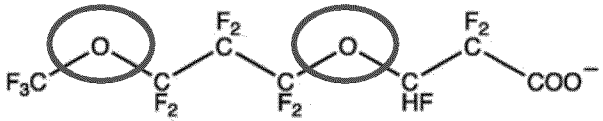
production of legacy compounds pushed to developing world;

almost no information on toxicity, transport and fate, bioaccumulation and, persistence of replacement compounds

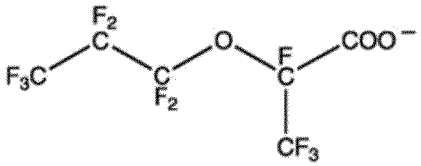
Z. Wang et al. / Environment International 60 (2013) 242–248

Fluoropolymer manufacture

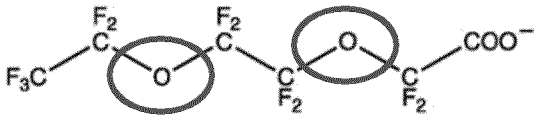
ADONA (CAS No. 958445-44-8)



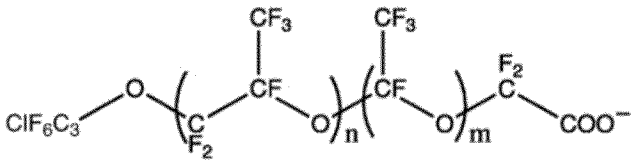
GenX (CAS No. 62037-80-3)



Asahi's product (CAS No. 908020-52-0)

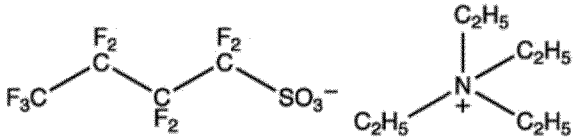


Solvay's product (CAS No. 329238-24-6)

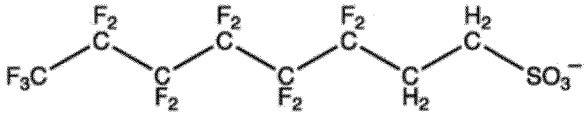


Metal plating

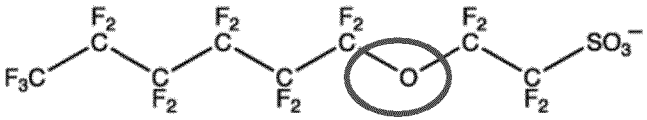
N(Et)$_4$-PFBS (CAS No. 25628-08-4)



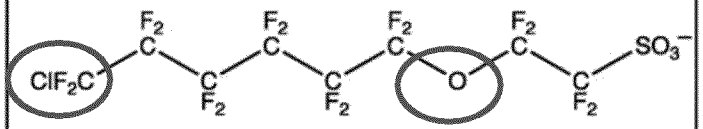
6:2 FTSA (CAS No. 27619-97-2)



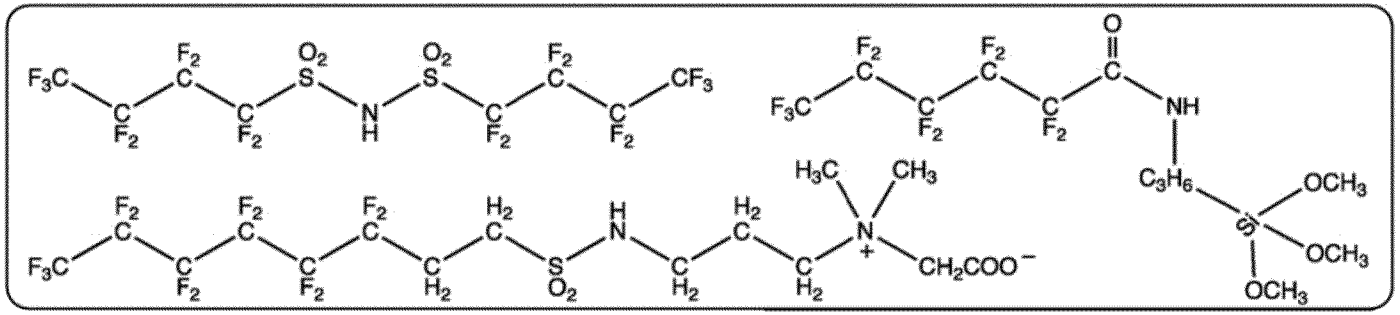
F-53 (CAS No. 754925-54-7)



F-53B (CAS No. 73606-19-6)



Fire fighting foams and miscellaneous



Unknown characteristics of “alternative” fluorinated compounds

- Actual identities of alternatives unknown in industrial sectors and geographical regions that are not well regulated

- Data on environmental and human health effects are incomplete (at best) and more often nonexistent

- Data on degradability, bioaccumulation, and toxicity (environmental and human) are incomplete (at best) or completely lacking

- Information on production volume and environmental emissions not available

Z. Wang et al. / Environment International 60 (2013) 242–248

US Environmental Protection Agency Drinking Water Activities

PFOS and PFOA on the Candidate Contaminant List (CCL) for possible regulation by National Primary Drinking Water Regulations

Unregulated Contaminant Monitoring Regulation 3 (UCMR3) announced in March 2011, approved by OMB in March 2012

UCMR selects candidate pollutants for national screening every five years

All PWS serving over 10,000 people and 800 PWS serving fewer than 10,000 people required to monitor for compounds for a continuous 12 month period between January 2013 and December 2015

UCMR 3 includes: PFOS (40 ng/L), PFOA (20 ng/L), PFNA (20 ng/L), PFHxS (30 ng/L), PFHpA (10 ng/L), PFBS (90 ng/L)

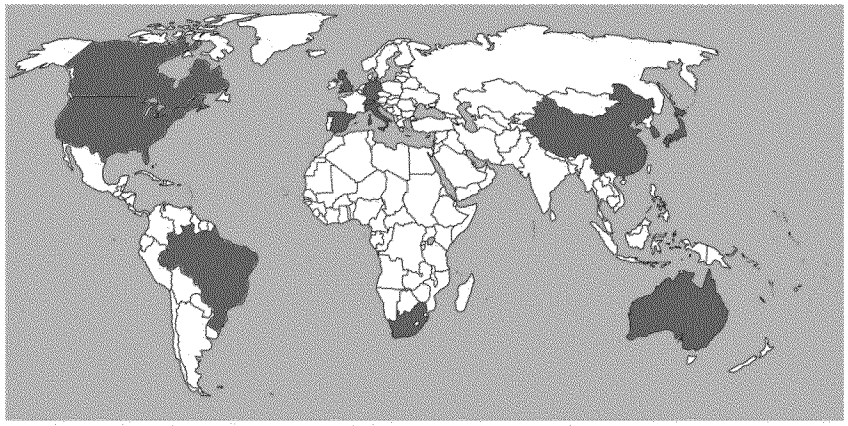
Unregulated Contaminant Monitoring Regulation 3 (UCMR3)

Interim results most recently released January 2015
http://water.epa.gov/lawsregs/rulesregs/sdwa/ucmr/data.cfm

3,605 Public Water Systems (PWS) with results so far

185 records with PFOS above the Reporting Limit of 40 ng/L,
68 PWS with levels above 40 ng/L

220 records with PFOA above the Reporting Limit of 20 ng/L
72 PWS with levels above 20 ng/L



PFASs have been detected worldwide in surface water, wastewater, groundwater, drinking water, and landfill leachates.

UCMR3 requires monitoring for six PFASs in US drinking water. Monitoring began in 2013, and latest data release was January 2015.

PFAS	MRL (ng/L)	Occurrence (%)	Max (ng/L)
C7	10	0.66	82 (NY, DE, PA)
C8	20	0.96	349 (PA)
C9	20	0.05	55.8 (NJ, PA)
PFBS	90	0.03	150 (CO, PA, AL)
PFHxS	30	0.61	680 (DE, PA, CO)
PFOS	40	0.81	1,800 (DE, CO, PA)

To date: 22,942 samples from 3,605 PWSs

PFAS detects: 351 samples (1.5%) from 132 PWSs (3.7%)

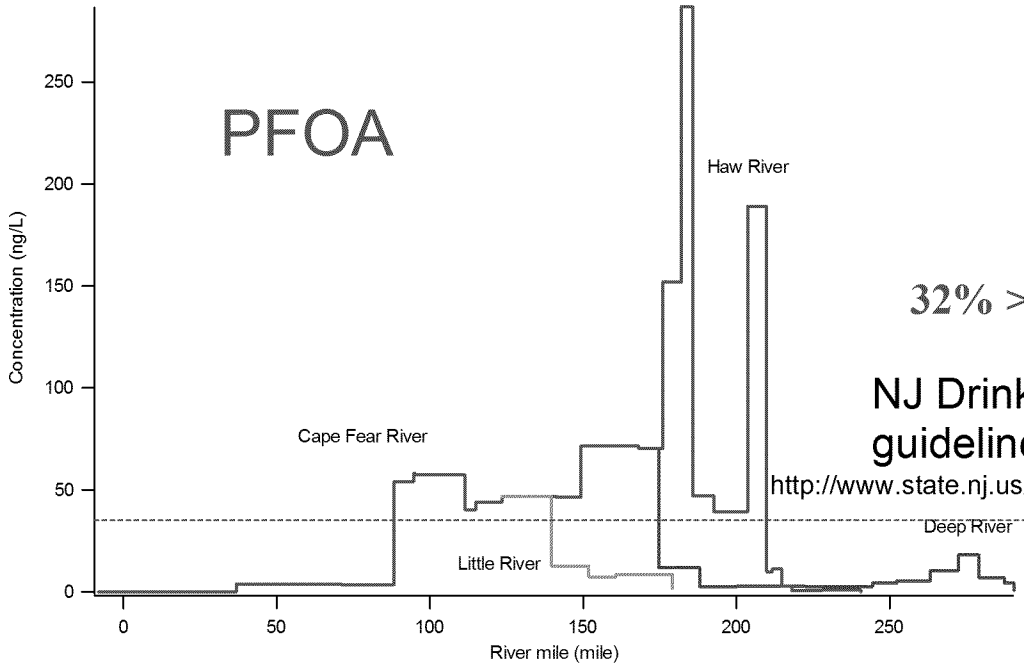
Of samples with PFAS detects: 22.8% derived from surface water

UCMR3 data for the State of North Carolina as of January 1, 2015

	A	B	C	D	E	F
1	PWSName	FacilityName	Date	PFAS	ng/L	State
495	City of Albemarle	Tuckertown	10/2/2013	PFHpA	10	NC
496	City of Albemarle	Town	10/2/2013	PFHpA	10	NC
497	Fayetteville Public Works Commission	Hoffner WTP	1/17/2013	PFHpA	10	NC
498	Old North Utilities Services - Fort Bragg	Fayetteville/Harnett Co. Intertie	1/15/2014	PFHpA	11	NC
499	Moore County Public Utilities - Pinehurst	EMWD Intertie	9/25/2013	PFHpA	12	NC
500	CFPUA - Wilmington	Sweeny WTP	6/9/2014	PFHpA	12	NC
501	Brunswick Regional Water & Sewer H2GO	Brunswick County Intertie	7/15/2014	PFHpA	12.03	NC
502	Old North Utilities Services - Fort Bragg	Fayetteville/Harnett Co. Intertie	10/17/2013	PFHpA	13	NC
503	CFPUA - Wilmington	Sweeny WTP	12/5/2013	PFHpA	14	NC
504	Fayetteville Public Works Commission	Glenville WTP	10/28/2013	PFHpA	15	NC
505	Moore County Public Utilities - Pinehurst	EMWD Intertie	2/28/2013	PFHpA	16	NC
506	Brookwood Comm. Water System	Well #34	2/24/2014	PFOA	20	NC
507	Town of Holly Springs	Harnett County Water Intertie	10/23/2013	PFHpA	20	NC
508	Harnett County Department of Public Utilit	Clearwell	6/11/2014	PFHpA	20	NC
509	Harnett County Department of Public Utilit	Clearwell	9/17/2014	PFOA	20	NC
510	Brookwood Comm. Water System	Well #39	2/25/2014	PFOA	21	NC
511	Moore County Public Utilities - Pinehurst	EMWD Intertie	5/16/2013	PFOA	23	NC
512	Moore County Public Utilities - Pinehurst	EMWD Intertie	11/26/2013	PFHpA	25	NC
513	CFPUA - Wilmington	Sweeny WTP	9/8/2014	PFHpA	27	NC
514	City of Sanford	Plant Clearwell	12/19/2013	PFHpA	29	NC
515	Town of Fuquay-Varina	Harnett County Water Intertie	9/23/2014	PFOA	30	NC
516	Orange Water & Sewer Authority	Plant Clearwell	2/11/2014	PFOA	30	NC
517	Moore County Public Utilities - Pinehurst	Fire Station #1	9/12/2013	PFHxS	40	NC
518	Harnett County Department of Public Utilit	Clearwell	9/17/2014	PFHpA	40	NC
519	Cliffdale West	Well #4	9/9/2014	PFOS	41	NC
520	Moore County Public Utilities - Pinehurst	Fire Station #1	2/27/2013	PFHxS	42	NC
521	City of Greensboro	Mitchell Filter	6/3/2014	PFOS	44	NC
522	Town of Fuquay-Varina	Harnett County Water Intertie	9/23/2014	PFHpA	50	NC
523	Bell Arthur Water Corp.	Well #6 (Otter Creek)	1/6/2014	PFOS	50	NC
524	City of Greensboro	Mitchell Filter	9/9/2014	PFOS	64	NC
525	Moore County Public Utilities - Pinehurst	Fire Station #1	9/12/2013	PFOS	73	NC
526	Moore County Public Utilities - Pinehurst	Fire Station #1	2/27/2013	PFOS	76	NC
527	Dover Water Department	Griffin Well Treatment Plant (Mast	3/24/2014	PFHpA	15	NH

The Cape Fear River Basin

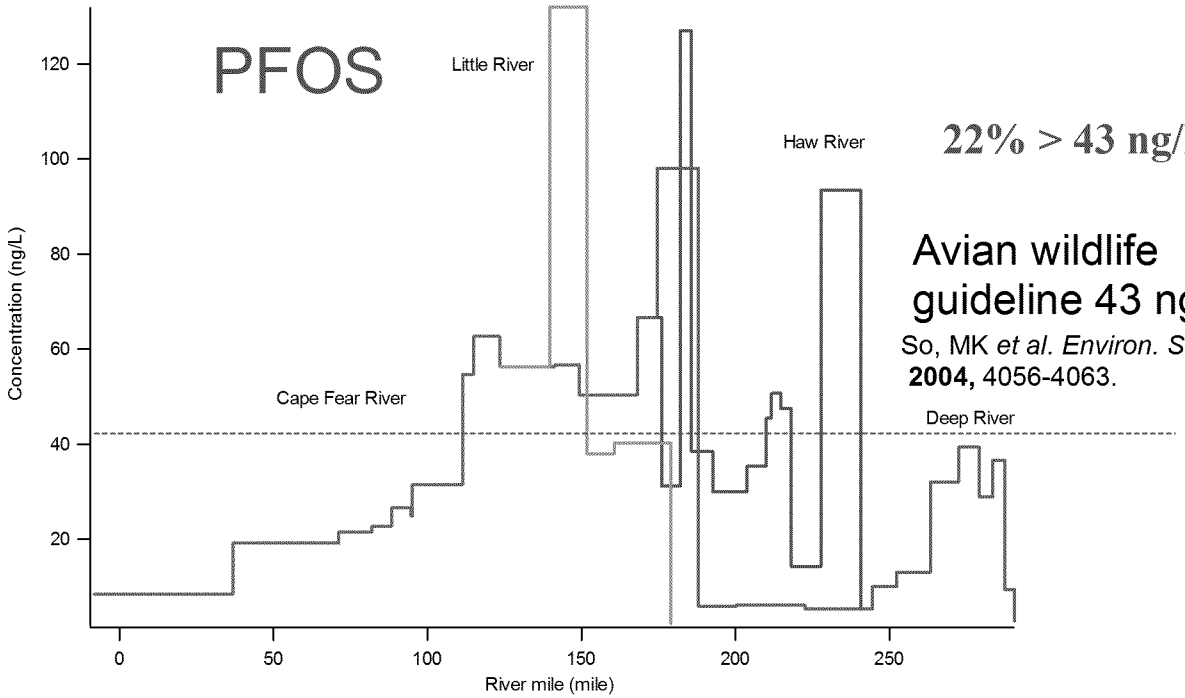
PFAS profiles in the Cape Fear Drainage Basin, North Carolina, USA



32% > 40 ng/L

NJ Drinking water guideline 40 ng/L

http://www.state.nj.us/dep/watersupply/pfoa.htm



22% > 43 ng/L

Avian wildlife guideline 43 ng/L

So, MK *et al. Environ. Sci. Technol.* **2004,** 4056-4063.

Nakayama *et al. Environ. Sci. Technol.* **2007,** 41: 5271-5276

Environmental Science & Technology

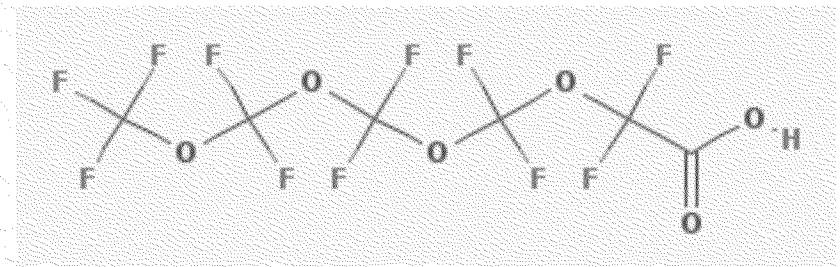
Article

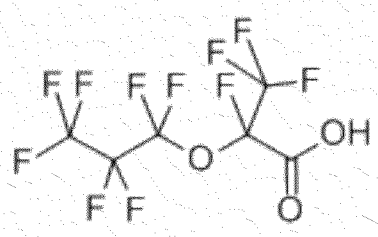
pubs.acs.org/est

Identification of Novel Perfluoroalkyl Ether Carboxylic Acids (PFECAs) and Sulfonic Acids (PFESAs) in Natural Waters Using Accurate Mass Time-of-Flight Mass Spectrometry (TOFMS)

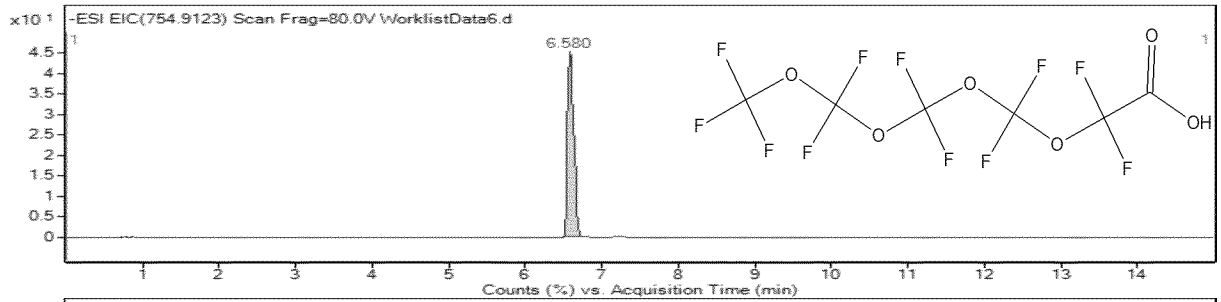
Mark Strynar,*[,†] Sonia Dagnino,[†,‡] Rebecca McMahen,[†,‡] Shuang Liang,[†,‡] Andrew Lindstrom,[†] Erik Andersen,[†] Larry McMillan,[§] Michael Thurman,[∥] Imma Ferrer,[∥] and Carol Ball[⊥]

[†]National Exposure Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina 27711, United States

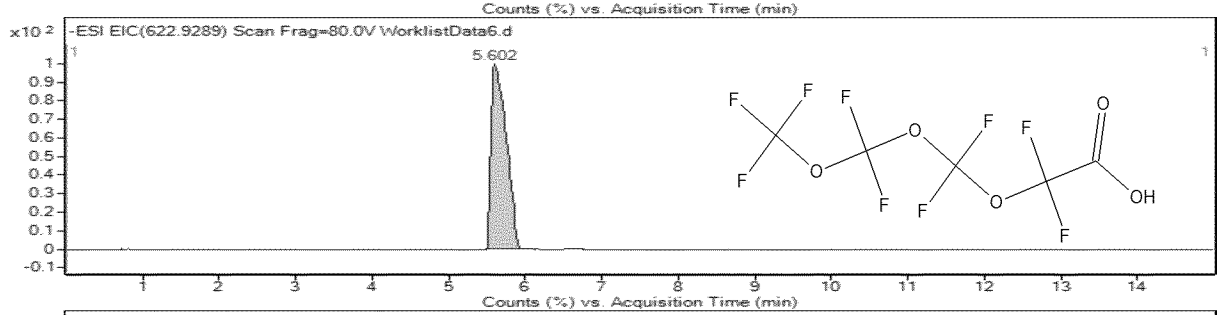




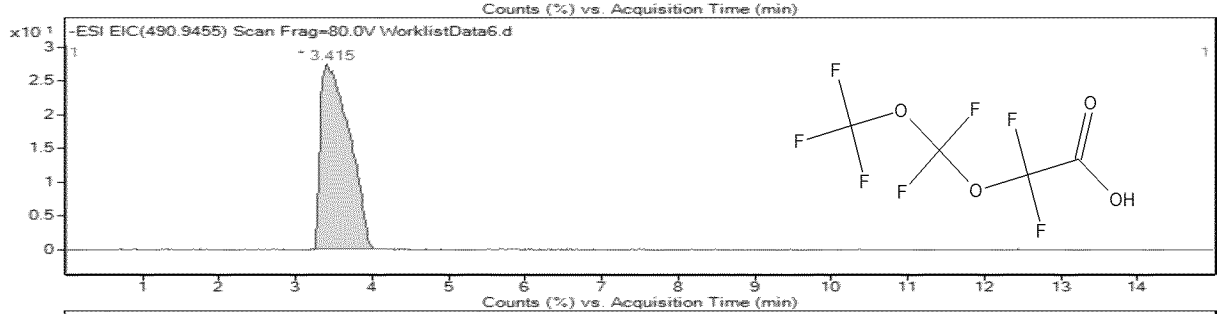
Replacement compounds found in the Cape Fear River, North Carolina, most likely source is fluorochemical production facility, source water for Wilmington and other cities near the coast



Molecular Formula = $C_6HF_{11}O_6$
Monoisotopic Mass = 377.959748 Da
[M-H]- = 376.952472 Da



Molecular Formula = $C_5HF_9O_5$
Monoisotopic Mass = 311.968027 Da
[M-H]- = 310.96075 Da



Molecular Formula = $C_4HF_7O_4$
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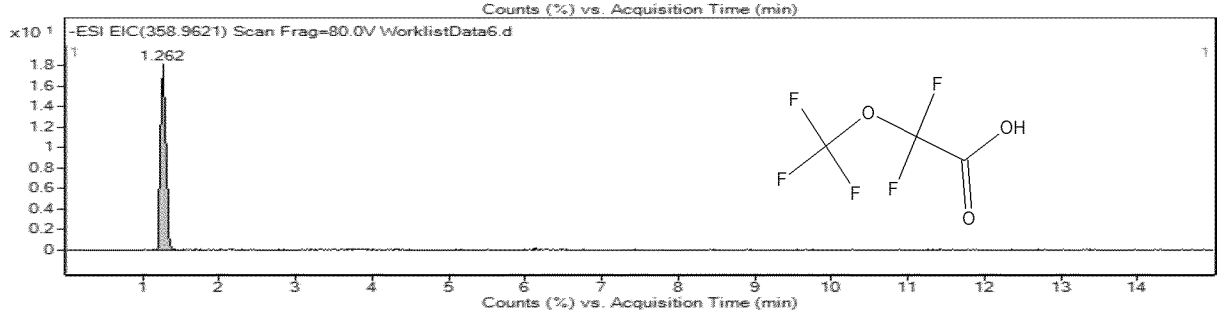
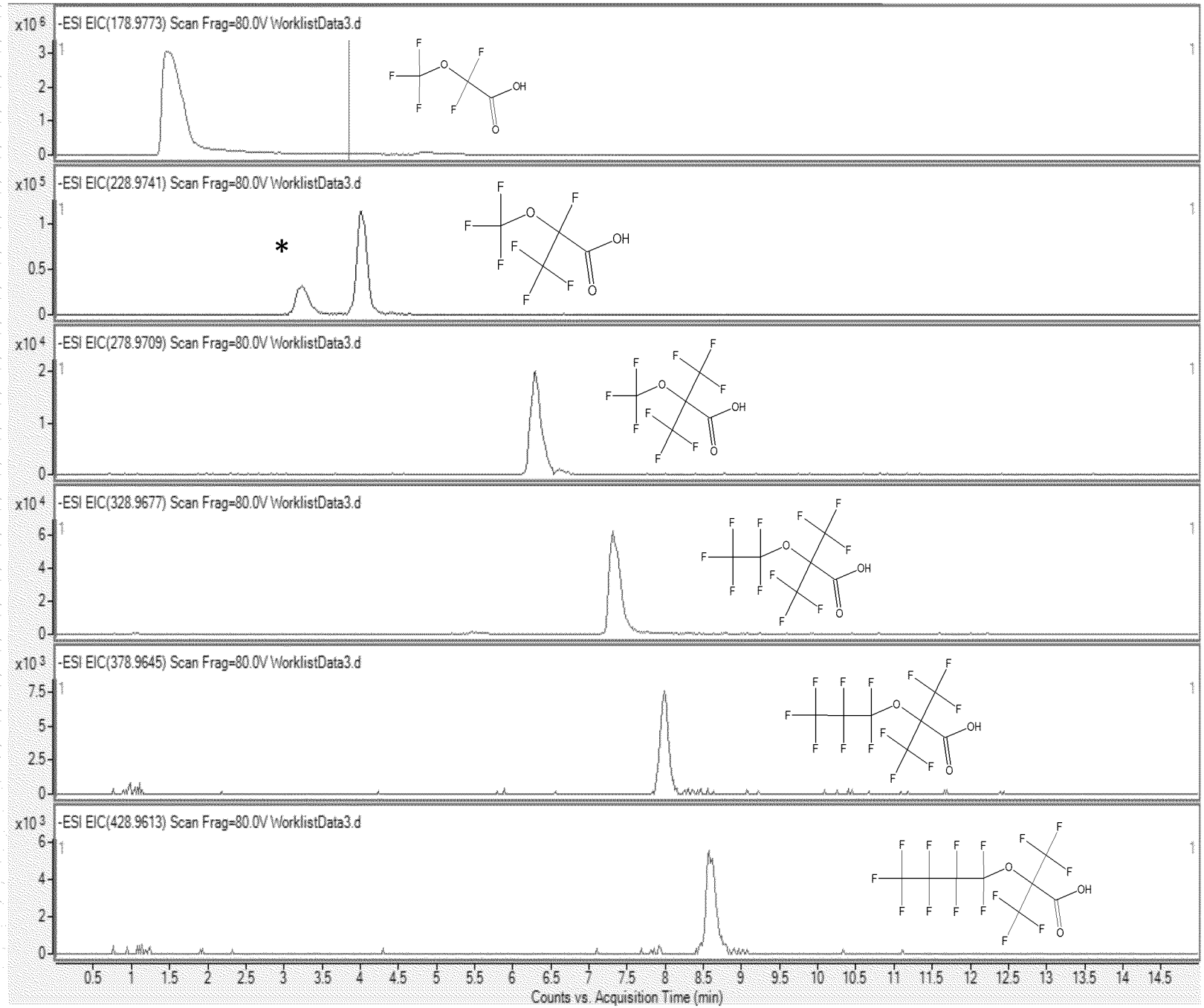
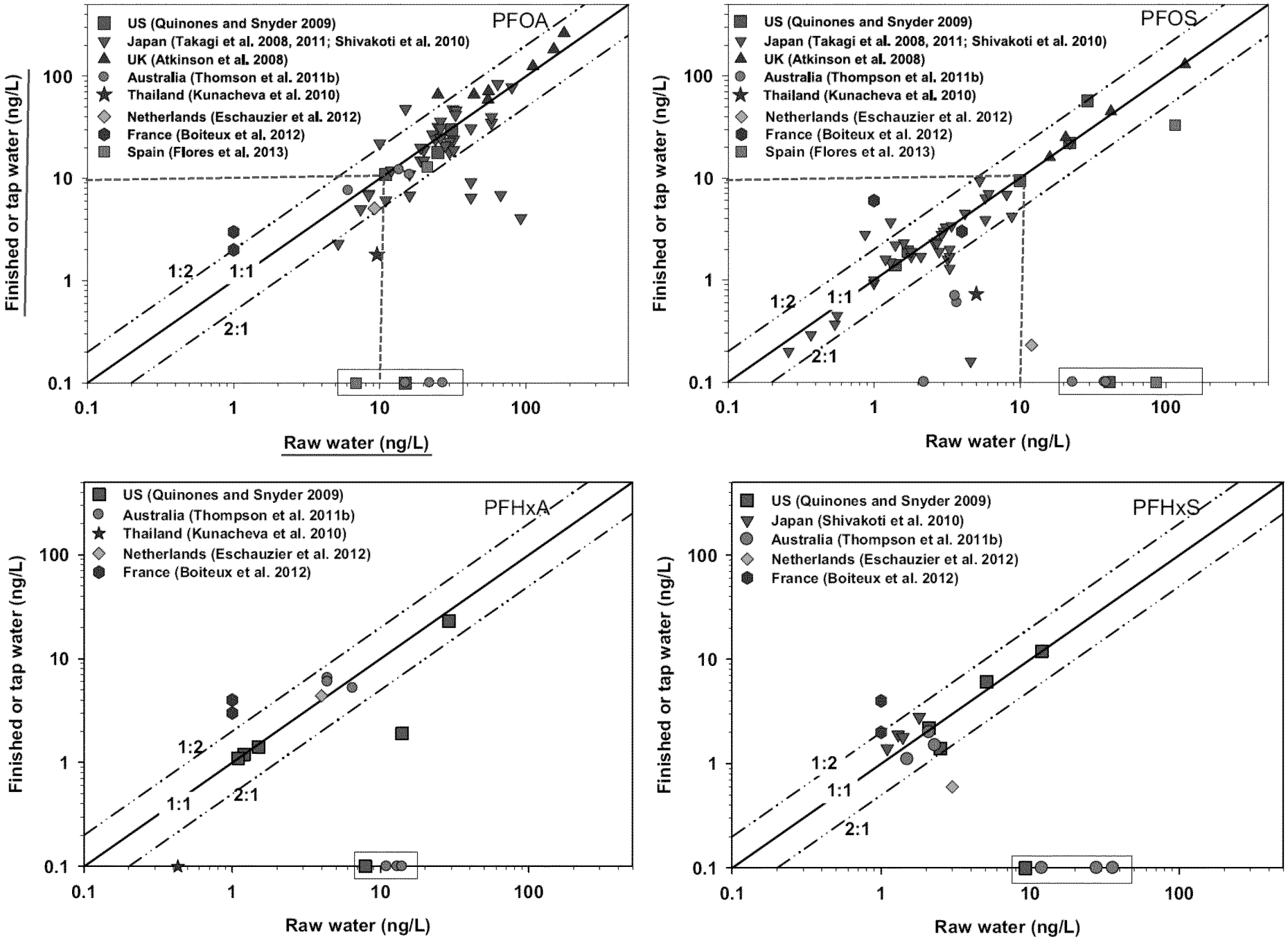
[M-H]- = 244.969029 Da



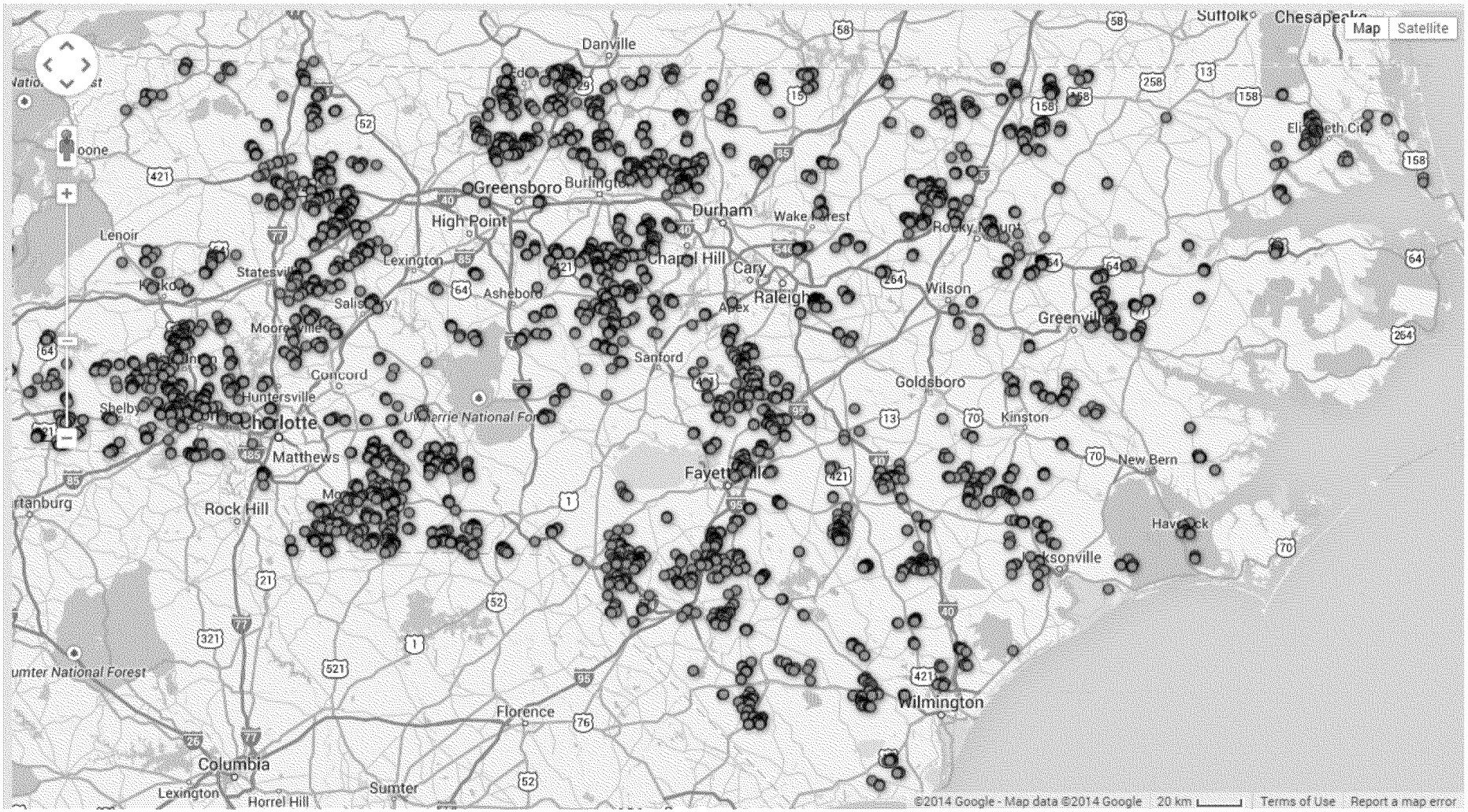
Molecular Formula = $C_3HF_5O_3$
Monoisotopic Mass = 179.984585 Da
[M-H]- = 178.977308 Da


x10 6 -ESI EIC(178.9773) Scan Frag=80.0V WorklistData3.d
x10 5 -ESI EIC(228.9741) Scan Frag=80.0V WorklistData3.d
*
x10 4 -ESI EIC(278.9709) Scan Frag=80.0V WorklistData3.d
x10 4 -ESI EIC(328.9677) Scan Frag=80.0V WorklistData3.d
x10 3 -ESI EIC(378.9645) Scan Frag=80.0V WorklistData3.d
x10 3 -ESI EIC(428.9613) Scan Frag=80.0V WorklistData3.d
Counts vs. Acquisition Time (min)

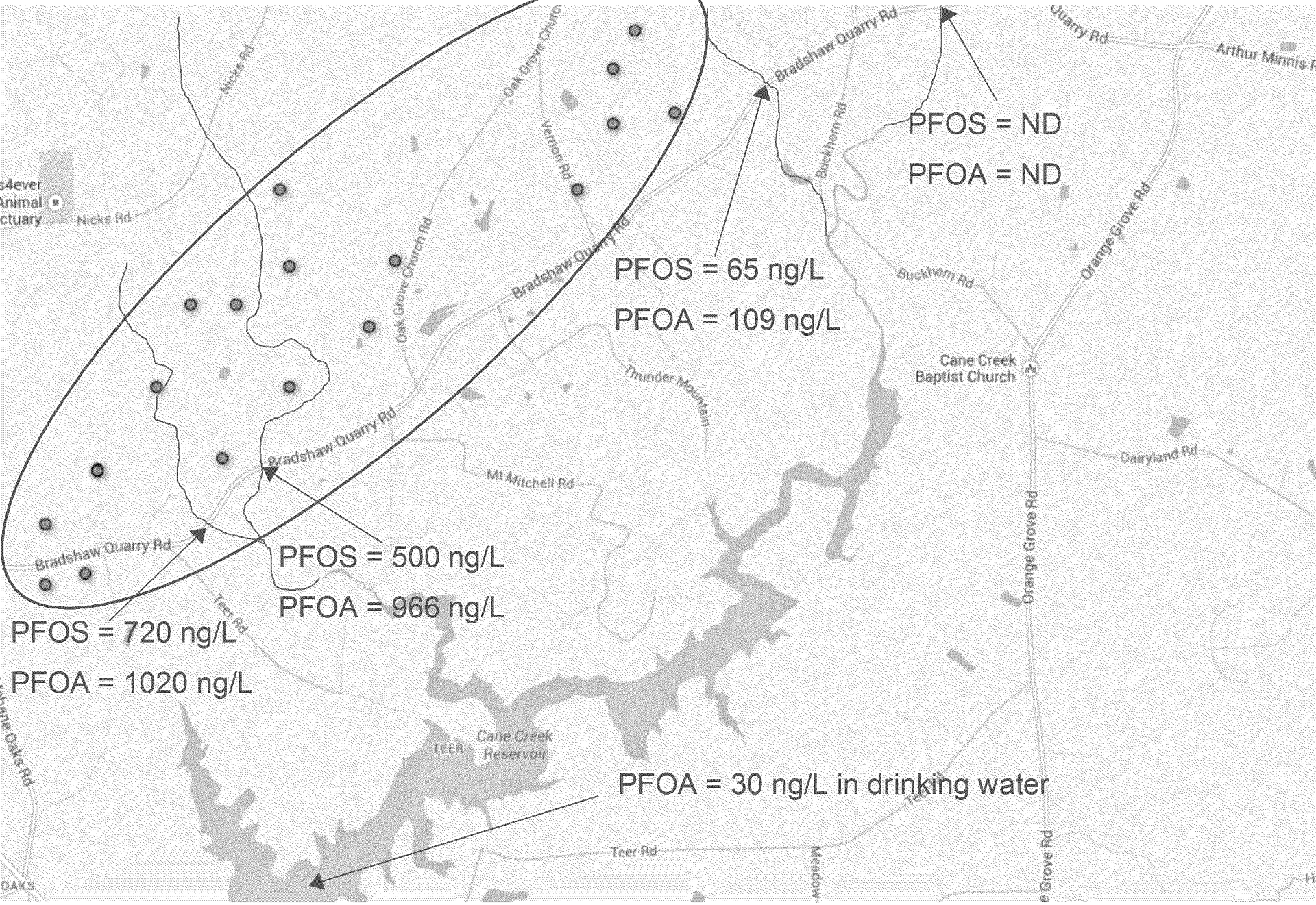


Rahman et al., (2014) *Water Research,* 50:318-340

4,146 permitted sludge application sites in North Carolina

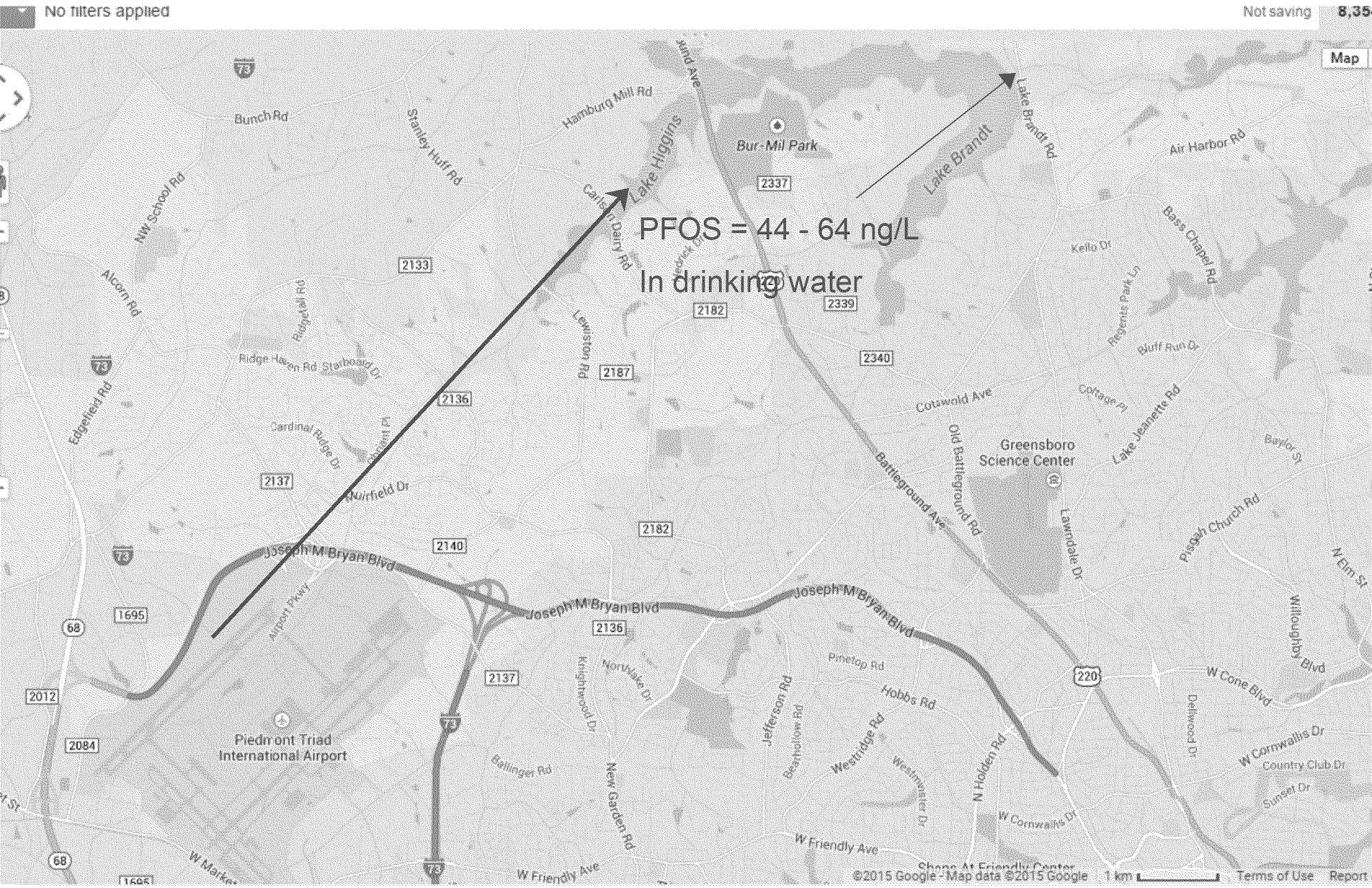


Sewage sludge in Cane Creek Resevoir basin - Serving Orange County, NC

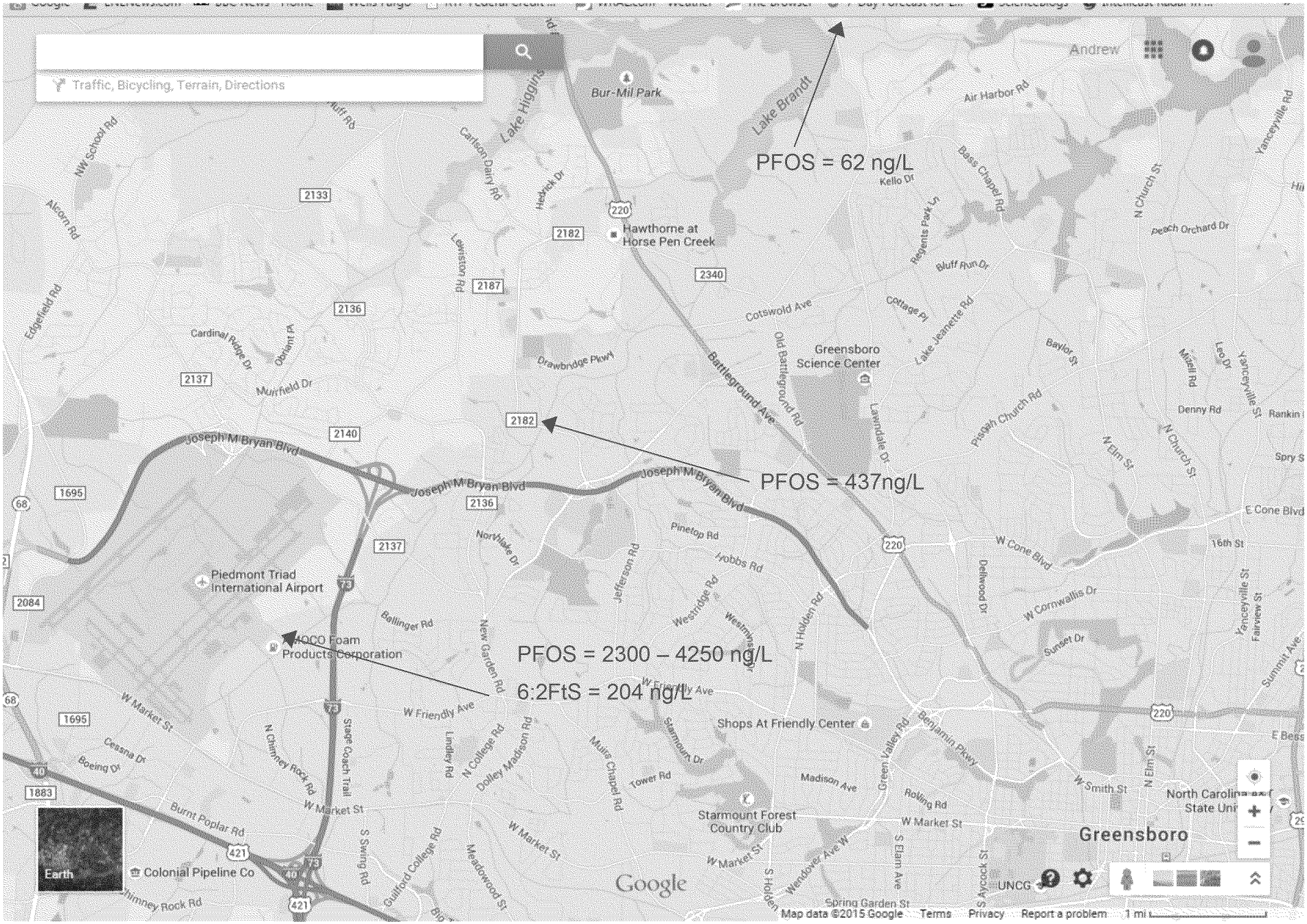


Traffic, Bicycling, Directions
Andrew
Oak Grove Church Rd
Bradshaw Quarry Rd
1115
1114
1116
1100
Grange Grove Rd
Cane Creek
Map
Google
Imagery ©2015 Google, Map data ©2015 Google
Terms
Privacy
Report a problem
2000 ft

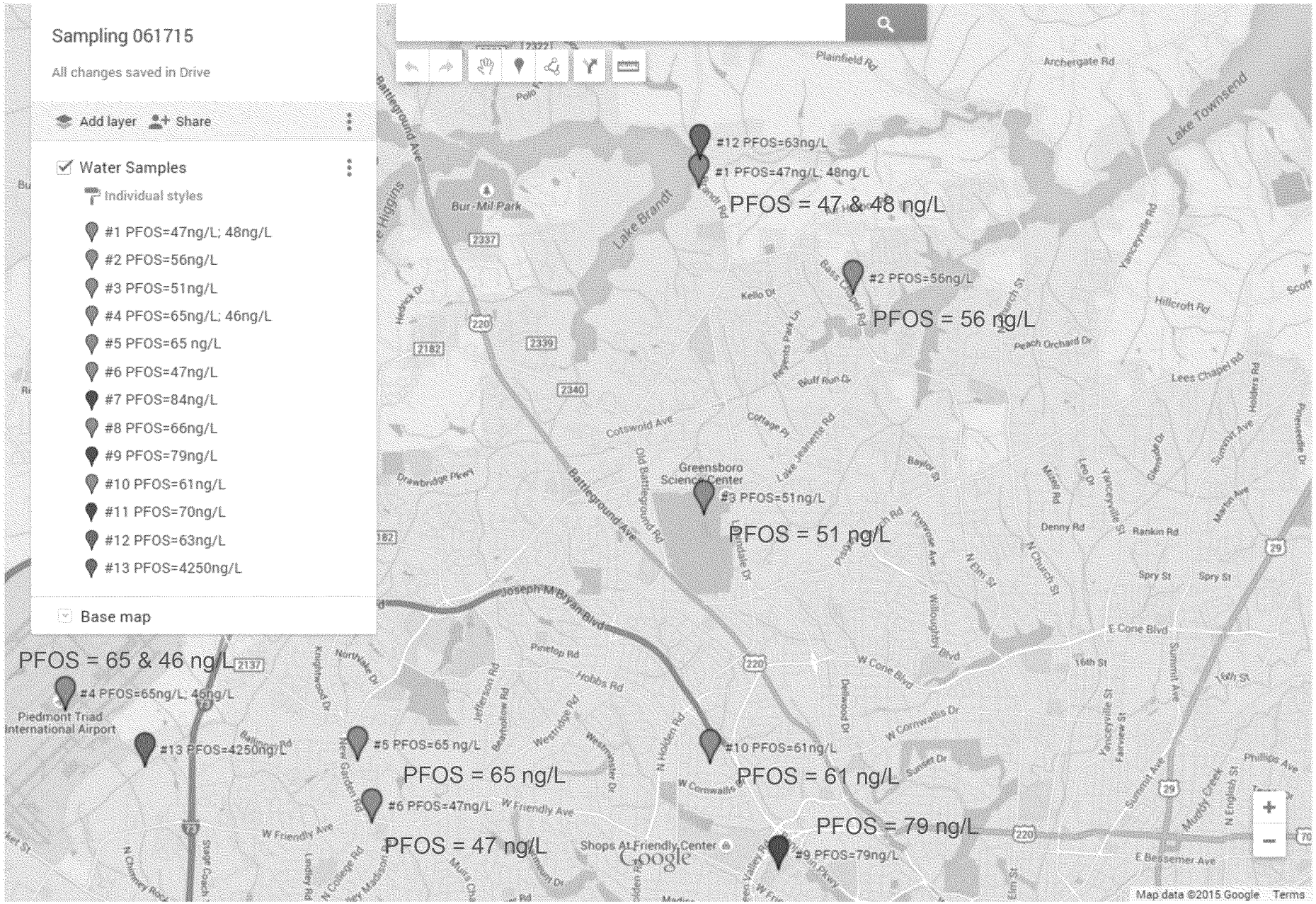
Near Piedmont Triad International Airport, Greensboro, North Carolina



PFOS in Surface water, Greensboro, North Carolina



PFOS in drinking water (ng/L), Greensboro, North Carolina



UCMR3 data for the State of Alabama of January 1, 2015 page 1

	A	B	C	D	E	F
1	PWSName	FacilityName	Date	PFAS	ng/L	State
2	Southside Waterworks	Gadsden Water Intertie	1/21/2013	PFHpA	10	AL
3	Southside Waterworks	Gadsden Water Intertie	4/15/2013	PFHpA	10	AL
4	Rainbow City Utilities Board	Gadsden Water Intertie	7/22/2013	PFHpA	10	AL
5	Rainbow City Utilities Board	Gadsden Water Intertie	4/15/2013	PFHpA	10	AL
6	Northeast Alabama Water System	Centre Water	6/12/2014	PFHpA	10	AL
7	Shelby County Water System	Talladega/Shelby WTP	10/17/2014	PFHpA	10	AL
8	Southside Waterworks	Gadsden Water Intertie	10/21/2013	PFHpA	20	AL
9	Southside Waterworks	Gadsden Water Intertie	7/8/2013	PFOA	20	AL
10	VAW Water System, Inc.	West Morgan-East Lawrence Wa	8/5/2014	PFOA	20	AL
11	VAW Water System, Inc.	West Morgan-East Lawrence Wa	2/18/2014	PFHpA	20	AL
12	VAW Water System, Inc.	West Morgan-East Lawrence Wa	5/28/2014	PFOA	20	AL
13	Rainbow City Utilities Board	Gadsden Water Intertie	1/15/2014	PFOA	20	AL
14	Rainbow City Utilities Board	Gadsden Water Intertie	10/21/2013	PFHpA	20	AL
15	Colbert County Rural Water System	West Lawrence	6/16/2014	PFOA	20	AL
16	Colbert County Rural Water System	Muscle Shoals	6/16/2014	PFOA	20	AL
17	Colbert County Rural Water System	Cherokee	6/16/2014	PFOA	20	AL
18	Florence Water-Wastewater Departmer	Wilson Lake + Treatment Plant	4/16/2014	PFOA	20	AL
19	Gadsden Waterworks & Sewer Board	Water Treatment Plant	10/2/2014	PFHpA	30	AL
20	Southside Waterworks	Gadsden Water Intertie	1/21/2013	PFOA	30	AL
21	Southside Waterworks	Gadsden Water Intertie	10/21/2013	PFOA	30	AL
22	Rainbow City Utilities Board	Gadsden Water Intertie	4/15/2013	PFOA	30	AL
23	Rainbow City Utilities Board	Gadsden Water Intertie	10/21/2013	PFOA	30	AL
24	West Morgan - East Lawrence Water Au	Tennessee River + Treatment Pla	2/5/2014	PFHpA	30	AL
25	Northeast Alabama Water System	Centre Water	6/12/2014	PFOA	30	AL
26	Gadsden Waterworks & Sewer Board	Water Treatment Plant	10/2/2014	PFOA	40	AL
27	Southside Waterworks	Gadsden Water Intertie	4/15/2013	PFOA	40	AL
28	Southside Waterworks	Gadsden Water Intertie	4/15/2013	PFOS	40	AL
29	VAW Water System, Inc.	West Morgan-East Lawrence Wa	11/4/2014	PFOS	40	AL
30	VAW Water System, Inc.	West Morgan-East Lawrence Wa	8/5/2014	PFOS	40	AL
31	Rainbow City Utilities Board	Gadsden Water Intertie	7/22/2013	PFOA	40	AL
32	Colbert County Rural Water System	Water Treatment Plant	6/16/2014	PFOS	40	AL
33	Colbert County Rural Water System	West Lawrence	6/16/2014	PFOS	40	AL
34	Colbert County Rural Water System	Muscle Shoals	6/16/2014	PFOS	40	AL
35	Florence Water-Wastewater Departmer	Wilson Lake + Treatment Plant	4/16/2014	PFOS	40	AL
36	Gadsden Waterworks & Sewer Board	Water Treatment Plant	10/2/2014	PFOS	50	AL
37	Southside Waterworks	Gadsden Water Intertie	1/21/2013	PFOS	50	AL
38	Colbert County Rural Water System	Cherokee	6/16/2014	PFOS	50	AL
39	West Morgan - East Lawrence Water Au	Tennessee River + Treatment Pla	8/28/2013	PFOS	50	AL
40	Northeast Alabama Water System	Centre Water	6/12/2014	PFOS	50	AL
41	VAW Water System, Inc.	West Morgan-East Lawrence Wa	5/28/2014	PFOS	60	AL
42	Rainbow City Utilities Board	Gadsden Water Intertie	7/22/2013	PFOS	60	AL
43	Florence Water-Wastewater Departmer	Cypress Creek + Treatment Plant	10/15/2013	PFOS	60	AL
44	VAW Water System, Inc.	West Morgan-East Lawrence Wa	2/18/2014	PFOA	90	AL
45	Rainbow City Utilities Board	Gadsden Water Intertie	7/22/2013	PFBS	100	AL
46	West Morgan - East Lawrence Water Au	Tennessee River + Treatment Pla	2/5/2014	PFOA	100	AL
47	West Morgan - East Lawrence Water Au	Tennessee River + Treatment Pla	2/5/2014	PFOS	130	AL
48	VAW Water System, Inc.	West Morgan-East Lawrence Wa	2/18/2014	PFOS	180	AL
49	City of Tempe	EPDS004- ID#09353	1/14/2014	PFHpA	11	AZ

UCMR3 data for the State of California of January 1, 2015 page 1

1	PWSName	FacilityName	Date	PFAS	ng/L	State
78	City of Corona	19579 Temescal Canyon Road	1/16/2014	PFHpA	10	CA
79	City of Corona	R-3 Blend (SB-9)	1/16/2014	PFHpA	11	CA
80	City of Orange	Well 15	5/14/2014	PFHpA	11.5	CA
81	CA Water Service - Visalia	Well 77-1	6/11/2014	PFHpA	13	CA
82	City of Corona	R-3 Blend (SB-9)	7/10/2014	PFHpA	13	CA
83	Elsinore Valley Municipal Water District	Canyon Lake WTP	9/19/2013	PFHpA	13	CA
84	Santa Clarita Water Division	Santa Clara Well	4/9/2014	PFHpA	14	CA
85	Elsinore Valley Municipal Water District	Canyon Lake WTP	6/19/2014	PFHpA	14	CA
86	City of Lathrop	Well 21	10/15/2013	PFOA	20	CA
87	City of Pico Rivera Water Department	Well 7	7/30/2014	PFOA	20	CA
88	City of Pico Rivera Water Department	Well 11	1/24/2013	PFOA	20	CA
89	Camp Pendleton (South)	Chlorination Station 410618	11/13/2013	PFOA	21	CA
90	City of Pico Rivera Water Department	Well 12	1/24/2013	PFOA	21	CA
91	Orchard Dale Water District	Mills	7/21/2014	PFOA	21	CA
92	Eastern Municipal Water District	Well 59 (Indian Ave.)	6/18/2013	PFHpA	22	CA
93	Eastern Municipal Water District	Well 59 (Indian Ave.)	10/8/2013	PFHpA	22	CA
94	City of Corona	19579 Temescal Canyon Road	10/9/2013	PFOA	23	CA
95	Valencia Water Company	Well S-7	3/12/2014	PFOA	23	CA
96	Valencia Water Company	Well S-6	3/12/2014	PFOA	24	CA
97	Valencia Water Company	Well N	5/28/2014	PFOA	24	CA
98	Yorba Linda Water District	Highland Reservoir	1/9/2013	PFOA	24.1	CA
99	City of Lathrop	Well 21	4/17/2013	PFOA	25	CA
100	Yorba Linda Water District	Highland Reservoir	7/10/2013	PFOA	25.9	CA
101	City of Corona	Lester SWTP	1/15/2014	PFOA	26	CA
102	City of Orange	Well 15	5/14/2014	PFOA	26.1	CA
103	City of Pico Rivera Water Department	Well 6	6/4/2014	PFOA	27	CA
104	Santa Clarita Water Division	Santa Clara Well	4/9/2014	PFOA	27	CA
105	Camp Pendleton (South)	Chlorination Station 410618	5/21/2014	PFOA	28	CA
106	Atascadero Mutual Water Company	Treatment Building C	10/30/2013	PFOA	28	CA
107	City of Pico Rivera Water Department	Well 4	7/30/2014	PFOA	29	CA
108	City of Corona	Ontario/Garretson Zone 3 (SB-1	10/23/2013	PFOA	29	CA
109	City of Orange	Well 3	5/14/2014	PFOA	29.1	CA
110	City of Pleasanton	Well 6	6/17/2014	PFHxS	32	CA
111	CA Water Service - Chico	Well 7-4	8/21/2013	PFHxS	35	CA
112	Sacramento Suburban Water District	Well 46 (Jonas/Sierra Mills)	8/11/2014	PFHxS	35	CA
113	Camp Pendleton (South)	I & M Plant 22035	11/26/2013	PFHxS	36	CA
114	City of Pleasanton	Well 6	11/4/2013	PFHxS	36	CA
115	Sacramento Suburban Water District	Well 46 (Jonas/Sierra Mills)	2/5/2014	PFHxS	36	CA
116	City of Corona	19579 Temescal Canyon Road	1/16/2014	PFOA	36	CA
117	City of Corona	R-3 Blend (SB-9)	1/16/2014	PFOA	38	CA
118	Elsinore Valley Municipal Water District	Canyon Lake WTP	9/19/2013	PFOA	38	CA
119	Elsinore Valley Municipal Water District	Canyon Lake WTP	6/19/2014	PFOA	38	CA
120	Camp Pendleton (South)	Chlorination Station 410618	11/13/2013	PFHxS	41	CA
121	City of Corona	19579 Temescal Canyon Road	1/16/2014	PFOS	41	CA
122	City of Fullerton	Well 3A	4/15/2014	PFOS	41.2	CA
123	City of Pico Rivera Water Department	Well 1	1/24/2013	PFOS	42	CA
124	CA Water Service - Chico	Well 7-4	2/11/2014	PFHxS	42	CA
125	City of Corona	R-3 Blend (SB-9)	7/10/2014	PFOA	42	CA
126	Golden State Water Co. - Norwalk	Pioneer Well 3	1/17/2014	PFOS	43	CA
127	City of Corona	Ontario/Garretson Zone 3 (SB-1	10/23/2013	PFOS	43	CA
128	City of Garden Grove	Well 19	5/14/2013	PFOS	44	CA

UCMR3 data for the State of California of January 1, 2015 page 2

	A	B	C	D	E	F
1	PWSName	FacilityName	Date	PFAS	ng/L	State
129	Eastern Municipal Water District	Well 59 (Indian Ave.)	10/8/2013	PFOA	44	CA
130	Orchard Dale Water District	Mills	7/21/2014	PFOS	45	CA
131	Park Water Company - Bellflower/Norw a	Well 46C	6/18/2013	PFOS	45	CA
132	City of Corona	R-3 Blend (SB-9)	1/16/2014	PFOS	45	CA
133	City of Orange	Well 15	5/14/2014	PFOS	45.3	CA
134	Montebello Land & Water Company	Pumping Plant (Wells 7, 8A, 9, 10	6/26/2014	PFOS	46	CA
135	City of Corona	R-3 Blend (SB-9)	7/10/2014	PFOS	46	CA
136	Montebello Land & Water Company	Well 11A	6/26/2014	PFOS	47	CA
137	Yorba Linda Water District	Highland Reservoir	7/10/2013	PFOS	47	CA
138	Yorba Linda Water District	Highland Reservoir	1/9/2013	PFOS	47.4	CA
139	City of Pico Rivera Water Department	Well 8	1/24/2013	PFOS	48	CA
140	City of Garden Grove	Well 23	5/15/2013	PFOS	48	CA
141	Park Water Company - Bellflower/Norw a	Well 41A	6/18/2013	PFOS	49	CA
142	Park Water Company - Bellflower/Norw a	Well 41A	3/7/2014	PFOS	49	CA
143	City of Garden Grove	Well 19	11/13/2013	PFOS	49.3	CA
144	City of Garden Grove	Well 23	11/13/2013	PFOS	49.6	CA
145	City of Pico Rivera Water Department	Well 8	7/30/2013	PFOS	50	CA
146	CA Water Service - East Los Angeles	Well 38-2	9/22/2014	PFOS	50	CA
147	Park Water Company - Bellflower/Norw a	Well 28B	6/18/2013	PFOS	51	CA
148	Eastern Municipal Water District	Well 59 (Indian Ave.)	6/18/2013	PFOA	53	CA
149	CA Water Service - East Los Angeles	Well 38-2	2/11/2014	PFOS	54	CA
150	CA Water Service - Chico	Well 41-1	2/19/2014	PFOS	57	CA
151	City of Lathrop	Well 21	10/15/2013	PFOS	58	CA
152	CA Water Service - Chico	Well 7-4	8/21/2013	PFOS	59	CA
153	Camp Pendleton (South)	Chlorination Station 410618	5/21/2014	PFHxS	62	CA
154	City of Pico Rivera Water Department	Well 7	7/30/2014	PFOS	62	CA
155	City of Orange	Well 3	5/14/2014	PFOS	65.9	CA
156	City of Lathrop	Well 21	4/17/2013	PFOS	76	CA
157	CA Water Service - Chico	Well 7-4	2/11/2014	PFOS	76	CA
158	Eastern Municipal Water District	Well 59 (Indian Ave.)	10/8/2013	PFOS	81	CA
159	Eastern Municipal Water District	Well 59 (Indian Ave.)	6/18/2013	PFOS	82	CA
160	CA Water Service - Visalia	Well 77-1	6/11/2014	PFHxS	92	CA
161	Eastern Municipal Water District	Well 59 (Indian Ave.)	6/18/2013	PFHxS	110	CA
162	CA Water Service - Visalia	Well 77-1	6/11/2014	PFOS	120	CA
163	Eastern Municipal Water District	Well 59 (Indian Ave.)	10/8/2013	PFHxS	120	CA

UCMR3 data for the State of Colorado of January 1, 2015 page 1

	A	B	C	D	E	F
1	PWSName	FacilityName	Date	PFAS	ng/L	State
164	City of Fountain	Chlorination for Well 2	1/15/2013	PFHpA	10	CO
165	City of Fountain	Chlorination for Well 4	1/15/2013	PFHpA	10	CO
166	City of Fountain	Chlorination for Well 2	7/17/2013	PFHpA	10	CO
167	Security WSD	FVA Intertie	7/30/2014	PFHpA	10	CO
168	Widefield WSD	Chlorinator & Aeration for Wells W	6/19/2014	PFHpA	11	CO
169	Widefield WSD	Chlorinator & Aeration for Wells W	11/20/2013	PFHpA	16	CO
170	Widefield WSD	Chlorinator for C36 Well	11/6/2013	PFHpA	18	CO
171	Widefield WSD	Chlorinator for W3 Well	11/12/2013	PFHpA	18	CO
172	Widefield WSD	Chlorinator for W4 Well	11/12/2013	PFHpA	19	CO
173	City of Fountain	Fountain Valley Authority Intertie	4/16/2013	PFHpA	20	CO
174	City of Fountain	Chlorination for Well 3	1/15/2013	PFOA	20	CO
175	City of Fountain	Chlorination for Well 1	1/15/2013	PFHpA	20	CO
176	Security WSD	Chlorinator for S7 Well	7/30/2014	PFHpA	20	CO
177	Security WSD	Chlorinator for S8 Well	7/30/2014	PFHpA	20	CO
178	Security WSD	Chlorinator for FV4 Well	7/29/2014	PFHpA	20	CO
179	Security WSD	Chlorinator for FV4 Well	1/14/2014	PFHpA	20	CO
180	Security WSD	Chlorinator for S11 Well	7/30/2014	PFHpA	20	CO
181	Security WSD	Chlorinator for S11 Well	4/22/2014	PFHpA	20	CO
182	Security WSD	Chlorinator for Well S10	7/30/2014	PFHpA	20	CO
183	Security WSD	Chlorinator for S2 Well	7/29/2014	PFHpA	20	CO
184	Security WSD	Chlorinator for S9 Well	7/29/2014	PFHpA	20	CO
185	Security WSD	Chlorinator for S2 Well	1/21/2014	PFHpA	20	CO
186	Security WSD	Chlorinator for S7 Well	1/22/2014	PFHpA	20	CO
187	Security WSD	Chlorinator for R1 Well	1/14/2014	PFHpA	20	CO
188	Security WSD	Chlorinator for S4 Well	7/30/2014	PFHpA	20	CO
189	Security WSD	Chlorinator for S4 Well	2/10/2014	PFHpA	20	CO
190	Security WSD	Chlorinator for R1 Well	8/11/2014	PFHpA	20	CO
191	Security WSD	Chlorinator for R2 Well	7/29/2014	PFHpA	20	CO
192	Security WSD	Chlorinator for Well S10	1/14/2014	PFHpA	20	CO
193	Widefield WSD	Chlorinator for E2 Well	5/14/2014	PFHpA	20	CO
194	Widefield WSD	Chlorinator & Aeration for Wells W	6/19/2014	PFOA	21	CO
195	Widefield WSD	Chlorinator for W7 Well	5/14/2014	PFHpA	22	CO
196	Widefield WSD	Chlorinator for W3 Well	5/14/2014	PFHpA	24	CO
197	Widefield WSD	Chlorinator for W4 Well	5/19/2014	PFHpA	24	CO
198	Widefield WSD	Chlorinator & Aeration for Wells W	11/20/2013	PFOA	24	CO
199	City of Fountain	Chlorination for Well 3	7/17/2013	PFOA	30	CO
200	City of Fountain	Fountain Valley Authority Intertie	7/17/2013	PFHxS	30	CO
201	City of Fountain	Chlorination for Well 4	1/15/2013	PFOA	30	CO
202	Security WSD	Chlorinator for S4 Well	7/30/2014	PFOA	30	CO
203	Security WSD	Chlorinator for S7 Well	7/30/2014	PFOA	30	CO
204	Security WSD	FVA Intertie	7/30/2014	PFOA	30	CO
205	Security WSD	CT 150,000 Gallon Steel Tank	1/22/2014	PFHpA	30	CO
206	Security WSD	Chlorinator for S16 Well	7/30/2014	PFHpA	30	CO
207	Security WSD	Chlorinator for S11 Well	4/22/2014	PFOA	30	CO
208	Security WSD	Chlorinator for S9 Well	1/14/2014	PFHpA	30	CO
209	Security WSD	Chlorinator for S16 Well	1/22/2014	PFHpA	30	CO
210	Security WSD	Chlorinator for S4 Well	2/10/2014	PFOA	30	CO
211	Security WSD	Chlorinator for S15 Well	7/30/2014	PFHpA	30	CO
212	Security WSD	Chlorinator for R2 Well	1/14/2014	PFHpA	30	CO
213	Security WSD	Chlorinator for S15 Well	1/21/2014	PFHpA	30	CO
214	Security WSD	Chlorinator for S11 Well	7/30/2014	PFOA	30	CO

UCMR3 data for the State of Colorado of January 1, 2015 page 2

	A	B	C	D	E	F	G
1	PWSName	FacilityName	Date	PFAS	ng/L	State	
215	Security WSD	CT 150,000 Gallon Steel Tank	7/15/2014	PFHpA	30	CO	
216	Security WSD	Chlorinator for S7 Well	1/22/2014	PFOA	30	CO	
217	Widefield WSD	Chlorinator for C36 Well	11/6/2013	PFOA	30	CO	
218	Widefield WSD	Chlorinator for W3 Well	11/12/2013	PFOA	31	CO	
219	Widefield WSD	Chlorinator for W7 Well	5/14/2014	PFOA	36	CO	
220	Widefield WSD	Chlorinator for E2 Well	5/14/2014	PFOA	36	CO	
221	Widefield WSD	Chlorinator for W4 Well	11/12/2013	PFOA	37	CO	
222	City of Fountain	Chlorination for Well 2	1/15/2013	PFOA	40	CO	
223	City of Fountain	Chlorination for Well 4	7/17/2013	PFOA	40	CO	
224	City of Fountain	Chlorination for Well 1	1/15/2013	PFOA	40	CO	
225	City of Fountain	Chlorination for Well 1	1/15/2013	PFOS	40	CO	
226	City of Fountain	Chlorination for Well 1	7/17/2013	PFOA	40	CO	
227	City of Fountain	Chlorination for Well 2	1/15/2013	PFOS	40	CO	
228	City of Fountain	Fountain Valley Authority Intertie	4/16/2013	PFOA	40	CO	
229	City of Fountain	Chlorination for Well 2	7/17/2013	PFOA	40	CO	
230	City of Fountain	Chlorination for Well 3	1/15/2013	PFHxS	40	CO	
231	City of Fountain	Chlorination for Well 3	1/15/2013	PFOS	40	CO	
232	Security WSD	Chlorinator for R1 Well	1/14/2014	PFOS	40	CO	
233	Security WSD	Chlorinator for R1 Well	1/14/2014	PFOA	40	CO	
234	Security WSD	Chlorinator for S2 Well	7/29/2014	PFOS	40	CO	
235	Security WSD	Chlorinator for W12 Well	7/30/2014	PFHpA	40	CO	
236	Security WSD	Chlorinator for S9 Well	7/29/2014	PFOA	40	CO	
237	Security WSD	Chlorinator for Well S10	1/14/2014	PFOA	40	CO	
238	Security WSD	Chlorinator for Well S10	7/30/2014	PFOA	40	CO	
239	Security WSD	Chlorinator for S15 Well	7/30/2014	PFOS	40	CO	
240	Security WSD	Chlorinator for S8 Well	7/30/2014	PFOA	40	CO	
241	Security WSD	FVA Intertie	1/22/2014	PFOS	40	CO	
242	Security WSD	Chlorinator for W12 Well	1/15/2014	PFHpA	40	CO	
243	Security WSD	Chlorinator for W12 Well	1/15/2014	PFOA	40	CO	
244	Security WSD	Air Stripping Plant	7/31/2014	PFHpA	40	CO	
245	Security WSD	Air Stripping Plant	1/29/2014	PFHpA	40	CO	
246	Security WSD	Chlorinator for R1 Well	8/11/2014	PFOA	40	CO	
247	Security WSD	Chlorinator for S2 Well	1/21/2014	PFOS	40	CO	
248	Widefield WSD	Chlorinator & Aeration for Wells W	11/20/2013	PFOS	42	CO	
249	Widefield WSD	Chlorinator for W4 Well	5/19/2014	PFOA	45	CO	
250	Widefield WSD	Chlorinator for E2 Well	11/13/2013	PFHxS	46	CO	
251	Widefield WSD	Chlorinator for W3 Well	5/14/2014	PFOA	48	CO	
252	Widefield WSD	Chlorinator & Aeration for Wells W	6/19/2014	PFOS	49	CO	
253	City of Fountain	Chlorination for Well 1	7/17/2013	PFHxS	50	CO	
254	City of Fountain	Chlorination for Well 3	7/17/2013	PFHxS	50	CO	
255	City of Fountain	Chlorination for Well 4	7/17/2013	PFHxS	50	CO	
256	Security WSD	Chlorinator for W12 Well	7/30/2014	PFOA	50	CO	
257	Security WSD	Chlorinator for FV4 Well	7/29/2014	PFOA	50	CO	
258	Security WSD	Chlorinator for FV4 Well	1/14/2014	PFOA	50	CO	
259	Security WSD	Chlorinator for S16 Well	1/22/2014	PFOS	50	CO	
260	Security WSD	Chlorinator for S2 Well	1/21/2014	PFOA	50	CO	
261	Security WSD	Chlorinator for S2 Well	7/29/2014	PFOA	50	CO	
262	Security WSD	Chlorinator for R1 Well	8/11/2014	PFOS	50	CO	
263	Security WSD	Chlorinator for S9 Well	1/14/2014	PFOA	50	CO	
264	Security WSD	Chlorinator for R2 Well	1/14/2014	PFOS	50	CO	
265	Widefield WSD	Chlorinator for W4 Well	5/19/2014	PFOS	51	CO	

UCMR3 data for the State of Colorado of January 1, 2015 page 3

1	PWSName	FacilityName	Date	PFAS	ng/L	State
266	Widefield WSD	Chlorinator for W3 Well	5/14/2014	PFOS	57	CO
267	City of Fountain	Chlorination for Well 2	7/17/2013	PFHxS	60	CO
268	Security WSD	Chlorinator for FV4 Well	7/29/2014	PFOS	60	CO
269	Security WSD	CT 150,000 Gallon Steel Tank	7/15/2014	PFOS	60	CO
270	Security WSD	CT 150,000 Gallon Steel Tank	1/22/2014	PFOS	60	CO
271	Security WSD	Chlorinator for W3 Well	1/15/2014	PFHpA	60	CO
272	Security WSD	FVA Intertie	7/30/2014	PFOS	60	CO
273	Security WSD	FVA Intertie	4/21/2014	PFOS	60	CO
274	Security WSD	FVA Intertie	1/22/2014	PFHxS	60	CO
275	Security WSD	Chlorinator for R2 Well	1/14/2014	PFOA	60	CO
276	Security WSD	Chlorinator for FV4 Well	1/14/2014	PFOS	60	CO
277	Security WSD	Chlorinator for S16 Well	7/30/2014	PFOS	60	CO
278	Security WSD	Chlorinator for S16 Well	1/22/2014	PFOA	60	CO
279	Security WSD	Chlorinator for S15 Well	1/21/2014	PFOA	60	CO
280	Security WSD	Chlorinator for R2 Well	7/29/2014	PFOS	60	CO
281	Security WSD	Chlorinator for R2 Well	7/29/2014	PFOA	60	CO
282	Security WSD	FVA Intertie	4/21/2014	PFHxS	60	CO
283	Widefield WSD	Chlorinator & Aeration for Wells W	6/19/2014	PFHxS	69	CO
284	Security WSD	Chlorinator for Well W8	1/15/2014	PFHpA	70	CO
285	Security WSD	CT 150,000 Gallon Steel Tank	7/15/2014	PFOA	70	CO
286	Security WSD	Chlorinator for S16 Well	7/30/2014	PFOA	70	CO
287	Security WSD	Chlorinator for S15 Well	7/30/2014	PFOA	70	CO
288	Security WSD	Chlorinator for Well W8	7/31/2014	PFHpA	70	CO
289	Security WSD	Chlorinator for W3 Well	1/15/2014	PFOA	70	CO
290	Security WSD	CT 150,000 Gallon Steel Tank	1/22/2014	PFOA	70	CO
291	Security WSD	Chlorinator for S9 Well	1/14/2014	PFOS	70	CO
292	Security WSD	Chlorinator for W3 Well	7/30/2014	PFHpA	70	CO
293	Widefield WSD	Chlorinator for W7 Well	11/12/2013	PFHxS	71	CO
294	Widefield WSD	Chlorinator & Aeration for Wells W	11/20/2013	PFHxS	75	CO
295	City of Fountain	Chlorination for Well 1	1/15/2013	PFHxS	80	CO
296	City of Fountain	Chlorination for Well 4	1/15/2013	PFHxS	80	CO
297	City of Fountain	Chlorination for Well 2	1/15/2013	PFHxS	80	CO
298	Security WSD	Chlorinator for S8 Well	7/30/2014	PFOS	80	CO
299	Security WSD	Chlorinator for W3 Well	7/30/2014	PFOA	80	CO
300	Security WSD	Chlorinator for Well W8	7/31/2014	PFOA	80	CO
301	Security WSD	Chlorinator for S9 Well	7/29/2014	PFOS	80	CO
302	Security WSD	Air Stripping Plant	1/29/2014	PFOA	80	CO
303	Security WSD	Chlorinator for Well W8	1/15/2014	PFOA	80	CO
304	Security WSD	Chlorinator for S7 Well	1/22/2014	PFOS	90	CO
305	Security WSD	Chlorinator for S4 Well	2/10/2014	PFOS	90	CO
306	Security WSD	Chlorinator for S7 Well	7/30/2014	PFOS	90	CO
307	Security WSD	Chlorinator for Well S10	7/30/2014	PFOS	90	CO
308	Security WSD	Chlorinator for S11 Well	4/22/2014	PFHxS	90	CO
309	Security WSD	Chlorinator for Well S10	1/14/2014	PFOS	90	CO
310	Security WSD	Chlorinator for S11 Well	4/22/2014	PFOS	90	CO
311	Security WSD	Chlorinator for Well W8	7/31/2014	PFBS	90	CO
312	Security WSD	Air Stripping Plant	7/31/2014	PFOA	90	CO
313	City of Fountain	Fountain Valley Authority Intertie	4/16/2013	PFOS	100	CO
314	Security WSD	Chlorinator for S11 Well	7/30/2014	PFHxS	100	CO
315	Security WSD	Chlorinator for S11 Well	7/30/2014	PFOS	100	CO
316	Security WSD	Chlorinator for S4 Well	7/30/2014	PFOS	100	CO

UCMR3 data for the State of Colorado of January 1, 2015 page 4

1	PWSName	FacilityName	Date	PFAS	ng/L	State
319	Security WSD	Chlorinator for S4 Well	2/10/2014	PFHxS	110	CO
320	Security WSD	Chlorinator for S7 Well	1/22/2014	PFHxS	130	CO
321	Security WSD	Chlorinator for Well S10	1/14/2014	PFHxS	130	CO
322	Widefield WSD	Chlorinator for E2 Well	5/14/2014	PFHxS	130	CO
323	Security WSD	Chlorinator for Well S10	7/30/2014	PFHxS	140	CO
324	Security WSD	Chlorinator for S4 Well	7/30/2014	PFHxS	140	CO
325	Security WSD	Chlorinator for S7 Well	7/30/2014	PFHxS	140	CO
326	Security WSD	Air Stripping Plant	1/29/2014	PFOS	140	CO
327	Security WSD	Air Stripping Plant	7/31/2014	PFBS	150	CO
328	Security WSD	Chlorinator for FV4 Well	1/14/2014	PFHxS	160	CO
329	Widefield WSD	Chlorinator for W7 Well	5/14/2014	PFHxS	160	CO
330	Security WSD	Chlorinator for S9 Well	7/29/2014	PFHxS	180	CO
331	Security WSD	Chlorinator for S8 Well	7/30/2014	PFHxS	180	CO
332	Security WSD	FVA Intertie	7/30/2014	PFHxS	180	CO
333	Widefield WSD	Chlorinator for W3 Well	11/12/2013	PFHxS	180	CO
334	Widefield WSD	Chlorinator for E2 Well	5/14/2014	PFOS	180	CO
335	City of Fountain	Fountain Valley Authority Intertie	4/16/2013	PFHxS	190	CO
336	Security WSD	Chlorinator for FV4 Well	7/29/2014	PFHxS	200	CO
337	Security WSD	Chlorinator for S9 Well	1/14/2014	PFHxS	210	CO
338	Security WSD	Chlorinator for S2 Well	7/29/2014	PFHxS	210	CO
339	Security WSD	Air Stripping Plant	7/31/2014	PFOS	210	CO
340	Security WSD	Chlorinator for S2 Well	1/21/2014	PFHxS	210	CO
341	Widefield WSD	Chlorinator for W7 Well	5/14/2014	PFOS	210	CO
342	Widefield WSD	Chlorinator for C36 Well	11/6/2013	PFHxS	210	CO
343	Widefield WSD	Chlorinator for W4 Well	11/12/2013	PFHxS	220	CO
344	Security WSD	Chlorinator for S15 Well	1/21/2014	PFHxS	230	CO
345	Security WSD	Chlorinator for W12 Well	7/30/2014	PFHxS	240	CO
346	Security WSD	Chlorinator for W12 Well	1/15/2014	PFHxS	240	CO
347	Widefield WSD	Chlorinator for W3 Well	5/14/2014	PFHxS	270	CO
348	Security WSD	Chlorinator for S15 Well	7/30/2014	PFHxS	290	CO
349	Security WSD	Chlorinator for S16 Well	1/22/2014	PFHxS	300	CO
350	Security WSD	Chlorinator for Well W8	1/15/2014	PFHxS	310	CO
351	Security WSD	Chlorinator for R1 Well	8/11/2014	PFHxS	310	CO
352	Security WSD	Chlorinator for W9 Well	1/15/2014	PFHxS	310	CO
353	Security WSD	Chlorinator for R1 Well	1/14/2014	PFHxS	320	CO
354	Security WSD	Chlorinator for R2 Well	7/29/2014	PFHxS	330	CO
355	Widefield WSD	Chlorinator for W4 Well	5/19/2014	PFHxS	330	CO
356	Security WSD	Chlorinator for W9 Well	7/30/2014	PFHxS	340	CO
357	Security WSD	Chlorinator for R2 Well	1/14/2014	PFHxS	340	CO
358	Security WSD	Chlorinator for Well W8	7/31/2014	PFHxS	350	CO
359	Security WSD	CT 150,000 Gallon Steel Tank	7/15/2014	PFHxS	360	CO
360	Security WSD	CT 150,000 Gallon Steel Tank	1/22/2014	PFHxS	380	CO
361	Security WSD	Chlorinator for S16 Well	7/30/2014	PFHxS	420	CO
362	Security WSD	Air Stripping Plant	1/29/2014	PFHxS	440	CO
363	Security WSD	Chlorinator for Well W8	1/15/2014	PFOS	580	CO
364	Security WSD	Chlorinator for Well W8	7/31/2014	PFOS	580	CO
365	Security WSD	Chlorinator for W12 Well	7/30/2014	PFOS	590	CO
366	Security WSD	Air Stripping Plant	7/31/2014	PFHxS	590	CO
367	Security WSD	Chlorinator for W12 Well	1/15/2014	PFOS	600	CO
368	Security WSD	Chlorinator for W9 Well	7/30/2014	PFOS	650	CO
369	Security WSD	Chlorinator for W9 Well	1/15/2014	PFOS	1300	CO

UCMR3 data for the State of Pennsylvania of January 1, 2015 page 1

1	PWSName	FacilityName	Date	PFAS	ng/L	State
611	Warminster Municipal Authority	Well 2	11/19/2013	PFHpA	10.5	PA
612	Aqua PA - Hatboro	Well 17	6/25/2014	PFHpA	11	PA
613	Ambler Borough Water Department	Loch Alsh Well #2	8/20/2013	PFHpA	12	PA
614	Warminster Municipal Authority	Well 13	6/9/2014	PFHpA	15.9	PA
615	Warminster Municipal Authority	Well 13	11/19/2013	PFHpA	19.9	PA
616	Aqua PA - Bristol	Filter Plant 2	6/20/2014	PFOA	20	PA
617	Horsham Water & Sewer Authority	Well #10	6/24/2014	PFOA	20	PA
618	Warminster Municipal Authority	Well 5	6/9/2014	PFOA	20.1	PA
619	Warminster Municipal Authority	Well 9	11/19/2013	PFOA	20.3	PA
620	Warminster Municipal Authority	Well 2	6/9/2014	PFOA	21.6	PA
621	Emmaus Borough Public Water	Waterworks Building	3/24/2014	PFNA	22	PA
622	Warminster Municipal Authority	Well 14	11/19/2013	PFOA	22.8	PA
623	Horsham Water & Sewer Authority	Well #17	6/24/2014	PFOA	23	PA
624	Warminster Municipal Authority	Well 5	11/19/2013	PFOA	23.1	PA
625	United Water PA	Airport	2/4/2014	PFHpA	23.4	PA
626	Warminster Municipal Authority	Well 10	11/19/2013	PFHpA	23.8	PA
627	Warminster Municipal Authority	Well 10	6/9/2014	PFHpA	23.9	PA
628	Warminster Municipal Authority	Well 15	11/19/2013	PFOA	24.6	PA
629	Aqua PA - Bristol	Filter Plant 2	12/20/2013	PFOA	26	PA
630	Ambler Borough Water Department	Loch Alsh Well #2	8/20/2013	PFNA	29	PA
631	Warminster Municipal Authority	Well 2	11/19/2013	PFHxS	30.8	PA
632	Warminster Municipal Authority	Well 14	11/19/2013	PFHxS	31.1	PA
633	Quakertown Boro	Well 13	6/17/2014	PFNA	32.3	PA
634	Horsham Water & Sewer Authority	Well #26	6/24/2014	PFHpA	33	PA
635	Horsham Water & Sewer Authority	Well #10	6/24/2014	PFHxS	33	PA
636	Warminster Municipal Authority	Well 2	11/19/2013	PFOA	34.3	PA
637	Warminster Municipal Authority	Well 15	11/19/2013	PFHxS	35.9	PA
638	United Water PA	Airport	2/4/2014	PFOA	38	PA
639	Horsham Water & Sewer Authority	Well #40	6/24/2014	PFHpA	39	PA
640	Horsham Water & Sewer Authority	Well #17	6/24/2014	PFHxS	40	PA
641	Horsham Water & Sewer Authority	Well #10	6/24/2014	PFOS	41	PA
642	Warminster Municipal Authority	Well 2	6/9/2014	PFOS	41.3	PA
643	United Water PA	Airport	2/4/2014	PFNA	46.6	PA
644	Warminster Municipal Authority	Well 14	11/19/2013	PFOS	55.3	PA
645	Warminster Municipal Authority	Well 26	11/19/2013	PFHpA	56.1	PA
646	Warminster Municipal Authority	Well 2	11/19/2013	PFOS	57.6	PA
647	Warminster Municipal Authority	Well 13	6/9/2014	PFHxS	59.5	PA
648	Horsham Water & Sewer Authority	Well #40	6/24/2014	PFOA	63	PA
649	Warminster Municipal Authority	Well 15	11/19/2013	PFOS	64.5	PA
650	Warminster Municipal Authority	Well 26	6/9/2014	PFHpA	71	PA
651	Horsham Water & Sewer Authority	Well #17	6/24/2014	PFOS	74	PA
652	Warminster Municipal Authority	Well 10	6/9/2014	PFHxS	76.1	PA
653	Horsham Water & Sewer Authority	Well #21	6/24/2014	PFHxS	79	PA
654	Warminster Municipal Authority	Well 13	6/9/2014	PFOA	79.6	PA
655	Warminster Municipal Authority	Well 10	6/9/2014	PFOA	82.2	PA
656	Warminster Municipal Authority	Well 13	11/19/2013	PFHxS	87.9	PA
657	Warminster Municipal Authority	Well 10	11/19/2013	PFOA	88.5	PA
658	Warminster Municipal Authority	Well 10	11/19/2013	PFHxS	100	PA
659	Horsham Water & Sewer Authority	Well #40	6/24/2014	PFBS	110	PA
660	Warminster Municipal Authority	Well 13	11/19/2013	PFOA	122	PA
661	Warminster Municipal Authority	Well 13	6/9/2014	PFOS	138	PA

UCMR3 data for the State of Pennsylvania of January 1, 2015 page 2

	A	B	C	D	E	F
1	PWSName	FacilityName	Date	PFAS	ng/L	State
662	Horsham Water & Sewer Authority	Well #21	6/24/2014	PFOS	140	PA
663	Warminster Municipal Authority	Well 13	11/19/2013	PFOS	160	PA
664	Warminster Municipal Authority	Well 10	6/9/2014	PFOS	176	PA
665	Warminster Municipal Authority	Well 10	11/19/2013	PFOS	193	PA
666	United Water PA	Airport	2/4/2014	PFHxS	209	PA
667	Horsham Water & Sewer Authority	Well #26	6/24/2014	PFOA	290	PA
668	Warminster Municipal Authority	Well 26	11/19/2013	PFOA	291	PA
669	Warminster Municipal Authority	Well 26	11/19/2013	PFHxS	334	PA
670	Warminster Municipal Authority	Well 26	6/9/2014	PFOA	349	PA
671	United Water PA	Airport	2/4/2014	PFOS	363	PA
672	Warminster Municipal Authority	Well 26	6/9/2014	PFHxS	389	PA
673	Horsham Water & Sewer Authority	Well #26	6/24/2014	PFHxS	390	PA
674	Horsham Water & Sewer Authority	Well #40	6/24/2014	PFHxS	590	PA
675	Horsham Water & Sewer Authority	Well #26	6/24/2014	PFOS	700	PA
676	Warminster Municipal Authority	Well 26	11/19/2013	PFOS	791	PA
677	Horsham Water & Sewer Authority	Well #40	6/24/2014	PFOS	1000	PA
678	Warminster Municipal Authority	Well 26	6/9/2014	PFOS	1090	PA
679	Pierre	Treat Site #8	1/22/2014	PFHxS	40	SD

Each record in this database represents the occurrence of PFAS in finished drinking water but likely to represent a complicated story involving different sources, histories, and populations

Drinking water in UCMR3 is likely to be the tip of the iceberg, may indicate widespread environmental contamination including: agricultural commodities, fisheries and wildlife resources, soil contamination, airborne exposures

UCMR3 results should be used for many things:

Help identify and categorize sources of legacy contamination: industrial chemical production, sewage sludge, AFFF, landfills…

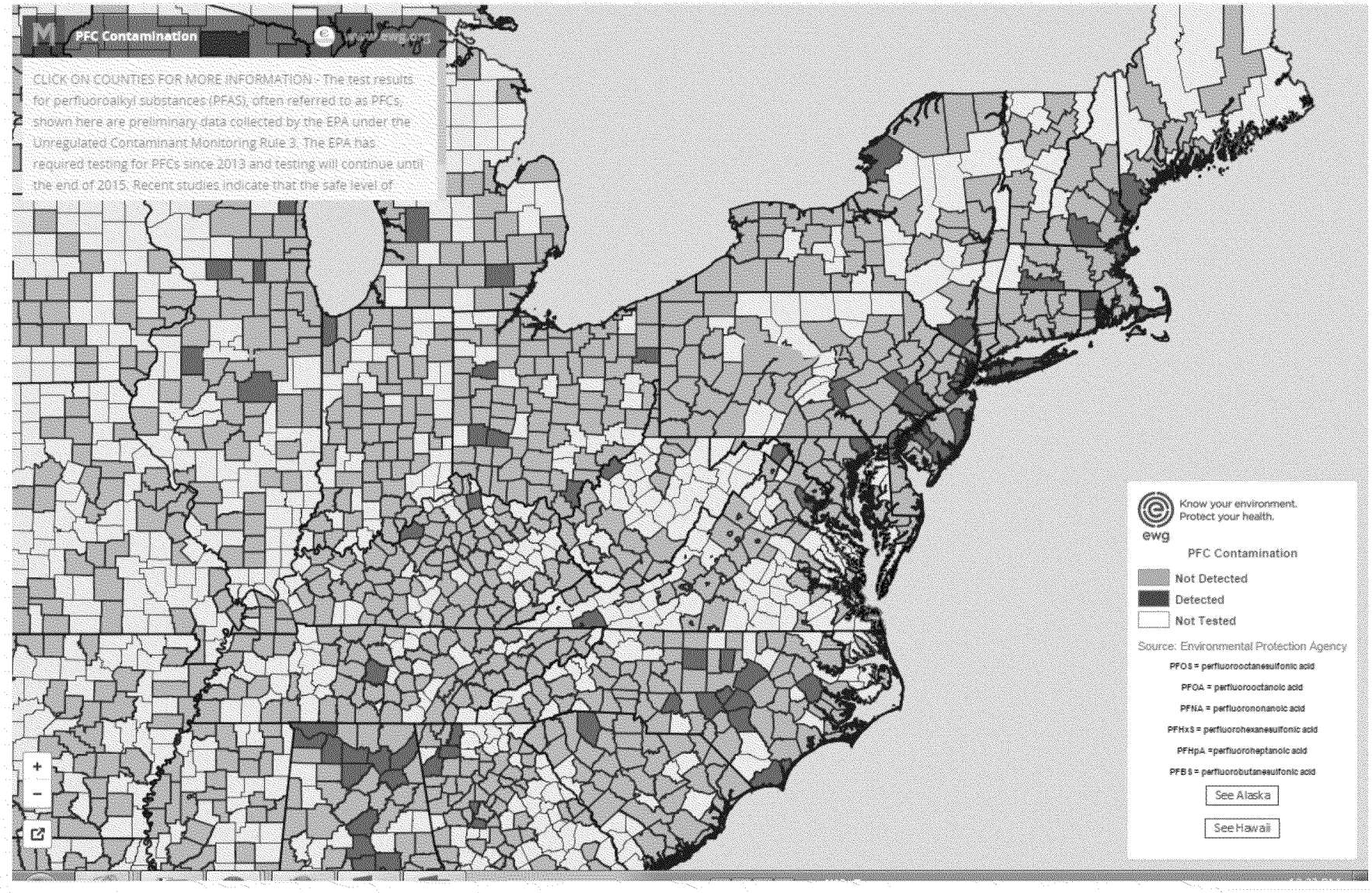
Identify and populations that are exposed to PFAS, provide rational & location for specific epidemiological studies

Verify and track community exposures with blood and water measurements - efforts underway in Wilmington and Greensboro, NC

Help municipalities identify sources of contamination, assess control technologies

Use UCMR3 results to target additional studies to monitor for replacement PFAS

Need detailed maps of UCMR3 results to facilitate this research



http://www.ewg.org/research/teflon-chemical-harmful-at-smallest-doses

30 records with PFOS higher than the Provisional Health Advisory (200 ng/L)

1	PWSID	PWSName	FacilityName	CollectionDate	Contaminant	ng/L	ug/L	State
48	CO0121775	Security WSD	Air Stripping Plant	7/31/2014	PFOS	210	0.21	CO
49	CO0121900	Widefield WSD	Chlorinator for W7 Well	5/14/2014	PFOS	210	0.21	CO
50	MA4020004	Hyannis Water System	Mary Dunn Well #3	5/22/2014	PFOS	210	0.21	MA
51	OH2903412	Wright-Patterson AFB Area A/C	Building 10855 - STU 1	10/7/2014	PFOS	210	0.21	OH
52	AZ0407046	Liberty Water LPSCO	Well 02 Airline	5/29/2014	PFOS	220	0.22	AZ
53	GU0000006	GU Waterworks Authority - Northern System	Well A-25	3/25/2015	PFOS	220	0.22	GU
54	ME0091410	Sanford Water District	Treatment Plant 5	8/7/2013	PFOS	250	0.25	ME
55	FL1170525	Emerald Coast Utilities Authority	Hagler	6/17/2014	PFOS	270	0.27	FL
56	ME0091410	Sanford Water District	Treatment Plant 5	4/9/2013	PFOS	290	0.29	ME
57	AZ0408001	Oatman Water Company	Well #1	4/22/2014	PFOS	300	0.3	AZ
58	NY5110526	Suffolk County Water Authority	Gus Guerrera Wellfield	9/8/2014	PFOS	330	0.33	NY
59	PA7220015	United Water PA	Airport	2/4/2014	PFOS	363	0.363	PA
60	FL1170525	Emerald Coast Utilities Authority	Hagler	11/11/2014	PFOS	380	0.38	FL
61	MA4020004	Hyannis Water System	Mary Dunn Well #2	5/22/2014	PFOS	430	0.43	MA
62	MN1820016	Oakdale	Well #7	1/5/2015	PFOS	435	0.435	MN
63	WA5336350	Issaquah Water System	Well #4	1/8/2014	PFOS	514	0.514	WA
64	NY5110526	Suffolk County Water Authority	Oxhead Rd. Wellfield	12/29/2014	PFOS	530	0.53	NY
65	CO0121775	Security WSD	Chlorinator for Well W8	1/15/2014	PFOS	580	0.58	CO
66	CO0121775	Security WSD	Chlorinator for Well W8	7/31/2014	PFOS	580	0.58	CO
67	CO0121775	Security WSD	Chlorinator for W12 Well	7/30/2014	PFOS	590	0.59	CO
68	CO0121775	Security WSD	Chlorinator for W12 Well	1/15/2014	PFOS	600	0.6	CO
69	WA5336350	Issaquah Water System	Well #4	7/22/2013	PFOS	600	0.6	WA
70	CO0121775	Security WSD	Chlorinator for W9 Well	7/30/2014	PFOS	650	0.65	CO
71	PA1090070	Warrington Township Water & Sewer Department	Wells 1, 2 & 6	11/11/2014	PFOS	666.66	0.66666	PA
72	PA1460033	Horsham Water & Sewer Authority	Well #26	6/24/2014	PFOS	700	0.7	PA
73	PA1090069	Warminster Municipal Authority	Well 26	11/19/2013	PFOS	791	0.791	PA
74	DE0000552	Artesian Water Company	Wilm. Manor 3 Plant	7/17/2013	PFOS	930	0.93	DE
75	PA1460033	Horsham Water & Sewer Authority	Well #40	6/24/2014	PFOS	1000	1	PA
76	PA1090069	Warminster Municipal Authority	Well 26	6/9/2014	PFOS	1090	1.09	PA
77	CO0121775	Security WSD	Chlorinator for W9 Well	1/15/2014	PFOS	1300	1.3	CO
78	DE0000552	Artesian Water Company	Wilm. Manor 3 Plant	1/28/2014	PFOS	1800	1.8	DE

Questions?

Email: lindstrom.andrew@epa.gov

To: Carroll, Gregory[Carroll.Gregory@epa.gov]
From: BNA Highlights
Sent: Wed 9/16/2015 8:18:55 PM
Subject: Sep 16 -- BNA, Inc. Daily Environment Report - Afternoon Briefing

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Afternoon Briefing - Your Preview of Today's News

The following news provides a snapshot of what Bloomberg BNA is working on today. Read the full version of all the stories in the final issue, published each night.

Climate Pledges a Third of What Is Needed, UN Official Says

Posted September 16, 2015, 3:56 P.M. ET

By Dean Scott

The carbon emissions cuts that countries have pledged to make ahead of the end-of-year Paris climate summit would provide only about one-third of what is needed to keep global temperatures from rising more than 2 degrees Celsius later this century, a top United Nations official said today.

Pledges from the U.S., European Union, China and other large emitters would cut a total of 5 gigatons “from where we would have been without them” by 2030, a “marked improvement” from a business-as-usual trajectory without those pledges, said Christiana Figueres, who oversees the talks as executive director of the UN Framework Convention on Climate Change.

“But we have to be very clear that if we want to be on a 2 degree pathway by 2030, we would have to reduce [emissions] 15 gigatons,” Figueres said on a press call held by the Union of Concerned Scientists.

The pledges thus far from more than 60 nations ahead of the Paris talks, where negotiators from developed and developing nations alike hope to conclude the climate deal, “is movement in the right direction,” she said. “But it does not get us to the 2 degree pathway,” she said. The Paris accord could include provisions allowing nations to ratchet down their emissions further in future years without reopening the entire agreement, she said.

EPA Under Court Mandate to Revise Stormwater Rules

Posted September 16, 2015, 2:31 P.M. ET

By Amena Saiyid

The Environmental Protection Agency is bound to revise stormwater regulations for cities and suburbs with populations of fewer than 100,000 under an agreement reached with environmental groups and approved yesterday by the U.S. Court of Appeals for the Ninth Circuit.

Under the settlement, the EPA is required to propose revised stormwater runoff rules by Dec. 17 and issue them in

final form by Nov. 17, 2016. By next May, it must also determine whether to regulate stormwater runoff from forest roads.

The settlement resolves a lawsuit challenging 1999 regulations that was brought by the Environmental Defense Center Inc. and the Natural Resources Defense Council, which claimed the EPA failed to comply with a Ninth Circuit ruling from 2003.

McConnell Calls for Ending Crude Oil Export Ban

Posted September 16, 2015, 11:06 A.M. ET

By Ari Natter

Senate Majority Leader Mitch McConnell (R-Ky.) today threw his weight behind ending the 40-year-old ban on crude oil exports, calling the trade prohibition “a relic of the '70s,” according to a spokesman for the lawmaker.

“He pointed out that if Iran will be able to export its oil under the president’s deal with Iran, “why not us?' ” spokesman Don Stewart told Bloomberg BNA in an e-mail, characterizing McConnell's remarks before the Washington-based Business Roundtable.

Legislation (H.R. 702) that would lift the oil export ban is expected to receive a vote in the House as soon as this month, though the Senate has yet to announce if and when the upper chamber would take up such a bill.

Pompeo Considering RFS, Ozone Rule Study Amendment

Posted September 16, 2015, 2:38 P.M. ET

By Ari Natter

A federal study on the impact the renewable fuel standard and ozone regulations are having on refiners is among the amendments Rep. Mike Pompeo (R-Kan.) is considering filing to oil export legislation being marked up by a House committee tomorrow.

“As we move forward and pull all the strings that are impacting our energy markets, we need to look at everything,” Pompeo said in a phone interview. It is unclear if the amendment will be considered germane to the underlying bill, which would repeal the 40-year ban on crude oil exports.

Pompeo said he decided against another amendment to H.R. 702 that would have required a study of the legislation's implications for the Jones Act, which requires ships moving between U.S. ports to be U.S.-built and U.S.-crewed.

The bill, by Rep. Joe Barton (R-Texas), is being voted on by the House and Energy and Commerce Committee.

Senators: EPA Must Engage on Animas Superfund Request

Posted September 16, 2015, 4:14 P.M. ET

By Rachel Leven

Senators urged today the Environmental Protection Agency to engage a broad range of stakeholders in determining whether to grant the Navajo Nation's request to designate the area where the EPA triggered a 3 million gallon mining waste release a Superfund site.

Sen. Michael Bennet (D-Colo.) called for the agency to engage people from throughout Colorado in the potential Upper Animas River Mining District designation, while Sen. Martin Heinrich (D-N.M.) said the agency should engage individuals around the entire watershed. Both senators highlighted the “sensitivity” of the placement of the area on the National Priorities List, with Heinrich saying there are concerns that it could hurt tourism but that he hasn't found that to be true with certain designated locations in, for example, New Mexico.

EPA Administrator Gina McCarthy told the senators that the agency will take the designation request “seriously.”

“It's extremely important. It's something we have been soliciting interest of for many years because we think it is the

only way you are going to get significant federal funding to support those cleanup efforts, and short of other congressional action, there is simply to pass that job off to somebody else," McCarthy told the Senate Indian Affairs Committee at a hearing on the recent Gold King Mine spill.

Bring Chemical Safety Bill to Floor, Coalition Says

Posted September 16, 2015, 3:46 P.M. ET

By Pat Rizzuto

The Senate's most widely supported chemical safety bill should be brought to the floor for consideration, a coalition of unions, environmental and health, and animal welfare organizations said today.

"American desperately needs this legislation, which would require safety evaluations of all new and existing chemicals, give EPA stronger testing authority, modernize testing methods, and remove the barriers that have prevented EPA from regulating known dangers like asbestos," wrote Jennifer Howse, president of the March of Dimes; Collin O'Mara, president of the National Wildlife Federation; and other leaders of a coalition said to represent 25 million Americans.

The coalition wrote Majority Leader Mitch McConnell (R-Ky.) and Minority Leader Harry Reid (D-Nev.) asking that the Frank R. Lautenberg Chemical Safety for the 21st Century Act, S. 697, to be brought to the Senate floor as soon as possible. The bill would be the first overhaul of the U.S.'s primary chemicals legislation, the Toxic Substances Control Act, since 1976.

S. 697 is supported by 52 senators representing 33 states.

Tea Party Groups Urge Ryan Against Wind Credit Extension

Posted September 16, 2015, 11:40 A.M. ET

By Ari Natter

The House should leave an extension of the wind production tax credit out of tax extender's legislation, a coalition of conservative groups said in a letter today to House Ways and Means Chairman Paul Ryan (R-Wis.).

"Your colleagues in the Senate have already passed a tax extenders package that includes the wind PTC for another two years," said the letter, which was signed by groups such as Heritage Action for America. "The House now has a chance to take a different path by rejecting corporate welfare and business-as-usual tax policy that is hurting our country."

The groups, which also included the Club for Growth and Americans for Prosperity, said they may record how members vote on tax extenders legislation on their respective "legislative scorecards" if an extension of the 2.3 cent per kilowatt hour tax credit for wind and other renewable sources is included. The letter is available at http://americanenergyalliance.org/wp-content/uploads/2015/09/PTC-Coalition-Letter.pdf.

Clean Power Plan Sent Sept. 4 to Federal Register

Posted September 16, 2015, 2:33 P.M. ET

By Anthony Adragna

Environmental Protection Agency Administrator Gina McCarthy told the Senate Environment and Public Works Committee that her agency sent its final Clean Power Plan to the Federal Register for formal publication Sept. 4 and still expects it to be published next month.

Formal publication of the regulation to reduce carbon dioxide emissions from the nation's fleet of existing power plants is important because it kicks off a 60-day period in which legal challenges can be filed and a period in which Congress can attempt to block it through the Congressional Review Act. The slow publication was noted by committee Republicans.

"Are you aware that delaying publication until the end of October interferes with the ability of Congress and the public

to challenge the rules before the big show in Paris?" Sen. Jim Inhofe (R-Okla.) asked during the hearing, which focused on the agency's response to the August spill from Colorado's Gold King Mine.

White House Announces $120M in Solar Tech Funding

Posted September 16, 2015, 10:11 A.M. ET

By Rebecca Kern

The White House announced today it is investing more than $120 million to scale up the growth of solar energy technologies in 24 states.

Vice President Joe Biden will highlight these investments at the Solar Power International Conference in Anaheim, Calif., later today.

The funding includes $30 million from the Energy Department as part of its second annual round of the technology-to-market program, which supports the development of tools and technologies to reduce the costs of solar energy systems, the White House said in an fact sheet.

The Energy Department is also providing $20 million to support its photovoltaics research and development program, funding 35 projects to improve photovoltaic performance.

An additional $52 million comes from the department's SunShot Initiative, which seeks to make solar technologies more cost competitive. Also, the Department of Agriculture is awarding grants totaling $8 million to assist in lowering solar energy costs for residents in rural areas.

Senators to Introduce Good Samaritan Mine Cleanup Bill

Posted September 16, 2015, 10:40 A.M. ET

By Rachel Leven

Sens. Michael Bennet (D-Colo.) and Cory Gardner (R-Colo.) plan to introduce good Samaritan legislation in the wake of the Environmental Protection Agency-triggered Gold King Mine spill of 3 million gallons of mining waste into Colorado waterways, Bennet said today.

Bennet and Sen. Tom Udall (D-N.M.) introduced good Samaritan legislation—a type of legislation that would allow individuals and entities to clean up abandoned and inactive mines and be shielded from liability—during the last Congress. Bennet recently said that the spill that reached the Animas River presents an opportunity to make the case for this type of bill, and the National Mining Association and others have come out in support of this type of policy action.

Bennet made his announcement at the Senate Environment and Public Works Committee hearing on the spill, where EPA Administrator Gina McCarthy is set to speak soon. The August spill occurred when contractors for the EPA tried to open the blocked Gold King Mine near Silverton, Colo., to address leaks, accidentally releasing the toxic mining waste water into the Animas River.

UN Meetings Seen as Opportunity on Growth, Environment

Posted September 16, 2015, 2:27 P.M. ET

By Andrea Vittorio

A pair of upcoming United Nations gatherings focused on solving some of the world's biggest problems, including climate change, offer an "unprecedented" opportunity to show that economic growth and development don't have to come at the expense of the environment, a former World Bank economist said today.

Next week, about 190 countries will meet at UN headquarters in New York to sign off on a sweeping new sustainable development agenda that not only spans social and economic issues but also emphasizes environmental issues like never before. They will meet again in Paris at year's end to hammer out a global agreement to fight climate change.

"This is a truly unprecedented opportunity to redefine the vision, the model for economic development for the next generation," Manish Bapna, executive vice president and managing director of the World Resources Institute think tank, said in a briefing with reporters. "I think we're moving from a model that has historically framed economic growth and the environment as in conflict to one that sees economic growth and environmental sustainability as inextricably linked, a recognition that you can't have one without the other."

The UN's sustainable development agenda includes 17 goals that will replace the expiring set of goals that have guided development efforts since 2000.

Judges Question Sulfur Nonattainment Designations

Posted September 16, 2015, 12:34 P.M. ET

By Andrew Childers

Federal appellate judges questioned whether the Environmental Protection Agency improperly applied its revised sulfur dioxide air quality standard when it relied on past data to designate portions of Montana as not in compliance with the new requirements.

The Treasure State Resource Industry Association, a Montana industry group, had argued to the U.S. Court of Appeals for the District of Columbia Circuit today that the EPA improperly considered air quality data from 2010, which showed Yellowstone County was in compliance with the previous standard, when the agency revised its national ambient air quality standard for sulfur dioxide.

"It seems like they're put on probation now for what happened before," Judge Patricia Millett said.

The EPA's nonattainment designations, which found that 29 areas are not in compliance with the updated hourly sulfur dioxide standard of 75 parts per billion, also were challenged by U.S. Steel. That company challenged the EPA's decision to designate Wayne County, Mich., as a nonattainment area but not include a nearby coal-fired power plant in Monroe County as a source contributing to the violation.

DOE Issues Road Map to Double Energy Efficiency by 2030

Posted September 16, 2015, 2:47 P.M. ET

By Rebecca Kern

The Energy Department released a report today outlining strategies on how businesses, state and local governments, utilities, and consumers can reach President Barack Obama's goal of doubling energy efficiency in the U.S. by 2030.

The report was released as part of a partnership between the Energy Department, the Alliance to Save Energy and Council on Competitiveness at the Accelerate Energy Productivity 2030 summit today in Washington.

Energy Department Secretary Ernest Moniz said the report was the agency's "most ambitious innovation strategy to improve energy efficiency" and it required an "all hands on deck" approach from state and local governments, businesses, and research organizations to reach the goal by 2030.

Strategies include states setting and updating vehicle and product codes and standards, and providing energy performance information to consumers; utilities and regulators designing rates and related policies that align energy efficiency with utility business models; and businesses reinvesting avoided energy costs, according to the report.

EPA Plans to Have Draft Hard Rock Mine Rule in 2016

Posted September 16, 2015, 11:33 A.M. ET

By Rachel Leven

The Environmental Protection Agency plans to have a draft of its financial assurance rule for hard rock mines completed by August 2016, EPA Administrator Gina McCarthy said today.

The agency is under a court order to complete the rule by December 2017. The mining industry is opposed to the

rule, which aims to ensure that mine owners and operators pay for environmental cleanup efforts. The agency will also consult with other agencies prior to completing its draft to ensure that the rule (RIN 2050-AG61) is "solid and appropriate," McCarthy told the Senate Environment and Public Works Committee at its hearing on the Gold King Mine spill in Colorado.

"[My] understanding is that we do have an ability to require financial responsibility for our existing and active sites," McCarthy said. "The challenge for us are these legacy sites that we're talking about like the Gold King Mine, where we do not have a responsible party that we can turn to—that we will not be able to address those issues with this particular rulemaking."

The proposal discussed would be issued under Section 108(b) of Comprehensive Environmental Response, Compensation and Liability Act, also known as the Superfund law.

China Announces Steps to Clean Urban Surface Water

Posted September 16, 2015, 1:33 P.M. ET

By Michael Standaert

Environmental and urban management authorities in China have announced steps to address polluted urban surface water such as small river stretches, streams and ponds that it terms "black and smelly" water.

Several government ministries last week released guidelines on cleaning up such bodies of polluted urban water, a follow-up on implementation of a nationwide clean water action plan the State Council issued in April.

The guidelines target removal of large floating debris, accumulated garbage and illegal sewage outlets flowing to such bodies of water. China wants noticeable improvement by 2017 and elimination of the problem in all urban areas by 2030.

Environmental Group Leaks Preliminary EGA Product List

Posted September 16, 2015, 10:27 A.M. ET

By Bryce Baschuk

A European environmental advocacy group named Transport and Environment published a list of products being considered for inclusion in the Environmental Goods Agreement (EGA).

The document is an early compilation list of product nominations by the 17 World Trade Organization members currently involved in the EGA negotiations, according to one official participating in the negotiations. The preliminary list contains more than 2,400 environmental goods—like solar panels, bicycles, electric motors and hydraulic turbines—organized into more than 50 product categories.

The products included in the preliminary list have not yet been subjected to analysis, peer review, discussion and negotiation necessary to identify and agree on which products might be acceptable as part of an EGA outcome, the official told Bloomberg BNA. The current EGA product list, which has not been published, contains slightly more than 650 environmental goods organized into 10 categories, according to the European Union.

EU Chemicals Agency Moves on Flame Retardant Restriction

Posted September 16, 2015, 2:34 P.M. ET

By Stephen Gardner

A wide-ranging European Union restriction on the use of the flame retardant decabromodiphenyl ether (decaBDE) has moved a step closer with the finalization of an opinion in favor of the restriction by the European Chemical Agency's Socio-Economic Analysis Committee (SEAC).

The restriction on decaBDE, which is classified persistent, bioaccumulative and toxic, and very persistent and very bioaccumulative, would be a general prohibition of its use, except for some specific applications in the aviation industry, in road and non-road vehicles, and in spare parts for machinery.

The restriction will be adopted under the EU's REACH law (Regulation No. 1907/2006 on the registration, evaluation, and authorization of chemicals).

In addition, ECHA opened public consultations today on two other proposed REACH restrictions: on bisphenol A (BPA) and on pentadecafluorooctanoic acid (PFOA). The consultations are open through Nov. 16.

EU Backs Tighter Pollution Limits for Non-Road Vehicles

Posted September 16, 2015, 2:29 P.M. ET

By Stephen Gardner

Members of the European Parliament's environment committee have backed a European Commission proposal to tighten air pollutant emission limits in the European Union for “non-road mobile machinery,” a catch-all term that covers a range of equipment and machinery including construction and agricultural vehicles and trains.

Environment committee lawmakers voted 64–3 yesterday in favor of an amended version of the commission's proposal, published September 2014.

The committee made no major changes to the original proposal, but added provisions encouraging the retrofitting of existing non-road mobile machinery to reduce pollutant emissions and introduced amendments giving some waterway vessels more time to comply with new emission limits.

The law sets limits on emissions of carbon monoxide, hydrocarbons, nitrogen oxides and particulate matter that non-road machinery must comply with to be placed on the EU market.

The different classes of non-road mobile machinery are responsible for about 15 percent of EU nitrogen oxides emissions and about 5 percent of emissions of fine particles, according to the European Parliament.

Daily Environment Report

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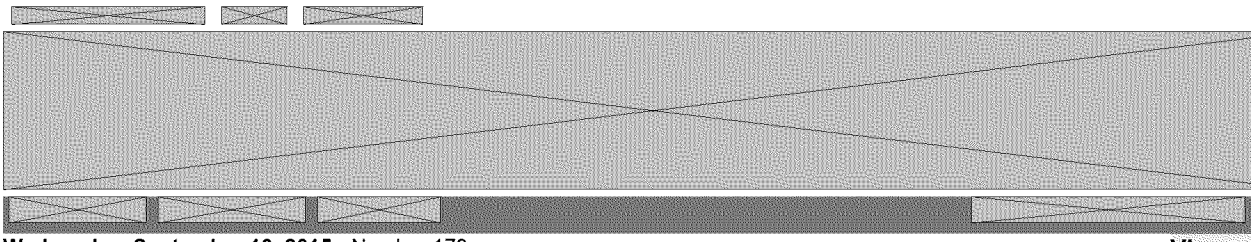

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NEWS

Air Pollution
Earthjustice: Industry Report Inflated Ozone Costs
A new economic analysis commissioned by Earthjustice alleges that an industry-funded study of the cost of implementing tighter national ozone standards contains a number of flaws and unfounded assumptions that "greatly overestimated"...

Air Pollution
Study: Diesel Cars Exceed EU Nitrogen Oxide Limits
New diesel cars on European Union roads exceed legal limits on nitrogen oxides in exhaust gases by a factor of five on average, according to research published by the advocacy group Transport & Environment (T&E)....

Biotechnology
Report: Scant Funding on Risks of Synthetic Biology
Less than 1 percent of the nearly $820 million the U.S. government invested in synthetic biology over six years has supported research on ethical, legal and social issues, according to a Woodrow Wilson Center report to be released Sept. 16....

Chemicals
EPA Inspector General Slams Green Chemistry Awards
The Environmental Protection Agency should stop using chemical companies' unverified pollution-prevention data to claim credit for having spurred cleaner air, less hazardous waste and other environmental benefits, the agency's...

Climate Change
House Republican Group to Push Climate Resolution
A group of 10 moderate House Republicans is poised to take what could be the first, small step to

rekindle the congressional debate over how the U.S. should confront climate change....

Climate Change
New Tool Finds Fossil Fuels in Retirement Savings
Environmentally conscious investors can now see whether there are fossil fuel stocks in their investment portfolios or retirement savings with a new online search tool launched Sept. 15....

Climate Change
UN Climate Chief Outlines Draft Text for Paris Summit
A text that will form the basis of an international climate deal, which could be signed at the United Nations climate summit in Paris later this year, has seen faster-than-expected progress in recent days and will be issued during the first week...

Coal Mining
Lawsuit Filed to Block Sale of Utah Coal for Export
An environmental group has sued the Interior and Agriculture departments to block the sale of more than 42 million tons of Utah coal to be shipped overseas (WildEarth Guardians v. Jewell, D. Colo., No. 15-CV-1984, 9/11/15. ...

Drinking Water
DuPont's Documents Under Scrutiny as PFOA Trial Opens
The future of DuPont Co. spinoff Chemours Corp. might hinge on an Ohio woman, who blames the chemical giant for her kidney cancer and says the words of the company's own lawyers support her claim (Bartlett v. E. I. du Pont de Nemours &...

Drinking Water
Water Groups Release Guidance on Wastewater Reuse
Faced with burgeoning demand and stressed supplies, four major water resource organizations released a framework document on how municipalities can convert wastewater to drinking water....

Elections
Gov. Brown Challenges GOP Candidates on Climate Change
As he struggles to bring under control devastating wildfires that he blames at least partly on global warming, California Gov. Jerry Brown (D) has an unwelcome message for the Republican presidential candidates gathering in his state for...

Elections
Sanders Wins Praise for Pro-Ethanol Stance
Bernie Sanders' admiration for corn-based fuel is winning him some praise in unusual places....

Endangered Species
Jewell Anticipates Decision Soon on Sage Grouse
The U.S. Fish and Wildlife Service will make its Endangered Species Act decision on the greater sage grouse "on or before" its Sept. 30 deadline, Interior Secretary Sally Jewell said Sept. 15....

Endangered Species
Navy to Limit Mammal Risks During Training, Testing
The U.S. Navy will limit the use of sonar and in-water explosives during training and testing in certain areas off the coasts of Southern California and Hawaii to protect marine mammals under a court settlement approved Sept. 14 (Conservation...

Endangered Species
Ontario Court to Examine Endangered Species Exemptions
The Ontario Court of Appeal will consider whether the province can continue to legally exempt

major industries from regulations intended to protect endangered species....

Energy
DOE to Launch Small Business Voucher Program With Labs
The Energy Department announced it will launch a small business voucher pilot program Sept. 23 to provide funding for small businesses to partner with national laboratories to develop clean energy technologies. ...

Energy
House Committee Plans Energy Bill Markup Sept. 17
The House Energy and Commerce Committee will vote on omnibus energy legislation Sept. 17, the committee announced. ...

Energy
Mexico Sets Minimum Payments for Offshore Oil Auction
Mexico will require royalty payments of 30 percent to 36 percent for companies that win rights to produce oil in the nine shallow-water fields up for auction this month, the country's finance ministry said Sept. 15....

International Climate
Chinese, U.S. Cities, States Unveil Emissions Pledges
Nearly a dozen Chinese cities and provinces vowed Sept. 15 to peak their carbon emissions as much as a decade sooner than the 2030 national target China President Xi Jinping announced last November. ...

Mining
Legacy Mines Cleanup Policy Solutions to Be Debated
The policy solutions for cleaning up legacy mines will be the focus of a wave of congressional hearings this week looking into the release by Environmental Protection Agency contractors of 3 million gallons of mining waste into Colorado's...

Mining
Plan to Mitigate Leaching From B.C. Mine Criticized
Plans to mitigate the leaching of selenium from British Columbia's Elk Valley coal mines into boundary waters shared with Montana won't work, the University of Montana's director of the Center for Integrated Research on the...

Oil & Gas
Hoeven to 'Demand' Crude Oil Export Amendment to TSCA
Sen. John Hoeven (R-N.D.) said Sept. 15 he will "absolutely demand" that legislation to lift the 40-year-old ban on most crude oil exports be added as an amendment to a widely supported chemical safety bill that may be brought to the...

Oil & Gas
White House Opposes Bill to Lift Crude Export Ban
The White House said Sept. 15 it "wouldn't support" House legislation (H.R. 702) that would end the ban on most crude oil exports, as House leadership announced it planned to bring the bill to the floor at the end of September. ...

Oil Spills
BP Had No Hazardous Waste Reporting Duty for Gulf Spill
BP Plc had no obligation to report a release of hazardous substances in the wake of the Deepwater Horizon explosion and oil spill, a federal district court ruled Sept. 14 (In re Oil Spill by the Oil Rig "Deepwater Horizon" in the Gulf of...

Regulatory Policy
House Readies Streamlined Permitting Bill
The House Rules Committee is slated to take up a bill Sept. 16 known as the RAPID Act that would streamline permitting requirements, setting up a vote on the House floor as early as next week....

Renewable Energy
U.S. Solar Heads for Slump in 2017 If Tax Incentive Cut
The U.S. solar industry will slump in 2017 if Congress allows a key federal tax credit to lapse, Bloomberg New Energy Finance said in a report....

Solid Waste
Backing Duke, Green Groups Claim Win Over Regulators
In an unusual alliance with Duke Energy Corp., environmental groups are claiming victory over a North Carolina agency assigned to oversee the cleanup of coal ash disposal ponds after a major spill last year....

Stormwater
Ohio Sewer District Can Manage Stormwater, Court Rules
An Ohio sewer district has the authority to create a stormwater management program and charge a fee to support it, according to the state's highest court (Ne. Ohio Reg'l Sewer Dist. v. Bath Twp., Ohio, No. 2013-1770, 9/15/15)....

Superfund
CERCLA Settlement Doesn't Bar New Party's Claim
A Superfund settlement of claims that contamination migrated from an automotive machine shop onto a neighboring property in California doesn't bar another neighbor's claims against the machine shop, a federal court has ruled (Mehrabian...

Superfund
No Duty to Defend Indian Tribes' Superfund Claims
A clause barring insurance coverage for Superfund actions brought by the government includes claims lodged by Indian tribes in their capacity as natural resource trustees, a federal district court ruled (Century Indem. Co. v. The Marine...

Vehicle Fuels
EPA's McCarthy Reassures Biofuels Backers
The head of the Environmental Protection Agency told an ethanol industry conference Sept. 15 that the agency's upcoming final rule on the renewable fuel standard should protect long-term investment in advanced biofuels....

Vehicle Fuels
EU Renews Tariffs on U.S. Biodiesel for Five Years
The European Union renewed tariffs Sept. 15 on biodiesel from the U.S. for another five years in a sign of persistent trade tensions over renewable energy....

Water Pollution
Fear of Contamination Insufficient for Nuisance Class
The fear of migrating contamination was insufficient for a proposed class of Missouri landowners to proceed with nuisance claims against Conoco Phillips, the U.S. Court of Appeals for the Eighth Circuit ruled Sept. 15 (Smith v. ConocoPhillips...

Water Pollution

Murray Energy to Pay $2.5 Million for 2009 Fish Kill
Murray Energy Corp. has agreed to pay $2.5 million to the Pennsylvania Fish & Game Commission for a 2009 water discharge from an underground coal mine that caused a major fish kill in a tributary of the Monongahela River. ...

CORRECTION
An article in the Aug. 3 report on nickel exposure and allergies contained European exposure information that had been superseded (148 DEN A-11, 8/3/15). The European Union REACH regulation (1907/2006/EC), which superseded the EU's...

REGULATORY AGENDA

Comment Deadlines

SEPTEMBER 15 FEDERAL REGISTER

SEPTEMBER 16 FEDERAL REGISTER

Daily Environment Report

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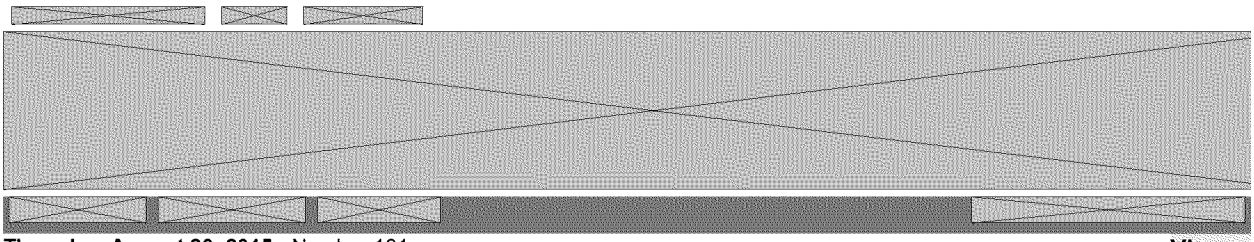

Thursday, August 20, 2015 · Number 161

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NEWS

Air Pollution
D.C. Circuit Halts Power Plant Startup Lawsuits
The U.S. Court of Appeals for the District of Columbia Circuit halted a pair of lawsuits over power plant startup provisions included in the mercury and air toxics standards, pending a decision on whether those standards should remain in place...

Air Pollution
Eight Areas Should Get Ozone Extension, EPA Finds
The Environmental Protection Agency proposed to grant eight nonattainment areas, including the Washington, D.C., and Houston metropolitan areas, an additional year to meet the current national ambient air quality standards for ozone....

Air Pollution
Lawsuit Seeks EPA Carbon Monoxide Review Documents
The Public Employees for Environmental Responsibility sued the Environmental Protection Agency's Office of Inspector General for failure to release documents related to a review of carbon monoxide exposure studies (Pub. Emp. for...

Climate Change
Antarctic Ice Melt Predictions Covered in Study
Antarctic ice melt could raise global sea levels by as much as 20 centimeters by the end of the century—enough to fill the Caspian Sea—and by close to 50 centimeters by 2200 under a worst-case scenario calculated by researchers...

Climate Change
Study: China's Carbon Emissions Lower Than Thought
China's carbon emissions since the turn of the century were significantly lower than estimated by

international organizations, the journal Nature reported Aug. 19, basing its finding in large part on a closer scrutiny of the type of coal...

Climate Regulation
Consolidation Denied in Clean Power Plan Lawsuits
A federal appellate court denied an emergency motion from 15 states seeking to consolidate their legal challenge to the Environmental Protection Agency's Clean Power Plan with other pending appeals (In re West Virginia, D.C. Cir. , No....

Coal Mining
Environmentalist Snaps Up Mines to Sell Coal With Carbon Credits From Planting Trees
An environmentalist is scooping up mines from bankrupt Patriot Coal Corp., betting he can turn the scorned fuel green and help revive the struggling Appalachian region....

Drinking Water
Drinking Water Levels of PFOA Unsafe, Group Says
Concentrations of perfluorooctanoic acid (PFOA) found in water systems serving 6.5 million Americans are harmful even though they are below a federal advisory level, according to a report the Environmental Working Group will release Aug....

Elections
Colorado Mine Spill Seeps Into Presidential Race
The spill of 3 million gallons of metals and sediment into Colorado's Animas River gushed into the presidential race conversation, as Republican contender Ben Carson toured the site and promised "a different kind" of Environmental...

Enforcement
Two More Executives Plead Guilty in Elk River Spill
The last of six Freedom Industries executives charged with triggering a 10-day drinking water crisis for 300,000 residents in West Virginia pleaded guilty Aug. 19 in federal court to three pollution charges United States v. Farrell, S.D....

Hazmat Transport
Judge Rejects Negligence Claims Against Norfolk, CSX
Norfolk Southern Railway Co. and CSX Transportation Inc. aren't liable for negligence in the derailment of a train carrying toxic cargo in Paulsboro, N.J., because neither company owned the train that derailed, employed the crew or operated...

Hydraulic Fracturing
Fracking Ban Cases Sent to Colorado Supreme Court
The Colorado Court of Appeals has sent challenges by the oil and gas industry of two local government hydraulic fracturing bans directly to the state Supreme Court for review (Colo. Oil and Gas Ass'n v. Longmont, Colo. Ct. App., No. 14CA1759,...

Oil & Gas
12 Senators Seek SEC Review of Offshore Drilling Risk Disclosures
Twelve senators opposed to offshore oil and gas drilling sent a letter Aug. 18 to the Securities and Exchange Commission asking the agency to review the risk disclosures of companies engaged in drilling on the Outer Continental Shelf....

Pesticides
USGS Finds Low Levels of Neonics in Numerous Waterways
Low levels of neonicotinoid insecticides were found in almost two-thirds of streams tested by U.S. Geological Survey researchers, according to a USGS study published online Aug. 18 in the journal

Environmental Chemistry....

Radioactive Waste
Yucca Would Have Small Impact on Groundwater, NRC Says
The proposed nuclear repository for spent nuclear fuel and high-level radioactive waste at Yucca Mountain would have a small impact on local groundwater, a Nuclear Regulatory Commission analysis found....

Regulatory Policy
California PUC Considers Expanding Public Record Access
The California Public Utilities Commission, under heat for cozy relationships with the regulated community, is proposing increased public access for all records submitted to regulators....

Renewable Energy
China Sees Consolidation of Solar Companies
China's government said Aug. 19 that it expects consolidation to accelerate among solar companies, which are struggling to cope with a shift in market conditions. Companies with the strongest technology, capital, management and brands...

Renewable Energy
SkyPower Lines Up $12B in Deals for Solar Power
Seven years ago, Kerry Adler watched as the disaster at Lehman Brothers Holdings Inc. scuttled his vision of renewable power riches. Today, he has reassembled assets from the collapsed investment bank, laying the foundation for a green energy...

Renewable Energy
Solar Developers Benefitting From Crowdfunding in China
When Kong Sun Holdings Ltd. went looking for money to build a small-scale solar power plant in Inner Mongolia, large banks weren't interested....

Renewable Energy
Wind Turbine Makers Become Top Stock Performers as Growth Surges
Europe's wind turbine makers are becoming top performers in the stock market as orders surge and the industry enjoys the fruits of cost cutting in years past....

Trade
Chinese Solar-Panel Maker Loses EU Duty Exemption
Chinese solar-panel producer Znshine PV-Tech Co. lost its exemption from European Union tariffs after EU trade authorities said the company breached the terms of a price-floor agreement....

Vehicle Fuels
Cheap Oil's Making It Tough for Ethanol to Pay the Bills
Cheap crude oil may make it hard for ethanol companies to pay their bills on time....

Water Pollution
Advisory Finally Lifted for Tennessee Creek Segment
A water contact advisory for 1.8 miles of Oostanaula Creek in Athens, Tenn., in place for more than 30 years, has been lifted. In an Aug. 17 announcement, the Tennessee Department of Environment and Conservation said the advisory was posted...

Water Pollution
Animas Spill Highlights Quest for Good Samaritan Bill
The Environmental Protection Agency's accidental spill of historic mining waste into the Animas River could improve chances for the passage of a bill in Congress to encourage good Samaritans

to clean up abandoned mines, Sen. Michael...

Water Pollution
Asphalt Oil Maker to Pay for Clean Water Act Violations
A Missouri manufacturer of asphalt oil will pay more than $200,000 to resolve charges that it violated the Clean Water Act and the Emergency Planning and Community Right-to-Know Act at a facility located next to the Eleven Point River in southern...

Water Pollution
Pavement Sealant Maker Fined $10M for Discharges
An asphalt sealant manufacturer in Atlanta faces a $10 million fine for water pollution violations, but a full site cleanup is more likely to result from a related lawsuit still pending against the property owner, according to the plaintiff,...

Water Resources
Plan for Calif. Desalination Plant Called 'Feasible'
An independent evaluation of water intake options for a proposed desalination plant in Orange County, Calif., appears to support a plan by the company developing the project to use existing pipes from an old power plant to withdraw seawater....

SPECIAL REPORT

Fuel Efficiency
California Has a Plan to End the Auto Industry as We Know It
Sergio Marchionne had a funny thing to say about the $32,500 battery-powered Fiat 500e that his company markets in California as "eco-chic."...

REGULATORY AGENDA

AUGUST 19 FEDERAL REGISTER

AUGUST 20 FEDERAL REGISTER

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To: Huff, Lisa[Huff.Lisa@epa.gov]
From: U.S. EPA
Sent: Thur 1/15/2015 6:23:09 PM
Subject: News Release: EPA's Actions to Restrict PFOA and Similar Chemicals Yield Significant Human Health and Environmental Benefits/Due to the agency's efforts, the CDC finds a 41 percent reduction in human blood-levels

CONTACT:

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202-564-4355

FOR IMMEDIATE RELEASE

January 15, 2015

EPA's Actions to Restrict PFOA and Similar Chemicals Yield Significant Human Health and Environmental Benefits

Due to the agency's efforts, the CDC finds a 41 percent reduction in human blood-levels

WASHINGTON – To further agency and industry achievements, the U.S. Environmental Protection Agency (EPA) today proposed measures to ensure that perfluorinated chemicals that have been phased out do not re-enter the marketplace without review.

"Through our environmental stewardship program, eight companies have helped us make real progress to reduce these chemicals as evidenced in the CDC findings" said Jim Jones, assistant administrator for EPA's Office of Chemical Safety and Pollution Prevention. "We will continue that progress now that all importers and other domestic manufacturers will be required to give EPA an opportunity to review and restrict uses of these perfluorinated chemicals."

Today's action builds on several EPA has taken since 2006, when reaching an agreement with companies to phase-out the chemicals by the end of 2015. Participating companies are on track to phase-out the chemicals by the end of 2015 and have successfully developed over 150 alternatives. EPA is also releasing the 2013 and 2104 companies' progress in meeting the 2015 phase-out goal.

These chemicals are used in a wide range of industrial applications and consumer goods, including cleaners, textiles, carpet, leather, paper and paints, fire-fighting foams, and wire insulation. These chemicals are toxic, persist in the environment worldwide, and can accumulate in people and animals.

EPA is proposing this Significant New Use Rule (SNUR) for Long-Chain Perfluoroalkyl Carboxylate chemicals in part in anticipation of this 2015 phase-out deadline. In 2013, EPA issued a final Significant

New Use Rule for use of perfluorinated chemicals in carpets and carpet aftercare products. EPA has also issued other Significant New Use Rules on perfluorinated chemicals, including perfluorooctane sulfonate (PFOS) and perfluoroalkyl sulfonates (PFAS) that were voluntarily phased-out of production.

This proposal requires that anyone who intends to import these perfluorinated chemicals, including in products, or domestically produce or process these chemicals for any new use submit a notification to EPA at least 90 days before beginning the activity. This notice will provide the agency with an opportunity to evaluate the new use and, if necessary, take action to prohibit or limit the activity.

Information on today's proposed rule and other actions EPA has taken on long-chain perfluorinated chemicals can be found at: http://www.epa.gov/oppt/existingchemicals/pubs/actionplans/pfcs.html

Information on progress on the 2010-2015 PFOA Stewardship Program can be found at: http://epa.gov/oppt/pfoa/pubs/stewardship/index.html

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NEWS

Air Pollution
French Study Says PM-10 Pollution Below EU Safety Level Still Significant Health Risk
Increased air pollution from coarse particles is linked to a "significant" rises in nonaccidental and cardiovascular deaths in French cities, even when pollution levels are below the European Commission's safety threshold,...

Air Pollution
New York Proposes Streamlined Air Regulations
The New York State Department of Environmental Conservation has proposed regulations to update and streamline rules to control emissions of criteria and non-criteria air pollutants. The regulations, which were published in the Dec. 31...

Chemicals
Drug Industry Groups Ask High Court To Review Case Involving Rx Disposal Law
Pharmaceutical industry groups have asked the U.S. Supreme Court to review an appeals court decision upholding a California county's drug disposal law (Pharm. Research & Mfrs. of Am. v. Cnty. of Alameda, U.S., No. 14-751, 12/24/14)....

Chemicals
EU Chemicals Agency Calls for Input On German-Norwegian Proposed PFOA Ban
The European Chemicals Agency is calling for comments through June 17 on a joint German-Norwegian proposal to prohibit the manufacture, use or sale in the European Union of perfluorooctanoic acid, its salts and substances that could degrade...

Chemicals
Japan to Classify Azo Compounds Hazardous
Japan will list two dozen azo compounds as hazardous substances requiring parties that use them to meet a set of detailed standards and tolerance levels starting April 1, the Ministry of Health, Labor and Welfare announced. The 24 substances...

Chemicals
No 'Every Exposure' Asbestos Testimony In Case Against Adhesive Product Maker
Bayer CropScience, Inc. (Amchem) secured the exclusion of specific causation opinions of a plaintiff's expert who asserted the adhesive maker was liable in an asbestos case under the "each and every exposure" theory, according...

Chesapeake Bay
Some Maryland Farmers Eligible for Rules Exemption
The Maryland Department of Agriculture (MDA) announced Jan. 7 the approval of final regulations that allow farmers who have voluntarily met their Chesapeake Bay total maximum daily load nutrient and sediment reduction requirements to obtain...

Climate Change
'All-Out Attack' Being Waged to Undermine Science on Environment, EPA Chief Says
An "all-out attack" is being waged in Washington, D.C., in an effort to undermine the scientific evidence on a changing global environment, the head of the Environmental Protection Agency said Jan. 7....

Climate Regulation
EPA Delays Timeline for Finalizing Carbon Standards for New, Existing Power Plants
The Environmental Protection Agency is delaying its schedule for issuing final carbon emissions standards for power plants, the agency announced Jan. 7....

Climate Regulation
Nonprofit Groups Sue Energy Department For Kemper Carbon Capture Project Records
Two free-market legal groups filed a lawsuit in federal district court seeking documentation about the status of the Southern Co.'s Kemper County Energy Facility that the Environmental Protection Agency cited as an example of "adequately...

Climate Science
Fossil Fuels Must Remain in Ground To Restrict Warming, Scientists Say
Two-thirds of the world's fossil-fuel reserves must remain unburnt to hold temperature increases below dangerous levels, according to researchers at University College London....

Congress
Highway Bill, Robust EPA Oversight Atop Inhofe 2015 Senate Committee Agenda
Reaching a "robust" highway bill agreement and conducting aggressive oversight of Environmental Protection Agency regulations are the top two priorities for Senate Environment and Public Works Committee Chairman James Inhofe...

Drinking Water
Elk River Spill Shows Better Tap Water

Flushing Method Is Needed, Scientists Say
Science-based methods to flush tap water after source water is contaminated should be developed to expedite recovery, minimize health effects and reduce contaminant concentrations in homes, according to research published in "Environmental...

Endangered Species
Interior Secretary Issues Order to Address
Wildfires Destroying Sage Grouse Habitat
Interior Secretary Sally Jewell issued a secretarial order (No. 3336) outlining a strategy to address more frequent and intense wildfires that are damaging landscapes critical to greater sage grouse and other sagebrush species. ...

Energy
Drop in Natural Gas Price Raises Risk
For Aging Coal, Nuclear Power Plants
A 37 percent drop in natural gas prices since June has lowered what U.S. nuclear and coal plants can charge for electricity, potentially speeding the demise of generators teetering on the brink of closing....

Energy Efficiency
Energy Department Seeks Common Definition
To Aid Development of Zero Energy Buildings
The Energy Department is seeking public comment on a proposed common definition for zero energy buildings (ZEBs)....

Energy
Infrastructure Recommendations Coming
As Efficiency Push Continues, Moniz Says
The Energy Department will release a series of recommendations for modernizing and improving the nation's energy infrastructure in February as the first installment of its Quadrennial Energy Review, Secretary Ernest Moniz said Jan....

Energy
Obama Keystone Veto Threat Sets Up
Many Battles on Republican Agenda
President Barack Obama has vetoed fewer bills than any U.S. president since James Garfield held the office for six months in 1881. With Republicans now in control of Congress, that probably will change....

Energy
White House Formally Threatens to Veto
House Bill to Approve Keystone XL Pipeline
The White House Jan. 7 formally threatened to veto legislation that would approve the Keystone XL pipeline, saying a House bill (H.R. 3) expected to be approved later this week "conflicts with longstanding Executive branch procedures...

Hazmat Transport
White House Reviewing Proposal Seeking
Minimum Crew Size for Trains Carrying Crude
The White House Jan. 7 received for interagency review a Federal Railroad Administration proposed rule that is expected to establish a two-person crew minimum for trains carrying crude oil....

Hydraulic Fracturing

Draft Virginia Fracking Rule Would Require Public Disclosure of All Fluid Ingredients
Companies using hydraulic fracturing to extract oil and natural gas in Virginia would have to disclose to the public all ingredients used in the process, including all ingredients in substances claimed as trade secrets, under a Department...

Insurance
Buffett's Commercial Insurer Plans To Offer New Pollution-Coverage Option
Berkshire Hathaway Inc., the firm controlled by billionaire Warren Buffett, is pushing to sell more insurance to energy and construction companies by offering as much as $25 million in new pollution coverage....

Invasive Species
EPA Says Small Vessels Need Ballast Water Coverage Despite Congressional Ban
Vessels shorter than 79 feet will need to seek Clean Water Act permits to cover discharges of ballast water regardless of the three-year moratorium imposed by Congress on the regulation of incidental discharges, according to the Environmental...

Oil & Gas
Colorado Oil, Gas Commission Stiffens Penalties for Rules Violations
The Colorado Oil and Gas Conservation Commission approved rule and policy changes (Rules 522 & 523) significantly increasing penalties for violations of the state's drilling regulations. ...

Pesticides
Industry Trying to Use Trans-Atlantic Talks To Weaken Pesticide Rules, Report Claims
The pesticide industry is trying to influence the outcome of a proposed trans-Atlantic trade agreement to weaken agricultural regulations, an environmental law firm charged in a report....

Regulatory Policy
House Resumes Push to Revamp Regulatory Procedures, Eyes Vote on Accountability Act
House Republicans Jan. 7 formally resumed their campaign to roll back regulatory burdens and revamp the rulemaking process with the introduction of the Regulatory Accountability Act, the first of several deregulation bills likely to get...

Renewable Energy
Cheap Solar Power Emerging From Mineral Named for Russian Count
Materials that may be cheaper and more efficient than silicon at converting the sun's rays into electricity could be key to the next generation of solar power, scientists say....

Renewable Energy
Utilities Cancel Power-Purchase Agreements After Cape Wind Misses Dec. 31 Deadline
Cape Wind, the $2.6 billion offshore wind farm that's been under development in Massachusetts for 13 years, is facing a significant threat as two utilities seek to terminate contracts to buy electricity....

Right to Know
Environmentalists File Lawsuit Over

Energy Companies' Toxics Release Data
A lawsuit filed Jan. 7 by a coalition of 10 environmental groups eventually could lead to new requirements on oil and gas extraction companies to report toxic pollution data to the Environmental Protection Agency (Envtl. Integrity Project...

Risk Assessment
EPA Draft Report on Inhaled Ethylene Oxide
Endorsed by Agency's Advisory Committee
A scientific advisory committee endorsed in a draft report released Jan. 7 the Environmental Protection Agency's draft assessment of the carcinogenicity of inhaled ethylene oxide....

Storage Tanks
West Virginia Water Crisis Spawned
Safeguards, But Some Tanks Called Unfit
In the year following West Virginia's drinking water emergency, the state has become a national model for aboveground storage tank regulation, yet it still is home to 1,100 chemical containers deemed "unfit for service," according...

Water Pollution
EPA Approves Trash Reduction Plan for River, Harbor
The Environmental Protection Agency and the Maryland Department of the Environment Jan. 6 announced a total maximum daily load program for trash in sections of Maryland's Patapsco River. The 39-mile-long river empties into the Baltimore...

Water Pollution
EPA to Hold Webinar on e-NPDES Reporting Rule
The Environmental Protection Agency on Jan. 20 will hold a webinar to discuss the supplemental notice to the proposed rule on electronic reporting under the Clean Water Act discharge permit program. The Dec. 1 supplemental notice sought to...

Water Pollution
Groups Ask Courts to Force EPA Responses
To Petitions on Two States' NPDES Authority
The Sierra Club and allied groups filed suit Jan. 7 to force the Environmental Protection Agency to respond to their petitions seeking federal takeover of Clean Water Act permitting programs in Kentucky and West Virginia....

INTERVIEW

Views on Mexico's New Energy Laws From Juan Acra Lopez,
President of COPARMEX's Energy Commission
Bloomberg BNA: What role do you envision Mexican enterprises playing in the upcoming energy auctions? ...

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JANUARY 8 FEDERAL REGISTER

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NEW YORK STATE OF OPPORTUNITY. | Department of Environmental Conservation | Department of Health

January 14, 2016

The Honorable Gina McCarthy
Administrator
USEPA Headquarters
William Jefferson Clinton Building
1200 Pennsylvania Avenue, N. W.
Mail Code: 1101A
Washington, DC 20460

Dear Administrator McCarthy:

We write to you to request that EPA take vigorous action to address the presence of perfluorooctanoic acid (PFOA) in drinking water and groundwater. Respectfully, we ask that EPA:

- lower its provisional health advisory of 400 parts per trillion (ppt) for PFOA drinking water to take into account the most current scientific evidence;
- act expeditiously to adopt a protective maximum contaminant level for PFOA;
- expeditiously list PFOA as a hazardous substance under the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA) to facilitate the cleanup of contaminated groundwater and other media; and
- review the remaining uses of PFOA under the Toxic Substances Control Act and curtail it whenever less toxic alternatives are available.

The New York State Department of Health has been working with the Village of Hoosick Falls and the Town of Hoosick for more than a year to address PFOA contamination of drinking water. PFOA in the Village of Hoosick Falls public water supply exceeds the provisional EPA health advisory of 400 ppt. Private wells in the Town of Hoosick have also shown signs of contamination, but at lower levels than in the municipal supply. The Department of Health, the Village, and Saint-Gobain Performance Plastics have collaborated to evaluate treatment options for the Village water supply, implement a bottled water program, and design and order a temporary treatment system to be installed in the coming weeks on the Village water supply. This temporary treatment system will remain in place until a planned permanent treatment system is operational later this year.

We write to you because this is not just a local issue. The presence of PFOA in drinking water is an emerging nation-wide issue.

NEW YORK STATE OF OPPORTUNITY. | Department of Environmental Conservation | Department of Health

As reported in the New York Times Magazine on January 10, 2016, several studies have asserted that the presence of PFOA in drinking water and groundwater may be more pervasive than originally thought and may subject people across the country to PFOA exposure since EPA first began working on this issue in 2001.

It is imperative that the federal government step forward and use the authority it already holds under federal law to comprehensively address this national issue. The State of New York stands ready to assist EPA in any way we can in this important effort to protect public health and the environment from PFOA.

Sincerely,

Howard Zucker M.D.

Dr. Howard Zucker
Commissioner
DOH

Basil Seggos
Acting Commissioner
DEC

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We write to you to request that EPA take vigorous action to address the presence of perfluorooctanoic acid (PFOA) in drinking water and groundwater. Respectfully, we ask that EPA:

- lower its provisional health advisory of 400 parts per trillion (ppt) for PFOA drinking water to take into account the most current scientific evidence;
- act expeditiously to adopt a protective maximum contaminant level for PFOA;
- expeditiously list PFOA as a hazardous substance under the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA) to facilitate the cleanup of contaminated groundwater and other media; and
- review the remaining uses of PFOA under the Toxic Substances Control Act and curtail it whenever less toxic alternatives are available.

The New York State Department of Health has been working with the Village of Hoosick Falls and the Town of Hoosick for more than a year to address PFOA contamination of drinking water. PFOA in the Village of Hoosick Falls public water supply exceeds the provisional EPA health advisory of 400 ppt. Private wells in the Town of Hoosick have also shown signs of contamination, but at lower levels than in the municipal supply. The Department of Health, the Village, and Saint-Gobain Performance Plastics have collaborated to evaluate treatment options for the Village water supply, implement a bottled water program, and design and order a temporary treatment system to be installed in the coming weeks on the Village water supply. This temporary treatment system will remain in place until a planned permanent treatment system is operational later this year.

We write to you because this is not just a local issue. The presence of PFOA in drinking water is an emerging nation-wide issue.

NEW YORK STATE OF OPPORTUNITY. | Department of Environmental Conservation | Department of Health

As reported in the New York Times Magazine on January 10, 2016, several studies have asserted that the presence of PFOA in drinking water and groundwater may be more pervasive than originally thought and may subject people across the country to PFOA exposure since EPA first began working on this issue in 2001.

It is imperative that the federal government step forward and use the authority it already holds under federal law to comprehensively address this national issue. The State of New York stands ready to assist EPA in any way we can in this important effort to protect public health and the environment from PFOA.

Sincerely,

Howard Zucker M.D.

Dr. Howard Zucker
Commissioner
DOH

Basil Seggos
Acting Commissioner
DEC

From: Owens, Jim
Required Attendees: CI ALL; Lindstrom, Andrew; Buckley, Timothy; ORD-NERL-IO; ORD-NERL-IO-RPCS; ORD-NERL-AMAD Feds and NonFeds; ORD-NERL-ERD; ORD-NERL-ESD; ORD-NERL-HEASD Feds and NonFeds
Optional Attendees: Sayles, Gregory; Gonzalez, Michael; Coffel, Debra; Ziser, Shelly; Cabezas, Heriberto; Dasu, Kavitha; Mills, Marc; Vazquez, Margie; Gonzalez, Alejandra; Harmon, Dave; Andrews, Jenifer; Gilliland, Alice; Wang, Bo; Finan, Katherine; Nelson, Khary; Vanselow, Deborah; Luebbering, Gregory; Morgan, Jeffrey; Cubbison, Christopher; Newhart, Gary; Barrett, William; Cain, Cathy; Flotemersch, Joseph; Murray, Daniel; Miller, Diana; Conner, Michele; Impellitteri, Christopher; Legare, Pamela; Melnyk, Lisa; Rahman, Maqsud; Heckman, Lee; Martin, Roy; Fulk, Florence; Adams, Tammy; Varughese, Eunice; Lu, Jingrang; Brown, Kendra; Parris, Brenda; Bain, Zeno; Martin, Todd; Lindquist, Alan; Plunkett, Trevor; Reid, Jon; Lyons, Terry; Hadley, Angela; Coggins, Cathy; Hutcherson, Chris; Griffin, Shannon; Gillespie, Andrew; Owens, Beth; Acheson, Carolyn; Carroll, Gregory; Brinkmiller, Michael; Lazorchak, Jim; Stelma, Gerard; See, Mary Jean; Haworth, Jerry; Simic, Melissa; Toth, Greg; Bagley, Mark; Glassmeyer, Susan; Troyer, Michael; Waters, Tom; Behymer, Thomas; Koumai, Ouro; Autrey, Brad; Parker, Randy; Rhodes, Eric; Batt, Angela; Shoemaker, Jody; Savage, Russell; Gatchett, Annette; Jackson, Toya; Villegas, Eric; Scola, Jennifer; Tettenhorst, Dan; Allen, Derrick; Ransick, Linda; Powers, Jeanna; Triantafyllidou, Simoni; Colon, Dalizza; Herwehe, Jerry; Newton, Seth; Jenkins, Tom; Pitchford, Ann; Kaushik, Surender; Stroup, Gene; Polite, Gwendolyn; Conner, Teri; Betowski, Don; Purucker, Tom; Schumacher, Brian; Medina-Vera, Myriam; Patel, Ashok; McCurdy, Tom; Hall, Eric; Starr, James; Weinstein, Jason; Weber, Eric; Magnuson, Matthew; Nees, Monica; Mills, Noelle; Baxter, Lisa; Bouchard, Dermont; Ulrich, Elin; Price, PaulS; Oliver, Karen; Biryol, Derya; Voit, Jim; Duvall, Rachelle; Johnston, JohnM; Dodmane, Puttappa; Mukerjee, Shaibal; Stanek, Lindsay; Tulve, Nicolle; Lewandowski, Michael; Cashdollar, Jennifer; Sobus, Jon; Nolte, Chris; Strynar, Mark; Noel, James; Kenneke, John; Speth, Thomas; Gallardo, Vincent; Tornero-Velez, Rogelio; Landis, Matthew; Hartzell, Evelyn; Personal Email / Ex. 6 McMahen, Rebecca; McMillan, Larry; Farrar, David
Location: AWBERC Auditorium
Importance: Normal
Subject: Seminar by Dr. Andrew Lindstrom (NERL-RTP): "Current research priorities for per and polyfluoroalkyl substances (PFAS): implications of UCMR3 results and industry reformulations" (presentation attached)
Start Date/Time: Tue 9/22/2015 6:00:00 PM
End Date/Time: Tue 9/22/2015 7:00:00 PM
Lindstrom Cincinnati 9-22-15.pptx

NERL VTC Locations:
Las Vegas - Exc Conference Room 212
RTP - E230A
Athens - no available rooms

Call-In Info:
US/Canada Dial-In Number: (866) 299-3188
Int'l/Local Dial-in #: Conference Code/ Ex. 6
Conference Code: Conference Code/ Ex. 6

Biography
Andrew B. Lindstrom has a Ph.D. from the University of North Carolina's School of Public Health and has been working for the United States Environmental Protection Agency's (USEPA) National Exposure Research Laboratory (NERL) for the past 25 years. He is currently working in the Exposure Measurements and Analysis Branch where his areas of expertise include measurement

of trace level contaminants in environmental and biological matrices and human exposure assessment.

Abstract

Current research priorities for per and polyfluoroalkyl substances (PFAS): implications of UCMR3 results and industry reformulations
With more than a decade of intensive scientific research and increasing regulatory pressure worldwide, the sources of per- and polyfluoroalkyl substances (PFAS) in the environment and routes of human exposure still need to be fully characterized. Research has documented that PFAS contamination in the environment is often associated with direct emissions from chemical production facilities, effluents from municipal wastewater treatment plants, and the application of sewage sludge in agricultural areas. International regulatory efforts have succeeded in reducing the traditional PFAS such as PFOS and PFOA in many areas, but they have also served to relocate the production of these legacy materials to regions with developing economies and less stringent regulation. Moreover, a whole new generation of PFAS has replaced the regulated materials previously produced in the Western world. Interim results from the USEPA's Unregulated Contaminant Monitoring Rule 3 (UCMR3) program now provide detailed information on the occurrence of six PFAS in finished drinking water across the US. These data serve as an important source of information to: 1) identify and classify the sources of current PFAS contamination, 2) assess, test, and improve mitigation and control technologies used by water providers, 3) assess the importance of drinking water as a route of human exposure in specific populations, and 4) identify communities to conduct epidemiological studies to investigate the origins of PFAS-related disease.

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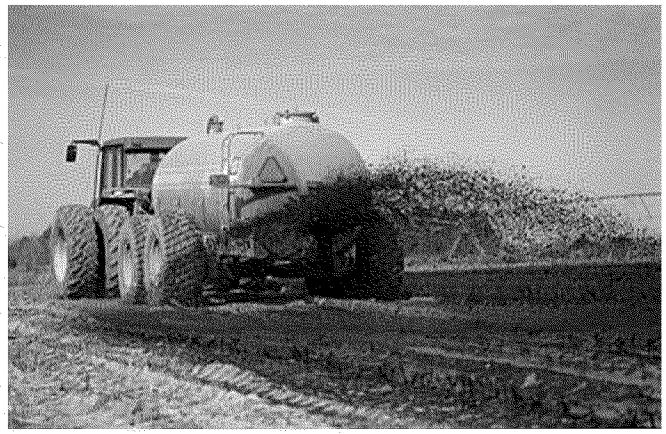
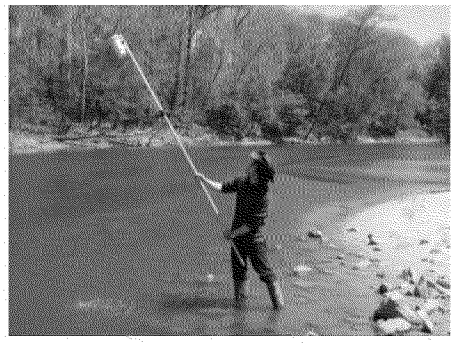
Cell: 919-302-6635
Email: lindstrom.andrew@epa.gov

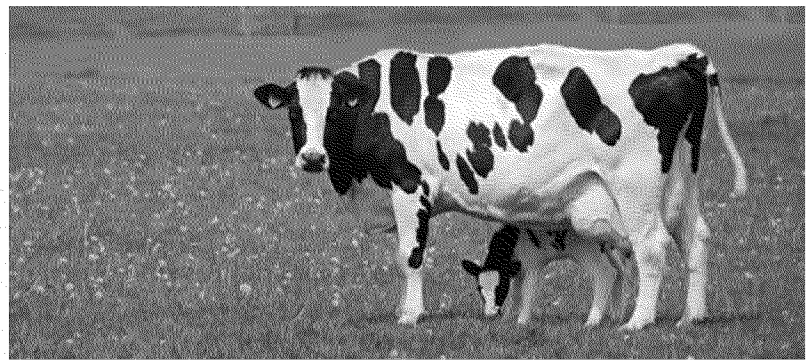
Current research priorities for per- and polyfluoroalkyl substances (PFAS): implications of UCMR3 results and industry reformulations

Andrew B. Lindstrom

U.S. Environmental Protection Agency, National Exposure Research Laboratory, Research Triangle Park, NC



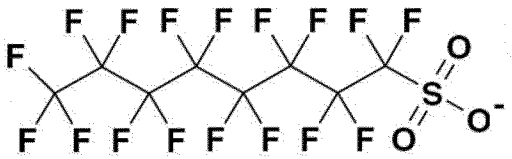




USEPA
AWBERC Auditorium
Cincinnati, OH
September 22, 2015

Presentation Outline

Discuss per- and polyfluoroalkyl substance (PFAS), their properties, toxicity, regulations



Discuss “replacement” PFAS

Summary of interim results from the UCMR3 program, implications, and opportunities

Long-term outlook regarding PFAS

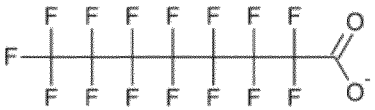
Three major considerations

Principle of Precaution…"where there are threats of serious or irreversible damage, lack of full scientific certainty shall not be used as a reason for postponing cost effective measures to prevent environmental degradation". *Commission of the European Communities: (2000)* *http://ec.europa.eu/dgs/health_consumer/library/pub/pub07_en.pdf*

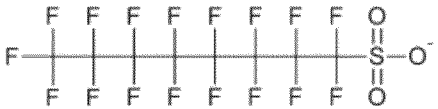
Green Chemistry…always strive to use the least toxic alternative available, with a preference for compounds that quickly and harmlessly degrade to their original starting materials
Anastas PT, Warner JC. 1998. Green Chemistry: Theory and Practice. New York, NY Oxford University Press

Environmental Justice…"the avoidance of hazards and acquisition of benefits through relationships that negatively impact the environment of others" *Steve Wing, (2015) University of North Carolina*

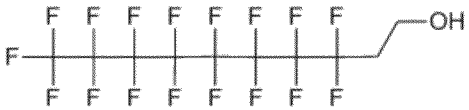
Some per- and polyfluoroalkyl substance (PFAS)



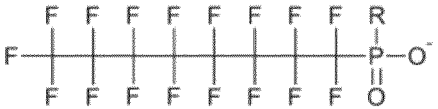
Perfluorocarboxylic acids
(ex. PFOA)



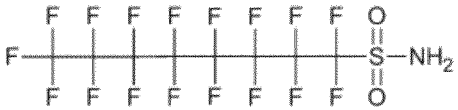
Perfluorosulfonic acids
(ex. PFOS)



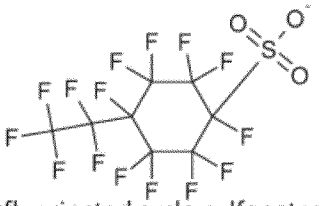
Fluorotelomer alcohol
(ex. 8:2 FTOH)



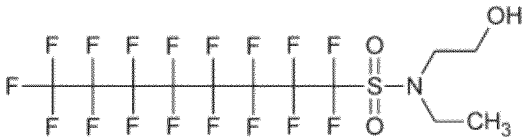
Perfluorophosphonic/phosphinic acids
(ex. If R=OH then PFOPA
If R=C8 perfluoroalkane then 8:8 PFPi)



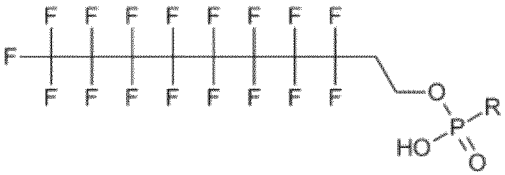
Perfluorosulfonamide
(ex. FOSA)



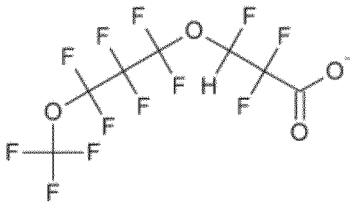
Perfluorinated cyclo sulfonates
(ex. PFECHS)



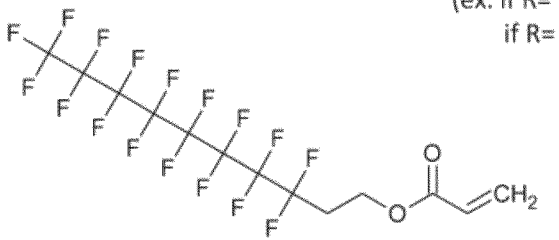
Perfluorosulfonamidoethanol
(ex. N-EtFOSE)



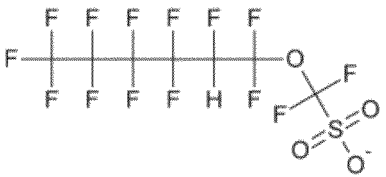
Fluorotelomer phosphate esters
(ex. if R= OH then 8:2 monoPAP
if R= 8:2 FTO ester then 8:2 diPAP)



Polyfluorinated ether carboxylates
(ex. 4,8-dioxa-3H-perfluorononanoate)



Polyfluorinated polymeric unit
(ex. 1H,1H,2H,2H-perfluorodecyl acrylate)

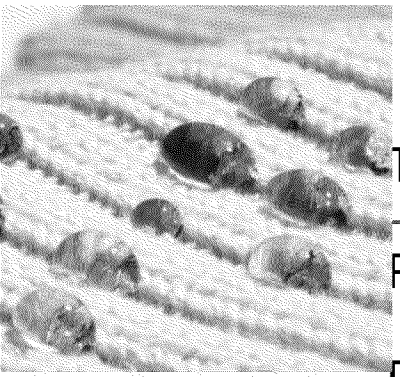


Polyfluorinated ether sulfonates
(ex. Perfluoro [hexyl ethyl ether sulfonate])

Figure 1. Generic structures for polyfluorinated compounds. The $n = 8$ linear carbon structures are shown for many of these examples, but $n = 4-14$ linear and/or branched carbon units are generally possible.

PFAS used in many products

Many PFAS are toxic, extremely persistent, and bioaccumulative



Textile treatments

Paints

Pesticides

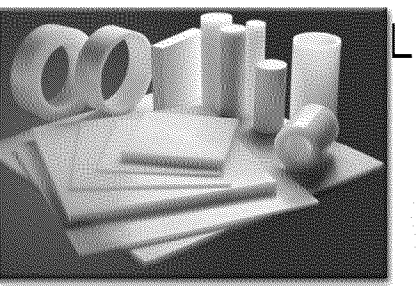
Floor polish

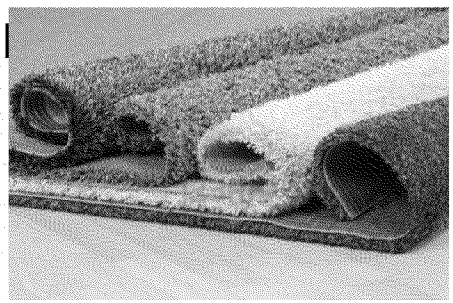
Denture cleaner

Polymers

Adhesives

Lul





Paper coatings

Surfactants

Fire-fighting foam

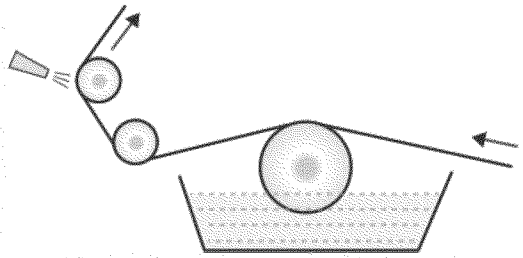
Photographic film

Shampoo

Non-stick cookware

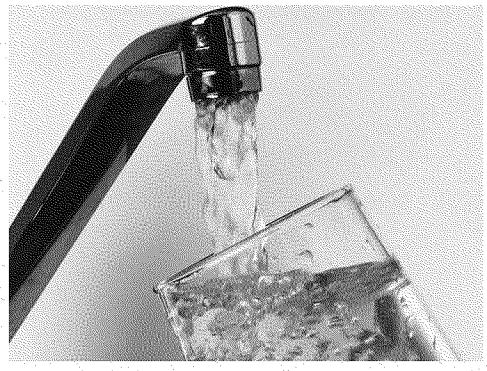
Caulks

Carpets



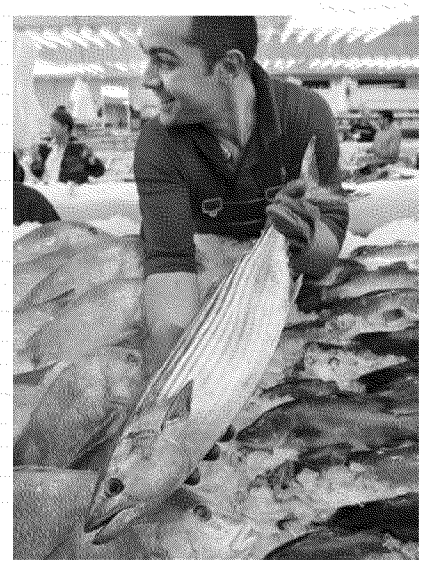
Sources of PFAS exposure for humans

Best documented source includes contaminated drinking water around industrial operations e.g., Cottage Grove, Minnesota; Parkersburg, West Virginia; Dalton, Georgia; Decatur, Alabama; Arnsberg, Germany; Osaka, Japan

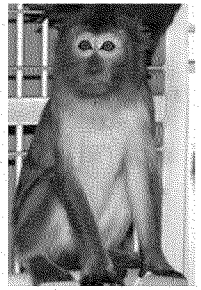


Food is also implicated in many studies (mostly modeling), but there are few good data on food items (complex matrices). Exception is fish, which is a well documented source.

Fromme et al. 2009, Inter. J. Hyg. & Envr. Heath (212) 239-270



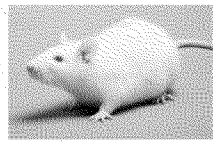
PFAS Health Effects Summary



Animal toxicity

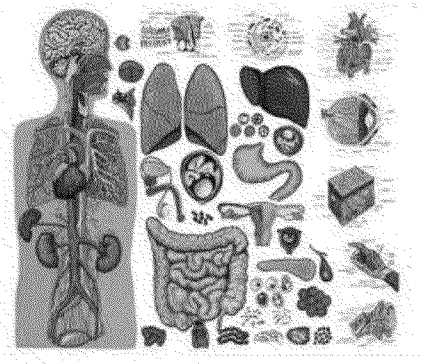
- Causes liver, immune system, developmental, endocrine, metabolic, and neurobehavioral toxicity.
- PFOA and PFOS caused tumors in chronic rat studies.

Post et al., (2012) Perfluorooctanoic acid (PFOA) , an emerging drinking water contaminant: A critical review of recent literature, *Environmental Research* (116) 93-117



Human health effects associated with PFAS in the general population and/or communities with contaminated drinking water include:

- ↑ cholesterol
- ↑ uric acid
- ↑ liver enzymes
- ↓ birth weight
- ↓ vaccine response
- Thyroid disease
- Osteoarthritis
- Diabetes
- Testicular and kidney cancer
- Pregnancy-induced hypertension
- Ulcerative colitis
- Effects in young adulthood from prenatal exposures
  - *Obesity in young women.*
  - *↓ sperm count in young men.*



Steenland et al., (2014), *Epidemiology* (25) 167-169; http://www.c8sciencepanel.org/

2009 US Environmental Protection Agency Short-term Provisional Health Advisories

Provisional Health Advisory levels for PFOS and PFOA in drinking water

PFOS = 200 ng/L PFOA = 400 ng/L

Short term exposure only (months) – no long-term (chronic) standard set

State of New Jersey long-term chronic health-based drinking water guidance for PFOA of 40 ng/L issued in 2007

* Some experts calling for reduction in EPA's Provisional standards by a factor of 100 – 1000 to be truly protective for chronic exposures

PFOS = 2 ng/L PFOA = 4 ng/L

* Immunotoxicity of perfluorinated alkylates: calculation of benchmark doses based on serum concentrations in children Grandjean, P ; Budtz-Jorgensen, E ;Environmental Health (12:35) DOI: 10.1186/1476-069X-12-35, APR 19 2013

US Environmental Protection Agency
PFOA Stewardship Program

In January 2006, USEPA started this program to help minimize impact of PFOA in the environment

Eight major international companies have agreed to participate (including 3M, DuPont, Asahi Glass, Daikin)

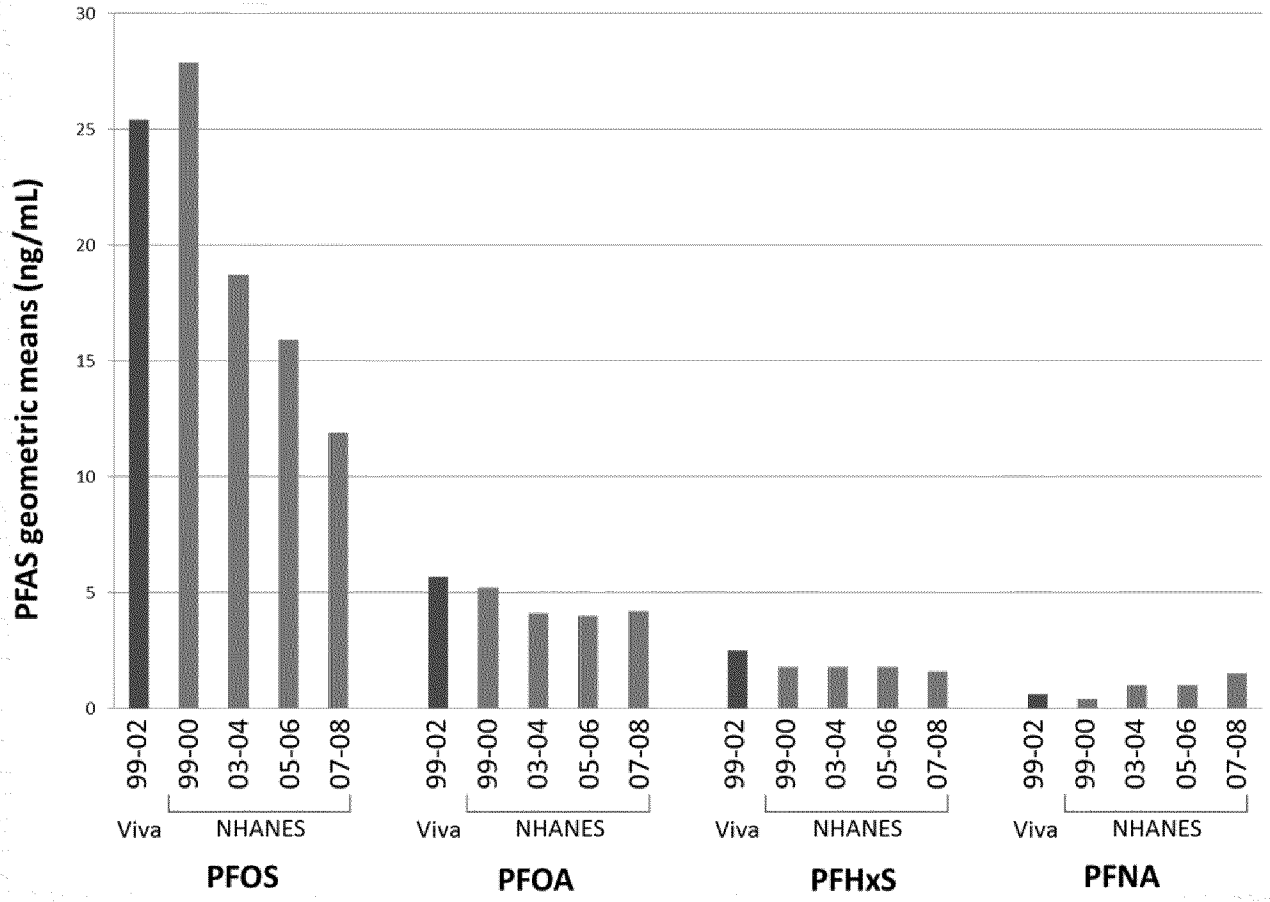
Agreement to voluntarily reduce factory emissions and product content of PFOA and related compounds* on a global basis by 95% no later than 2010

Agreement to work toward total elimination of emissions and product content of these compounds by 2015

Based on emissions and content determinations made for 2006

* Includes PFOA, precursor chemicals that can break down to PFOA, higher homologues (C9 and larger)

Trends in PFAS Serum Levels in US



Sagiv et al. (2015) *Environ. Sci. Technol.* DOI: 10.1021/acs.est.5b02489

Table 2. Geometric mean and 95% confidence interval and selected percentiles of PFOS, PFOA, PFHxS, and PFNA serum concentrations (ng/mL) for the U.S. population 12 years of age and older: Data from NHANES 2011-2012 [a]

	Geometric Mean (95% Confidence Interval)		Selected Percentiles			
			50th	75th	90th	95th
PFHxS	1.28	1.15-1.43	1.27	2.26	3.81	5.43
PFOS	6.31	5.83-6.82	6.51	10.48	15.62	21.68
PFOA	2.08	1.95-2.22	2.08	3.02	4.35	5.67
PFNA	0.88	0.80-0.97	0.86	1.30	1.95	2.54

Perspectives | Brief Communication

The Madrid Statement on Poly- and Perfluoroalkyl Substances (PFASs)

http://dx.doi.org/10.1289/ehp.1509934

As scientists and other professionals from a variety of disciplines, we are concerned about the production and release into the environment of an increasing number of poly- and perfluoroalkyl substances (PFASs) for the following reasons:

1. PFASs are man-made and found everywhere. PFASs are highly

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Environmental Health Perspectives • VOLUME 123 | NUMBER 5 | May 2015

Legacy PFAS continue to be a problem – persistent;

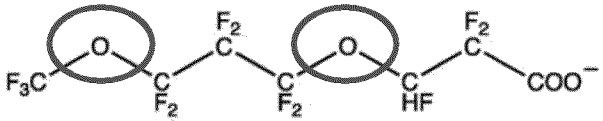
production of legacy compounds pushed to developing world;

almost no information on toxicity, transport and fate, bioaccumulation and, persistence of replacement compounds

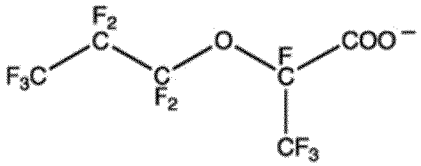
Z. Wang et al. / Environment International 60 (2013) 242–248

Fluoropolymer manufacture

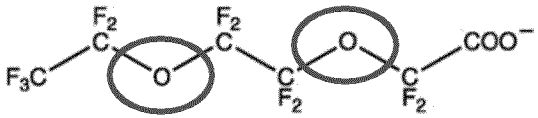
ADONA (CAS No. 958445-44-8)



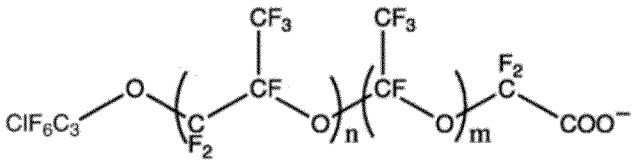
GenX (CAS No. 62037-80-3)



Asahi's product (CAS No. 908020-52-0)

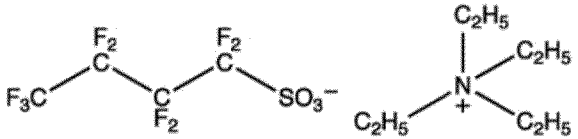


Solvay's product (CAS No. 329238-24-6)

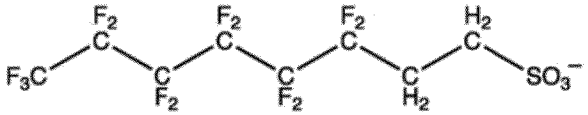


Metal plating

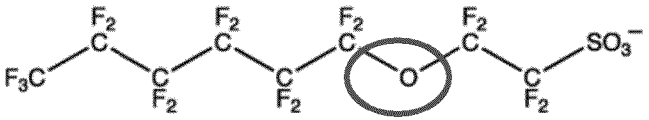
N(Et)$_4$-PFBS (CAS No. 25628-08-4)



6:2 FTSA (CAS No. 27619-97-2)



F-53 (CAS No. 754925-54-7)



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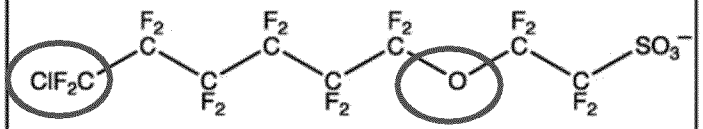
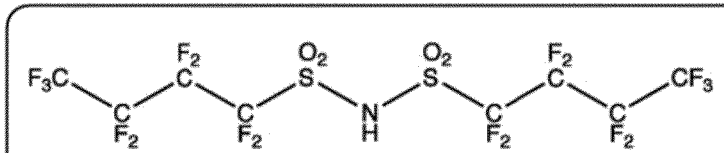
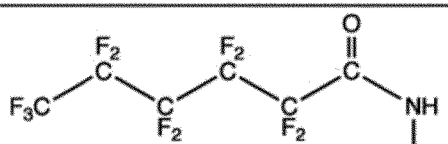
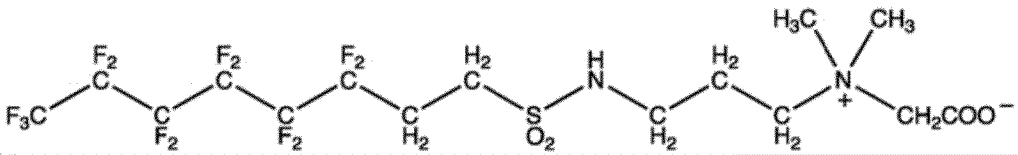
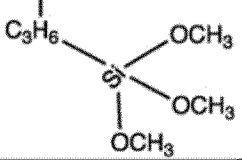


Fire fighting foams and miscellaneous









Unknown characteristics of “alternative” fluorinated compounds

- Actual identities of alternatives unknown in industrial sectors and geographical regions that are not well regulated

- Data on environmental and human health effects are incomplete (at best) and more often nonexistent

- Data on degradability, bioaccumulation, and toxicity (environmental and human) are incomplete (at best) or completely lacking

- Information on production volume and environmental emissions not available

Z. Wang et al. / Environment International 60 (2013) 242–248

US Environmental Protection Agency Drinking Water Activities

PFOS and PFOA on the Candidate Contaminant List (CCL) for possible regulation by National Primary Drinking Water Regulations

Unregulated Contaminant Monitoring Regulation 3 (UCMR3) announced in March 2011, approved by OMB in March 2012

UCMR selects candidate pollutants for national screening every five years

All PWS serving over 10,000 people and 800 PWS serving fewer than 10,000 people required to monitor for compounds for a continuous 12 month period between January 2013 and December 2015

UCMR 3 includes: PFOS (40 ng/L), PFOA (20 ng/L), PFNA (20 ng/L), PFHxS (30 ng/L), PFHpA (10 ng/L), PFBS (90 ng/L)

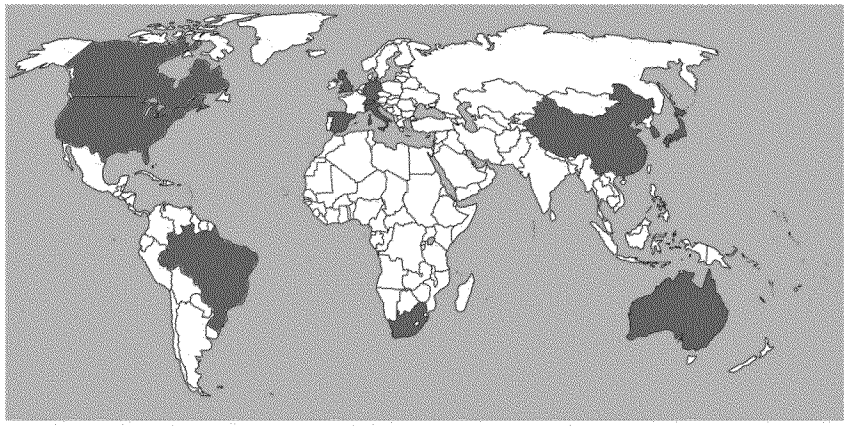
Unregulated Contaminant Monitoring Regulation 3 (UCMR3)

Interim results most recently released January 2015
http://water.epa.gov/lawsregs/rulesregs/sdwa/ucmr/data.cfm

3,605 Public Water Systems (PWS) with results so far

185 records with PFOS above the Reporting Limit of 40 ng/L,
68 PWS with levels above 40 ng/L

220 records with PFOA above the Reporting Limit of 20 ng/L
72 PWS with levels above 20 ng/L



PFASs have been detected worldwide in surface water, wastewater, groundwater, drinking water, and landfill leachates.

UCMR3 requires monitoring for six PFASs in US drinking water. Monitoring began in 2013, and latest data release was January 2015.

PFAS	MRL (ng/L)	Occurrence (%)	Max (ng/L)
C7	10	0.66	82 (NY, DE, PA)
C8	20	0.96	349 (PA)
C9	20	0.05	55.8 (NJ, PA)
PFBS	90	0.03	150 (CO, PA, AL)
PFHxS	30	0.61	680 (DE, PA, CO)
PFOS	40	0.81	1,800 (DE, CO, PA)

To date: 22,942 samples from 3,605 PWSs

PFAS detects: 351 samples (1.5%) from 132 PWSs (3.7%)

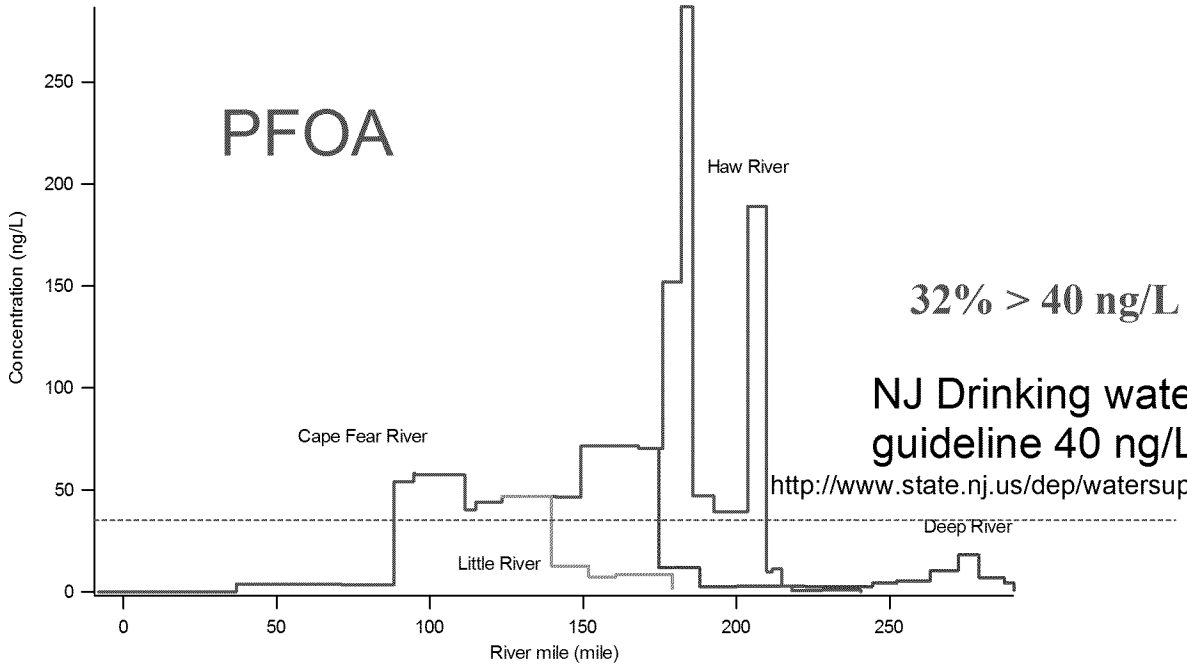
Of samples with PFAS detects: 22.8% derived from surface water

UCMR3 data for the State of North Carolina as of January 1, 2015

	A	B	C	D	E	F
1	PWSName	FacilityName	Date	PFAS	ng/L	State
495	City of Albemarle	Tuckertown	10/2/2013	PFHpA	10	NC
496	City of Albemarle	Town	10/2/2013	PFHpA	10	NC
497	Fayetteville Public Works Commission	Hoffner WTP	1/17/2013	PFHpA	10	NC
498	Old North Utilities Services - Fort Bragg	Fayetteville/Harnett Co. Intertie	1/15/2014	PFHpA	11	NC
499	Moore County Public Utilities - Pinehurst	EMWD Intertie	9/25/2013	PFHpA	12	NC
500	CFPUA - Wilmington	Sweeny WTP	6/9/2014	PFHpA	12	NC
501	Brunswick Regional Water & Sewer H2GO	Brunswick County Intertie	7/15/2014	PFHpA	12.03	NC
502	Old North Utilities Services - Fort Bragg	Fayetteville/Harnett Co. Intertie	10/17/2013	PFHpA	13	NC
503	CFPUA - Wilmington	Sweeny WTP	12/5/2013	PFHpA	14	NC
504	Fayetteville Public Works Commission	Glenville WTP	10/28/2013	PFHpA	15	NC
505	Moore County Public Utilities - Pinehurst	EMWD Intertie	2/28/2013	PFHpA	16	NC
506	Brookwood Comm. Water System	Well #34	2/24/2014	PFOA	20	NC
507	Town of Holly Springs	Harnett County Water Intertie	10/23/2013	PFHpA	20	NC
508	Harnett County Department of Public Utilit	Clearwell	6/11/2014	PFHpA	20	NC
509	Harnett County Department of Public Utilit	Clearwell	9/17/2014	PFOA	20	NC
510	Brookwood Comm. Water System	Well #39	2/25/2014	PFOA	21	NC
511	Moore County Public Utilities - Pinehurst	EMWD Intertie	5/16/2013	PFOA	23	NC
512	Moore County Public Utilities - Pinehurst	EMWD Intertie	11/26/2013	PFHpA	25	NC
513	CFPUA - Wilmington	Sweeny WTP	9/8/2014	PFHpA	27	NC
514	City of Sanford	Plant Clearwell	12/19/2013	PFHpA	29	NC
515	Town of Fuquay-Varina	Harnett County Water Intertie	9/23/2014	PFOA	30	NC
516	Orange Water & Sewer Authority	Plant Clearwell	2/11/2014	PFOA	30	NC
517	Moore County Public Utilities - Pinehurst	Fire Station #1	9/12/2013	PFHxS	40	NC
518	Harnett County Department of Public Utilit	Clearwell	9/17/2014	PFHpA	40	NC
519	Cliffdale West	Well #4	9/9/2014	PFOS	41	NC
520	Moore County Public Utilities - Pinehurst	Fire Station #1	2/27/2013	PFHxS	42	NC
521	City of Greensboro	Mitchell Filter	6/3/2014	PFOS	44	NC
522	Town of Fuquay-Varina	Harnett County Water Intertie	9/23/2014	PFHpA	50	NC
523	Bell Arthur Water Corp.	Well #6 (Otter Creek)	1/6/2014	PFOS	50	NC
524	City of Greensboro	Mitchell Filter	9/9/2014	PFOS	64	NC
525	Moore County Public Utilities - Pinehurst	Fire Station #1	9/12/2013	PFOS	73	NC
526	Moore County Public Utilities - Pinehurst	Fire Station #1	2/27/2013	PFOS	76	NC
527	Dover Water Department	Griffin Well Treatment Plant (Mast	3/24/2014	PFHpA	15	NH

The Cape Fear River Basin

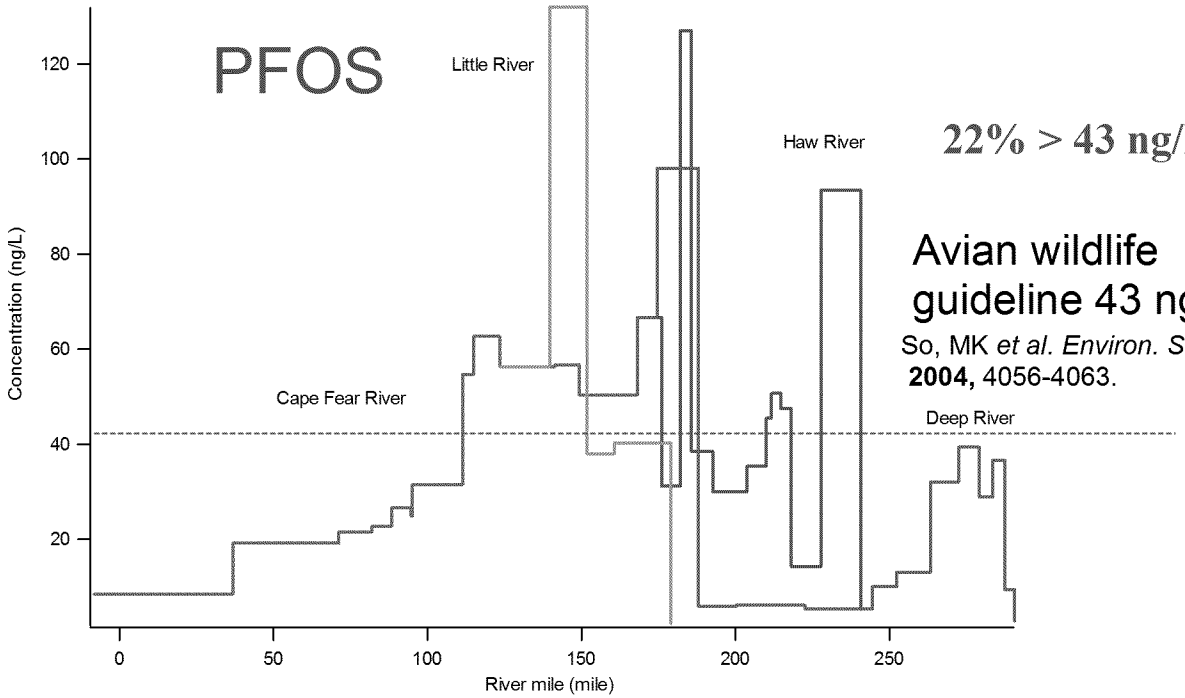
PFAS profiles in the Cape Fear Drainage Basin, North Carolina, USA



32% > 40 ng/L

NJ Drinking water guideline 40 ng/L

http://www.state.nj.us/dep/watersupply/pfoa.htm



22% > 43 ng/L

Avian wildlife guideline 43 ng/L

So, MK *et al. Environ. Sci. Technol.* **2004,** 4056-4063.

Nakayama *et al. Environ. Sci. Technol.* **2007,** 41: 5271-5276

Environmental Science & Technology

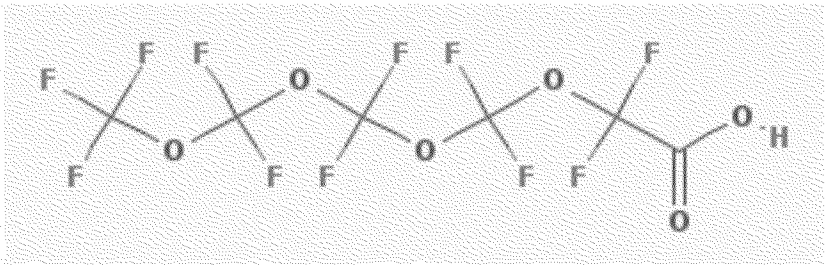
Article

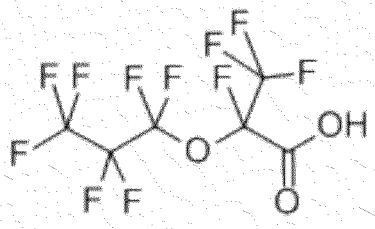
pubs.acs.org/est

Identification of Novel Perfluoroalkyl Ether Carboxylic Acids (PFECAs) and Sulfonic Acids (PFESAs) in Natural Waters Using Accurate Mass Time-of-Flight Mass Spectrometry (TOFMS)

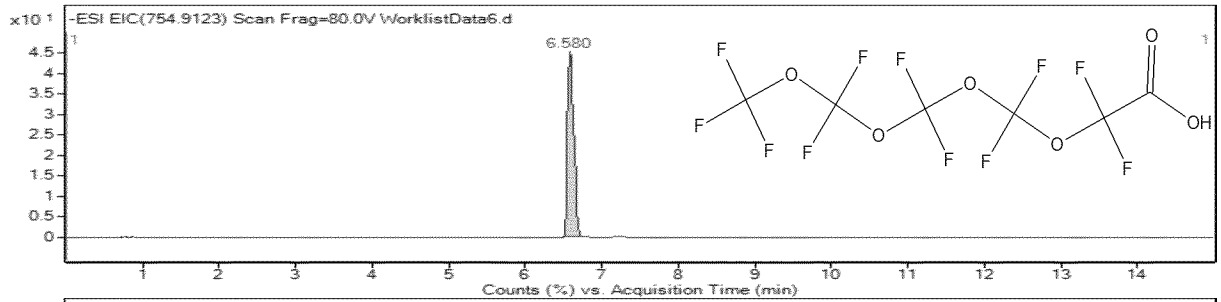
Mark Strynar,*[,†] Sonia Dagnino,[†,‡] Rebecca McMahen,[†,‡] Shuang Liang,[†,‡] Andrew Lindstrom,[†] Erik Andersen,[†] Larry McMillan,[§] Michael Thurman,[∥] Imma Ferrer,[∥] and Carol Ball[⊥]

[†]National Exposure Research Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina 27711, United States

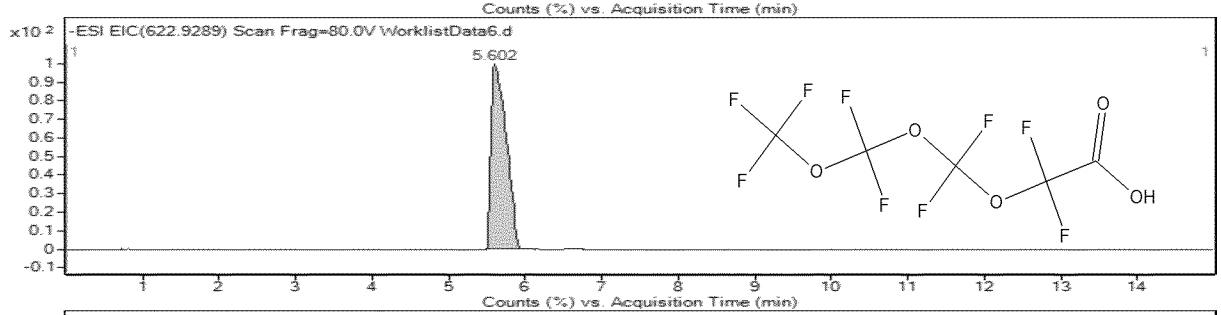




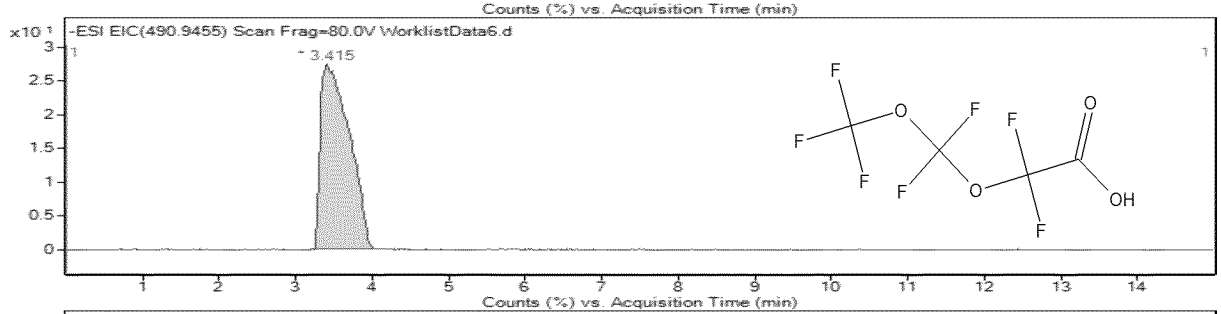
Replacement compounds found in the Cape Fear River, North Carolina, most likely source is fluorochemical production facility, source water for Wilmington and other cities near the coast



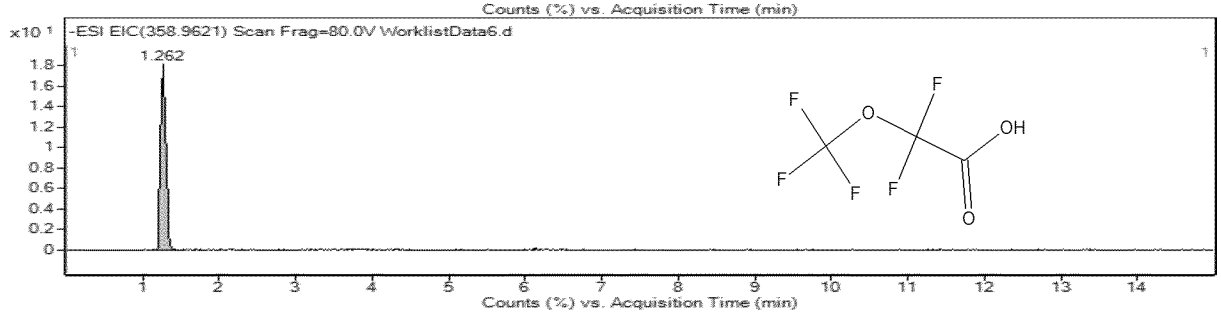
Molecular Formula = $C_6HF_{11}O_6$
Monoisotopic Mass = 377.959748 Da
[M-H]- = 376.952472 Da



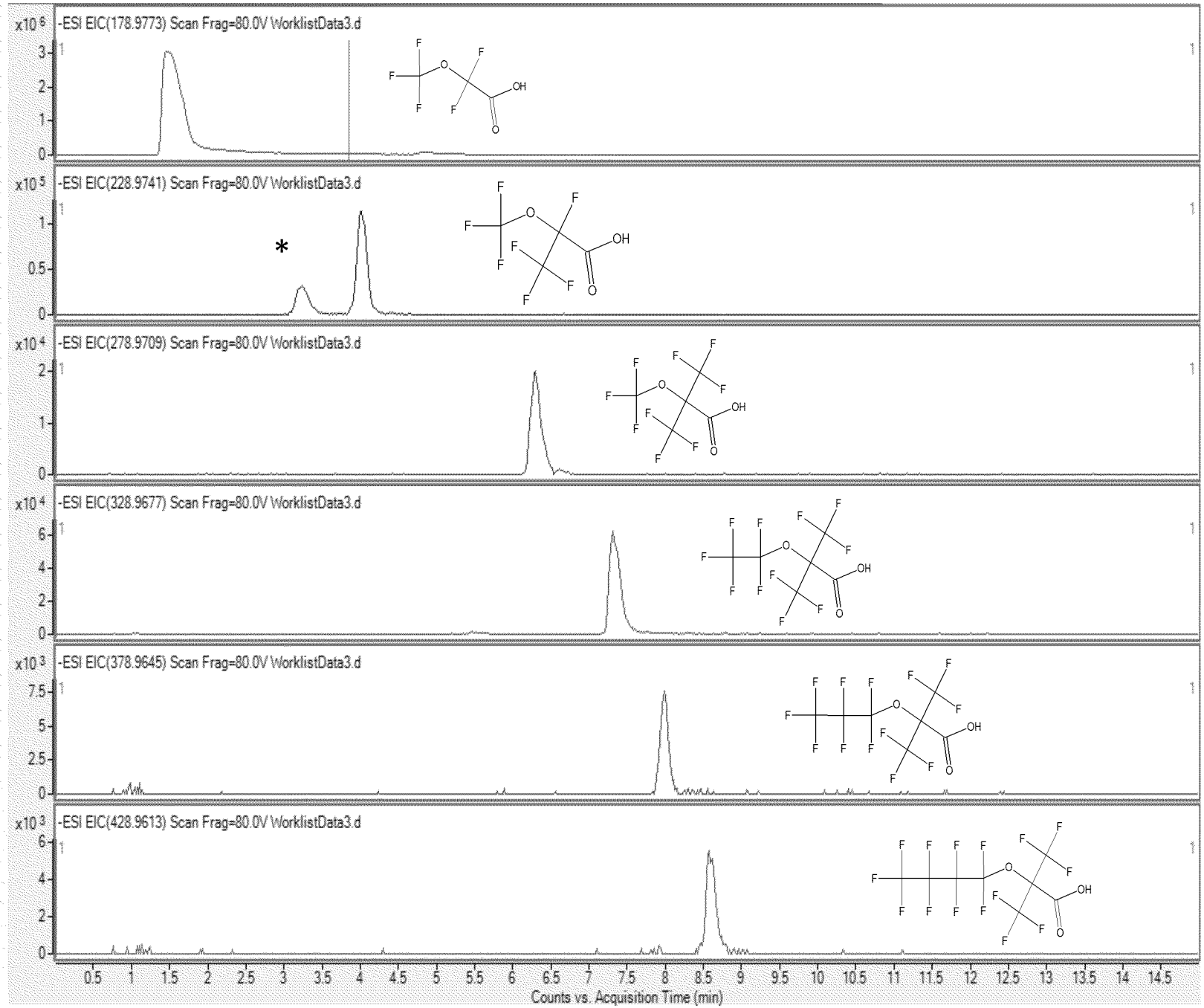
Molecular Formula = $C_5HF_9O_5$
Monoisotopic Mass = 311.968027 Da
[M-H]- = 310.96075 Da

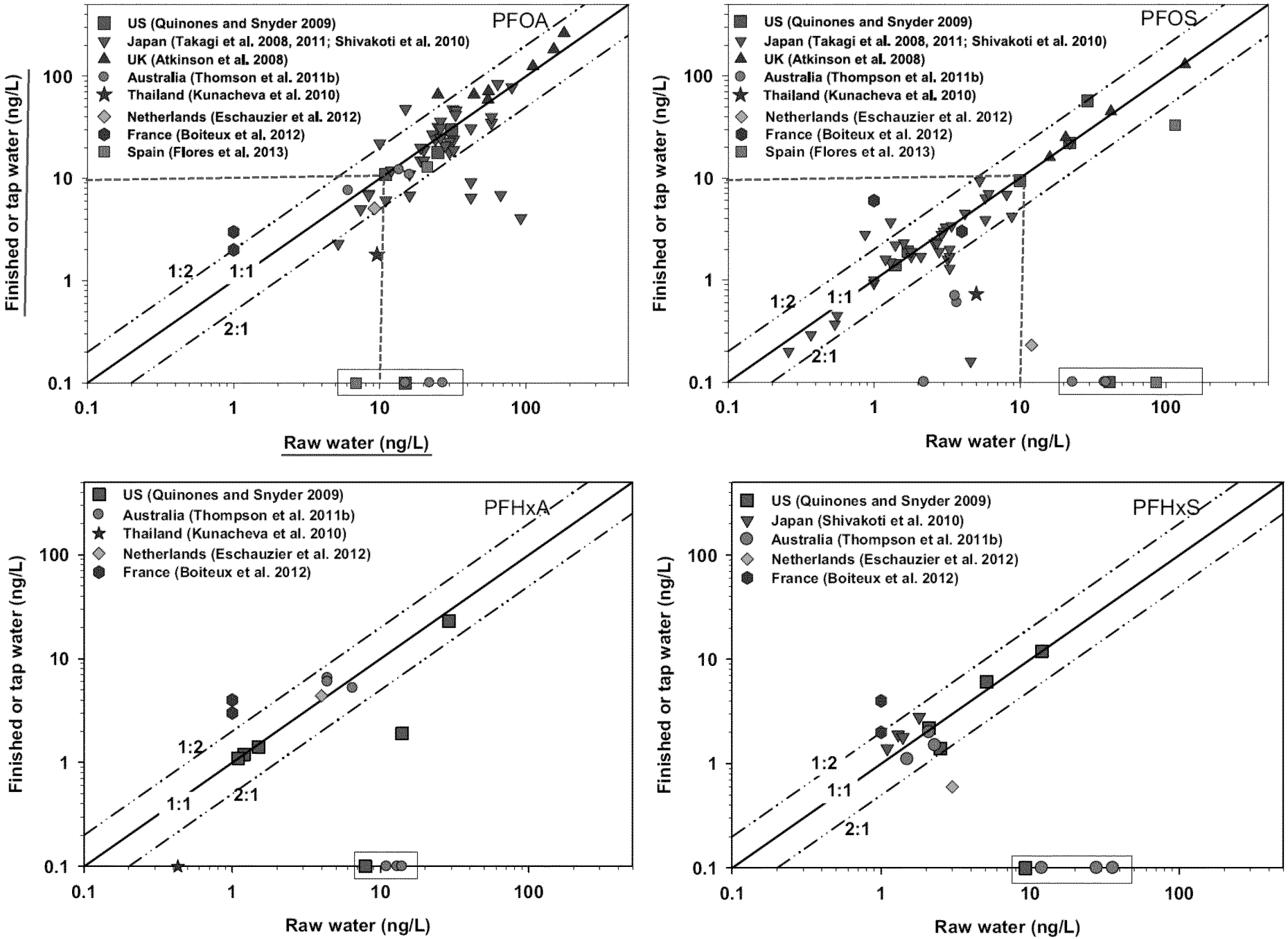


Molecular Formula = $C_4HF_7O_4$
Monoisotopic Mass = 245.976306 Da
[M-H]- = 244.969029 Da



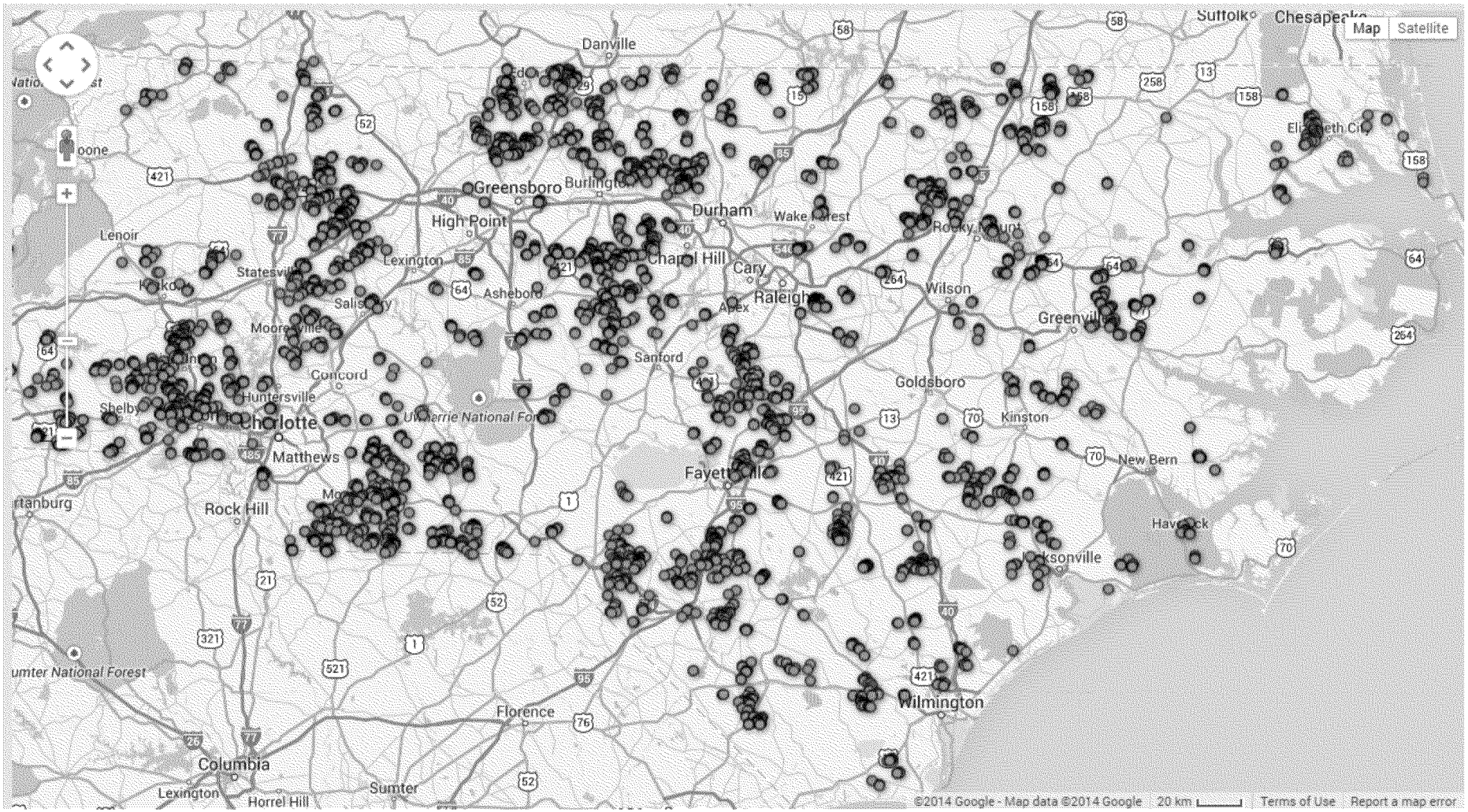
Molecular Formula = $C_3HF_5O_3$
Monoisotopic Mass = 179.984585 Da
[M-H]- = 178.977308 Da


x10 6 -ESI EIC(178.9773) Scan Frag=80.0V WorklistData3.d
x10 5 -ESI EIC(228.9741) Scan Frag=80.0V WorklistData3.d
*
x10 4 -ESI EIC(278.9709) Scan Frag=80.0V WorklistData3.d
x10 4 -ESI EIC(328.9677) Scan Frag=80.0V WorklistData3.d
x10 3 -ESI EIC(378.9645) Scan Frag=80.0V WorklistData3.d
x10 3 -ESI EIC(428.9613) Scan Frag=80.0V WorklistData3.d
Counts vs. Acquisition Time (min)

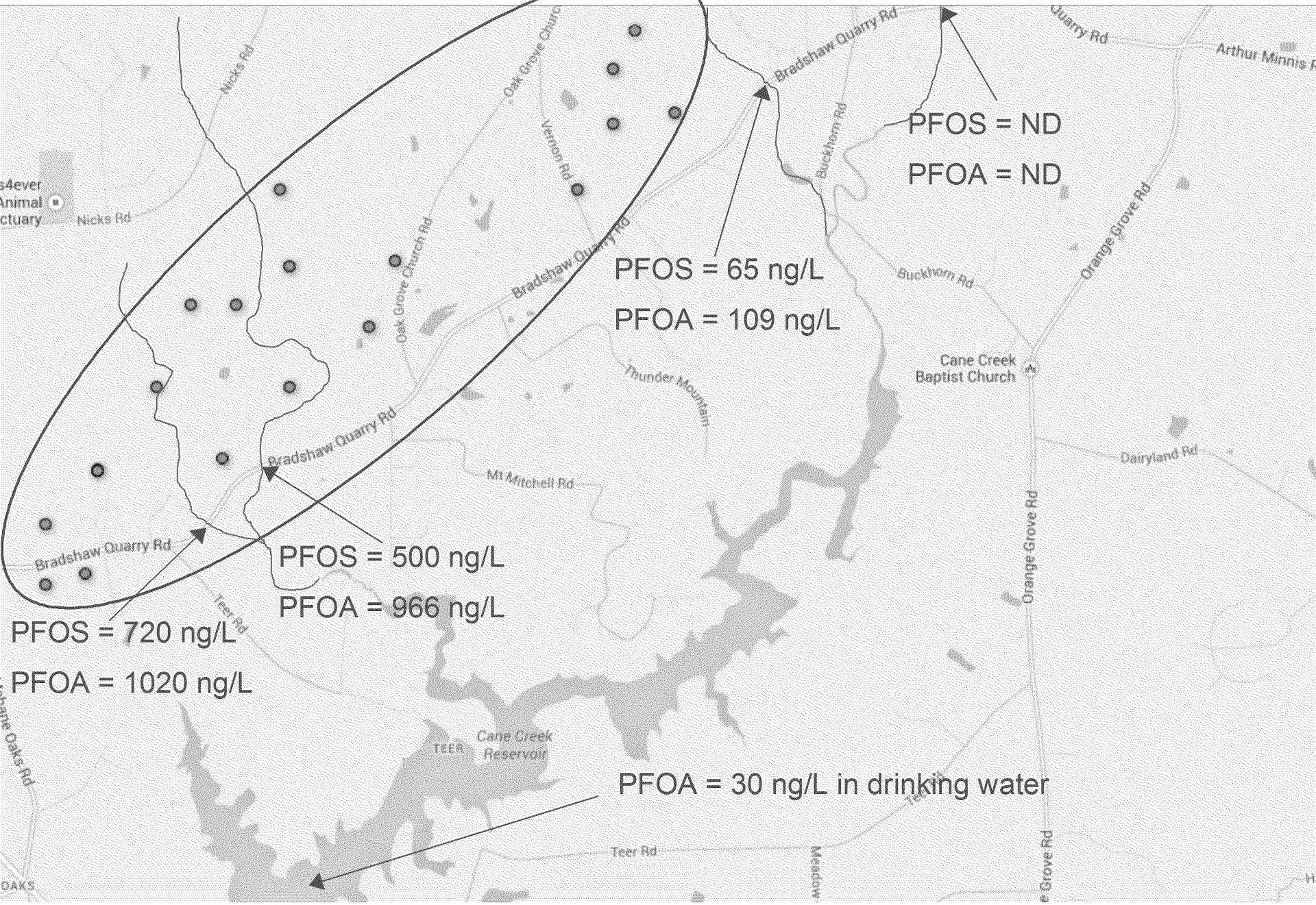


Rahman et al., (2014) *Water Research,* 50:318-340

4,146 permitted sludge application sites in North Carolina

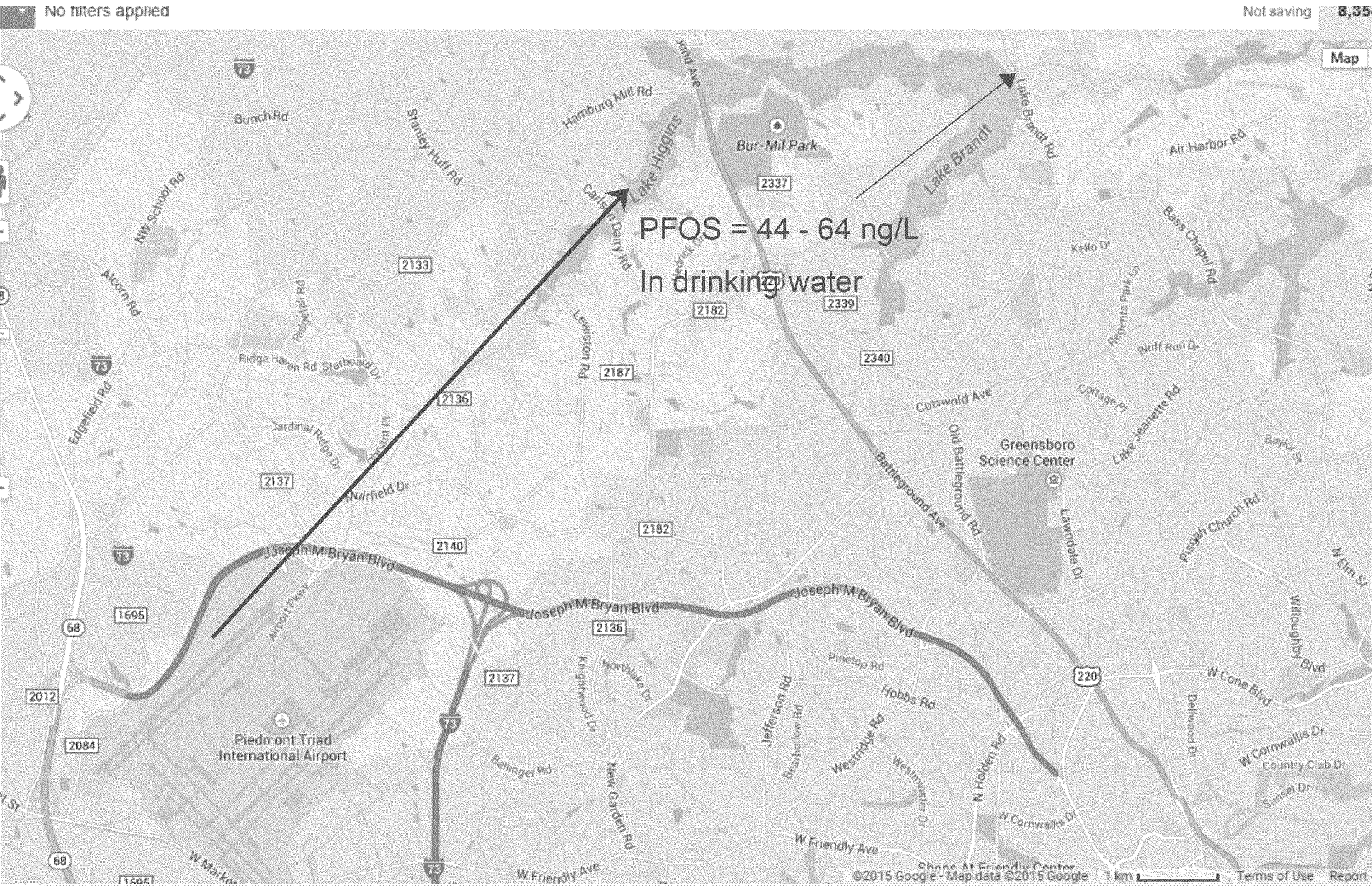


Sewage sludge in Cane Creek Resevoir basin - Serving Orange County, NC

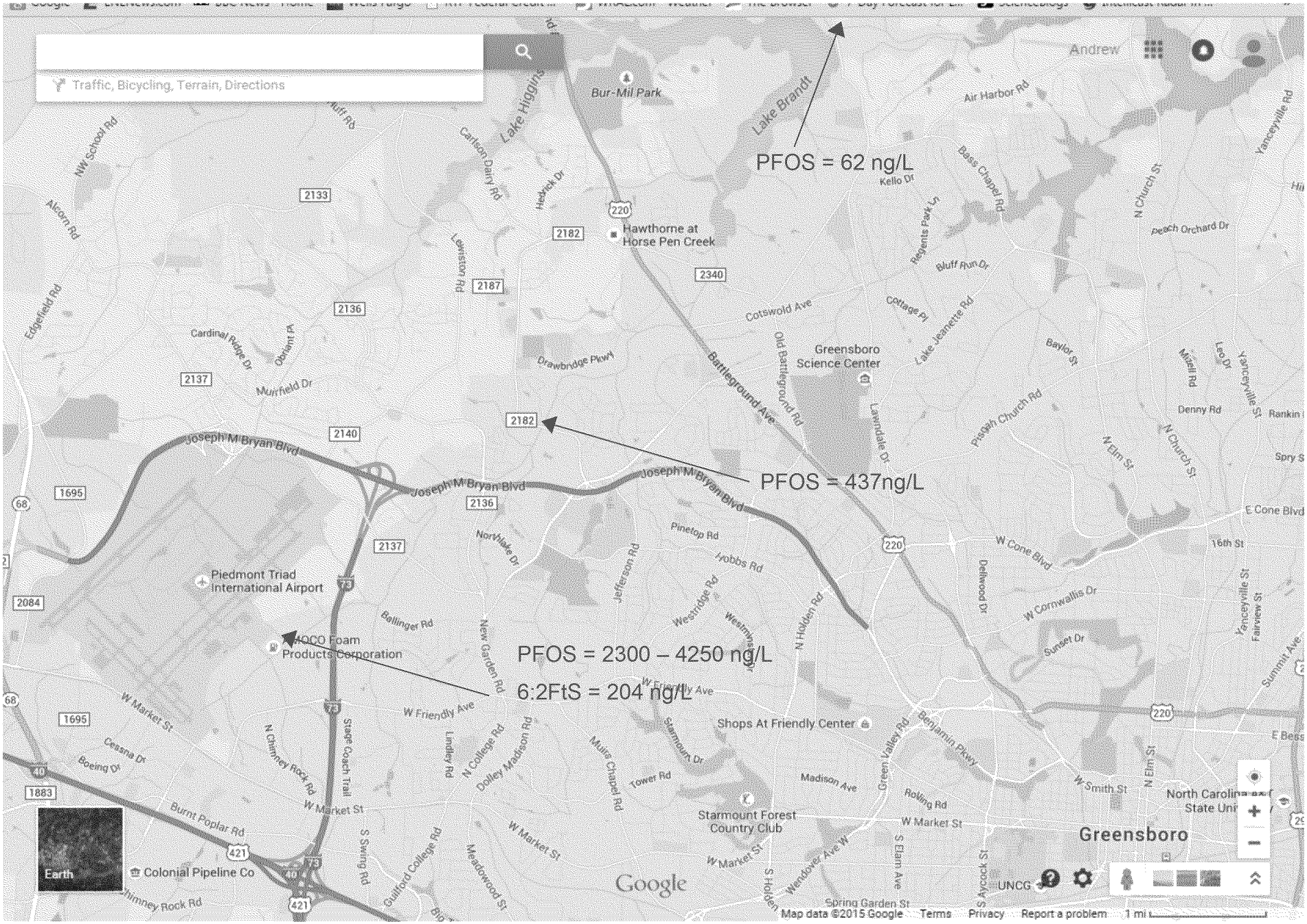


Traffic, Bicycling, Directions
Andrew
Oak Grove Church Rd
Bradshaw Quarry Rd
1115
1114
1116
Grange Grove Rd
Cane Creek
1100
1100
Map
Google
Imagery ©2015 Google, Map data ©2015 Google
Terms
Privacy
Report a problem
2000 ft

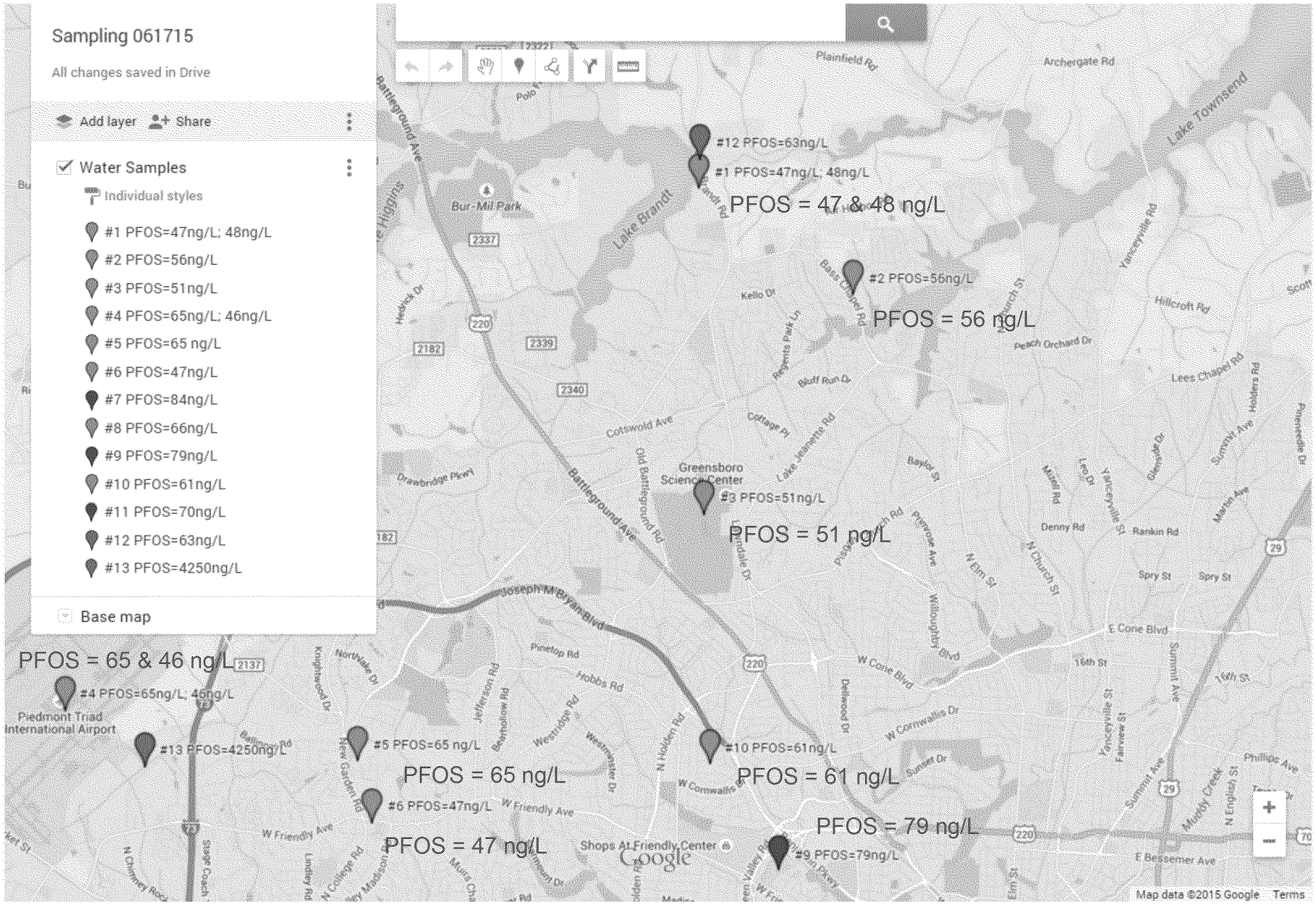
Near Piedmont Triad International Airport, Greensboro, North Carolina



PFOS in Surface water, Greensboro, North Carolina



PFOS in drinking water (ng/L), Greensboro, North Carolina



UCMR3 data for the State of Alabama of January 1, 2015 page 1

	A	B	C	D	E	F
1	PWSName	FacilityName	Date	PFAS	ng/L	State
2	Southside Waterworks	Gadsden Water Intertie	1/21/2013	PFHpA	10	AL
3	Southside Waterworks	Gadsden Water Intertie	4/15/2013	PFHpA	10	AL
4	Rainbow City Utilities Board	Gadsden Water Intertie	7/22/2013	PFHpA	10	AL
5	Rainbow City Utilities Board	Gadsden Water Intertie	4/15/2013	PFHpA	10	AL
6	Northeast Alabama Water System	Centre Water	6/12/2014	PFHpA	10	AL
7	Shelby County Water System	Talladega/Shelby WTP	10/17/2014	PFHpA	10	AL
8	Southside Waterworks	Gadsden Water Intertie	10/21/2013	PFHpA	20	AL
9	Southside Waterworks	Gadsden Water Intertie	7/8/2013	PFOA	20	AL
10	VAW Water System, Inc.	West Morgan-East Lawrence Wa	8/5/2014	PFOA	20	AL
11	VAW Water System, Inc.	West Morgan-East Lawrence Wa	2/18/2014	PFHpA	20	AL
12	VAW Water System, Inc.	West Morgan-East Lawrence Wa	5/28/2014	PFOA	20	AL
13	Rainbow City Utilities Board	Gadsden Water Intertie	1/15/2014	PFOA	20	AL
14	Rainbow City Utilities Board	Gadsden Water Intertie	10/21/2013	PFHpA	20	AL
15	Colbert County Rural Water System	West Lawrence	6/16/2014	PFOA	20	AL
16	Colbert County Rural Water System	Muscle Shoals	6/16/2014	PFOA	20	AL
17	Colbert County Rural Water System	Cherokee	6/16/2014	PFOA	20	AL
18	Florence Water-Wastewater Departmer	Wilson Lake + Treatment Plant	4/16/2014	PFOA	20	AL
19	Gadsden Waterworks & Sewer Board	Water Treatment Plant	10/2/2014	PFHpA	30	AL
20	Southside Waterworks	Gadsden Water Intertie	1/21/2013	PFOA	30	AL
21	Southside Waterworks	Gadsden Water Intertie	10/21/2013	PFOA	30	AL
22	Rainbow City Utilities Board	Gadsden Water Intertie	4/15/2013	PFOA	30	AL
23	Rainbow City Utilities Board	Gadsden Water Intertie	10/21/2013	PFOA	30	AL
24	West Morgan - East Lawrence Water Au	Tennessee River + Treatment Pla	2/5/2014	PFHpA	30	AL
25	Northeast Alabama Water System	Centre Water	6/12/2014	PFOA	30	AL
26	Gadsden Waterworks & Sewer Board	Water Treatment Plant	10/2/2014	PFOA	40	AL
27	Southside Waterworks	Gadsden Water Intertie	4/15/2013	PFOA	40	AL
28	Southside Waterworks	Gadsden Water Intertie	4/15/2013	PFOS	40	AL
29	VAW Water System, Inc.	West Morgan-East Lawrence Wa	11/4/2014	PFOS	40	AL
30	VAW Water System, Inc.	West Morgan-East Lawrence Wa	8/5/2014	PFOS	40	AL
31	Rainbow City Utilities Board	Gadsden Water Intertie	7/22/2013	PFOA	40	AL
32	Colbert County Rural Water System	Water Treatment Plant	6/16/2014	PFOS	40	AL
33	Colbert County Rural Water System	West Lawrence	6/16/2014	PFOS	40	AL
34	Colbert County Rural Water System	Muscle Shoals	6/16/2014	PFOS	40	AL
35	Florence Water-Wastewater Departmer	Wilson Lake + Treatment Plant	4/16/2014	PFOS	40	AL
36	Gadsden Waterworks & Sewer Board	Water Treatment Plant	10/2/2014	PFOS	50	AL
37	Southside Waterworks	Gadsden Water Intertie	1/21/2013	PFOS	50	AL
38	Colbert County Rural Water System	Cherokee	6/16/2014	PFOS	50	AL
39	West Morgan - East Lawrence Water Au	Tennessee River + Treatment Pla	8/28/2013	PFOS	50	AL
40	Northeast Alabama Water System	Centre Water	6/12/2014	PFOS	50	AL
41	VAW Water System, Inc.	West Morgan-East Lawrence Wa	5/28/2014	PFOS	60	AL
42	Rainbow City Utilities Board	Gadsden Water Intertie	7/22/2013	PFOS	60	AL
43	Florence Water-Wastewater Departmer	Cypress Creek + Treatment Plant	10/15/2013	PFOS	60	AL
44	VAW Water System, Inc.	West Morgan-East Lawrence Wa	2/18/2014	PFOA	90	AL
45	Rainbow City Utilities Board	Gadsden Water Intertie	7/22/2013	PFBS	100	AL
46	West Morgan - East Lawrence Water Au	Tennessee River + Treatment Pla	2/5/2014	PFOA	100	AL
47	West Morgan - East Lawrence Water Au	Tennessee River + Treatment Pla	2/5/2014	PFOS	130	AL
48	VAW Water System, Inc.	West Morgan-East Lawrence Wa	2/18/2014	PFOS	180	AL
49	City of Tempe	EPDS004- ID#09353	1/14/2014	PFHpA	11	AZ

UCMR3 data for the State of California of January 1, 2015 page 1

1	PWSName	FacilityName	Date	PFAS	ng/L	State
78	City of Corona	19579 Temescal Canyon Road	1/16/2014	PFHpA	10	CA
79	City of Corona	R-3 Blend (SB-9)	1/16/2014	PFHpA	11	CA
80	City of Orange	Well 15	5/14/2014	PFHpA	11.5	CA
81	CA Water Service - Visalia	Well 77-1	6/11/2014	PFHpA	13	CA
82	City of Corona	R-3 Blend (SB-9)	7/10/2014	PFHpA	13	CA
83	Elsinore Valley Municipal Water District	Canyon Lake WTP	9/19/2013	PFHpA	13	CA
84	Santa Clarita Water Division	Santa Clara Well	4/9/2014	PFHpA	14	CA
85	Elsinore Valley Municipal Water District	Canyon Lake WTP	6/19/2014	PFHpA	14	CA
86	City of Lathrop	Well 21	10/15/2013	PFOA	20	CA
87	City of Pico Rivera Water Department	Well 7	7/30/2014	PFOA	20	CA
88	City of Pico Rivera Water Department	Well 11	1/24/2013	PFOA	20	CA
89	Camp Pendleton (South)	Chlorination Station 410618	11/13/2013	PFOA	21	CA
90	City of Pico Rivera Water Department	Well 12	1/24/2013	PFOA	21	CA
91	Orchard Dale Water District	Mills	7/21/2014	PFOA	21	CA
92	Eastern Municipal Water District	Well 59 (Indian Ave.)	6/18/2013	PFHpA	22	CA
93	Eastern Municipal Water District	Well 59 (Indian Ave.)	10/8/2013	PFHpA	22	CA
94	City of Corona	19579 Temescal Canyon Road	10/9/2013	PFOA	23	CA
95	Valencia Water Company	Well S-7	3/12/2014	PFOA	23	CA
96	Valencia Water Company	Well S-6	3/12/2014	PFOA	24	CA
97	Valencia Water Company	Well N	5/28/2014	PFOA	24	CA
98	Yorba Linda Water District	Highland Reservoir	1/9/2013	PFOA	24.1	CA
99	City of Lathrop	Well 21	4/17/2013	PFOA	25	CA
100	Yorba Linda Water District	Highland Reservoir	7/10/2013	PFOA	25.9	CA
101	City of Corona	Lester SWTP	1/15/2014	PFOA	26	CA
102	City of Orange	Well 15	5/14/2014	PFOA	26.1	CA
103	City of Pico Rivera Water Department	Well 6	6/4/2014	PFOA	27	CA
104	Santa Clarita Water Division	Santa Clara Well	4/9/2014	PFOA	27	CA
105	Camp Pendleton (South)	Chlorination Station 410618	5/21/2014	PFOA	28	CA
106	Atascadero Mutual Water Company	Treatment Building C	10/30/2013	PFOA	28	CA
107	City of Pico Rivera Water Department	Well 4	7/30/2014	PFOA	29	CA
108	City of Corona	Ontario/Garretson Zone 3 (SB-1	10/23/2013	PFOA	29	CA
109	City of Orange	Well 3	5/14/2014	PFOA	29.1	CA
110	City of Pleasanton	Well 6	6/17/2014	PFHxS	32	CA
111	CA Water Service - Chico	Well 7-4	8/21/2013	PFHxS	35	CA
112	Sacramento Suburban Water District	Well 46 (Jonas/Sierra Mills)	8/11/2014	PFHxS	35	CA
113	Camp Pendleton (South)	I & M Plant 22035	11/26/2013	PFHxS	36	CA
114	City of Pleasanton	Well 6	11/4/2013	PFHxS	36	CA
115	Sacramento Suburban Water District	Well 46 (Jonas/Sierra Mills)	2/5/2014	PFHxS	36	CA
116	City of Corona	19579 Temescal Canyon Road	1/16/2014	PFOA	36	CA
117	City of Corona	R-3 Blend (SB-9)	1/16/2014	PFOA	38	CA
118	Elsinore Valley Municipal Water District	Canyon Lake WTP	9/19/2013	PFOA	38	CA
119	Elsinore Valley Municipal Water District	Canyon Lake WTP	6/19/2014	PFOA	38	CA
120	Camp Pendleton (South)	Chlorination Station 410618	11/13/2013	PFHxS	41	CA
121	City of Corona	19579 Temescal Canyon Road	1/16/2014	PFOS	41	CA
122	City of Fullerton	Well 3A	4/15/2014	PFOS	41.2	CA
123	City of Pico Rivera Water Department	Well 1	1/24/2013	PFOS	42	CA
124	CA Water Service - Chico	Well 7-4	2/11/2014	PFHxS	42	CA
125	City of Corona	R-3 Blend (SB-9)	7/10/2014	PFOA	42	CA
126	Golden State Water Co. - Norwalk	Pioneer Well 3	1/17/2014	PFOS	43	CA
127	City of Corona	Ontario/Garretson Zone 3 (SB-1	10/23/2013	PFOS	43	CA
128	City of Garden Grove	Well 19	5/14/2013	PFOS	44	CA

UCMR3 data for the State of California of January 1, 2015 page 2

	A	B	C	D	E	F
1	PWSName	FacilityName	Date	PFAS	ng/L	State
129	Eastern Municipal Water District	Well 59 (Indian Ave.)	10/8/2013	PFOA	44	CA
130	Orchard Dale Water District	Mills	7/21/2014	PFOS	45	CA
131	Park Water Company - Bellflower/Norw a	Well 46C	6/18/2013	PFOS	45	CA
132	City of Corona	R-3 Blend (SB-9)	1/16/2014	PFOS	45	CA
133	City of Orange	Well 15	5/14/2014	PFOS	45.3	CA
134	Montebello Land & Water Company	Pumping Plant (Wells 7, 8A, 9, 10	6/26/2014	PFOS	46	CA
135	City of Corona	R-3 Blend (SB-9)	7/10/2014	PFOS	46	CA
136	Montebello Land & Water Company	Well 11A	6/26/2014	PFOS	47	CA
137	Yorba Linda Water District	Highland Reservoir	7/10/2013	PFOS	47	CA
138	Yorba Linda Water District	Highland Reservoir	1/9/2013	PFOS	47.4	CA
139	City of Pico Rivera Water Department	Well 8	1/24/2013	PFOS	48	CA
140	City of Garden Grove	Well 23	5/15/2013	PFOS	48	CA
141	Park Water Company - Bellflower/Norw a	Well 41A	6/18/2013	PFOS	49	CA
142	Park Water Company - Bellflower/Norw a	Well 41A	3/7/2014	PFOS	49	CA
143	City of Garden Grove	Well 19	11/13/2013	PFOS	49.3	CA
144	City of Garden Grove	Well 23	11/13/2013	PFOS	49.6	CA
145	City of Pico Rivera Water Department	Well 8	7/30/2013	PFOS	50	CA
146	CA Water Service - East Los Angeles	Well 38-2	9/22/2014	PFOS	50	CA
147	Park Water Company - Bellflower/Norw a	Well 28B	6/18/2013	PFOS	51	CA
148	Eastern Municipal Water District	Well 59 (Indian Ave.)	6/18/2013	PFOA	53	CA
149	CA Water Service - East Los Angeles	Well 38-2	2/11/2014	PFOS	54	CA
150	CA Water Service - Chico	Well 41-1	2/19/2014	PFOS	57	CA
151	City of Lathrop	Well 21	10/15/2013	PFOS	58	CA
152	CA Water Service - Chico	Well 7-4	8/21/2013	PFOS	59	CA
153	Camp Pendleton (South)	Chlorination Station 410618	5/21/2014	PFHxS	62	CA
154	City of Pico Rivera Water Department	Well 7	7/30/2014	PFOS	62	CA
155	City of Orange	Well 3	5/14/2014	PFOS	65.9	CA
156	City of Lathrop	Well 21	4/17/2013	PFOS	76	CA
157	CA Water Service - Chico	Well 7-4	2/11/2014	PFOS	76	CA
158	Eastern Municipal Water District	Well 59 (Indian Ave.)	10/8/2013	PFOS	81	CA
159	Eastern Municipal Water District	Well 59 (Indian Ave.)	6/18/2013	PFOS	82	CA
160	CA Water Service - Visalia	Well 77-1	6/11/2014	PFHxS	92	CA
161	Eastern Municipal Water District	Well 59 (Indian Ave.)	6/18/2013	PFHxS	110	CA
162	CA Water Service - Visalia	Well 77-1	6/11/2014	PFOS	120	CA
163	Eastern Municipal Water District	Well 59 (Indian Ave.)	10/8/2013	PFHxS	120	CA

UCMR3 data for the State of Colorado of January 1, 2015 page 1

	A	B	C	D	E	F
1	PWSName	FacilityName	Date	PFAS	ng/L	State
164	City of Fountain	Chlorination for Well 2	1/15/2013	PFHpA	10	CO
165	City of Fountain	Chlorination for Well 4	1/15/2013	PFHpA	10	CO
166	City of Fountain	Chlorination for Well 2	7/17/2013	PFHpA	10	CO
167	Security WSD	FVA Intertie	7/30/2014	PFHpA	10	CO
168	Widefield WSD	Chlorinator & Aeration for Wells W	6/19/2014	PFHpA	11	CO
169	Widefield WSD	Chlorinator & Aeration for Wells W	11/20/2013	PFHpA	16	CO
170	Widefield WSD	Chlorinator for C36 Well	11/6/2013	PFHpA	18	CO
171	Widefield WSD	Chlorinator for W3 Well	11/12/2013	PFHpA	18	CO
172	Widefield WSD	Chlorinator for W4 Well	11/12/2013	PFHpA	19	CO
173	City of Fountain	Fountain Valley Authority Intertie	4/16/2013	PFHpA	20	CO
174	City of Fountain	Chlorination for Well 3	1/15/2013	PFOA	20	CO
175	City of Fountain	Chlorination for Well 1	1/15/2013	PFHpA	20	CO
176	Security WSD	Chlorinator for S7 Well	7/30/2014	PFHpA	20	CO
177	Security WSD	Chlorinator for S8 Well	7/30/2014	PFHpA	20	CO
178	Security WSD	Chlorinator for FV4 Well	7/29/2014	PFHpA	20	CO
179	Security WSD	Chlorinator for FV4 Well	1/14/2014	PFHpA	20	CO
180	Security WSD	Chlorinator for S11 Well	7/30/2014	PFHpA	20	CO
181	Security WSD	Chlorinator for S11 Well	4/22/2014	PFHpA	20	CO
182	Security WSD	Chlorinator for Well S10	7/30/2014	PFHpA	20	CO
183	Security WSD	Chlorinator for S2 Well	7/29/2014	PFHpA	20	CO
184	Security WSD	Chlorinator for S9 Well	7/29/2014	PFHpA	20	CO
185	Security WSD	Chlorinator for S2 Well	1/21/2014	PFHpA	20	CO
186	Security WSD	Chlorinator for S7 Well	1/22/2014	PFHpA	20	CO
187	Security WSD	Chlorinator for R1 Well	1/14/2014	PFHpA	20	CO
188	Security WSD	Chlorinator for S4 Well	7/30/2014	PFHpA	20	CO
189	Security WSD	Chlorinator for S4 Well	2/10/2014	PFHpA	20	CO
190	Security WSD	Chlorinator for R1 Well	8/11/2014	PFHpA	20	CO
191	Security WSD	Chlorinator for R2 Well	7/29/2014	PFHpA	20	CO
192	Security WSD	Chlorinator for Well S10	1/14/2014	PFHpA	20	CO
193	Widefield WSD	Chlorinator for E2 Well	5/14/2014	PFHpA	20	CO
194	Widefield WSD	Chlorinator & Aeration for Wells W	6/19/2014	PFOA	21	CO
195	Widefield WSD	Chlorinator for W7 Well	5/14/2014	PFHpA	22	CO
196	Widefield WSD	Chlorinator for W3 Well	5/14/2014	PFHpA	24	CO
197	Widefield WSD	Chlorinator for W4 Well	5/19/2014	PFHpA	24	CO
198	Widefield WSD	Chlorinator & Aeration for Wells W	11/20/2013	PFOA	24	CO
199	City of Fountain	Chlorination for Well 3	7/17/2013	PFOA	30	CO
200	City of Fountain	Fountain Valley Authority Intertie	7/17/2013	PFHxS	30	CO
201	City of Fountain	Chlorination for Well 4	1/15/2013	PFOA	30	CO
202	Security WSD	Chlorinator for S4 Well	7/30/2014	PFOA	30	CO
203	Security WSD	Chlorinator for S7 Well	7/30/2014	PFOA	30	CO
204	Security WSD	FVA Intertie	7/30/2014	PFOA	30	CO
205	Security WSD	CT 150,000 Gallon Steel Tank	1/22/2014	PFHpA	30	CO
206	Security WSD	Chlorinator for S16 Well	7/30/2014	PFHpA	30	CO
207	Security WSD	Chlorinator for S11 Well	4/22/2014	PFOA	30	CO
208	Security WSD	Chlorinator for S9 Well	1/14/2014	PFHpA	30	CO
209	Security WSD	Chlorinator for S16 Well	1/22/2014	PFHpA	30	CO
210	Security WSD	Chlorinator for S4 Well	2/10/2014	PFOA	30	CO
211	Security WSD	Chlorinator for S15 Well	7/30/2014	PFHpA	30	CO
212	Security WSD	Chlorinator for R2 Well	1/14/2014	PFHpA	30	CO
213	Security WSD	Chlorinator for S15 Well	1/21/2014	PFHpA	30	CO
214	Security WSD	Chlorinator for S11 Well	7/30/2014	PFOA	30	CO

UCMR3 data for the State of Colorado of January 1, 2015 page 2

	A	B	C	D	E	F	G
1	PWSName	FacilityName	Date	PFAS	ng/L	State	
215	Security WSD	CT 150,000 Gallon Steel Tank	7/15/2014	PFHpA	30	CO	
216	Security WSD	Chlorinator for S7 Well	1/22/2014	PFOA	30	CO	
217	Widefield WSD	Chlorinator for C36 Well	11/6/2013	PFOA	30	CO	
218	Widefield WSD	Chlorinator for W3 Well	11/12/2013	PFOA	31	CO	
219	Widefield WSD	Chlorinator for W7 Well	5/14/2014	PFOA	36	CO	
220	Widefield WSD	Chlorinator for E2 Well	5/14/2014	PFOA	36	CO	
221	Widefield WSD	Chlorinator for W4 Well	11/12/2013	PFOA	37	CO	
222	City of Fountain	Chlorination for Well 2	1/15/2013	PFOA	40	CO	
223	City of Fountain	Chlorination for Well 4	7/17/2013	PFOA	40	CO	
224	City of Fountain	Chlorination for Well 1	1/15/2013	PFOA	40	CO	
225	City of Fountain	Chlorination for Well 1	1/15/2013	PFOS	40	CO	
226	City of Fountain	Chlorination for Well 1	7/17/2013	PFOA	40	CO	
227	City of Fountain	Chlorination for Well 2	1/15/2013	PFOS	40	CO	
228	City of Fountain	Fountain Valley Authority Intertie	4/16/2013	PFOA	40	CO	
229	City of Fountain	Chlorination for Well 2	7/17/2013	PFOA	40	CO	
230	City of Fountain	Chlorination for Well 3	1/15/2013	PFHxS	40	CO	
231	City of Fountain	Chlorination for Well 3	1/15/2013	PFOS	40	CO	
232	Security WSD	Chlorinator for R1 Well	1/14/2014	PFOS	40	CO	
233	Security WSD	Chlorinator for R1 Well	1/14/2014	PFOA	40	CO	
234	Security WSD	Chlorinator for S2 Well	7/29/2014	PFOS	40	CO	
235	Security WSD	Chlorinator for W12 Well	7/30/2014	PFHpA	40	CO	
236	Security WSD	Chlorinator for S9 Well	7/29/2014	PFOA	40	CO	
237	Security WSD	Chlorinator for Well S10	1/14/2014	PFOA	40	CO	
238	Security WSD	Chlorinator for Well S10	7/30/2014	PFOA	40	CO	
239	Security WSD	Chlorinator for S15 Well	7/30/2014	PFOS	40	CO	
240	Security WSD	Chlorinator for S8 Well	7/30/2014	PFOA	40	CO	
241	Security WSD	FVA Intertie	1/22/2014	PFOS	40	CO	
242	Security WSD	Chlorinator for W12 Well	1/15/2014	PFHpA	40	CO	
243	Security WSD	Chlorinator for W12 Well	1/15/2014	PFOA	40	CO	
244	Security WSD	Air Stripping Plant	7/31/2014	PFHpA	40	CO	
245	Security WSD	Air Stripping Plant	1/29/2014	PFHpA	40	CO	
246	Security WSD	Chlorinator for R1 Well	8/11/2014	PFOA	40	CO	
247	Security WSD	Chlorinator for S2 Well	1/21/2014	PFOS	40	CO	
248	Widefield WSD	Chlorinator & Aeration for Wells W	11/20/2013	PFOS	42	CO	
249	Widefield WSD	Chlorinator for W4 Well	5/19/2014	PFOA	45	CO	
250	Widefield WSD	Chlorinator for E2 Well	11/13/2013	PFHxS	46	CO	
251	Widefield WSD	Chlorinator for W3 Well	5/14/2014	PFOA	48	CO	
252	Widefield WSD	Chlorinator & Aeration for Wells W	6/19/2014	PFOS	49	CO	
253	City of Fountain	Chlorination for Well 1	7/17/2013	PFHxS	50	CO	
254	City of Fountain	Chlorination for Well 3	7/17/2013	PFHxS	50	CO	
255	City of Fountain	Chlorination for Well 4	7/17/2013	PFHxS	50	CO	
256	Security WSD	Chlorinator for W12 Well	7/30/2014	PFOA	50	CO	
257	Security WSD	Chlorinator for FV4 Well	7/29/2014	PFOA	50	CO	
258	Security WSD	Chlorinator for FV4 Well	1/14/2014	PFOA	50	CO	
259	Security WSD	Chlorinator for S16 Well	1/22/2014	PFOS	50	CO	
260	Security WSD	Chlorinator for S2 Well	1/21/2014	PFOA	50	CO	
261	Security WSD	Chlorinator for S2 Well	7/29/2014	PFOA	50	CO	
262	Security WSD	Chlorinator for R1 Well	8/11/2014	PFOS	50	CO	
263	Security WSD	Chlorinator for S9 Well	1/14/2014	PFOA	50	CO	
264	Security WSD	Chlorinator for R2 Well	1/14/2014	PFOS	50	CO	
265	Widefield WSD	Chlorinator for W4 Well	5/19/2014	PFOS	51	CO	

UCMR3 data for the State of Colorado of January 1, 2015 page 3

1	PWSName	FacilityName	Date	PFAS	ng/L	State
266	Widefield WSD	Chlorinator for W3 Well	5/14/2014	PFOS	57	CO
267	City of Fountain	Chlorination for Well 2	7/17/2013	PFHxS	60	CO
268	Security WSD	Chlorinator for FV4 Well	7/29/2014	PFOS	60	CO
269	Security WSD	CT 150,000 Gallon Steel Tank	7/15/2014	PFOS	60	CO
270	Security WSD	CT 150,000 Gallon Steel Tank	1/22/2014	PFOS	60	CO
271	Security WSD	Chlorinator for W9 Well	1/15/2014	PFHpA	60	CO
272	Security WSD	FVA Intertie	7/30/2014	PFOS	60	CO
273	Security WSD	FVA Intertie	4/21/2014	PFOS	60	CO
274	Security WSD	FVA Intertie	1/22/2014	PFHxS	60	CO
275	Security WSD	Chlorinator for R2 Well	1/14/2014	PFOA	60	CO
276	Security WSD	Chlorinator for FV4 Well	1/14/2014	PFOS	60	CO
277	Security WSD	Chlorinator for S16 Well	7/30/2014	PFOS	60	CO
278	Security WSD	Chlorinator for S16 Well	1/22/2014	PFOA	60	CO
279	Security WSD	Chlorinator for S15 Well	1/21/2014	PFOA	60	CO
280	Security WSD	Chlorinator for R2 Well	7/29/2014	PFOS	60	CO
281	Security WSD	Chlorinator for R2 Well	7/29/2014	PFOA	60	CO
282	Security WSD	FVA Intertie	4/21/2014	PFHxS	60	CO
283	Widefield WSD	Chlorinator & Aeration for Wells W	6/19/2014	PFHxS	69	CO
284	Security WSD	Chlorinator for Well W8	1/15/2014	PFHpA	70	CO
285	Security WSD	CT 150,000 Gallon Steel Tank	7/15/2014	PFOA	70	CO
286	Security WSD	Chlorinator for S16 Well	7/30/2014	PFOA	70	CO
287	Security WSD	Chlorinator for S15 Well	7/30/2014	PFOA	70	CO
288	Security WSD	Chlorinator for Well W8	7/31/2014	PFHpA	70	CO
289	Security WSD	Chlorinator for W9 Well	1/15/2014	PFOA	70	CO
290	Security WSD	CT 150,000 Gallon Steel Tank	1/22/2014	PFOA	70	CO
291	Security WSD	Chlorinator for S9 Well	1/14/2014	PFOS	70	CO
292	Security WSD	Chlorinator for W9 Well	7/30/2014	PFHpA	70	CO
293	Widefield WSD	Chlorinator for W7 Well	11/12/2013	PFHxS	71	CO
294	Widefield WSD	Chlorinator & Aeration for Wells W	11/20/2013	PFHxS	75	CO
295	City of Fountain	Chlorination for Well 1	1/15/2013	PFHxS	80	CO
296	City of Fountain	Chlorination for Well 4	1/15/2013	PFHxS	80	CO
297	City of Fountain	Chlorination for Well 2	1/15/2013	PFHxS	80	CO
298	Security WSD	Chlorinator for S8 Well	7/30/2014	PFOS	80	CO
299	Security WSD	Chlorinator for W9 Well	7/30/2014	PFOA	80	CO
300	Security WSD	Chlorinator for Well W8	7/31/2014	PFOA	80	CO
301	Security WSD	Chlorinator for S9 Well	7/29/2014	PFOS	80	CO
302	Security WSD	Air Stripping Plant	1/29/2014	PFOA	80	CO
303	Security WSD	Chlorinator for Well W8	1/15/2014	PFOA	80	CO
304	Security WSD	Chlorinator for S7 Well	1/22/2014	PFOS	90	CO
305	Security WSD	Chlorinator for S4 Well	2/10/2014	PFOS	90	CO
306	Security WSD	Chlorinator for S7 Well	7/30/2014	PFOS	90	CO
307	Security WSD	Chlorinator for Well S10	7/30/2014	PFOS	90	CO
308	Security WSD	Chlorinator for S11 Well	4/22/2014	PFHxS	90	CO
309	Security WSD	Chlorinator for Well S10	1/14/2014	PFOS	90	CO
310	Security WSD	Chlorinator for S11 Well	4/22/2014	PFOS	90	CO
311	Security WSD	Chlorinator for Well W8	7/31/2014	PFBS	90	CO
312	Security WSD	Air Stripping Plant	7/31/2014	PFOA	90	CO
313	City of Fountain	Fountain Valley Authority Intertie	4/16/2013	PFOS	100	CO
314	Security WSD	Chlorinator for S11 Well	7/30/2014	PFHxS	100	CO
315	Security WSD	Chlorinator for S11 Well	7/30/2014	PFOS	100	CO
316	Security WSD	Chlorinator for S4 Well	7/30/2014	PFOS	100	CO

UCMR3 data for the State of Colorado of January 1, 2015 page 4

	A	B	C	D	E	F
1	PWSName	FacilityName	Date	PFAS	ng/L	State
319	Security WSD	Chlorinator for S4 Well	2/10/2014	PFHxS	110	CO
320	Security WSD	Chlorinator for S7 Well	1/22/2014	PFHxS	130	CO
321	Security WSD	Chlorinator for Well S10	1/14/2014	PFHxS	130	CO
322	Widefield WSD	Chlorinator for E2 Well	5/14/2014	PFHxS	130	CO
323	Security WSD	Chlorinator for Well S10	7/30/2014	PFHxS	140	CO
324	Security WSD	Chlorinator for S4 Well	7/30/2014	PFHxS	140	CO
325	Security WSD	Chlorinator for S7 Well	7/30/2014	PFHxS	140	CO
326	Security WSD	Air Stripping Plant	1/29/2014	PFOS	140	CO
327	Security WSD	Air Stripping Plant	7/31/2014	PFBS	150	CO
328	Security WSD	Chlorinator for FV4 Well	1/14/2014	PFHxS	160	CO
329	Widefield WSD	Chlorinator for W7 Well	5/14/2014	PFHxS	160	CO
330	Security WSD	Chlorinator for S9 Well	7/29/2014	PFHxS	180	CO
331	Security WSD	Chlorinator for S8 Well	7/30/2014	PFHxS	180	CO
332	Security WSD	FVA Intertie	7/30/2014	PFHxS	180	CO
333	Widefield WSD	Chlorinator for W3 Well	11/12/2013	PFHxS	180	CO
334	Widefield WSD	Chlorinator for E2 Well	5/14/2014	PFOS	180	CO
335	City of Fountain	Fountain Valley Authority Intertie	4/16/2013	PFHxS	190	CO
336	Security WSD	Chlorinator for FV4 Well	7/29/2014	PFHxS	200	CO
337	Security WSD	Chlorinator for S9 Well	1/14/2014	PFHxS	210	CO
338	Security WSD	Chlorinator for S2 Well	7/29/2014	PFHxS	210	CO
339	Security WSD	Air Stripping Plant	7/31/2014	PFOS	210	CO
340	Security WSD	Chlorinator for S2 Well	1/21/2014	PFHxS	210	CO
341	Widefield WSD	Chlorinator for W7 Well	5/14/2014	PFOS	210	CO
342	Widefield WSD	Chlorinator for C36 Well	11/6/2013	PFHxS	210	CO
343	Widefield WSD	Chlorinator for W4 Well	11/12/2013	PFHxS	220	CO
344	Security WSD	Chlorinator for S15 Well	1/21/2014	PFHxS	230	CO
345	Security WSD	Chlorinator for W12 Well	7/30/2014	PFHxS	240	CO
346	Security WSD	Chlorinator for W12 Well	1/15/2014	PFHxS	240	CO
347	Widefield WSD	Chlorinator for W3 Well	5/14/2014	PFHxS	270	CO
348	Security WSD	Chlorinator for S15 Well	7/30/2014	PFHxS	290	CO
349	Security WSD	Chlorinator for S16 Well	1/22/2014	PFHxS	300	CO
350	Security WSD	Chlorinator for Well W8	1/15/2014	PFHxS	310	CO
351	Security WSD	Chlorinator for R1 Well	8/11/2014	PFHxS	310	CO
352	Security WSD	Chlorinator for W9 Well	1/15/2014	PFHxS	310	CO
353	Security WSD	Chlorinator for R1 Well	1/14/2014	PFHxS	320	CO
354	Security WSD	Chlorinator for R2 Well	7/29/2014	PFHxS	330	CO
355	Widefield WSD	Chlorinator for W4 Well	5/19/2014	PFHxS	330	CO
356	Security WSD	Chlorinator for W9 Well	7/30/2014	PFHxS	340	CO
357	Security WSD	Chlorinator for R2 Well	1/14/2014	PFHxS	340	CO
358	Security WSD	Chlorinator for Well W8	7/31/2014	PFHxS	350	CO
359	Security WSD	CT 150,000 Gallon Steel Tank	7/15/2014	PFHxS	360	CO
360	Security WSD	CT 150,000 Gallon Steel Tank	1/22/2014	PFHxS	380	CO
361	Security WSD	Chlorinator for S16 Well	7/30/2014	PFHxS	420	CO
362	Security WSD	Air Stripping Plant	1/29/2014	PFHxS	440	CO
363	Security WSD	Chlorinator for Well W8	1/15/2014	PFOS	580	CO
364	Security WSD	Chlorinator for Well W8	7/31/2014	PFOS	580	CO
365	Security WSD	Chlorinator for W12 Well	7/30/2014	PFOS	590	CO
366	Security WSD	Air Stripping Plant	7/31/2014	PFHxS	590	CO
367	Security WSD	Chlorinator for W12 Well	1/15/2014	PFOS	600	CO
368	Security WSD	Chlorinator for W9 Well	7/30/2014	PFOS	650	CO
369	Security WSD	Chlorinator for W9 Well	1/15/2014	PFOS	1300	CO

UCMR3 data for the State of Pennsylvania of January 1, 2015 page 1

1	PWSName	FacilityName	Date	PFAS	ng/L	State
611	Warminster Municipal Authority	Well 2	11/19/2013	PFHpA	10.5	PA
612	Aqua PA - Hatboro	Well 17	6/25/2014	PFHpA	11	PA
613	Ambler Borough Water Department	Loch Alsh Well #2	8/20/2013	PFHpA	12	PA
614	Warminster Municipal Authority	Well 13	6/9/2014	PFHpA	15.9	PA
615	Warminster Municipal Authority	Well 13	11/19/2013	PFHpA	19.9	PA
616	Aqua PA - Bristol	Filter Plant 2	6/20/2014	PFOA	20	PA
617	Horsham Water & Sewer Authority	Well #10	6/24/2014	PFOA	20	PA
618	Warminster Municipal Authority	Well 5	6/9/2014	PFOA	20.1	PA
619	Warminster Municipal Authority	Well 9	11/19/2013	PFOA	20.3	PA
620	Warminster Municipal Authority	Well 2	6/9/2014	PFOA	21.6	PA
621	Emmaus Borough Public Water	Waterworks Building	3/24/2014	PFNA	22	PA
622	Warminster Municipal Authority	Well 14	11/19/2013	PFOA	22.8	PA
623	Horsham Water & Sewer Authority	Well #17	6/24/2014	PFOA	23	PA
624	Warminster Municipal Authority	Well 5	11/19/2013	PFOA	23.1	PA
625	United Water PA	Airport	2/4/2014	PFHpA	23.4	PA
626	Warminster Municipal Authority	Well 10	11/19/2013	PFHpA	23.8	PA
627	Warminster Municipal Authority	Well 10	6/9/2014	PFHpA	23.9	PA
628	Warminster Municipal Authority	Well 15	11/19/2013	PFOA	24.6	PA
629	Aqua PA - Bristol	Filter Plant 2	12/20/2013	PFOA	26	PA
630	Ambler Borough Water Department	Loch Alsh Well #2	8/20/2013	PFNA	29	PA
631	Warminster Municipal Authority	Well 2	11/19/2013	PFHxS	30.8	PA
632	Warminster Municipal Authority	Well 14	11/19/2013	PFHxS	31.1	PA
633	Quakertown Boro	Well 13	6/17/2014	PFNA	32.3	PA
634	Horsham Water & Sewer Authority	Well #26	6/24/2014	PFHpA	33	PA
635	Horsham Water & Sewer Authority	Well #10	6/24/2014	PFHxS	33	PA
636	Warminster Municipal Authority	Well 2	11/19/2013	PFOA	34.3	PA
637	Warminster Municipal Authority	Well 15	11/19/2013	PFHxS	35.9	PA
638	United Water PA	Airport	2/4/2014	PFOA	38	PA
639	Horsham Water & Sewer Authority	Well #40	6/24/2014	PFHpA	39	PA
640	Horsham Water & Sewer Authority	Well #17	6/24/2014	PFHxS	40	PA
641	Horsham Water & Sewer Authority	Well #10	6/24/2014	PFOS	41	PA
642	Warminster Municipal Authority	Well 2	6/9/2014	PFOS	41.3	PA
643	United Water PA	Airport	2/4/2014	PFNA	46.6	PA
644	Warminster Municipal Authority	Well 14	11/19/2013	PFOS	55.3	PA
645	Warminster Municipal Authority	Well 26	11/19/2013	PFHpA	56.1	PA
646	Warminster Municipal Authority	Well 2	11/19/2013	PFOS	57.6	PA
647	Warminster Municipal Authority	Well 13	6/9/2014	PFHxS	59.5	PA
648	Horsham Water & Sewer Authority	Well #40	6/24/2014	PFOA	63	PA
649	Warminster Municipal Authority	Well 15	11/19/2013	PFOS	64.5	PA
650	Warminster Municipal Authority	Well 26	6/9/2014	PFHpA	71	PA
651	Horsham Water & Sewer Authority	Well #17	6/24/2014	PFOS	74	PA
652	Warminster Municipal Authority	Well 10	6/9/2014	PFHxS	76.1	PA
653	Horsham Water & Sewer Authority	Well #21	6/24/2014	PFHxS	79	PA
654	Warminster Municipal Authority	Well 13	6/9/2014	PFOA	79.6	PA
655	Warminster Municipal Authority	Well 10	6/9/2014	PFOA	82.2	PA
656	Warminster Municipal Authority	Well 13	11/19/2013	PFHxS	87.9	PA
657	Warminster Municipal Authority	Well 10	11/19/2013	PFOA	88.5	PA
658	Warminster Municipal Authority	Well 10	11/19/2013	PFHxS	100	PA
659	Horsham Water & Sewer Authority	Well #40	6/24/2014	PFBS	110	PA
660	Warminster Municipal Authority	Well 13	11/19/2013	PFOA	122	PA
661	Warminster Municipal Authority	Well 13	6/9/2014	PFOS	138	PA

UCMR3 data for the State of Pennsylvania of January 1, 2015 page 2

	A	B	C	D	E	F
1	PWSName	FacilityName	Date	PFAS	ng/L	State
662	Horsham Water & Sewer Authority	Well #21	6/24/2014	PFOS	140	PA
663	Warminster Municipal Authority	Well 13	11/19/2013	PFOS	160	PA
664	Warminster Municipal Authority	Well 10	6/9/2014	PFOS	176	PA
665	Warminster Municipal Authority	Well 10	11/19/2013	PFOS	193	PA
666	United Water PA	Airport	2/4/2014	PFHxS	209	PA
667	Horsham Water & Sewer Authority	Well #26	6/24/2014	PFOA	290	PA
668	Warminster Municipal Authority	Well 26	11/19/2013	PFOA	291	PA
669	Warminster Municipal Authority	Well 26	11/19/2013	PFHxS	334	PA
670	Warminster Municipal Authority	Well 26	6/9/2014	PFOA	349	PA
671	United Water PA	Airport	2/4/2014	PFOS	363	PA
672	Warminster Municipal Authority	Well 26	6/9/2014	PFHxS	389	PA
673	Horsham Water & Sewer Authority	Well #26	6/24/2014	PFHxS	390	PA
674	Horsham Water & Sewer Authority	Well #40	6/24/2014	PFHxS	590	PA
675	Horsham Water & Sewer Authority	Well #26	6/24/2014	PFOS	700	PA
676	Warminster Municipal Authority	Well 26	11/19/2013	PFOS	791	PA
677	Horsham Water & Sewer Authority	Well #40	6/24/2014	PFOS	1000	PA
678	Warminster Municipal Authority	Well 26	6/9/2014	PFOS	1090	PA
679	Pierre	Treat Site #8	1/22/2014	PFHxS	40	SD

Each record in this database represents the occurrence of PFAS in finished drinking water but likely to represent a complicated story involving different sources, histories, and populations

Drinking water in UCMR3 is likely to be the tip of the iceberg, may indicate widespread environmental contamination including: agricultural commodities, fisheries and wildlife resources, soil contamination, airborne exposures

UCMR3 results should be used for many things:

Help identify and categorize sources of legacy contamination: industrial chemical production, sewage sludge, AFFF, landfills…

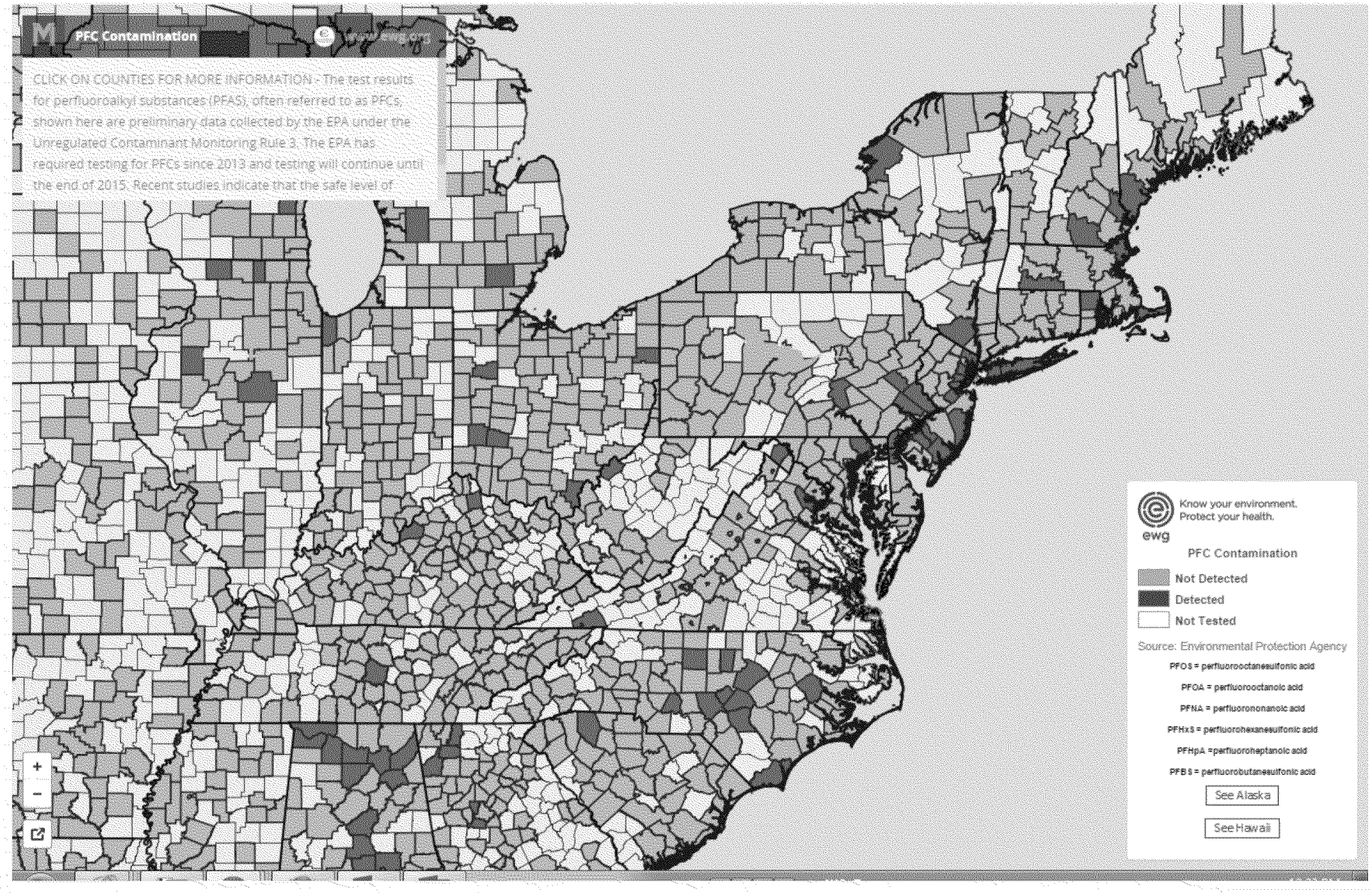
Identify and populations that are exposed to PFAS, provide rational & location for specific epidemiological studies

Verify and track community exposures with blood and water measurements - efforts underway in Wilmington and Greensboro, NC

Help municipalities identify sources of contamination, assess control technologies

Use UCMR3 results to target additional studies to monitor for replacement PFAS

Need detailed maps of UCMR3 results to facilitate this research



http://www.ewg.org/research/teflon-chemical-harmful-at-smallest-doses

30 records with PFOS higher than the Provisional Health Advisory (200 ng/L)

1	PWSID	PWSName	FacilityName	CollectionDate	Contaminant	ng/L	ug/L	State
48	CO0121775	Security WSD	Air Stripping Plant	7/31/2014	PFOS	210	0.21	CO
49	CO0121900	Widefield WSD	Chlorinator for W7 Well	5/14/2014	PFOS	210	0.21	CO
50	MA4020004	Hyannis Water System	Mary Dunn Well #3	5/22/2014	PFOS	210	0.21	MA
51	OH2903412	Wright-Patterson AFB Area A/C	Building 10855 - STU 1	10/7/2014	PFOS	210	0.21	OH
52	AZ0407046	Liberty Water LPSCO	Well 02 Airline	5/29/2014	PFOS	220	0.22	AZ
53	GU0000006	GU Waterworks Authority - Northern System	Well A-25	3/25/2015	PFOS	220	0.22	GU
54	ME0091410	Sanford Water District	Treatment Plant 5	8/7/2013	PFOS	250	0.25	ME
55	FL1170525	Emerald Coast Utilities Authority	Hagler	6/17/2014	PFOS	270	0.27	FL
56	ME0091410	Sanford Water District	Treatment Plant 5	4/9/2013	PFOS	290	0.29	ME
57	AZ0408001	Oatman Water Company	Well #1	4/22/2014	PFOS	300	0.3	AZ
58	NY5110526	Suffolk County Water Authority	Gus Guerrera Wellfield	9/8/2014	PFOS	330	0.33	NY
59	PA7220015	United Water PA	Airport	2/4/2014	PFOS	363	0.363	PA
60	FL1170525	Emerald Coast Utilities Authority	Hagler	11/11/2014	PFOS	380	0.38	FL
61	MA4020004	Hyannis Water System	Mary Dunn Well #2	5/22/2014	PFOS	430	0.43	MA
62	MN1820016	Oakdale	Well #7	1/5/2015	PFOS	435	0.435	MN
63	WA5336350	Issaquah Water System	Well #4	1/8/2014	PFOS	514	0.514	WA
64	NY5110526	Suffolk County Water Authority	Oxhead Rd. Wellfield	12/29/2014	PFOS	530	0.53	NY
65	CO0121775	Security WSD	Chlorinator for Well W8	1/15/2014	PFOS	580	0.58	CO
66	CO0121775	Security WSD	Chlorinator for Well W8	7/31/2014	PFOS	580	0.58	CO
67	CO0121775	Security WSD	Chlorinator for W12 Well	7/30/2014	PFOS	590	0.59	CO
68	CO0121775	Security WSD	Chlorinator for W12 Well	1/15/2014	PFOS	600	0.6	CO
69	WA5336350	Issaquah Water System	Well #4	7/22/2013	PFOS	600	0.6	WA
70	CO0121775	Security WSD	Chlorinator for W9 Well	7/30/2014	PFOS	650	0.65	CO
71	PA1090070	Warrington Township Water & Sewer Department	Wells 1, 2 & 6	11/11/2014	PFOS	666.66	0.66666	PA
72	PA1460033	Horsham Water & Sewer Authority	Well #26	6/24/2014	PFOS	700	0.7	PA
73	PA1090069	Warminster Municipal Authority	Well 26	11/19/2013	PFOS	791	0.791	PA
74	DE0000552	Artesian Water Company	Wilm. Manor 3 Plant	7/17/2013	PFOS	930	0.93	DE
75	PA1460033	Horsham Water & Sewer Authority	Well #40	6/24/2014	PFOS	1000	1	PA
76	PA1090069	Warminster Municipal Authority	Well 26	6/9/2014	PFOS	1090	1.09	PA
77	CO0121775	Security WSD	Chlorinator for W9 Well	1/15/2014	PFOS	1300	1.3	CO
78	DE0000552	Artesian Water Company	Wilm. Manor 3 Plant	1/28/2014	PFOS	1800	1.8	DE

Questions?

Email: lindstrom.andrew@epa.gov

From: Burneson, Eric
Required Attendees: Burneson, Eric; Lovely, William; Grevatt, Peter; sarah.pillsbury@des.nh.gov
Optional Attendees: Greene, Ashley; Spalding, Curt; Olson, Bryan; Barmakian, Nancy; Lindsay, Jane; Gray, Stuart; Szaro, Deb
Location: 866-299-3188 code 202-564-5250
Importance: Normal
Subject: Monitoring Results PFOA
Start Date/Time: Fri 3/18/2016 3:45:00 PM
End Date/Time: Fri 3/18/2016 4:15:00 PM

Confirming the call at 11:45. Note that the call in code originally sent was incorrect. The code above is 202 564 5250 is the correct code.

From: Burneson, Eric
Required Attendees: Christ, Lisa (Christ.Lisa@epa.gov); Huff, Lisa; Behl, Betsy
Location: Call in number: 866-299-3188 code: 212-637-3324# or WJC South 6204 for VTC.
Importance: Normal
Subject: Call in to FFLC Meeting EPA/DoD Session
Start Date/Time: Wed 3/2/2016 8:00:00 PM
End Date/Time: Wed 3/2/2016 10:30:00 PM

Greg

DoD questions:

1. What is EPA doing regarding all UCMR 3 exceedances of PFOA and PFOS values (e.g. PHA or other) nationally? What about the other 28 UCMR 3 constituents?
2. How is or will EPA be addressing the existing PHAs and potential changes to PHAs for PFOA and PFOS at public water systems (PWS)?
3. Does EPA have an expected timeframe when these existing PHAs may change?
4. Will EPA be developing MCLs for PFOA and PFOS and any other PFCs?
5. Where there are UCMR 3 exceedances of PHAs at PWSs, what actions is EPA taking? Is this consistent whether there is a DoD installation nearby or not?

Draft Agenda for FFLC Meeting/EPA-DoD Session:

Wednesday, March 2, 2016

12:00 - 12:15	**DoD - Introductions and Opening Remarks**		EPA, DoD
12:15 - 1:15	**Partnering efforts** ✓ Discussion expanding on the partnering discussion at the October FFLC	I, C	DoD, Region 3
1:15 - 1:45	**Climate Change** ✓ Discussion related to the Climate Change project. ✓ Update from EPA on the EPA-wide effort and how it relates to cleanup sites	I, C	FFRRO/DoD
1:45 - 2:30	**Performance Based Contracting**	I,C	EPA, DoD
2:30 - 2:45	**BREAK**		
2:45 - 3:15	**ATSDR Involvement**	I, C	DoD

3:15 - 5:30 (includes 10 min break)	**PFCs - General Discussion** ✓ Dover success story (R3) ✓ Discussion of UCMR3 ✓ EPA and DoD share what they are currently doing to address PFCs on their sites ✓ How does DoD plan to address PFCs moving forward on their sites?	I, C	DoD, EPA - EPA Region 3 - EPA Office of Water - EPA and DoD - DoD (open discussion per DoD component?)	
5:30	**Day 2 Wrap Up**			

****
Greg Gervais
Acting Deputy Director
Federal Facilities Restoration & Reuse Office
USEPA OLEM
202-564-4409 (office)
****

To: Burneson, Eric[Burneson.Eric@epa.gov]
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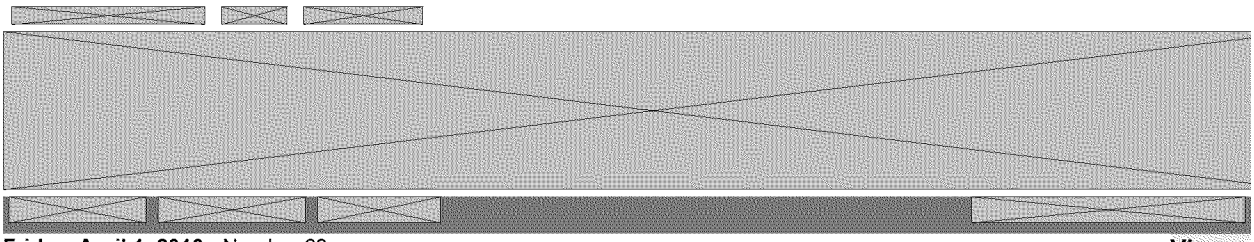

Friday, April 1, 2016 · Number 63

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NEWS

Agriculture
Groups File Suit to Reverse FDA Approval of GE Salmon
A coalition of consumer and environmental groups are suing the Food and Drug Administration to reverse the agency's approval of genetically modified salmon for sale and consumption (Institute for Fisheries Resources v. Burwell, No....

Air Pollution
DOJ Expects to Have 'Something' by Judge's VW Deadline
A top Justice Department official told Bloomberg BNA that he expects the federal government and Volkswagen to have "something" to tell a federal court judge by an April 21 deadline to present a plan to address about 580,000 noncompliant...

Air Pollution
Modi's Air Quality Plan Resisted by Indian Power Generators
India's effort to clean up the world's worst air is facing resistance from power producers who say the government is asking them to spend too much and revamp old plants too quickly....

Asbestos
Dismissal of Asbestos Cases Against Four Companies Upheld
Asbestos exposure claims brought by the survivor of a South Carolina shipyard worker don't have enough evidentiary support and must be dismissed, the U.S. Court of Appeals for the Fourth Circuit ruled (Pace v. Air & Liquid Sys. Corp.,...

Asbestos
No Product Liability for Shipbuilders in Asbestos Case
The heirs of a man who allegedly contracted mesothelioma while working on Navy warships containing asbestos can't proceed with strict product liability claims against the shipbuilders, the

Ninth Circuit ruled March 31 (McIndoe v. Huntington...

Biotechnology
Biotech Framework Update Should Focus on Data Burden
The multiple federal agencies that regulate biotechnology and its products should create more certainty for the regulated community with clear review triggers and data standards, and a single information package they could submit to all...

Chemicals
Eight Chemical Firms No Longer Make, Use PFOA
The Arkema Group, BASF Corp., Daikin Industries, DuPont successor Chemours and four other chemical manufacturers that previously made or used perfluorooctanoic acid (PFOA) and related perfluorinated chemicals ceased their production...

Chemicals
Groups Demand Answer From FDA on Perchlorate
Six advocacy groups filed a lawsuit March 31 against the U.S. Food and Drug Administration in an attempt to force the agency to respond to its calls for a ban on perchlorate in food packaging (Breast Cancer Fund v. FDA, 9th Cir., No. 16-70878, 3/31/16)....

Climate Change
Panel Wraps Up First Phase on Company Climate Disclosure
A panel convened to bring about greater transparency to the way companies disclose the risks they face from climate change has concluded the first phase of its work as it moves toward proposing a set of voluntary reporting standards....

Climate Regulation
Administration Rushing Methane Rules to Boost Legacy: API
President Barack Obama's administration is rushing out a suite of rules meant to curb methane emissions as a way to bolster his climate change legacy, the American Petroleum Institute said March 31....

Climate Regulation
Former EPA Administrators Defend Clean Power Plan
The Clean Power Plan falls squarely within the Environmental Protection Agency's authority under the "broadly worded" provisions of the Clean Air Act, two past agency administrators said in defense of the rule (West Virginia...

Climate Regulation
White House Reviewing De Minimis Permitting Rule
The White House Office of Management and Budget is reviewing an Environmental Protection Agency proposal to set a de mininis standard for greenhouse gas emissions, below which large industrial facilities would not need to obtain a permit...

Drinking Water
EPA Attorney: Flint Shows Need to Improve Communication
The Flint water crisis illustrates a need for regulators to think "long and hard" about how best to communicate public health risks, the Environmental Protection Agency's top attorney said. ...

Drinking Water
EPA Must Resolve Oil, Gas Wells Oversight Issues: GAO
The Environmental Protection Agency hasn't ensured that underground sources of drinking water are being adequately protected from certain oil and gas underground injection activities, the Government Accountability Office said...

Drinking Water
New York Says Hoosick Falls Water Free of PFOA
Repeated tests of the municipal water system of Hoosick Falls, N.Y., have detected no perfluorooctanoic acid contamination, the New York Health Department said March 30. The village's drinking water is now safe for all uses, including...

Energy
Mexico's First Power Auction Hailed as Success
Mexico is one step closer to meeting its ambitious clean energy targets, thanks to its first wholesale electricity auction on March 29, in which 11 companies won long-term contracts to supply renewable energy and clean energy certificates...

Energy
Ohio OKs Rate Hikes, Subsidizes Coal, Nuclear Plants
Electric bills will go up for millions following the Public Utility Commission of Ohio's approval of two utilities' rate stability plans that subsidize older power plants....

Enforcement
SEC Sues Navistar and Ex-CEO for Truck Engine Lies
A former Navistar International Corp. chief executive officer is facing Securities and Exchange Commission allegations that he misled investors about the company's development of an advanced technology truck engine (SEC v. Ustian,...

Hydraulic Fracturing
Nebraska Governor Signs Bill on Fracking Disposal Wells
Nebraska Gov. Pete Ricketts (R) signed a bill March 30 that imposes additional disclosure regulations on operators of wastewater disposal wells used in hydraulic fracturing....

International Climate
Obama, Xi Push Early Entry Into Force of Paris Climate Deal
A U.S.-China pledge to quickly sign and implement the Paris climate pact—and prod other countries to follow suit—could get the deal formally in force well before 2020, Obama administration officials said March 31....

Mining
Molycorp OK to Exit Chapter 11 Without Troubled Mine
Molycorp Inc. will be able to exit bankruptcy protection with its profitable mineral-processing business, while leaving its money-losing California rare-earths mine behind (In re Molycorp Inc., Bankr. D. Del., No. 15-bk-11357, 3/30/16)....

Natural Gas
Japanese Ambassador Urges Canada LNG Exports
The window for Canada to begin exporting liquefied natural gas is closing and may not open again for a decade or more, Japan's ambassador to Canada recently warned Prime Minister Justin Trudeau and environmental officials....

Oil & Gas
BP Signs First Shale-Gas Exploration Deal in China with CNPC
BP Plc will explore for shale-gas in China with the country's biggest oil company China National Petroleum Corp., the British producer's first such contract in the nation estimated to hold the world's biggest resources...

Oil & Gas

Exxon, California Reach Agreement in Refinery Unit Restart
Exxon Mobil Corp. and California regulators reached a preliminary agreement to restart a unit at the Torrance refinery, a key step toward closing the facility's pending sale to PBF Energy Inc. ...

Oil & Gas
Oil Industry Urged to Focus on Long Term During Downturn
Even though the current oil-gas downturn is a normal part of the industry's cycle and less severe than the 1980s slump, companies need to focus on a long-term business strategy, panelists at a forum in Houston said March 31....

Oil Spills
Cruden: Judges' OK Would Lock Down BP Settlement
Judicial approval of a multibillion-dollar settlement between the federal government and BP Plc over the Deepwater Horizon oil spill would be a powerful tool to ensure that the oil and gas company meets its obligations, a top Justice Department...

Pipeline Safety
Return of Gas Pipeline Rules Opposed by Industry
Safety regulations previously opposed by natural gas pipeline operators and others in the industry have returned in a Department of Transportation proposed rule that would set new requirements for both main and gathering pipelines. ...

Radioactive Waste
N.M. Deal Aims to Enhance Los Alamos Cleanup
New Mexico's Environment Department on March 30 released a draft consent order for comment governing the cleanup of legacy waste at Los Alamos National Laboratory. The state initiated public discussion and feedback five months ago on...

Renewable Energy
$2.6 Billion Buying Binge Pushed SunEdison to Brink
Just nine months ago, SunEdison Inc. was Wall Street's favorite clean-energy company. It sopped up every dollar it could come by to finance a breathtaking buying binge of wind and solar farms, and in the process became the world's...

Renewable Energy
The U.K. Propped Up Europe's Ailing Solar Market Last Year
If it wasn't for the U.K., Europe's solar power market would have flat-lined last year, according to new industry figures that reveal the world's biggest market for the technology is struggling to retain its scale....

Renewable Energy
U.K. Carbon Pollution Fell in 2015 as Renewables Replace Coal
U.K. greenhouse gas pollution fell last year after generation from coal plants dropped and clean energy use rose, according to government figures published March 31....

Risk Assessment
Chemical Facility Auditing Proposal Defended by EPA's Giles
An Environmental Protection Agency proposal that would require chemical plants to hire independent third parties to perform compliance audits under the agency's Risk Management Program is supported by evidence that those audits are...

Superfund
Buyer of Contaminated Parcel Can't Enforce Consent Decree
A company that bought property at an auction set up under a Superfund consent decree lacks

standing to enforce the decree, the U.S. District Court for the District of Colorado ruled March 29 (United States v. Colorado & Eastern R.R., 2016...

Superfund
EPA, NOAA Differ on Hudson River Dredging Project
Two federal agencies have issued conflicting studies on the nearly completed project to dredge polychlorinated biphenyls from the Hudson River, with the National Oceanic and Atmospheric Administration calling for additional dredging...

Superfund
OIG Flays EPA Superfund, RCRA Financial Assurances
The Environmental Protection Agency is jeopardizing taxpayer dollars, public health and environmental protections by failing to lock in place financial assurances from companies that operate Comprehensive Environmental Response,...

Water Pollution
Des Moines Utility's Battle Over Farm Runoff Causes Rift
In the early spring of 2014, two lab workers for the Des Moines Water Works climbed into a truck and drove north. They pulled over on Highway 20 where the road crosses Cedar Creek, made their way to the water's edge, dunked a cup attached to...

Water Pollution
EPA Power Plant Data Not Available Under Clean Water Act
The Clean Water Act does not preempt the Environmental Protection Agency's ability under the Freedom of Information Act to withhold data it obtained as part of a survey about pollution from steam-driven power plants, a federal district...

Wetlands
U.S.-State Study Charts Long Island Sound Wetlands Loss
Nearly a third of the wetlands along Long Island Sound have disappeared since the 1880s, representing a serious loss for coastal communities, fish and wildlife, according to a federal-state study....

Analytical Methods to Enforce EU's REACH
A compendium of analytical methods that European Union member state authorities can use to enforce manufacturers' compliance with restrictions the European Commission has approved for certain chemicals was announced by the European...

REGULATORY AGENDA

APRIL 1 FEDERAL REGISTER

Comment Deadlines

MARCH 31 FEDERAL REGISTER

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To: Burneson, Eric[Burneson.Eric@epa.gov]
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Afternoon Briefing - Your Preview of Today's News

The following news provides a snapshot of what Bloomberg BNA is working on today. Read the full version of all the stories in the final issue, published each night.

U.S.-China Pledge May Speed Up Paris Climate Deal Implementation

Posted March 31, 2016, 12:41 P.M. ET

By Dean Scott

A U.S.-China pledge today to quickly sign and implement the Paris climate deal—and prod other countries to follow suit—could get the deal formally in force well before 2020, the top U.S. climate negotiator said today.

President Barack Obama and China President Xi Jinping, meeting today in Washington, D.C., announced the two top-emitting nations will sign the climate accord in New York on April 22 and “take their respective domestic steps” at home to join the pact “as early as possible” in 2016.

The 2015 climate pact is to enter into force no later than 2020, but today's announcement may have “precisely the effect” of it being implemented “significantly earlier”—perhaps even this year—U.S. Special Envoy for Climate Change Todd Stern told reporters on a press call. At least 55 nations accounting for 55 percent of emissions must ratify or otherwise approve the accord for it to enter into effect, but the U.S. and China alone account for roughly 40 percent of global emissions, Stern said.

DOJ Expects to Have ‘Something' by Judge's VW Deadline

Posted March 31, 2016, 3:08 P.M. ET

By Patrick Ambrosio

A top Justice Department official told Bloomberg BNA today that he expects the federal government and Volkswagen to have “something” to tell a federal court judge by an April 21 deadline to present a plan to address about 580,000 noncompliant diesel vehicles.

“I expect that we'll have something to tell the court by April 21,” said John Cruden, assistant attorney general for environment and natural resources. “I can't tell you what that is because we're still working on it, but I expect that we'll have something to tell the court.”

Cruden, during an interview at an American Bar Association conference in Austin, Texas, said the Environmental

Protection Agency and California regulators continue to work on a plan to address Volkswagen's diesel vehicles, which were equipped with illegal technology that allowed the vehicles to pass emissions tests despite emitting more pollution than allowed. U.S. District Court Judge Charles Breyer, at a March 24 hearing, gave the automaker a deadline to present a plan to present a concrete proposal that would address the over-emitting diesels, which remain on the road more than six months after the emissions issues were first announced to the public.

“They are diligently working right now on those issues,” Cruden said of the EPA and the California Air Resources Board. “It's complicated how you handle those cars.”

Eight Chemical Manufacturers No Longer Make, Use PFOA

Posted March 31, 2016, 3:03 P.M. ET

By Pat Rizzuto

The Arkema Group, BASF Corp., DuPont successor Chemours and five other chemical manufacturers that previously made or used perfluorooctanoic acid (PFOA) and related perfluorinated chemicals ceased their production or use of them by the end of 2015, the Environmental Protection Agency told Bloomberg BNA today.

The EPA updated Bloomberg BNA on the status of 2010/2015 PFOA Stewardship Program.

Under that voluntary 2006 program, eight major U.S. fluoropolymer and telomer manufacturers committed to reduce their production or use of PFOA and other chemicals that can break down to PFOA. They also pledged to work toward the elimination of PFOA from their emissions and products by 2015. Those pledges were met, the EPA confirmed.

PFOA has been used to make fluoropolymers, which are used to make many consumer and industrial products including non-stick cookware. The association of some perfluorinated chemicals, particularly PFOA, with kidney and testicular cancer, thyroid disease and other diseases has prompted thousands of lawsuits against DuPont and Chemours.

Notwithstanding the cessation of PFOA's production and use, the chemical persists for many years in the environment and human bodies. It is being found in drinking water systems in New Hampshire, New York state and elsewhere due to that persistence.

EPA Falls Short on Superfund, RCRA Financial Assurances: OIG

Posted March 31, 2016, 3:17 P.M. ET

By Brian Dabbs

The Environmental Protection Agency's Office of Inspector General lashed into the agency's alleged failure to lock in place required financial assurances from companies that operate Comprehensive Environmental Response, Compensation, and Liability Act and Resource Conservation and Recovery Act sites.

“If the EPA cannot determine if it has secured valid and sufficient financial assurance instruments from those, taxpayers are at risk for paying significant amounts of those parties' financial obligations,” said the report, which says $577 million in corporate self-insurance is expired and billions more are undocumented. “In addition, public health protections may be delayed or deferred.”

The EPA disputed the claims as overblown, according to the report. OIG officials described improvement as “time-critical,” providing EPA 30 days to respond.

Former EPA Administrators Defend Clean Power Plan

Posted March 31, 2016, 12:21 P.M. ET

By Andrew Childers

The Environmental Protection Agency's Clean Power Plan falls squarely within its authority under the “broadly worded” provisions of the Clean Air Act, two past agency administrators who were appointed by Republican

presidents said in defense of the rule.

Congress intentionally made many of the Clean Air Act's provisions broad to give the EPA the flexibility to address emerging air pollution problems without having to ask for the law to be updated, former EPA Administrators William D. Ruckelshaus and William K. Reilly said an amicus brief to be filed today in the U.S. Court of Appeals for the District of Columbia Circuit.

"Many of the Clean Air Act's central terms are famously capacious precisely because Congress in drafting the law anticipated EPA's need to address environmental issues as they emerged and evolved over time, in ways not specifically identified at the time of enactment," they said. "In that light, the current administrator's interpretation of the act in support of the Clean Power Plan falls well within her authority under the Act to make discretionary judgments in adapting its provisions to new challenges."

Ruckelshaus was the EPA's first administrator, appointed by President Richard Nixon. He later served in the same capacity for President Ronald Reagan. Reilly was EPA administrator under President George H.W. Bush.

Panel Wraps Up First Phase on Company Climate Disclosure

Posted March 31, 2016, 12:23 P.M. ET

By Chisaki Watanabe and Gareth Allan

A panel convened to bring about greater transparency to the way companies disclose the risks they face from climate change has concluded the first phase of its work as it moves toward proposing a set of voluntary reporting standards.

The Task Force on Climate-Related Disclosures was established in December by the Financial Stability Board to address the lack of a coherent framework for disclosure among Group of 20 countries, Mary Schapiro, former chair of the U.S. Securities and Exchange Commission, said today at a briefing in Tokyo.

The panel also wants to provide access to "decision-useful, clear and comparable information" on climate-related risks to investors, lenders and insurers, she said. Regulators will also benefit from understanding such risks.

The group concluded phase one today after surveying how information has been disclosed in Group of 20 countries, according to Schapiro. The panel submitted a progress report today to the FSB plenary session, she said. Phase two will convene in April, with the task force scheduled to conclude its work in December.

Michael Bloomberg, founder and majority owner of Bloomberg News parent Bloomberg LP, serves as chairman for the task force.

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Consumer Group Sues to Reverse GE Salmon Approval

Posted March 31, 2016, 11:49 A.M. ET

By Casey Wooten

A coalition led by the Center for Food Safety has filed a lawsuit against the Food and Drug Administration over the agency's approval of genetically engineered salmon for commercial sale and consumption.

Filed yesterday in the U.S. District Court for the Northern District of California, the lawsuit seeks to reverse the November 2015 approval, saying that the FDA did not adequately assess the full range of environmental and ecological effects farming genetically engineered salmon may create.

The salmon, made by AquaBounty Technologies Inc., would be the first mass-produced genetically engineered animal cleared for human consumption, the lawsuit said.

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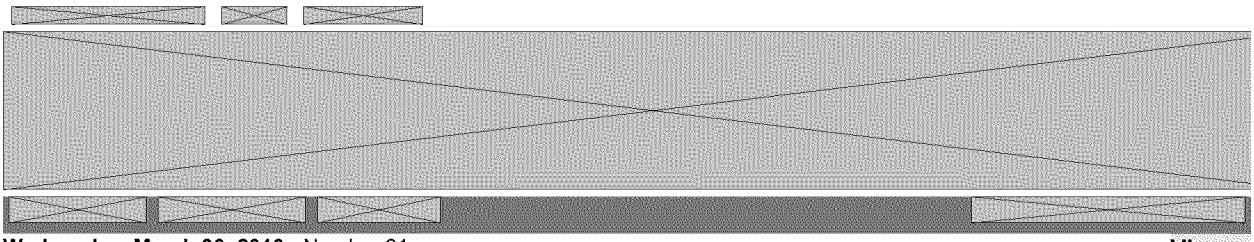

Wednesday, March 30, 2016 · Number 61

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NEWS

Air Pollution
Advocates Challenge EPA's Method to Meet Toxics Obligations
Environmental petitioners are challenging the Environmental Protection Agency's use of surrogate pollutants to meet its Clean Air Act obligations to set emissions standards for industrial sectors that emit certain hazardous air...

Air Pollution
FTC Sues Volkswagen Over Deceptive 'Clean Diesel' Marketing
The Federal Trade Commission filed a lawsuit in federal district court against Volkswagen Group of America, alleging violations of federal law related to the automaker's marketing of its TDI "Clean Diesel" vehicles (FTC v....

Air Pollution
IG Faults EPA Explanation of Tier 3 Rule Cost Estimates
Employees at the Environmental Protection Agency did not accurately or thoroughly explain the basis for cost estimates associated with the Tier 3 standards for gasoline and vehicles, but those lapses were unintentional, according to the...

Asbestos
33-Year-Old Asbestos Memo Sealed in Wrongful Death Case
A 33-year-old memorandum concerning asbestos risks in the pipe industry is playing an outsize role in an asbestos suit on behalf of a pipe worker who died from mesothelioma (Lee v. Certainteed Corp., E.D.N.C., No. 5:13-cv-00826, 3/28/16...

Asbestos
Hillshire Failed to Meet Asbestos Lawsuit Limitation
An asbestos wrongful death suit will return to state court because a Tyson Foods subsidiary waited

too long to remove the action, the Northern District of California ruled March 28 (Lopez v. Allied Packing & Supply Inc., N.D. Cal., No. 16-cv-00371,...

Climate Change
Arctic Sea Ice Shrinks to Lowest on Record After Warm Winter
Ice covered the smallest extent of ocean in the Arctic for a second year in a row this winter after "crazy" high temperatures stopped large swaths of sea from freezing over....

Climate Change
Attorneys General May Join Forces on Climate Probe
State attorneys general looking into allegations that fossil fuel companies misled the public and investors about climate change may be joining forces....

Climate Regulation
EPA Proposes to Curtail Use of Certain HFCs
Various new limitations would be placed on the use of certain ozone-depleting substances in multiple industrial sectors under a proposed Environmental Protection Agency regulation. ...

Climate Regulation
States Defend EPA Clean Power Plan Authority
A court decision to overturn the Environmental Protection Agency's Clean Power Plan on the grounds that it would cause shifts in the nation's electricity mix would effectively bar the agency from regulating all power plant emissions,...

Coal Mining
China's Top Coal-Producing Province Seeks Overseas Market
China’s biggest coal region has a fix for the country’s glut that may send shivers through miners from the U.S. Appalachian Mountains to Australia’s Hunter Basin....

Coal Mining
WildEarth Drops Lawsuit Over Colorado Coal Mine
An environmental group has agreed to dismiss a lawsuit against two Interior Department agencies as well as an administrative appeal of a permit granted by the Environmental Protection Agency for a coal mine in northwest Colorado (WildEarth...

Drinking Water
Ayotte Pushes McCarthy to Address PFOA Water Contamination
Sen. Kelly Ayotte (R-N.H.) prodded the Environmental Protection Agency to expedite the release of a health advisory for perfluorooctanoic acid drinking water contamination in a March 28 letter to agency Administrator Gina McCarthy. New...

Energy
NRC Commissioner: Greater Urgency Needed in Staff Reductions
While the Nuclear Regulatory Commission is meeting its goals to reduce staff as part of its streamlining initiative, an NRC commissioner raised concerns that the agency isn't moving fast enough. ...

Enforcement
Blankenship Should Face Maximum One-Year Sentence: U.S.
Former coal baron Donald Blankenship should face the maximum sentence of one year in prison for flouting mine-safety rules, prosecutors said, urging a judge to send a message to the coal industry (United States v. Blankenship, S.D. W.Va.,...

EPA

Purchase Card Program at EPA Deemed 'Low Risk'
Efforts to implement internal controls of the purchase card program at the Environmental Protection Agency have been successful, and it is now "at a low risk for illegal, improper or erroneous purchases and payments," according...

Hydraulic Fracturing
Study Cites Water Contamination by Fracking
Two researchers at Stanford University said March 29 that an Environmental Protection Agency investigation in 2010 and 2011 provided evidence that hydraulic fracturing near Pavillion, Wyo., had contaminated underground sources of potential...

International Climate
Papua New Guinea First Nation to File Climate Plan
Papua New Guinea became the first country to file a national climate change action plan under the international Paris Agreement, a first step toward applying the terms agreed to by nearly 200 countries last December....

Ozone Depletion
Chemical Makers Say EPA Reversed Course With HFC Ban
The Environmental Protection Agency improperly compared hydrofluorocarbons to later-generation chemicals when it chose to ban the use of some HFCs in favor of substances with less climate change impact, chemical manufacturers told an...

Pesticides
Judges Close Out Enlist Duo Case, Pending EPA Review
A panel of federal appeals court judges closed out a lawsuit over the Dow Chemical Co. pesticide Enlist Duo, although the legal wrangling over this weed killer may not have ended yet....

Renewable Energy
Bills to Purge Energy Credits Stall in Oklahoma Legislature
Four bills before the Oklahoma Legislature seeking the elimination of wind energy tax credits in addition to a set percentage of natural gas use in the state's energy mix will go no further this year, following failures to meet their respective...

Risk Assessment
Industry, Advocates Split on Risk Management Proposal
After digesting the Environmental Protection Agency Risk Management Program proposal for roughly a month, not much has changed in the minds of the chemical and manufacturing industry community....

Solid Waste
WhatsApp-Wielding Vigilantes Want Trash Off India's Streets
If you were a pile of garbage on the street in India, here's what might happen to you now: A concerned citizen takes your picture, then sends it by WhatsApp to the smartphone of the garbage police. Khaki-clad cops jump in their vehicles,...

Superfund
Ninth Circuit Set to Hear Novel Superfund Appeal
The Superfund law, treaties and predictions of limitless liability are likely to animate oral arguments before the U.S. Court of Appeals for the Ninth Circuit April 6 (Pakootas v. Teck Cominco Metals, Ltd., 9th Cir., No. 15-35228, 3/26/15)....

Superfund
Union Pacific Allowed Final Judgment on Superfund Claims

Union Pacific Railroad Co. won't have to wait for Superfund litigation against other defendants to get a final judgment that claims lodged by Asarco LLC are time-barred, the U.S. District Court for the Eastern District of Missouri ruled...

Sustainability
Sensor Technology, Big Data Help 'Smart Cities' Go Green
Stockholm slashed carbon emissions 15 percent, in part by monitoring and taxing road congestion. The Mediterranean island of Malta cut energy use by tracking it and informing residents of their consumption habits. In the Miami area, devices...

Vehicle Fuels
BYD Sees Electric-Car Sales Tripling in Market Coveted by Tesla
BYD Co., the Warren Buffett-backed Chinese car-and-battery manufacturer, may boost deliveries of electric vehicles by as much as three times this year in its home market, underscoring the rising demand that's prompting Tesla Motors...

Water Pollution
EPA Starts Hudson River PCB Project Review
The Environmental Protection Agency announced March 29 it has begun its expedited review of the project to dredge polychlorinated biphenyls from the Hudson River, examining whether the goals of the project have been met....

Water Pollution
Hawaii Imposes Statewide Ban on Cesspools
Newly implemented statewide rules in Hawaii bar construction of new cesspools and offer tax credits to help owners of existing cesspools located near sources of water connect to sewer systems or install septic tanks. ...

Water Pollution
Practical Impact to Be Focus of Water Act Oral Argument
The practical implications of delaying judicial review of a determination that a parcel of land is covered by the Clean Water Act will be front and center when the Supreme Court hears arguments March 30 over whether such a determination is final...

Water Pollution
Real Cost of Water Rule Unknown, ABA Panelists Say
While concerns persist over the potential impacts of a federal rule seeking to define which waters can be regulated under the Clean Water Act, ambiguities remain about its actual costs, panelists said at a conference March 29. ...

Utah Governor Signs Bill Funding California Coal Port
Utah Gov. Gary Herbert (R) signed a bill (S.B. 246) March 22 committing $53 million in state funds to helping build a coal port in Oakland, Calif., with the goal of aiding in the sale of Utah coal overseas. The measure, "Funding for Infrastructure...

INTERVIEW

Sustainability
A More Circular Economy:
A G-7 Focus on Curbing Use of Raw Materials
The Environmental Protection Agency's Mathy Stanislaus coordinates the U.S. response to an alliance launched last year by the Group of Seven leading industrialized countries to address a key

source of global greenhouse gas emissions:...

REGULATORY AGENDA

Comment Deadlines

MARCH 29 FEDERAL REGISTER

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NEWS

Air Pollution
California Adopts Diisocyanate Airborne Exposure Levels
California's Office of Environmental Health Hazard Assessment adopted airborne concentrations of toluene diisocyanate and methylene diphenyl diisocyanate state regulators will use in assessing exposure risks for the two toxic...

Air Pollution
Cincinnati Air Standards Litigation Denied Review
The U.S. Supreme Court will not review a lower court decision that vacated the Environment Protection Agency's redesignation in 2011 of Greater Cincinnati, Ohio, as meeting national air standards for particulate matter (Ohio v. Sierra...

Air Pollution
Litigation Tracker: Ozone Briefing to Begin, Refinery Litigation Halted
Briefing on the legality of the Environmental Protection Agency's national ozone standards issued in 2015 will begin in April and continue through the summer of 2016....

Air Pollution
OMB Reviewing Ozone, Particulate Analysis Guidance
The White House Office of Management and Budget is reviewing a guidance document on permitting issues under ozone and fine particulate matter air quality standards, according to the office's website. The guidance document (RIN 2060-ZA24),...

Climate Regulation
Allowance Allocations Key for Clean Power Plan: Paper
How and to whom states allocate carbon dioxide emissions allowances as part of any mass-based compliance strategy for the Environmental Protection Agency's Clean Power Plan will depend on

the goals state regulators hope to achieve,...

Climate Regulation
EPA Defends Generation Shifting in Clean Power Plan
A Clean Air Act requirement that the Environmental Protection Agency determine the "best system" for cutting carbon dioxide emissions is broad enough to encompass the reductions anticipated in the Clean Power Plan, the agency argued...

Drinking Water
EPA to Release Nonbinding Heath Advisory for PFOA
The Environmental Protection Agency is poised to release a nonbinding health advisory for perfluorooctanoic acid this spring, EPA Region III Administrator Shawn M. Garvin said in a March 25 letter in response to pressure to act on West Virginia...

Drinking Water
FEMA Extends Emergency Status in Flint Until August
The Federal Emergency Management Agency has extended its emergency declaration for Flint, Mich., through Aug. 14 in response to a March 14 request by the state. The agency said in a March 25 letter released by Michigan Gov. Rick Snyder (R) that...

Drinking Water
Halliburton Energy Consent Order Bars Suit Over Well Water
A consent order between Oklahoma and Halliburton Energy Services authorizing the state to address a groundwater plume bars a lawsuit to expedite the cleanup of well-water contamination, the U.S. District Court for the Western District of...

Endangered Species
Federal Protections Defended for Sage Grouse
The Idaho governor and state Legislature lack legal standing to sue over federal protections for the greater sage grouse because the plaintiffs cannot claim any injury until federal agencies get down to the details of blocking projects or...

EPA
House Science Panel Presses McCabe Again on Air Travel
Republicans on the House Science, Space and Technology Committee are reiterating demands for information on the travel habits of the Environmental Protection Agency's top air official, warning they may subpoena the records....

Forests
High Court Refuses Tongass Roadless Case Review
The U.S. Supreme Court refused March 28 to review an appeals court decision affirming tough restrictions on road construction in the Tongass National Forest in Alaska (Alaska v. Organized Vill. of Kake, U.S., No. 15-467, 3/28/16). The Roadless...

Hazardous Waste
Connecticut Companies Settle on Alleged PCB Violations
Two Connecticut companies have settled claims by the Environmental Protection Agency that they violated federal laws on toxic substances in their handling of waste contaminated by polychlorinated biphenyls, the agency announced March...

Hydraulic Fracturing
Quake Outlook Includes Human-Induced Temblors for First Time
The U.S. agency responsible for monitoring earthquakes has for the first time issued a short-term seismic forecast that includes both natural and human-induced risks. ...

International Issues

Canada Builders Get Environmental Details for Cement

The Cement Association of Canada voluntarily registered an environmental product declaration for general-use and portland-limestone cement, Canada's independent industrial standards organization, the CSA Group, said March 23....

Pesticides

EPA Inspector General to Look Into 'Superweeds'

The Environmental Protection Agency's inspector general is launching an evaluation of how effective the agency is at delaying or preventing the spreading of herbicide-resistant weeds....

Pipeline Safety

Industry Seeks Comment Extension for Pipeline Rule

The Transportation Department should extend the 60-day comment period for its recently proposed rule for new pipeline safety requirements, the American Petroleum Institute, American Gas Association and other trade groups wrote in a letter...

Water Pollution

South Dakota Senator Seeks Override of Buffer Bill Veto

South Dakota state Sen. Jim Peterson (D) is leading an effort to override a veto of legislation that would have allowed farmers to reclassify some of their property if they created buffer strips along the state's public waters....

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NEWS

Air Pollution
Eighth Circuit Affirms EPA Haze Plan for Minnesota
The Eighth U.S. Circuit Court of Appeals again has upheld an Environmental Protection Agency-approved plan for reducing haze in the Boundary Waters Canoe Area Wilderness in northeastern Minnesota (Nat'l Conservation Ass'n v....

Air Pollution
Volkswagen Sued for $3.7B in Germany Over Diesel Scandal
Volkswagen AG is being sued for 3.3 billion euros ($3.7 billion) over the cover-up of its polluting diesel engines, its biggest legal challenge in Germany after a wave of lawsuits in the U.S. centered on the scandal....

Air Pollution
Volkswagen U.S. Unit Destroyed Evidence, Ex-Worker Suit Says
Personnel at Volkswagen AG's U.S. unit in Michigan destroyed evidence after the U.S. announced last year that the company had installed illegal devices on hundreds of thousands of vehicles to cheat emissions tests, a former employee...

Air Pollution
White House Threatens Veto of Coal Refuse Emissions Bill
If Congress passes a bill (H.R. 3797) that would alter the way coal refuse power plants are regulated under a pair of Environmental Protection Agency air pollution rules, White House staff would recommend that President Barack Obama veto the...

Chemicals
Geneticist to Testify at Birth Defect Trial Against AES
A geneticist may testify in a trial set to begin April 4 that severe congenital abnormalities in children

were caused by in utero exposure to coal ash waste illegally deposited by AES Corp. near a beach in the Dominican Republic (Pallano v. AES...

Chesapeake Bay
Maryland Phosphorus Rules Based on Soil Testing
Two-thirds of farm fields on Maryland's Lower Eastern Shore will be subject to new regulations intended to limit phosphorus runoff into the Chesapeake Bay, the state Department of Agriculture announced March 14. Of those two-thirds,...

Congress
Senate Action Seen on GMO, Energy Bills Before Break
Senate Republican leaders are working to pass a half-dozen or more items before lawmakers leave for a two-week break March 17, including a long-delayed comprehensive energy bill and a fix for Michigan's drinking water crisis....

Drinking Water
Capitol Hill, Officials Point Fingers Before Flint Hearings
In the days before the House Oversight and Government Reform Committee convenes what may be its highest profile hearings yet on the drinking water crisis in Flint, Mich., entities on all sides sought to put their ducks in a row in the public's...

Drinking Water
PFOA Undetectable in Hoosick Falls Water, Cuomo Says
Filtration of the water supply in Hoosick Falls, N.Y., has brought perfluorooctanoic acid (PFOA) contamination to undetectable levels, New York Gov. Andrew M. Cuomo (D) announced. The state Health Department will continue to test water...

Energy
Companies Demand Fix for Investment Tax Credit Extension
A year-end budget deal extending a prized renewable energy tax break had one major flaw, industry lobbyists say: It mistakenly cut out technologies such as geothermal, fuel cells and smaller wind projects. Now, dozens of energy companies...

Energy Efficiency
DOE Rule Would Increase Commercial Boiler Energy Efficiency
An Energy Department proposed rule would slightly increase the energy efficiency for commercial packaged boilers used to heat businesses, but energy efficiency advocates say the proposed standards aren't strong enough....

Energy
House Passes Two FERC Bills, One Energy Efficiency Bill
The House passed three energy bills by voice vote March 14, including the Fair RATES Act (H.R. 2984), which would amend the Federal Power Act so that proposed rate changes would go into effect after 60 days if the Federal Energy Regulatory Commission...

Enforcement
Canada Finalizes Fines for Offshore Environmental Damage
Canada finalized a series of regulations to allow for fines of up to C$100,000 ($74,000) a day for environmental damage caused by offshore oil and gas activity, to boost "no fault" liability limits for offshore operators to C$1 billion...

Enforcement
Lawyers: Flint Criminal Probe Likely Excludes Water Charges
Everyone from members of the general public to presidential hopefuls have called for those

responsible for the Flint, Mich., drinking water crisis to be identified and held accountable, but few have offered specifics on how to harness the...

EPA
Inspector General Examines Information Security at EPA
The Environmental Protection Agency's Office of Inspector General announced March 14 it would examine how well the agency protects personally identifiable and national security information. ...

Hydraulic Fracturing
$4.2 Million Fracking Verdict Likely to Spark More Suits
A recent jury verdict and $4.2 million award in favor of two Pennsylvania families who alleged fracking operations contaminated their wellwater is likely to trigger the filing of more, similar suits, sources told Bloomberg BNA....

Hydraulic Fracturing
Ohio Water Methane Linked to Coal, Not to Fracking
An independent academic study of methane pollution in water wells in Ohio concluded that naturally occurring coalbed methane, not hydraulic fracturing, is the culprit....

Oil & Gas
Keep Atlantic Coast in Leasing Plan: Oil-Gas Groups
Lobbyists have been hearing that the next stage of a planned five-year offshore leasing program for oil and natural gas exploration may be issued this week, galvanizing industry associations to renew their calls for Atlantic exploration...

Oil & Gas
Offshore Drilling Battleground Virginia to Learn Fate
From the shores of Savannah, Ga., to the Beaufort, N.C. beachfront, coastal communities in conservative Southern states have locked arms in opposition to oil and gas drilling in the Atlantic waters lapping their shores....

Radioactive Waste
Grijalva Presses EPA to Tackle South Florida Waste Leakage
The top Democrat on the House Natural Resources Committee is pushing the Environmental Protection Agency to stop a South Florida power plant from leaking radioactive waste into surrounding waters following a scathing local government report...

Renewable Energy
Solar Debate Heads to Foggy Maine; Panels Are Scarce
Despite long winters, a famously foggy coastline and relatively few solar panels in operation, Maine is emerging as a pivotal U.S. state for determining how consumers will pay for power generated by the sun....

Renewable Energy
Toyota, Partners Begin Hydrogen Supply Chain Project
Toyota Motor Corp. and partners will begin a project to use hydrogen produced from renewable energy to power forklifts and test the feasibility of a low-carbon hydrogen supply chain....

Risk Assessment
EPA Publishes Risk Management Program Revision
The Environmental Protection Agency published a revision (RIN 2050-AG82) to its Risk Management Program in the Federal Register (81 Fed. Reg. 13,638) on March 14. The agency on Feb. 25 floated the proposal, which amends the RMP's accident...

Trade
Canada Shut Out of Solar Case
The governments of Canada and eight of its provinces cannot take part in litigation challenging U.S. countervailing duties on Chinese solar panels, the Court of International Trade ruled March 14 (Changzhou Trina Solar Energy Co. v. United...

Trade
DOE Proposed Import Reporting Requirement Criticized
A proposal to require importers of products or equipment subject to an energy conservation standard to provide the Energy Department with certain electronic data before items may be imported into the U.S. came under fire by the Retail Industry...

Water Pollution
Power Plants Petition for Water Rule Rehearing
The Utility Water Act Group asked the full U.S. Court of Appeals for the Sixth Circuit to rehear a petition on whether a federal appeals court is the appropriate venue to hear challenges to the Clean Water Rule (Ohio v. U.S. Army Corps of Eng'rs...

INTERVIEW

Coal Mining
Joe Pizarchik: From Coal-Country Kid to Top Coal Regulator
The Octagon Room served as the nerve center for British and U.S. military chiefs during World War II and was used to plan the defeat of Nazi Germany. The historic office now belongs to Joe Pizarchik, who says working there gives him some perspective...

REGULATORY AGENDA

Comment Deadlines

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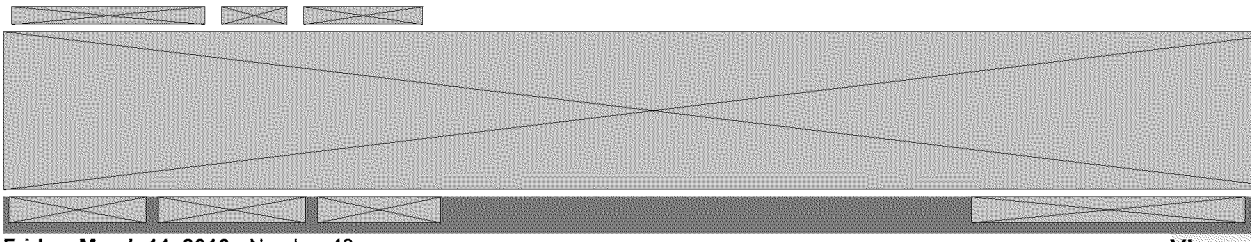

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NEWS

Air Pollution
Briefing in Ozone Litigation to Begin in April
Briefing on the legality of the Environmental Protection Agency's 2015 national ozone standards of 70 parts per billion will begin in April and conclude in September, according to court documents (Murray Energy Corp. v. EPA, D.C. Cir.,...

Air Pollution
Dallas Officials Want Portion of Volkswagen Settlement
Dallas-area transportation officials want to ensure that any settlement with Volkswagen AG over the company's use of illegal technology in its diesel vehicles provides funding to air quality projects in nonattainment areas that saw...

Air Pollution
Eighth Circuit Agrees to Rehear Minnesota Regional Haze Dispute
The Eighth U.S. Circuit Court of Appeals March 10 agreed to rehear a case challenging Minnesota's plan to reduce regional haze (Nat'l Parks Conservation Ass'n v. EPA, 8th Cir., No. 12-2910, 3/10/16)....

Air Pollution
House Rules to Consider Coal Refuse Emissions Bill
The House Rules Committee is set to consider a bill (H.R. 3797) that would alter the way coal refuse power plants are regulated under the Cross-State Air Pollution Rule and the Mercury and Air Toxics Standards. The legislation, introduced by...

Air Pollution
Los Angeles Area Emissions Trading Plan Challenged

Southern California air quality officials violated state law in approving industry-backed changes to a regional emissions trading program, environmental advocates said in a lawsuit filed late March 9 (Cmtys. for a Better Env't v. S....

Air Pollution
Ontario Reports Significant Smog Improvements
Ontario's air quality is significantly better than it was a decade ago, with notable decreases in smog-causing pollutants, the province's Ministry of the Environment and Climate Change said March 9. There were no smog advisories...

Asbestos
Tennessee Lawmakers Approve Asbestos Claim Limits
Plaintiffs filing lawsuits in Tennessee over harm from asbestos exposure may face new hurdles in seeking monetary relief, under a bill approved by the state Legislature March 10....

Biotechnology
India Cuts Monsanto Modified Cotton-Seed Royalty Fees by 70%
India cut royalties for genetically-modified cotton seeds, defying Monsanto Co., which has said such a move would cause it to reevaluate its business in the country, the largest grower of the fiber....

Brownfields
Wisconsin Expands Brownfield Liability Protections
Environmental remediation projects in Wisconsin targeting sediment underlying state waterways will be less costly, complicated and risky under a new law enacted by state lawmakers, an environmental attorney told Bloomberg BNA March 10....

Budget
House Appropriators Discuss Use of Equity Formula
The House Appropriations Committee is considering the use of an equity formula in upcoming appropriations bill language that would direct a portion of funds toward impoverished communities. ...

Chemical Testing
New Software Expected to Help Chemical Safety Reviews
An updated chemical data access and management system the European Chemical Industry Council expects to release within days will make it easier for companies to comply with regulations governing commercial chemicals, cosmetics and other...

Chesapeake Bay
Lawsuit: Problems Seen With Baywide Pollution Limits
The adoption of a cleanup plan for the Chesapeake Bay as a whole shouldn't relieve Maryland of its obligation to develop localized pollution-reduction strategies for other bay tributaries listed as impaired under the Clean Water Act,...

Climate Change
California Updates Plan for Extreme Weather, Sea Level Rise
The California Natural Resources Agency (CNRA) has updated its plans for addressing sea level rise and extreme weather events caused by climate change across a variety of sectors. ...

Climate Change
Canada to Reduce Oil, Gas Methane Emissions Up to 45 Percent
Canada will introduce regulations to reduce methane emissions from the oil and gas sector by 40 percent to 45 percent from 2012 levels by 2025 and harmonize the nation's regime with that of the

U.S., Environment and Climate Change Canada...

Climate Change
Seattle to Electrify Transport, Add Electric Vehicles
Seattle Mayor Ed Murray (D) announced a sweeping series of measures March 10 to reduce the city's carbon footprint by electrifying the city's transportation sector using the Northwest's abundant hydropower....

Climate Regulation
Clean Power Plan Opponents Turn Ginsburg's Words on EPA
Opponents of the Clean Power Plan are turning the words of a U.S. Supreme Court justice widely viewed as sympathetic to the Environmental Protection Agency into another weapon to attack the carbon dioxide standards for power plants....

Climate Regulation
EPA Moving With 'Urgency' on Methane From Existing Wells
The Environmental Protection Agency will solicit better methane emissions data from existing oil and gas wells as it pursues regulations for the industry long sought by environmental advocates, the White House announced March 10....

Climate Regulation
Inhofe: EPA Pressuring States on Power Plan Compliance
The Environmental Protection Agency is using the prospect of truncated deadlines to force states to continue compliance efforts for the Clean Power Plan even though the rule has been stayed by the U.S. Supreme Court, Sen. James Inhofe (R-Okla.)...

Coal Mining
Arch Coal to Abandon Montana Mine
Citing financial difficulty, Arch Coal said March 10 that it will no longer seek a mining permit for the Otter Creek coal reserves in southeastern Montana....

Coal Mining
Cantwell Urges Interior on Banning Self-Bonding in Mining
Intensifying her efforts to probe self-bonding in the mining industry, Sen. Maria Cantwell (D-Wash.) asked the Interior Department March 10 to consider prohibiting the practice....

Congress
Republicans See Window to Nullify Late-Term Obama Rules
A confluence of political factors could open the door for Republicans to successfully nullify Obama administration environmental regulations issued in the final months of his presidency, Senate lawmakers told Bloomberg BNA. ...

Drinking Water
EPA's McCarthy: Broader Justice, Water Debate Needed
While the Environmental Protection Agency is placing much of its efforts on helping Flint, Mich., residents deal with the ramifications of the city's lead-contaminated drinking water "disaster," the incident also has raised...

Drinking Water
Governors Call for Federal PFOA Contamination Guidance
The Environmental Protection Agency should establish national guidance addressing perfluorooctanoic acid (PFOA) contamination to help states assess the safety of their drinking water, three Democratic governors said in a March 10 letter....

Drinking Water
Ohio Seeks Faster Notification of Lead-in-Water Issues
Public utilities in Ohio should notify their customers within two days, not 30, if sampling shows lead contamination in the water supply, the state's environmental regulator is recommending. ...

Drinking Water
Unsafe Lead Levels in Water at 30 Newark Schools
Investigators are still searching for the source of high levels of lead found in the water at 30 schools in Newark, N.J., the New Jersey Department of Environmental Protection told Bloomberg BNA March 10....

Energy
Cornyn: McConnell May File For Cloture on Flint Bill
Senate Majority Leader Mitch McConnell (R-Ky.) may move to bring an energy bill and legislation that would provide aid to Flint, Mich., using procedural moves to sidestep senators who have been blocking the measure by using unanimous consent,...

Energy Efficiency
Government Seeks Companies' Help on Energy Goal
The nation's chief sustainability officer has called on companies such as Johnson Controls and AECOM to help meet the federal government's energy savings goal before the Obama administration ends. ...

Energy
U.S. Nuclear Industry Largely Safer After Fukushima Accident
The U.S. nuclear industry is safer after making fleet-wide improvements over the past five years since the meltdown of three nuclear reactors at the Fukushima Dai-Ichi nuclear plant in Japan, industry, regulators and safety advocates say....

Enforcement
EPA Limited in Next Steps on Flint Water Crisis,
After a slow start, the Environmental Protection Agency is doing what it can to make sure municipal and state officials resolve the Flint, Mich., lead-tainted drinking water crisis, and further actions such as penalties or a federal takeover...

General Policy
Green Policies Boost Industry, Not Pollution Havens: OECD
Stringent environmental policies can boost industrial innovation without diverting trade to less regulated markets, the Organization for Economic Cooperation and Development said....

Hazardous Waste
China Soil Pollution Cleanup Plans Face New Delays
Deliberation on China's long-sought Soil Pollution Prevention and Treatment Law to address severe contamination of the country's farmlands and areas around industrial sites will likely run into 2017, a top government official...

Hazmat Transport
Senate FAA Bill Bans Lithium Battery Shipments on Planes
The bulk transport of lithium ion batteries on passenger planes would be banned under Senate legislation reauthorizing the Federal Aviation Administration. The bill, unveiled March 9 by the Senate Commerce Committee, would also prohibit...

Hydraulic Fracturing
Fracking Court Issue: What Does FLPMA Authorize?

In the legal fight over regulation of hydraulic fracturing on federal lands, states have grounded their argument in a basic position: federal land management and leasing laws are about apportioning various uses of the surface and mineral...

International Climate
Obama, Trudeau Pledge Quick Paris Deal Implementation
The U.S. and Canada agreed to speed up implementation of the Paris climate agreement and lead by example in completing in 2016 plans that will outline how they will put themselves on a low-emissions development trajectory between now and 2050....

Oil & Gas
New York Gas Line Delayed Pending Environmental Permits
The opening of Williams Partners LP's proposed $925-million Constitution natural gas pipeline will be delayed to the second half of 2017 as the developer awaits a permit to clear trees in New York and other environmental approvals....

Pesticides
Canada Moves to End Pesticide Testing on Dogs
Canada will soon end a requirement that pesticides used on crops be tested on dogs for toxicity and safety, bringing rules in line with those in the U.S., Canada's health department said March 9....

Renewable Energy
Florida Voters to Decide Tax Break for Solar Panels
A proposal to exempt renewable energy equipment installed on commercial and industrial buildings from property tax assessments will go before Florida voters in August. ...

Risk Assessment
EPA: Lower Doses of Explosive May Harm Health
A military explosive found in the soil and groundwater of some hazardous waste sites can harm the nervous system at lower doses than previously estimated, according to draft assessment the Environmental Protection Agency released March...

Storage Tanks
EPA Inspector General to Vet Tribal Storage Tank Programs
The Environmental Protection Agency's Office of Inspector General is launching a review into the agency's underground storage tank programs in Indian country, according to an OIG memo released March 9....

Superfund
Superfund Claim Tossed in Hazmat Personal Injury Case
The Superfund law can't be used to establish a negligence claim over trichloroethylene that leached from a ball bearing factory into the groundwater of a nearby residential community, a federal court has ruled (Kirk v. Schaeffler Grp....

Toxic Substances
Canada Cites Impact of Foreign Sources on Mercury
Mercury remains a risk to Canadian ecosystems and human health, Environment and Climate Change Canada said March 8, in releasing the country's first comprehensive evaluation of scientific research on mercury. ...

Worker Safety
Federal Workers Given Zika Travel Warning
The top personnel management official in the U.S. issued a warning to all federal employees at risk

of contracting the Zika virus currently spreading in the Americas....

INTERVIEW

Risk Assessment
EPA Science Adviser Discusses Public Health Role
Bloomberg BNA's Steven Gibb interviewed EPA Science Adviser and Deputy Assistant Administrator for the Office of Research and Development Thomas Burke* March 4 on the role he envisions for public health in environmental decision-making....

REGULATORY AGENDA

Comment Deadlines

MARCH 10 FEDERAL REGISTER

MARCH 11 FEDERAL REGISTER

Daily Environment Report

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To: Burneson, Eric[Burneson.Eric@epa.gov]
From: BNA Highlights
Sent: Thur 3/10/2016 2:11:40 AM
Subject: Mar. 10 -- BNA, Inc. Daily Environment Report

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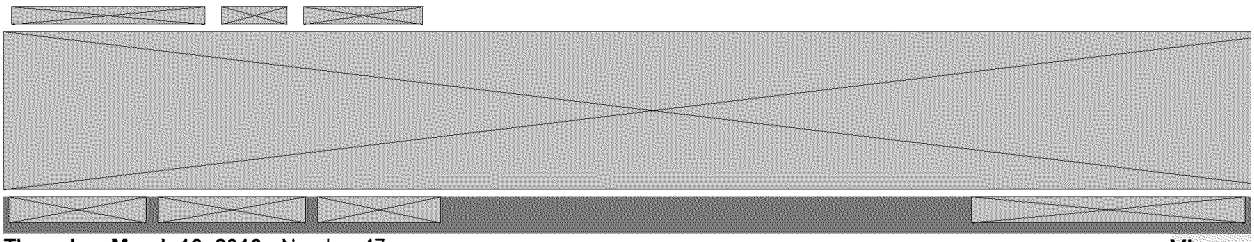

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Archive

NEWS

Agriculture
Bayer to Take on Monsanto in Brazil With GMO Soybean Seeds
Bayer AG and BASF SE are planning to introduce new genetically modified soybean seeds this year in Brazil as the companies seek to break Monsanto Co.'s decade-long dominance in the South American country, the world's second-largest...

Air Pollution
EPA: Dismiss Reporting Exemption Rule Challenge
The Environmental Protection Agency hit back at a challenge to the agency's Reporting Exemption Rule, saying the regulation complies with statutory latitude on reporting exemptions and arguing the Waterkeeper Alliance and other petitioners...

Air Pollution
Volkswagen's Top U.S. Official Leaves Company
Volkswagen Group of America announced that its top U.S. official has left the company, effective immediately....

Brownfields
New York Agency Revises Proposed Brownfields Criteria
The definition of "underutilized" would be broadened in screening properties to qualify for tangible property tax credits set by the New York Brownfields law, under a March 9 state Department of Environmental Conservation proposal....

Chemicals
California Proposes New Cancer Risk Factor for PCE Emissions
California's Office of Environmental Health Hazard Assessment has proposed to tighten the toxicity value that air quality regulators use to calculate cancer risk from exposure to perchloroethylene

emissions. ...

Climate Change
Paris Provides Signal to Transform Global Economy
The Paris Agreement has become a powerful pivot point in efforts to transform the global economy into a more sustainable model, participants said at the Climate Leadership Conference in Seattle....

Climate Regulation
Senators Offer Companion Bill on Race Car Rule
Four Republican senators have introduced legislation to clarify that race cars are exempt from Clean Air Act prohibitions on tampering with pollution controls. Sens. Richard Burr (N.C.), Shelley Moore Capito (W.Va.), Dean Heller (Nev.)...

Congress
Senate Must Consider Supreme Court Pick: Environmentalists
Seventeen environmental organizations have asked the Senate to conduct hearings and hold a "timely vote" on President Barack Obama's forthcoming pick to the U.S. Supreme Court....

Drinking Water
Flint Water Response Garners Local Leaders' Support
Flint, Mich., is amassing support from city and local leaders nationwide for revamping its drinking water infrastructure following the crisis that potentially exposed more than 8,000 children to high lead concentrations....

Drinking Water
Stabenow: 'Conceptual Agreement' Reached on Flint Deal
Lawmakers have reached a "conceptual agreement" over how to pay for federal aid for the drinking water crisis in Flint, Mich., Sen. Debbie Stabenow (D-Mich.) told Bloomberg BNA March 9, a development that could allow a stalled legislative...

Drinking Water
Town to Get Filtration for PFOA-Tainted Water
A plastics company has agreed to install a carbon filtration system for the drinking water supply in Petersburgh, N.Y., to address contamination with perfluorooctanoic acid (PFOA), state agencies announced March 9. The finding of contamination...

Drinking Water
Wisconsin Groups Demand Solution to Water Crisis
For the second time in 18 months, a coalition of Wisconsin environmental groups called on the Environmental Protection Agency to take emergency action to provide clean water to residents of a rural county east of Green Bay threatened by groundwater...

Energy
Southern Co. Tops Utility Lobbying Spenders in 2015
Southern Co.'s projects to build a "clean coal" facility and an advanced nuclear plant have been both over-budget and behind schedule. That hasn't been good news for the company, but it has been for one group: Washington...

Enforcement
Pesticide Enforcement Targets Data Submissions
The Environmental Protection Agency increasingly will focus on whether pesticide registrants have submitted the toxicity, efficacy and other data they must provide the agency, a senior EPA enforcement official said March 9....

Environmental Justice
Lawmaker Presses Civil Rights Group on Coal Ash
The Environmental Protection Agency's 2015 coal ash rule (RIN-2050-AE81) unfairly threatens minority, low-income and other communities by exempting municipal solid waste (MSW) facilities from disposal requirements, Rep. Hank...

Environmental Reviews
New York Environmental Review Rules Expected
New York state, after more than three years of work, is expected to issue regulations shortly to amend its environmental review process under the State Environmental Quality Review Act (SEQR)....

EPA
States Spar Over EPA Relationships at Senate Hearing
State environmental regulators clashed March 9 before the Senate's environmental panel about how well the federal Environmental Protection Agency works with them in developing regulations. ...

General Policy
Chinese Premier: 'Protect the Environment While Pursuing Development'
China's Premier Li Keqiang declared a "war on pollution" at the opening of the country's annual National People's Congress meetings in March two years ago and President Xi Jinping pledged an "iron hand" against...

Groundwater
Calif. Water Agency's Public Trust Powers Before Court
California's water rights agency has the authority under the public trust doctrine to regulate groundwater withdrawals that affect trust resources and such regulation is not precluded by the state's groundwater management law,...

Hydraulic Fracturing
Sierra Club Suit Seeks Fracking Quake Monitoring
Oil and gas companies in Oklahoma must be ordered to substantially reduce underground injection of fracking waste liquids to help stem a 300-fold increase in earthquakes since 2009, according to a recent lawsuit filed by the Sierra Club in...

International Climate
Alberta to Form Oil-Sands Advisory for Carbon Policy
Alberta will rely on an oil-sands industry advisory group to help the government implement its carbon emissions reductions policy and ensure that industry is consulted, Premier Rachel Notley said March 8. The group, whose members will be...

Oil & Gas
Oklahoma Launches Second Wastewater Injection Cutback
Oklahoma's energy and gas regulator is giving oil and gas well operators in the central portion of the state until May 28 to become fully compliant with a plan to cut saltwater disposal volumes to 40 percent below the levels of 2014. ...

Radioactive Waste
Spending Bill to Allow Interim Nuclear Waste Storage
Senate appropriators again will include language that would allow the Energy Department to develop interim nuclear waste storage sites as part of their fiscal year 2017 energy and water spending bill, Sen. Lamar Alexander (R-Tenn.), chairman...

Renewable Energy
U.S. Solar Growth Will More Than Double in 2016, Study Finds
U.S. solar installations are expected to more than double this year, driven by falling equipment prices and federal tax credits....

Sustainability
Green-Mining Baron Sets Sights on Europe's Biggest Steel Mill
From Andrew Carnegie and J.P. Morgan to Wilbur Ross and Lakshmi Mittal, rolling up steel mills has created some of the world's best-known fortunes....

Water Infrastructure
Water District in SEC Hot Water for 'Enron Accounting'
California's largest agricultural water district misled investors who bought $77 million of municipal bonds by using accounting tricks to mask how much its finances were being squeezed by a drought gripping the state, the U.S. Securities...

Water Resources
Request for Corps at Odds With Rhetoric: Waterways Council
A "huge disconnect" exists between the Obama administration's push for investment in infrastructure and its budget request for the U.S. Army Corps of Engineers, the president of the Waterways Council Inc. said at a March 9 news...

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Comment Deadlines

MARCH 10 FEDERAL REGISTER

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To: Jack Adams[JAdams@calgoncarbon.com]; Burneson, Eric[Burneson.Eric@epa.gov]
Cc: Greene, Ashley[Greene.Ashley@epa.gov]; Strong, Jamie[Strong.Jamie@epa.gov]
From: Lape, Jeff
Sent: Wed 4/13/2016 12:32:39 AM
Subject: RE: PFOA Highlights

Jack,
Thanks... Eric and his team are the right folks. If you do have a meeting, pls include Jamie Strong (and me) on the invite.
Best,
Jeff

-----Original Message-----
From: Jack Adams [mailto:JAdams@calgoncarbon.com]
Sent: Tuesday, April 12, 2016 11:20 AM
To: Burneson, Eric <Burneson.Eric@epa.gov>; Lape, Jeff <lape.jeff@epa.gov>
Subject: PFOA Highlights

Eric - Jeff

Greetings !

The Strategic Marketing Director for our largest business unit - Matt Adomaitis - is in DC with me this week.

He has been very involved in the Hoosick Falls (and other) PFOA situation.

Would you have a few minutes (together or separately) to meet with Matt so he can provide an update of our activities there ?

Wednesday afternoon or any time on Thursday work best for us.

Let me know know what works best for you.

Best Regards

Jack Adams
Director | Government Affairs
Calgon Carbon Corporation
412-551-6389

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To: Burneson, Eric[Burneson.Eric@epa.gov]; Lape, Jeff[lape.jeff@epa.gov]
From: Jack Adams
Sent: Tue 4/12/2016 3:19:50 PM
Subject: PFOA Highlights

Eric - Jeff

Greetings !

The Strategic Marketing Director for our largest business unit - Matt Adomaitis - is in DC with me this week.

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Jack Adams
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To: Burneson, Eric[Burneson.Eric@epa.gov]
From: Pillsbury, Sarah
Sent: Fri 3/18/2016 5:53:48 PM
Subject: RE: Invitation to a call at 11:45

Thanks. See you soon.

Sarah Pillsbury, Administrator

Drinking Water & Groundwater Bureau, NHDES

29 Hazen Drive, PO Box 95, Concord, NH 03302

Tel: (603) 271-1168 | Fax: (603) 271-5171

NHDES Be Well Informed Private Well App - An interactive web application for private well owners to interpret water quality test results and identify appropriate water treatment options

From: Burneson, Eric [mailto:Burneson.Eric@epa.gov]
Sent: Friday, March 18, 2016 1:22 PM
To: Pillsbury, Sarah
Cc: Freise, Clark; Burack, Thomas; Grevatt, Peter; Lovely, William
Subject: RE: Invitation to a call at 11:45

Sarah:

Following up on your question about what constitutes a "short term" for purposes of the provisional health advisory. The Health advisory states:

"The relevant period of exposure for the Health Advisory is a short-term exposure. This time period is consistent with the toxicity data used for PFOA and PFOS, both of which rely upon subchronic data." See https://www.epa.gov/sites/production/files/2015-09/documents/pfoa-pfos-provisional.pdf.

However the fact sheet we prepared in 2014 clarifies that short term means "weeks to months"

see https://www.epa.gov/sites/production/files/2015-09/documents/peer-review-of-health-effects-documents-for-pfoa-and-pfos-factsheet-february-2014.pdf.

I hope this information is helpful and look forward to speaking with you next week at the ASDWA Conference.

Eric Burneson, P.E.

Director of Standards and Risk Management

Office of Ground Water and Drinking Water

US Environmental Protection Agency

Phone: 202-564-5250

Fax: 202 564 3760

From: Pillsbury, Sarah [mailto:Sarah.Pillsbury@des.nh.gov]
Sent: Friday, March 18, 2016 11:23 AM
To: Burneson, Eric <Burneson.Eric@epa.gov>
Cc: Freise, Clark <Clark.Freise@des.nh.gov>; Burack, Thomas <Thomas.Burack@des.nh.gov>
Subject: RE: Invitation to a call at 11:45

Yes. Thank you. I am copying my Commissioner and Assistant Commissioner who may also join the call..

Sarah Pillsbury, Administrator

Drinking Water & Groundwater Bureau, NHDES

29 Hazen Drive, PO Box 95, Concord, NH 03302

Tel: (603) 271-1168 | Fax: (603) 271-5171

NHDES Be Well Informed Private Well App - An interactive web application for private well owners to interpret water quality test results and identify appropriate water treatment options

From: Burneson, Eric [mailto:Burneson.Eric@epa.gov]
Sent: Friday, March 18, 2016 11:18 AM
To: Pillsbury, Sarah
Subject: Invitation to a call at 11:45

Sarah:

I just sent an invitation to a call about the PFOA monitoring results for 11:45. Can you participate at this time? The call in number is 1 866 299 3188 code 202 564 5250

Eric Burneson, P.E.

Director of Standards and Risk Management

Office of Ground Water and Drinking Water

US Environmental Protection Agency

Phone: 202-564-5250

Fax: 202 564 3760

From: Pillsbury, Sarah
Location: 866-299-3188 code 202-564-5250
Importance: Normal
Subject: Accepted: Monitoring Results PFOA
Start Date/Time: Fri 3/18/2016 3:45:00 PM
End Date/Time: Fri 3/18/2016 4:15:00 PM

;

To: Burneson, Eric[Burneson.Eric@epa.gov]
Cc: Freise, Clark[Clark.Freise@des.nh.gov]; Burack, Thomas[Thomas.Burack@des.nh.gov]
From: Pillsbury, Sarah
Sent: Fri 3/18/2016 3:22:53 PM
Subject: RE: Invitation to a call at 11:45

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Sarah Pillsbury, Administrator

Drinking Water & Groundwater Bureau, NHDES

29 Hazen Drive, PO Box 95, Concord, NH 03302

Tel: (603) 271-1168 | Fax: (603) 271-5171

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Sent: Friday, March 18, 2016 11:18 AM
To: Pillsbury, Sarah
Subject: Invitation to a call at 11:45

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Eric Burneson, P.E.

Director of Standards and Risk Management

Office of Ground Water and Drinking Water

US Environmental Protection Agency

Phone: 202-564-5250

Fax: 202 564 3760

To: sara.pillsbury@des.nh.gov[sara.pillsbury@des.nh.gov]
From: postmaster@nh.gov
Sent: Fri 3/18/2016 3:14:16 PM
Subject: Undeliverable: Canceled: Monitoring Results PFOA
Canceled: Monitoring Results PFOA

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 h=From:To:Date:Subject:Message-ID:Content-Type:MIME-Version;
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 15.01.0434.020; Fri, 18 Mar 2016 15:14:13 +0000
From: "Burneson, Eric" <Burneson.Eric@epa.gov>
To: "sara.pillsbury@des.nh.gov" <sara.pillsbury@des.nh.gov>
Subject: Canceled: Monitoring Results PFOA
Thread-Topic: Monitoring Results PFOA
Thread-Index: AdGBKAbCB3a2i4EWS/2RjXI/o6v7CwAAMueA
Importance: high
X-Priority: 1
Date: Fri, 18 Mar 2016 15:14:12 +0000
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Content-Language: en-US
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authentication-results: des.nh.gov; dkim=none (message not signed)
 header.d=none;des.nh.gov; dmarc=none action=none header.from=epa.gov;
x-ms-exchange-messagesentrepresentingtype: 1
x-originating-ip: [2620:117:5010:ca::d020]
x-ms-office365-filtering-correlation-id: 1ff45fa7-8de6-4a1e-7d1e-08d34f3ff6c5
x-microsoft-exchange-diagnostics:
1;DM2PR09MB0207;5:5dryHowgURYjCEr0K9+F769oER7Cm1TKVRJeyRs2lRv8pVg2T8M4TLVKOlrEovpppYCy7uRi
x-microsoft-antispam: UriScan:;BCL:0;PCL:0;RULEID:;SRVR:DM2PR09MB0207;
x-microsoft-antispam-prvs:
<DM2PR09MB02076CEA63DD33254F45AA48F18C0@DM2PR09MB0207.namprd09.prod.outlook.com>
x-exchange-antispam-report-test: UriScan:;
x-exchange-antispam-report-cfa-test:
BCL:0;PCL:0;RULEID:(601004)(2401047)(8121501046)(5005006)(10201501046)(3002001);SRVR:DM2PR
x-forefront-prvs: 088552DE73
x-forefront-antispam-report:
SFV:NSPM;SFS:(10019020)(6009001)(10400500002)(19580395003)(92566002)(189998001)(5004730100
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spamdiagnosticmetadata: NSPM
Content-Type: multipart/alternative;
          boundary="_000_DM2PR09MB0206743C2D3C57C29FD707D1F18C0DM2PR09MB0206namp_"
MIME-Version: 1.0
X-MS-Exchange-CrossTenant-originalarrivaltime: 18 Mar 2016 15:14:12.8856
 (UTC)
X-MS-Exchange-CrossTenant-fromentityheader: Hosted
X-MS-Exchange-CrossTenant-id: 88b378b3-6748-4867-acf9-76aacbeca6a7
X-MS-Exchange-Transport-CrossTenantHeadersStamped: DM2PR09MB0207
X-NAI-Spam-Flag: NO
X-NAI-Spam-Level:
X-NAI-Spam-Threshold: 4.5
X-NAI-Spam-Score: 0.9
X-NAI-Spam-Rules: 6 Rules triggered
          STOCK_SYM_PTRN=0.5, IMPORTANCE_HIGH=0.2, PHONE_866=0.2,
EDT_SA_DN_PASS=0,
          EDT_SA_RB_PASS=0, RV5614=0
X-NAI-Spam-Version: 2.3.0.9418 : core <5614> : inlines <4532> : streams
 <1604955> : uri <2169106>
Return-Path: Burneson.Eric@epa.gov

From: Burneson, Eric
Location: 866-299-3188 code 202-564-5250
Importance: High
Subject: Canceled: Monitoring Results PFOA
Start Date/Time: Fri 3/18/2016 3:45:00 PM
End Date/Time: Fri 3/18/2016 4:15:00 PM

;

To: sara.pillsbury@des.nh.gov[sara.pillsbury@des.nh.gov]
From: postmaster@nh.gov
Sent: Fri 3/18/2016 3:09:48 PM
Subject: Undeliverable: Monitoring Results PFOA
Monitoring Results PFOA

Delivery has failed to these recipients or groups:

sara.pillsbury@des.nh.gov (sara.pillsbury@des.nh.gov)
The e-mail address you entered couldn't be found. Please check the recipient's e-mail address and try to resend the message. If the problem continues, please contact your helpdesk.

Diagnostic information for administrators:

Generating server: nh.gov

sara.pillsbury@des.nh.gov
#550 5.1.1 RESOLVER.ADR.RecipNotFound; not found ##

Original message headers:

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Received: from hznsmtp1inf.oit.nh.gov (10.12.10.25) by
 HZNGRANMAIL1.granite.nhroot.int (10.6.13.23) with Microsoft SMTP Server (TLS)
 id 14.3.266.1; Fri, 18 Mar 2016 11:09:48 -0400
Received: from gcc01-CY1-obe.outbound.protection.outlook.com
 (mail-cy1gcc01on0104.outbound.protection.outlook.com [23.103.200.104]) by
 hznsmtp1inf.oit.nh.gov with smtp      (TLS:
 TLSv1/SSLv3,256bits,ECDHE-RSA-AES256-SHA)        id
 3f7d_6f63_f4aef470_d30a_4efc_8f9e_de7fba05910f; Fri, 18 Mar 2016 11:09:47
 -0400
DKIM-Signature: v=1; a=rsa-sha256; c=relaxed/relaxed; d=usepa.onmicrosoft.com;
 s=selector1-epa-gov;
 h=From:To:Date:Subject:Message-ID:Content-Type:MIME-Version;
 bh=P1vBMkKU0W2CuDJrP8zdDukpt8eHCDM09jvD7v4IGBw=;

b=ReabQc1xseHYXZgBihWglBUPElga8040EcbS4TuROPiC34DRacdAWDekHTRRJlZ9QlHVoIw+vA/GLaVSnOHlH28O
Received: from DM2PR09MB0206.namprd09.prod.outlook.com (10.160.92.140) by
 DM2PR09MB0271.namprd09.prod.outlook.com (10.160.96.140) with Microsoft SMTP
 Server (TLS) id 15.1.434.16; Fri, 18 Mar 2016 15:09:46 +0000
Received: from DM2PR09MB0206.namprd09.prod.outlook.com ([10.160.92.140]) by
 DM2PR09MB0206.namprd09.prod.outlook.com ([10.160.92.140]) with mapi id
 15.01.0434.020; Fri, 18 Mar 2016 15:09:45 +0000
From: "Burneson, Eric" <Burneson.Eric@epa.gov>
To: "Lovely, William" <Lovely.William@epa.gov>, "Grevatt, Peter"
          <Grevatt.Peter@epa.gov>, "sara.pillsbury@des.nh.gov"
          <sara.pillsbury@des.nh.gov>
CC: "Greene, Ashley" <Greene.Ashley@epa.gov>
Subject: Monitoring Results PFOA
Thread-Topic: Monitoring Results PFOA
Thread-Index: AdGBKAbCB3a2i4EWS/2RjXI/o6v7CwAABUVA
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Date: Fri, 18 Mar 2016 15:09:45 +0000
Message-ID:
<DM2PR09MB0206FBE8BCCAC6C96573823EF18C0@DM2PR09MB0206.namprd09.prod.outlook.com>
Accept-Language: en-US
Content-Language: en-US
X-MS-Has-Attach:
X-MS-TNEF-Correlator:
authentication-results: epa.gov; dkim=none (message not signed)
 header.d=none;epa.gov; dmarc=none action=none header.from=epa.gov;
x-ms-exchange-messagesentrepresentingtype: 1
x-originating-ip: [2620:117:5010:ca::d020]
x-ms-office365-filtering-correlation-id: 9e79051d-78d8-4916-e8e0-08d34f3f577e
x-microsoft-exchange-diagnostics:
1;DM2PR09MB0271;5:DIFJQF58myRG3gsooOckFHrQAi7ePJWZGCVD1m5XUrGHvdS9Eh2iIQ+4UoV3e3+1dAis17Y5
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<DM2PR09MB0271B81DC671F721D925ED22F18C0@DM2PR09MB0271.namprd09.prod.outlook.com>
x-exchange-antispam-report-test: UriScan:;
x-exchange-antispam-report-cfa-test:
BCL:0;PCL:0;RULEID:(601004)(2401047)(8121501046)(5005006)(10201501046)(3002001);SRVR:DM2PR
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SFV:NSPM;SFS:(10019020)(6009001)(3480700003)(5004730100002)(189998001)(5002640100001)(5003
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MIME-Version: 1.0
X-MS-Exchange-CrossTenant-originalarrivaltime: 18 Mar 2016 15:09:45.8059
 (UTC)
X-MS-Exchange-CrossTenant-fromentityheader: Hosted
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X-MS-Exchange-Transport-CrossTenantHeadersStamped: DM2PR09MB0271
X-NAI-Spam-Flag: NO
X-NAI-Spam-Level:
X-NAI-Spam-Threshold: 4.5
X-NAI-Spam-Score: 0.7
X-NAI-Spam-Rules: 5 Rules triggered
          STOCK_SYM_PTRN=0.5, PHONE_866=0.2, EDT_SA_DN_PASS=0,
EDT_SA_RB_PASS=0,
          RV5614=0
X-NAI-Spam-Version: 2.3.0.9418 : core <5614> : inlines <4532> : streams
 <1604954> : uri <2169101>
Return-Path: Burneson.Eric@epa.gov

From: Burneson, Eric
Location: 866-299-3188 code 202-564-5250
Importance: Normal
Subject: Monitoring Results PFOA
Start Date/Time: Fri 3/18/2016 3:45:00 PM
End Date/Time: Fri 3/18/2016 4:15:00 PM

;

To: sara.pillsbury@des.nh.gov[sara.pillsbury@des.nh.gov]
From: postmaster@nh.gov
Sent: Fri 3/18/2016 3:09:10 PM
Subject: Undeliverable: Monitoring Results PFOA
Monitoring Results PFOA

Delivery has failed to these recipients or groups:

sara.pillsbury@des.nh.gov (sara.pillsbury@des.nh.gov)
The e-mail address you entered couldn't be found. Please check the recipient's e-mail address and try to resend the message. If the problem continues, please contact your helpdesk.

Diagnostic information for administrators:

Generating server: nh.gov

sara.pillsbury@des.nh.gov
#550 5.1.1 RESOLVER.ADR.RecipNotFound; not found ##

Original message headers:

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 HZNGRANMAIL1.granite.nhroot.int (10.6.13.23) with Microsoft SMTP Server (TLS)
 id 14.3.266.1; Fri, 18 Mar 2016 11:09:10 -0400
Received: from gcc01-CY1-obe.outbound.protection.outlook.com
 (mail-cy1gcc01on0122.outbound.protection.outlook.com [23.103.200.122]) by
 hznsmtplinf.oit.nh.gov with smtp      (TLS:
 TLSv1/SSLv3,256bits,ECDHE-RSA-AES256-SHA)        id
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 -0400
DKIM-Signature: v=1; a=rsa-sha256; c=relaxed/relaxed; d=usepa.onmicrosoft.com;
 s=selector1-epa-gov;
 h=From:To:Date:Subject:Message-ID:Content-Type:MIME-Version;
 bh=aaPAeWEweAOYj+tnWuEB/3WT9zX6HcJafDRXw9Fe3x0=;

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Received: from DM2PR09MB0206.namprd09.prod.outlook.com (10.160.92.140) by
 DM2PR09MB0271.namprd09.prod.outlook.com (10.160.96.140) with Microsoft SMTP
 Server (TLS) id 15.1.434.16; Fri, 18 Mar 2016 15:09:07 +0000
Received: from DM2PR09MB0206.namprd09.prod.outlook.com ([10.160.92.140]) by
 DM2PR09MB0206.namprd09.prod.outlook.com ([10.160.92.140]) with mapi id
 15.01.0434.020; Fri, 18 Mar 2016 15:09:07 +0000
From: "Burneson, Eric" <Burneson.Eric@epa.gov>
To: "Lovely, William" <Lovely.William@epa.gov>, "Grevatt, Peter"
          <Grevatt.Peter@epa.gov>, "sara.pillsbury@des.nh.gov"
          <sara.pillsbury@des.nh.gov>
CC: "Greene, Ashley" <Greene.Ashley@epa.gov>
Subject: Monitoring Results PFOA
Thread-Topic: Monitoring Results PFOA
Thread-Index: AdGBKAbCB3a2i4EWS/2RjXI/o6v7CwAABUVA
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Date: Fri, 18 Mar 2016 15:09:07 +0000
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Content-Language: en-US
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X-MS-TNEF-Correlator:
authentication-results: epa.gov; dkim=none (message not signed)
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x-ms-exchange-messagesentrepresentingtype: 1
x-originating-ip: [2620:117:5010:ca::d020]
x-ms-office365-filtering-correlation-id: 0c48da52-bd9a-4163-422a-08d34f3f407c
x-microsoft-exchange-diagnostics:
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BCL:0;PCL:0;RULEID:(601004)(2401047)(8121501046)(5005006)(10201501046)(3002001);SRVR:DM2PR
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 (UTC)
X-MS-Exchange-CrossTenant-fromentityheader: Hosted
X-MS-Exchange-CrossTenant-id: 88b378b3-6748-4867-acf9-76aacbeca6a7
X-MS-Exchange-Transport-CrossTenantHeadersStamped: DM2PR09MB0271
X-NAI-Spam-Flag: NO
X-NAI-Spam-Level:
X-NAI-Spam-Threshold: 4.5
X-NAI-Spam-Score: 0.7
X-NAI-Spam-Rules: 5 Rules triggered
          STOCK_SYM_PTRN=0.5, PHONE_866=0.2, EDT_SA_DN_PASS=0,
EDT_SA_RB_PASS=0,
          RV5614=0
X-NAI-Spam-Version: 2.3.0.9418 : core <5614> : inlines <4532> : streams
 <1604954> : uri <2169101>
Return-Path: Burneson.Eric@epa.gov

From: Burneson, Eric
Location: 866-299-3188; 202-565-5350
Importance: Normal
Subject: Monitoring Results PFOA
Start Date/Time: Fri 3/18/2016 3:45:00 PM
End Date/Time: Fri 3/18/2016 4:15:00 PM

;

To: Burneson, Eric[Burneson.Eric@epa.gov]
From: BNA Highlights
Sent: Thur 3/3/2016 2:04:44 AM
Subject: Mar. 3 -- BNA, Inc. Daily Environment Report

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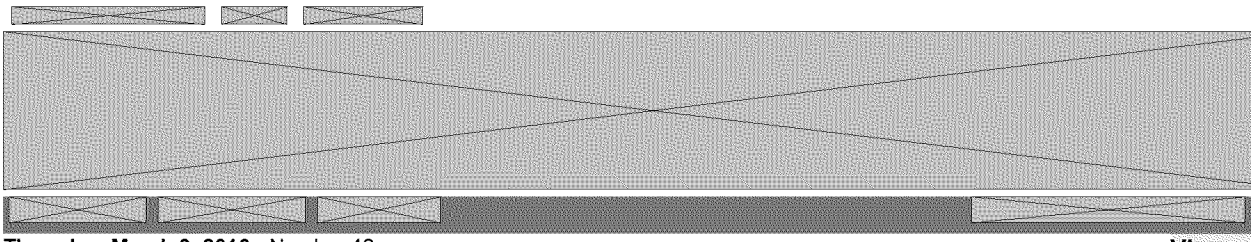

Thursday, March 3, 2016 · Number 42

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NEWS

Air Pollution
Court Sets May 11 Arguments on Los Angeles Ozone Plan
Oral arguments over the Environmental Protection Agency's approval of a plan to reduce ozone levels in the Los Angeles area will be heard on May 11 in San Francisco (Physicians for Soc. Responsibility-Los Angeles v. EPA, 9th. Cir., No....

Air Pollution
EPA: Conditions for Stay of Mercury Rule Not Met
A U.S. Supreme Court stay of the Environmental Protection Agency's Mercury and Air Toxics Standards for power plants would be unwarranted, the federal government argued in a March 2 court filing (Michigan v. EPA, U.S., No. 15A886, 3/2/16)....

Air Pollution
European Parliament 'Dieselgate' Inquiry Committee Starts Up
A European Parliament inquiry committee set up to investigate an alleged regulatory breakdown that allowed the VW "dieselgate" scandal to happen started a one-year investigation March 2 with the appointment of a chairwoman and...

Air Pollution
Veto Threatened for Bill to Block Brick Emissions Limits
President Barack Obama's administration "strongly opposes" and would veto legislation (H.R. 4557) that would delay compliance obligations for the brick industry under an Environmental Protection Agency toxic air emissions...

Chemicals
House, Senate Close in on Toxics Bill Minus PCB Off-Ramp
The Senate Environment and Public Works Committee is reviewing the House's proposed changes

to an update of the Toxic Substances Control Act and is close to resolving differences between the two chambers' bills, Sen. Jim Inhofe...

Chemicals
TSCA Bars Tort Claims for PCBs, Monsanto Successor Says
The existing Toxic Substances Control Act and a House bill that would update the law bar toxic tort claims involving polychlorinated biphenyls (PCBs), according to newly circulated legal documents filed in a case involving a corporate successor...

Chemicals
Uncommon EPA Notice Extends PMN Review Time
The Environmental Protection Agency needs more time to review information about a new solvent that chemical manufacturers would like to make in or import into the U.S., according to a March 2 Federal Register notice (81 Fed. Reg. 10,858). ...

Climate Change
Conference Speakers Call for Broad Carbon Pricing
Carbon pricing is the key to unlocking investment in clean energy technology as the world works to control climate change, several speakers told an environment and business conference in Vancouver March 2....

Climate Change
No Climate Policy Change Needed, EU Assessment Says
The European Union does not for the time being have to introduce any additional climate policies as a consequence of the United Nations Paris Agreement that was struck by nearly 200 nations in the French capital in December 2015, according to...

Climate Policy
Carbon Taxes Called Simpler, Broader Than Emissions Trading
Carbon taxes would be simpler to administer, capture wider swaths of the economy and, at least in the U.S., potentially garner conservative support, panelists said at a forum sponsored by Resources for the Future....

Climate Regulation
Flexibility Will Save Clean Power Plan, McCarthy Says
Flexibility built into the Clean Power Plan should inoculate the rule from challenges brought by states and industry groups, Environmental Protection Agency Administrator Gina McCarthy said, predicting the carbon dioxide standards for...

Congress
EPW Leaders, Advocates Spar Over Coal Ash Bill
Debate over legislation designed to strengthen state management of coal ash took center stage at a Senate Environment and Public Works Committee hearing on March 2, as Sen. Jim Inhofe (R-Okla.) expressed support, while ranking member Barbara...

Drinking Water
Stop Stalling, PFOA-Exposed West Virginians Tell EPA
West Virginians whose public water supplies are contaminated with perfluorooctanoic acid (PFOA) want action from state and federal regulators....

Energy
Kentucky Senate Passes Bill Allowing Nuclear
Kentucky's long-standing moratorium on nuclear power plants would be lifted under legislation (S.B. 89) passed March 1 by the state Senate. The measure would require power facilities to have

a nuclear waste storage plan rather than...

Energy
Trudeau: 'We Need Both Pipelines and Wind Turbines'
Canada pledged new investments of C$75 million ($55.7 million) to help municipalities address climate change infrastructure challenges, and C$50 million ($37.2 million) for green building projects, Prime Minister Justin Trudeau announced...

Hazmat Transport
Freight Train Spills Ethanol in N.Y. Derailment
A 16-car train derailment in upstate New York has resulted in a hazardous materials spill of ethanol, prompting the evacuation of nearby residences, Gov. Andrew M. Cuomo (D) announced March 2. Two of the cars on a Norfolk Southern Corp. freight...

Oil Spills
British Columbia Proposes Oil Spill Prevention Measure
British Columbia announced plans to amend its Environmental Management Act to address spill prevention and cleanup at sea and on land, among the province's conditions for approval of heavy oil pipelines through the jurisdiction....

Pesticides
European Commission: Glyphosate Reauthorization Not Decided
The European Commission said March 2 that it has not yet decided on a reauthorization in the European Union of the widely used herbicide glyphosate, despite circulating a draft regulation at the end of February under which glyphosate would...

Renewable Energy
Arizona Utility Seeks Lower Net-Metering Rates
In a case with potential impact on the solar energy industry in Arizona, Tucson-based UNS Electric Inc. is arguing before the Arizona Corporation Commission for lower net-metering rates for its rooftop solar customers....

Renewable Energy
China Urged to Avoid Excess Carbon Quotas in National Trading
Operators of two local pilot carbon-trading programs in China are urging designers of a national trading scheme to avoid allocating more emissions quotas than the market needs in order to avoid some of the supply imbalances the European Union...

Storage Tanks
N.C. Court Upholds Reduced Damages From Leak
A trial court didn't err in significantly lowering a jury award to the owners of a property contaminated by a leaking underground storage tank, the North Carolina Court of Appeals ruled (BSK Enter. Inc. v. Beroth Oil Co., N.C. Ct. App., No....

Superfund
Failure to Allege 'Disposal' Warrants Superfund Dismissal
Allegations that paper companies used hazardous substances that were later found at a Superfund site aren't enough to proceed with hazardous waste disposal claims, a federal court said (Garrett Day LLC v. Int'l Paper Co., S.D. Ohio...

Water Pollution
11th Circuit May Follow Precedent in Choosing Venue
The U.S. Court of Appeals for the Eleventh Circuit may follow its own legal precedent in resolving whether a district court or an appeals court should hear challenges to the water jurisdictional rule,

especially when it has ruled on a similar...

Water Pollution
Appeal Filed Against EPA's Malibu Creek TMDL
The owner of a wastewater reclamation facility appealed a district court ruling that the Environmental Protection Agency had the authority to issue in 2003 and revise in 2013 a total maximum daily load that set seasonal numeric targets for...

Water Pollution
EPA Must Act Fast on Sewage Dumped in Great Lakes: Senators
Three senators are urging the Environmental Protection Agency to quickly implement congressionally mandated requirements for municipal treatment facilities to notify the public of sewage dumped into the Great Lakes....

Water Pollution
EPA Ordered to Release List of Water Intake Rule Records
A U.S. appeals court ordered three federal agencies to release a list of previously undisclosed documents used to support their determination that a rule regulating industrial cooling water intake structures wouldn't jeopardize endangered...

Water Pollution
Latent Disease Claims Survive N.C. Repose Law Appeal
North Carolina's statute of repose—which extinguishes personal injury suits brought more than 10 years after the last act or omission of a defendant—doesn't apply to latent disease claims, the Fourth Circuit ruled...

Water Pollution
Northrop Wins Dismissal of N.Y. Water Contamination Case
A New York water provider's lawsuit against Northrop Grumman Corp. to recoup the cost of remediating ground water contamination is time-barred, the Eastern District of New York ruled Feb. 29 (Bethpage Water Dist. v. Northrop Grumman...

Water Resources
Senators Frustrated With Corps' Budget Request
Senate appropriators expressed frustration March 2 over the administration's refusal to seek enough funds for the U.S. Army Corps of Engineers and to spend the money already collected to maintain and replace locks in inland waterways....

Wetlands
Industry: Corps' Jurisdictional Determination Reviewable
A Clean Water Act jurisdictional determination is final agency action subject to judicial review, trade associations said in urging the U.S. Supreme Court to affirm a court of appeals ruling (U.S. Army Corps of Eng'rs v. Hawkes Co., U.S.,...

Deadline Extended for Comments on GMO Study
The Department of Agriculture is extending the amount of time the public can comment on its proposal to launch a study re-examining the way the agency regulates genetically modified crops. The USDA announced March 2 that it is adding an additional...

Lawmakers Head to Flint to Meet With Residents
Twenty-five members of Congress will go to Flint, Mich., March 4 to hear from residents on the local drinking water crisis. The trip will be led by Rep. Dan Kildee (D-Mich.) and will include Democrats such as House Democratic Leader Nancy Pelosi...

REGULATORY AGENDA

Comment Deadlines

MARCH 2 FEDERAL REGISTER

MARCH 3 FEDERAL REGISTER

Daily Environment Report

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To: Burneson, Eric[Burneson.Eric@epa.gov]
From: BNA Highlights
Sent: Tue 3/1/2016 3:14:31 AM
Subject: Mar. 1 -- BNA, Inc. Daily Environment Report

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Tuesday, March 1, 2016 · Number 40

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NEWS

Air Pollution
Comment Period Open on Sulfur Designations
The Environmental Protection Agency will accept comments through March 31 on its proposal to designate 12 areas as being in nonattainment of the 2012 national ambient air quality standards for sulfur dioxide. The agency on Feb. 17 released...

Air Pollution
Environmental Groups Seek to Defend Refinery Rule
A coalition of 11 environmental and public health groups want to help the Environmental Protection Agency defend its 2015 revised air pollution standards for refineries from industry lawsuits (Am. Fuel & Petrochemical Mfrs. v. EPA,...

Air Pollution
EPA Says Ozone Designations Consistent With Past
The process for designating areas under the 2015 national ozone standards will largely be consistent with the Environmental Protection Agency's process under past standards, the agency said in a guidance document....

Air Pollution
U.K. Could Face Tougher EU Pollution Rules if It Quits Bloc
If Britain departs from the European Union, the action could free up officials in Brussels to tighten air-pollution regulations everywhere in the region—including in the U.K.—because of a twist in the free-trade laws likely...

Air Pollution
VW Official Says Diesel Talks Could Still Take Months
Almost six months after Volkswagen AG's emissions cheating became public, Chief Executive

Officer Matthias Mueller said talks with U.S. regulators on fixing the rigged engines may take a few more weeks or months....

Chemicals
Buffalo Childhood Lead Poisoning Prevention Funded
A Buffalo community organization will receive $346,835 for hazard intervention and remediation programs to address lead in the city's homes, New York Attorney General Eric T. Schneiderman (D) announced Feb. 28....

Chemicals
Casey's Office Vows EPA Outreach Over Asbestos Site
Staff for Sen. Bob Casey (D-Pa.) plan to reach out to the Environmental Protection Agency to press constituent concerns over ongoing health risks posed by asbestos near former Superfund sites in Ambler, Pa., Casey's spokesman told Bloomberg...

Chemicals
Immunotoxicity From PFOA, PFOS Analyzed in Report
The National Toxicology Program will release for public comment by June 7 a draft monograph analyzing the immunotoxicity effects resulting from exposure to perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS). A peer review...

Chemicals
States to Focus on Flame Retardants, Chemical Disclosure
In 2016, states with Republican- and Democratic-controlled legislatures are expected to consider bills to phase out flame retardants, increase chemical disclosure and reduce children's exposure to lead, cadmium and other chemicals....

Chesapeake Bay
Supreme Court Declines to Hear Appeal to Chesapeake Bay Plan
The U.S. Supreme Court Feb. 29 declined to hear a petition by the American Farm Bureau Federation challenging the legality of a cleanup plan to restore the Chesapeake Bay (Am. Farm Bureau Fed'n v. EPA, U.S., 15-00599, 2/29/16)....

Climate Regulation
Report Warns of Increasing Petrochemical Emissions
Forty-four petrochemical facilities proposed or permitted in 2015 could increase greenhouse gas pollution by 86 million tons a year, according to a Feb. 29 report from the Environmental Integrity Project. Those emissions would be the equivalent...

Climate Regulation
Washington State Regulators to Rewrite Carbon Rule
Washington state business welcomed the announcement by the Department of Ecology that it is withdrawing for rewrite a draft rule that would require entities emitting at least 100,000 metric tons of greenhouse gases annually to cut carbon...

Coal Mining
Republicans Seek Interior Justification for Coal Lease Halt
The Interior Department must send its legal justification to Congress for a recent decision to halt most new coal leases and to reassess the environmental impacts of its coal program, several Republicans in Congress told Interior Secretary...

Drinking Water
Elevated PFOA Found in 29 Percent of Hoosick Falls Wells
The chemical perfluorooctanoic acid (PFOA) has been found at levels exceeding a federal advisory

level of 100 parts per trillion in 29 percent of the public and private wells recently tested by New York in Hoosick Falls, N.Y., the state Department...

Endangered Species
Idaho Details Objections to Sage Grouse Rules
Idaho's governor and Legislature filed a brief detailing their arguments that new federal protections for greater sage grouse birds violated federal laws on environmental analysis and land management (Otter v. Jewell, D.D.C., No....

Energy
Canada, China Agree to Cooperate on Clean Energy
Canada and China have agreed to cooperate on clean technology initiatives, Natural Resources Canada said. The Joint Declaration on Canada-China Clean Technology Cooperation calls for the countries to share best practices in setting clean...

Energy
Chinese Coal Use Drops Amid Nuclear, Renewables Rise
China consumed about 4 percent less coal last year while renewable energy from solar power grew almost three-quarters, underscoring President Xi Jinping's drive to reduce pollution and cut industrial overcapacity....

Energy
House Passes Advanced Nuclear, Other Energy Bills
The House voted Feb. 29 to pass legislation that would require the Energy Department to prioritize the development of advanced nuclear reactors....

Energy
India Doubles Coal Tax, Risks Higher Electricity Costs
To help reduce pollution, India doubled the clean-energy tax on coal, risking higher electricity-generation costs at a time when the government is seeking to revive ailing power distributors....

Enforcement
British Columbia Mines Penalties Could Rise to $1M
Proposed amendments to British Columbia's Mines Act introduced Feb. 25 would strengthen the province's regulatory oversight of the mining industry and give the provincial Ministry of Energy and Mines additional compliance and...

Enforcement
Lockheed to Pay $5 Million Over RCRA Violations
Lockheed Martin Corp. and two subsidiaries have agreed to pay $5 million in penalties over false claims submitted to the Energy Department for payments connected to contracted work at a uranium enrichment facility in Paducah, Ky., the Justice...

Enforcement
Ontario Gold Mine Fined for Illegally Dumping Effluent
An Ontario gold mine was fined C$350,000 ($259,000) for illegally dumping effluent containing cyanide and metals into a fish-bearing stream. The Ontario Court of Justice levied a C$175,000 ($129,500) fine against Wesdome Gold Mines Ltd....

Environmental Justice
Bill Would Incentivize Environmental Justice Efforts
Rep. John Lewis (D-Ga.) introduced a bill that would encourage universities and others to address environmental issues addressing low-income and minority communities. The Environmental Justice Act of 2016 (H.R. 4645) would establish...

Environmental Reviews
Voters Reject Mall That Skirted California Review
A regional mall that skirted state environmental review through the ballot initiative process has been rejected by voters in Carlsbad, Calif. "While we had hoped for a different outcome, we are proud of our effort, our plan, the integrity...

EPA
McCarthy Touts EPA's Role as Public Health Agency
The ongoing water contamination crisis in Flint, Mich., and the effects of a warming climate underscore the Environmental Protection Agency's role as a public health organization, Administrator Gina McCarthy said....

International Climate
Ontario Unveils Cap-and-Trade Details for 2017
Companies with more than 25,000 metric tons of greenhouse gas emissions annually will be required to take part in Ontario's emissions cap-and-trade program when it starts next year, the government announced....

Legislation
Cornyn: Holds Remain on Energy Bill, Flint Package
Multiple holds remain on a legislative deal that would bring stalled energy legislation and aid for Flint, Mich., to the Senate floor, Sen. John Cornyn (R-Texas) told reporters Feb. 29....

Pesticides
Colorado: Two Marijuana Growers Used Unsafe Pesticides
Colorado has issued public health and safety advisories after identifying potentially unsafe pesticide residues on marijuana products produced by two growers in the state....

Pipeline Safety
Cuomo Seeks Halt of Algonquin Gas Line for Safety Review
New York Gov. Andrew Cuomo urged federal energy regulators to suspend construction on Spectra Energy Corp.'s Algonquin natural gas pipeline until an analysis of the safety risks is completed....

Pipeline Safety
House Pipeline Safety Draft Differs From Senate Bill
A House subcommittee will consider March 1 draft pipeline safety reauthorization legislation that somewhat overlaps with a Senate bill expected to pass this week....

Radioactive Waste
Lawmakers Seek GAO Study of DOE's Ability to Complete Yucca
Reps. Fred Upton (R-Mich.) and John Shimkus (R-Ill.) asked the Government Accountability Office to study whether the Energy Department is able to complete the review of the Yucca Mountain nuclear waste license application. ...

Renewable Energy
Markey to Propose Extending Credit to Spur Offshore Wind
Sen. Edward J. Markey (D-Mass.) plans to introduce legislation "soon" to extend a federal tax credit that will spur wind-farm development in coastal waters....

Renewable Energy
Turbine Developer: U.S. Offshore Wind Must Think Big
Offshore wind companies need to install 500 megawatts annually for the next 15 years for the

technology to make economic sense in the U.S., according to Dong Energy A/S, the world's biggest developer of sea-based turbines....

Solid Waste
Cuomo Announces Plan to Widen N.Y. Solid Waste Controls
State controls would be extended over previously unregulated solid waste streams and rules for existing landfills and other waste facilities would be tightened, under a set of proposed regulations announced Feb. 29 by New York Gov. Andrew...

Superfund
Colorado Governor Seeks Superfund Cleanup of Gold King Mine
Colorado Gov. John Hickenlooper (D) asked the Environmental Protection Agency to expedite Superfund cleanup for the mining district near Silverton, Colo., where EPA investigators triggered the spill of some 3 million gallons of mining...

Trade
Canada-EU Amendments Address Environmental Regulation
Environment Minister Catherine McKenna Feb. 29 called the proposed Canada-European Union trade deal a "perfect example" of the complementary nature of environmental and economic policies. ...

Water Pollution
Inhofe Demands EPA Response Over Water Rule Promotion
Sen. James Inhofe (R-Okla.) wants to know why the Environmental Protection Agency still hasn't reported that it violated federal appropriations laws by promoting its Clean Water Act jurisdiction rule through social media....

Water Pollution
Rehearing Sought in Venue Ruling for Water Rule Lawsuits
The full U.S. Court of Appeals for the Sixth Circuit has been asked to consider a ruling by a three-judge panel of the court on whether it is the appropriate venue to hear challenges to the a rule clarifying the scope of Clean Water Act jurisdiction...

BNA INSIGHTS

An In-Depth Look Into the Role of Environmental Liability Transfers During Corporate Divestitures
The pace of change in market conditions today is moving at unprecedented speeds. New industrial technologies and cleaner energy solutions are forcing well-established corporations and traditional energy providers to pivot to accommodate...

SPECIAL REPORT

Chemicals
Making In Vitro Chemical Data Useful for Decisions
Scientists working for federal agencies, chemical manufacturers and other institutions are working to combine information from in vitro chemical bioactivity tests and computer models with exposure estimates to provide useful information...

INTERVIEW

Climate Change
Ted Lieu Sees Prosecution of Fossil Fuel Companies Ahead Over Climate Denial
Rep. Ted Lieu (D-Calif.) just arrived in the House in 2015 but has already been outspoken in urging the federal government to investigate whether fossil fuel companies intentionally hid the truth about the risks of climate change. In a Feb....

REGULATORY AGENDA

Water Pollution
Rehearing Sought in Venue Ruling for Water Rule Lawsuits
The full U.S. Court of Appeals for the Sixth Circuit has been asked to consider a ruling by a three-judge panel of the court on whether it is the appropriate venue to hear challenges to the a rule clarifying the scope of Clean Water Act jurisdiction...

Comment Deadlines

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MARCH 1 FEDERAL REGISTER

Daily Environment Report

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Afternoon Briefing - Your Preview of Today's News

The following news provides a snapshot of what Bloomberg BNA is working on today. Read the full version of all the stories in the final issue, published each night.

Methane From Oil, Gas Sector Higher Than Thought: EPA Chief

Posted February 24, 2016, 4:21 P.M. ET

By Nushin Huq

Methane emissions from the oil and gas sector are much higher than the Environmental Protection Agency previously thought, Administrator Gina McCarthy said today in Houston.

The most recent greenhouse gas inventory revealed higher levels from all parts of the energy sector, from storage to pipelines, McCarthy said. While emissions have not necessarily gone up, she said, the new report better captures data.

Voluntary agreements on emission reductions have not been as productive with the oil and gas industry compared to other industries, such as utilities and mining, McCarthy said at the IHS CERAWeek conference.

EPA will finalize its methane rules to regulate emissions from new oil and gas wells this spring, McCarthy said. When asked if this means the EPA will regulate natural gas more, she said, “We already are.”

20 States Ask U.S. Supreme Court to Stay EPA Mercury Rule

Posted February 24, 2016, 12:45 P.M. ET

By Patrick Ambrosio

A coalition of 20 states, led by Michigan, asked the U.S. Supreme Court to stay implementation of a multibillion-dollar rule limiting mercury emissions from power plants.

The states, in a petition filed today, said a stay is appropriate because the Supreme Court already decided in June 2015 that the Environmental Protection Agency violated the Clean Air Act when it failed to consider cost in making a finding that it was “appropriate and necessary” to regulate those emissions. That EPA finding triggered a requirement that the agency move ahead with a rulemaking process that resulted in promulgation of the Mercury and Air Toxics Standards.

The Supreme Court, in its 2015 opinion authored by Justice Antonin Scalia, remanded the rule back to the D.C. Circuit, which opted in December to leave the regulation in place while the EPA works to address the Supreme Court's holding.

“This is an unmistakable example of agency overreach,” the states said today. “An executive agency strayed far beyond the limited authority the legislative branch gave it, and then, when this court corrected the agency's error, the EPA requested on remand that the unauthorized, unlawful regulation should be left in place to have the force of law.”

The states argued that a stay is even more warranted for the MATS rule than it was for the Clean Power Plan, the Obama administration's carbon dioxide standards for power plants. The Supreme Court, in a 5-4 order issued prior to Scalia's death, granted a stay of the Clean Power Plan pending review by the D.C. Circuit and the Supreme Court.

Flint Legislation ‘Hotlined,' Inhofe Says

Posted February 24, 2016, 2:53 P.M. ET

By Ari Natter

Legislation that would provide funding to help Flint, Mich., deal with its lead-tainted drinking water and in the process allow stalled Senate energy legislation (S. 2012) to proceed has been “hotlined,” Sen. Jim Inhofe (R-Okla.) told Bloomberg BNA today.

The term refers to when the Senate majority leader and minority leader agree to bring a measure to the floor by unanimous consent, and use a special phone line to lawmakers' offices to see if there are any objections.

Inhofe said he expects to know if there are any objections later this afternoon. “I feel pretty optimistic that we’ll go ahead and do it,” Inhofe said. But he added that once the measure gets to the Senate floor: “It might not pass. It will take 60 votes.”

‘About 30’ Energy Bill Amendments Possible as Part of Flint Deal: Cornyn

Posted February 24, 2016, 1:30 P.M. ET

By Ari Natter

A deal in the works to move back to stalled Senate energy legislation (S. 2012) includes a request to hold a voice vote on “about 30 amendments,” Sen. John Cornyn (R-Texas) told reporters today.

In addition, “a handful of other items” would receive roll call votes, he said without elaborating.

Sen. John Thune (R-S.D.) said: “It sounds like they are close to reaching an agreement.”

Power Emissions to Decline Even Without Clean Power Plan: Analyst

Posted February 24, 2016, 4:13 P.M. ET

By Dean Scott

Carbon pollution from the U.S. power sector is likely to continue to fall sharply through 2025 with or without Obama administration carbon pollution limits recently put on hold by the U.S. Supreme Court, an analyst said today.

U.S. carbon dioxide emissions from the sector would total nearly 2.08 billion metric tons in 2025 if the high court ultimately decides to overturn the power sector limits, according to John Larsen, director of the Rhodium Group and a former official with the Energy Department.

But Larsen characterized that as a relatively modest impact compared to if the carbon limits were untouched by the court and fully implemented by the Environmental Protection Agency. That scenario would bring the sector's total down to about 1.76 billion tons in 2025.

The impact of leaving the EPA limits in place is so small—just over 300 million metric tons—largely because of other positive trends that have little to do with the regulations, Larsen said, speaking at a forum held by the New America

think tank and the Center for Climate and Energy Solutions.

Two big factors, Larsen said, are declining costs of solar and wind energy, which he said will only continue to fall, and the five year-extension of solar investment and wind production tax credits included in the omnibus funding bill enacted in December.

Companies Face Class Action Over PFOA Contamination in N.Y.

Posted February 24, 2016, 2:35 P.M. ET

By Amena H. Saiyid

Residents of Hoosick Falls, N.Y., filed a class action lawsuit against Saint-Gobain Performance Plastics Corp. and Honeywell International Inc., holding the two companies responsible for contaminating the aquifer underlying the village with the perfluorooctanoic acid (PFOA).

The lawsuit also accuses the two companies of being responsible for the presence of PFOA in the municipal water supply, private wells and local aquifer, “creating a public health crisis for residents and hurting property values.”

The Environmental Protection Agency on Jan. 28 recommended that people in the Town of Hoosick and Village of Hoosick Falls who have private wells with PFOA concentration of 100 parts per trillion or more use bottled water for cooking and drinking. The state of New York also in January declared the village to be a Superfund site, thereby activating emergency funds to address the problem.

The New York-based Weitz & Luxenberg P.C. filed the lawsuit on behalf of the village residents in U.S. District Court for the Northern District of New York.

Insurer Owes No Coverage to Condo Manager for Mold Cleanup

Posted February 24, 2016, 3:45 P.M. ET

By Peter Hayes

An insurer owes no coverage for a condominium management company's mold abatement because it had no legal obligation to conduct the cleanup, the Eighth Circuit ruled today.

Without a settled claim or a settlement or judgment arising from a suit, the management company wasn't “legally obligated to pay by reason of liability imposed by law,” the court said. As a result, there is no coverage under Missouri law.

The management company voluntarily conducted abatement of mold caused by vinyl wallpaper that trapped moisture. Consent forms signed by each owner didn't include an admission of liability, but didn't include a release or settlement of potential claims the property owner might have against the management company.

Judge Lavenski R. Smith wrote the opinion. Judges Diana E. Murphy and Michael Melloy were also on the panel.

Dowd & Bennett in St. Louis represented Busch Properties.

Foland & Wickens in Kansas City represented National Union.

Seven Chemicals May Cause Human Cancer, WHO Agency Says

Posted February 24, 2016, 11:48 A.M. ET

By Pat Rizzuto

Hydrazine, a rocket propellent; 2-mercaptobenzothiazole, a chemical used in rubber; and dimethylformamide, an acrylic-fiber chemical have all been classified as probably carcinogenic to humans, a World Health Organization agency announced today.

A majority of the scientists that WHO's International Agency for Research on Cancer (IARC) asked to review seven

chemicals also classified tetrabromobisphenol A, a widely used flame retardant, as probably carcinogenic to humans.

The panel classified 1-bromopropane, a vapor degreaser; 3-chloro-2- methylpropene, an industrial chemical and seed fumigant; and dimethyl-p- toluidine, which is used to make dental materials, bone cements, industrial glues and artificial fingernail preparations, as possibly carcinogenic to humans.

Albemarle Corp., the Dow Chemical Corp. and the Chevron Corp. are among the companies that make some of these chemicals, some or all of which are produced in volumes of one million pounds or more per year.

IARC's conclusions about the hazards chemicals pose can affect how chemicals are labeled and the safety information that must be provided to workers. Its conclusions on these seven chemicals, reached during a Feb. 2-9 meeting of scientists, were published in the Lancet.

Justices Debate Whether State Subsidy Enters FERC Turf

Posted February 24, 2016, 1:58 P.M. ET

By Rebecca Kern

U.S. Supreme Court justices today questioned whether a Maryland subsidy encouraging new electricity generation in the state crossed into the Federal Energy Regulatory Commission's authority over the wholesale energy markets.

Justice Sonia Sotomayor appeared to side with the federal government in saying that the case represented conflict preemption, with Maryland's subsidy program intruding into FERC's mechanism for setting wholesale electricity rates.

Maryland ordered utilities to enter into 20-year contracts with CPV Maryland LLC to build a new generation facility, and bid into PJM Interconnection LLC capacity markets, which FERC oversees.

Scott Strauss argued on behalf of the Maryland Public Service Commission that the contracts that bid and cleared into PJM's markets did not distort market pricing. Strauss also said states have the authority to regulate and build new power plants and facilities as they see fit, which Maryland did in this case.

Hughes v. Talen Energy Marketing LLC was heard before eight justices, and was among the first cases argued at the Supreme Court since the death of Justice Antonin Scalia. A decision in the case is expected as early as April, analysts say.

Inhofe Urges Congress to Block EPA on Fuel Standard

Posted February 24, 2016, 12:38 P.M. ET

By Renee Schoof

Congress should reconsider the renewable fuel standard by 2022 to prevent the Environmental Protection Agency from having full control over it, Sen. James Inhofe (R-Okla.) said today.

Targeted amounts of total renewable fuels that must be blended into the fuel supply, including advanced and conventional, are set by law through 2022. After that, under current law, the EPA would decide how to administer the program.

The EPA has mismanaged the renewable fuel standard and should not be allowed to run the program on its own, Inhofe, chairman of the Senate Environment and Public Works Committee, said during an oversight hearing.

Inhofe said he wants the renewable fuel standard repealed, but did not propose any legislation to eliminate or change it during the hearing. Republicans and Democrats from farm states defend the renewable fuel standard.

Lawmakers Soften Criticism of Water Project Work

Posted February 24, 2016, 3:05 P.M. ET

By Alan Kovski

The U.S. Army Corps of Engineers is getting better at reviewing potential water infrastructure projects and reporting them to Congress but still needs improvement, lawmakers told corps leaders today.

The corps also needs to do a better job of explaining to local officials the complexities of how to propose a project that can make it through daunting bureaucratic complexities, members of the House Transportation and Infrastructure Committee said.

Neither point was disputed by the two witnesses, Jo-Ellen Darcy, assistant secretary of the Army for civil works, and Lt. Gen. Thomas Bostick, chief of engineers.

The 2016 annual project report, delivered by the corps to Congress in the first week of February, listed enough new or revised projects ready for congressional approval that both Republicans and Democrats appear mollified, in contrast to the bipartisan anger they expressed after the first such report was delivered in 2015 under authority of a 2014 law.

A Water Resources Development Act of 2016, if written and passed as promised by lawmakers, is expected to approve many of the projects in the latest annual report, although funding will not necessarily follow in all cases.

Study: Microplastics in Water Despite Wastewater Treatment

Posted February 24, 2016, 12:12 P.M. ET

By Amena H. Saiyid

Thousands and perhaps millions of tiny shards of plastic that are less than 5 millimeters wide are entering urban rivers in Illinois every day by eluding capture at wastewater treatment plants, according to a new Loyola University study.

Released today by the American Geophysical Union, the findings by assistant professor Timothy Hoellein and his team show that wastewater treatment plants were a source of microplastics in 80 percent of the 10 urban rivers examined in Illinois, “regardless of the size of the river or the size and type of wastewater treatment plant.”

Hoellein estimates that between 15,000 and 4.5 million pieces of microplastics enter those rivers every day.

He told the AGU that the findings are significant because rivers are a source of drinking water for many communities and also a habitat for wildlife. Fish and other aquatic creatures eat the tiny pieces of plastic in rivers, which then make their way up the food chain–possibly ending up in people's dinner, according to Hoellein.

Some of those microplastics—microbeads in personal care products—are to be phased out starting July 1, 2017, under a new federal law.

U.S. Prevails in WTO Dispute Against India's Solar Program

Posted February 24, 2016, 12:35 P.M. ET

By Bryce Baschuk

A World Trade Organization dispute panel today agreed with U.S. trade officials that India's national solar program violated international trade rules and unfairly discriminated against imported solar cells and modules.

Specifically, the WTO ruled that the domestic content requirements of India's Jawaharlal Nehru National Solar Mission program are discriminatory because they require solar power developers to use Indian-manufactured cells and modules, according to its decision.

The WTO urged India to bring its measures into compliance with the national treatment obligations in Article 2.1 of the WTO Agreement on Trade-Related Investment Measures and Article III:4 of the General Agreement on Tariffs and Trade 1994.

“The United States strongly supports the rapid deployment of solar energy around the world—including in India,” said U.S. Trade Representative Michael Froman in a news release. “But discriminatory policies in the clean energy space in fact undermine our efforts to promote clean energy by requiring the use of more expensive and less efficient equipment, raising the cost of generating clean energy and making it more difficult for clean energy sources to be

competitive."

Investors Seek Vote on Climate Proposal at Exxon Mobil

Posted February 24, 2016, 11:23 A.M. ET

By Andrea Vittorio

Investors representing more than $1 billion in Exxon Mobil shares are urging the Securities and Exchange Commission to let a shareholder resolution on climate change onto the company's proxy.

The shareholder proposal, filed by New York state's comptroller and four other investors, reflects their ongoing concern over how a changing climate could impact Exxon Mobil's business, especially if policy makers live up to the goals of a recent international deal to fight it.

"As investors, we need to know how ExxonMobil's bottom line will be impacted by the global effort to reduce emissions and what the company plans to do about it," New York State Comptroller Thomas DiNapoli said in a statement today.

Exxon Mobil, which is simultaneously facing an inquiry from the state's attorney general into whether it misled the public and investors on climate change, has asked the SEC to block the proposal from making it to a vote at its annual meeting in the spring. The SEC declined to comment.

Conservative Groups Urge Congress to Oppose Carbon Tax

Posted February 24, 2016, 11:14 A.M. ET

By Anthony Adragna

Twenty conservative organizations, including FreedomWorks, the American Energy Alliance and Competitive Enterprise Institute, released a letter today urging Congress against pursuing any sort of carbon tax.

"Our nation's citizens expect – and deserve – their duly elected lawmakers to institute policies that move our economy forward and allow all Americans an equal opportunity to succeed," the groups wrote to House Majority Whip Steve Scalise (R-La.). "A carbon tax would fail resoundingly on both of these fronts."

Scalise introduced a resolution (H. Con. Res. 89) last year expressing the sense of Congress that a carbon tax would harm the U.S. economy. There has been little momentum for any sort of carbon tax among lawmakers, though Democratic Sens. Sheldon Whitehouse (R.I.) and Brian Schatz (Hawaii) have introduced legislation (S. 1548) in hopes of building support for one.

SAFE PIPES Act Would Cost $50 Million from 2017-2021: CBO

Posted February 23, 2016, 5:36 P.M. ET

By Rachel Leven

The Senate pipeline safety reauthorization bill would cost $50 million over the course of five years, the Congressional Budget Office said in its cost estimate released today.

The SAFE PIPES Act (S. 2276) would require $525 million in appropriations from 2017 to 2021 for the Pipeline and Hazardous Materials Safety Administration's pipeline safety functions, it said. The bill would generate $462 million in fees from pipeline owners and $17 million in revenue from new underground natural gas storage facility assessments.

The Senate Commerce, Science and Transportation Committee passed the SAFE PIPES Act on Dec. 9. The bill would lower maximum funding levels for pipeline safety programs and aim to allow PHMSA time to address leftover mandates from its 2012 pipeline safety authorization law (Pub. L. No. 112–90), which lapsed Sept. 30.

European Commission to Propose Glyphosate Reapproval

Posted February 24, 2016, 2:10 P.M. ET

By Stephen Gardner

The European Commission will propose to renew until 2031 the authorization in the European Union of the widely used pesticide glyphosate, according to a draft regulation obtained by Bloomberg BNA.

The proposed renewal is controversial because glyphosate has been labeled “probably carcinogenic” by the World Health Organization, though the European Food Safety Authority (EFSA) in November judged that the substance was “unlikely to pose a carcinogenic hazard to humans.”

According to the draft regulation to reauthorize glyphosate, the current authorization for the substance will expire June 30, and EFSA's assessment that it should not be classified as carcinogenic provides the basis for its reapproval as safe to use in the EU.

A committee of EU member state representatives will discuss and possibly vote on the commission's draft regulation on glyphosate at a March 7-8 meeting.

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NEWS

Air Pollution
Air Standards Set for Los Angeles Area Near Gas Leak
California state and local agencies released criteria late Feb. 16 for determining when emissions from Southern California Gas Co.'s underground natural gas storage field near Los Angeles no longer affect air quality in nearby neighborhoods....

Air Pollution
Canada Proposes Tighter Ambient Sulfur Dioxide Standard
Canada's health agency has proposed tightening the country's national ambient air quality objectives for sulfur dioxide, last updated more than two decades ago and more than four times higher than allowable levels across the border...

Air Pollution
EPA Proposes 12 Nonattainment Areas for Sulfur Standards
The Environmental Protection Agency will propose designating 12 areas in eight states as not achieving 2010 federal air quality standards for sulfur dioxide....

Chemicals
EPA to Release RDX Draft Analysis at May Meeting
The Environmental Protection Agency's Integrated Risk Information System Program will release a draft assessment for a chemical used in explosives at an upcoming May meeting, it announced Feb. 17. The program will release the draft...

Climate Change
High Temperatures on Pace to Break 2015 Record
Here we go again. For the surface of planet Earth, 2015 was the hottest year on record by a stunning margin. But already, 2016 is on track to beat it....

Corporate Responsibility
Apple's $1.5B Green Bond Seen Spurring Corporate Issuers
Apple Inc.'s new green bond may trigger other companies to follow suit, with more corporate issuance this year for investments in renewable energy and climate change projects, analysts examining the niche market said....

Drinking Water
EPA Didn't Enforce Flint Drinking Water Violations: CRS
The Environmental Protection Agency did not use its authority under the Safe Drinking Water Act to enforce violations against the public water utility in Flint, Mich., despite being aware of the elevated lead levels in the city's tap water,...

Energy
Ostendorff to Leave Nuclear Regulatory Commission
William Ostendorff, a Republican commissioner at the Nuclear Regulatory Commission, will leave the agency after his five-year term ends June 30....

Energy
South Carolina Offers Renewable Energy Tax Credits
South Carolina is providing income tax credits for certain solar energy and geothermal installations as part of legislation (H.3874) signed into law by Gov. Nikki Haley (R) Feb. 16....

Enforcement
Trial Begins for Former BP Manager Linked to 2010 Oil Spill
Federal prosecutors have what they expect to be their last chance to send someone to jail over the deadly 2010 BP Plc Gulf of Mexico well blowout and the worst offshore oil spill in U.S. history (United States v. Kaluza, E.D. La., No. 12-cr-00265,...

Environmental Justice
EPA Solicits Nominations for Justice Council
The Environmental Protection Agency is requesting nominations be submitted for its National Environmental Justice Advisory Council, according to a notice to be published in the Federal Register Feb. 18. The council offers recommendations...

EPA
Acting EPA Human Resources Chief to Depart Agency
The Environmental Protection Agency's acting leader of human resources, contract and grant issues will leave the agency for a teaching position at the University of Texas–Austin, Administrator Gina McCarthy said in an e-mail...

Hazardous Waste
Oil Pipeline Cases Stay in State Court After Plaintiffs Changed
The addition of substituted plaintiffs in a mass action suit in Louisiana over oil pipeline waste doesn't create a new "window" for transfer of the cases from state to federal court under the Class Action Fairness Act, a federal...

Oil & Gas
Oklahoma Announces Plan for Wastewater Injection Cutbacks
In a continuing effort to address increased earthquake activity, Oklahoma's energy and gas regulators unveiled what they called the "largest volume reduction plan yet" for underground wastewater disposal from oil and gas...

Renewable Energy

Ontario Investing $72M in Efficiency, Clean-Tech Programs
The Province of Ontario's Green Investment Fund will invest C$99 million ($72 million) in energy efficiency and clean-tech innovation programs. The government has committed C$74 million in the clean-tech initiative, designed to reduce...

Renewable Energy
Solar Group Pushes for Broad Eligibility for Tax Credits
The IRS should take a broad approach as it considers what types of renewable energy equipment are eligible for tax credits, a solar energy trade group said....

Risk Assessment
More Time Given to Provide Chlorinated Paraffin Use Data
Companies that process or use any of seven chlorinated paraffins as lubricants or flame retardants and for other purposes will have until March 23 to provide the Environmental Protection Agency information about how they use these chemicals,...

Stormwater
Trading a Boon When Using Green Infrastructure: NRDC
Cities using green infrastructure to manage stormwater will glean greater benefits by engaging in stormwater credit trading, according to a new issue brief by the nonprofit advocacy group the Natural Resources Defense Council....

Sustainability
Ford Drops Membership in ALEC Lobbying Group
Ford Motor Co. dropped its membership in the conservative lobbying group the American Legislative Exchange Council (ALEC), the company confirmed in an e-mail Feb. 17 to Bloomberg BNA. ...

Water Pollution
EPA: DC Water Waived Potomac River E. Coli TMDL Claims
The D.C. Water and Sewer Authority failed to state a claim when it filed a complaint challenging the total maximum daily load for E. coli that the Environmental Protection Agency approved for the Potomac River and its tributaries, the agency...

Water Pollution
New York Classifies PFOA as Hazardous
The chemical perfluorooctanoic acid (PFOA) was classified by New York state as a hazardous substance Feb. 17 in an emergency regulation published in the New York State Register....

Water Pollution
Toxins From Algae Found in Southeast U.S. Streams
Toxins from algae were found in 39 percent of small streams sampled across the southeastern U.S., the U.S. Geological Survey said Feb. 17....

SPECIAL REPORT

Water Resources
Groundwater Reserved Right Appeal Raises Doctrine Questions
Although water rights are generally products of state law—adjudicated in state courts and administered by state agencies—a federal common law doctrine creates a water right for federal lands, such as national forests, monuments...

REGULATORY AGENDA

Comment Deadlines

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NEWS

Air Pollution
China Proposes Car Interior Air Quality Testing
China's Ministry of Environmental Protection proposed mandatory testing of air quality inside new motor vehicles, according to a draft of the policy released for public comment Jan. 28. The amendment would replace a voluntary testing...

Air Pollution
Measure Would Halt New Injections at L.A. Gas Field
The California Senate approved a bill Jan. 28 to impose an immediate moratorium on natural gas injections at Southern California Gas Co.'s underground storage field near Los Angeles. The unanimous vote, 40-0, sends S.B. 380 to the Assembly...

Air Pollution
Ozone Rule Not Protective, Environmental Groups Argue
The Environmental Protection Agency erred when it rejected the advice of its own science advisers and set revised ozone standards that are insufficient to protect health or vegetation, environmental and public health groups told a federal...

Air Pollution
Utah Governor Pushing State's Refineries to Make Tier 3 Fuel
Utah Gov. Gary R. Herbert (R) said he continues to push the state's refineries to produce "as soon as possible" fuel under the Tier 3 rule, which cuts sulfur in gasoline and strengthens vehicle

emissions standards....

Climate Change
Amendment Eyes Fossil Fuel Disclosure on Climate
The push for better fossil fuel-company disclosure on climate change could get an added boost from Congress....

Climate Change
Maryland Bill Would Boost Greenhouse Gas Cuts
A Maryland Senate committee scheduled a Feb. 9 hearing on legislation (S.B. 323) that would boost the state's target for greenhouse gas emissions reductions to 40 percent below 2006 levels by 2030. The measure would codify the recommendation...

Climate Change
Northwest Territories Premier Talks Climate Change
Canada's Northwest Territories will use challenges presented by climate change and the current oil-price slump to build infrastructure and craft legislation on environmental assessments and hydraulic fracturing so the province...

Climate Regulation
21 Senate Democrats Urge New Methane Standards
The Environmental Protection Agency should expand its regulation of methane emissions to include existing sources from the oil and natural gas sectors, 21 members of the Senate Democratic caucus wrote Jan. 28. ...

Climate Regulation
Feb. 4 Deadline Set for EPA Power Plan Stay Response
The Environmental Protection Agency has a Feb. 4 deadline to respond to petitions to the U.S. Supreme Court seeking to stay implementation of carbon dioxide standards for power plants while the standards are being litigated, according to...

Climate Regulation
Proposal Would Alter Oil, Gas GHG Emissions Reporting
Oil and gas operations that are large emitters of greenhouse gases would be allowed to use new monitoring methods to detect leaks from their equipment under a proposed Environmental Protection Agency rule that would alter how those facilities...

Drinking Water
Bill to Authorize $400M for Flint Water Infrastructure
Legislation that would provide $400 million in federal emergency funding to help Flint, Mich., respond to its drinking water crisis was announced Jan. 28 by three Democratic members of the Michigan congressional delegation. ...

Drinking Water
Flint Emergency Extended, $28M Assistance OK'd
On the heels of a $28 million supplemental appropriation measure (H.B. 5220) for Flint passing the Michigan Senate, Gov. Rick Snyder (R) extended the city's state of emergency until April 14. ...

Energy Efficiency
California Adopts New Lighting Standards
California has set the nation's first energy efficiency standards for small lamps used in track lighting and general purpose light-emitting diodes, or LEDs....

Energy

Senate Adopts Energy Amendments on Nuclear, Research
Measures that would promote advanced nuclear energy projects and increase funding for energy research were among the first amendments adopted to a broad energy bill (S. 2012) on the Senate floor Jan. 28. ...

Environmental Reviews
Canada's Energy Project Review Process Lauded
Canada's new environmental assessment process for pipelines and other major energy projects is being widely lauded, despite questions about whether it is enough to help the country meet its international climate change commitments....

Hazmat Transport
UN Body Urges Ban on Lithium Batteries on Passenger Planes
The International Civil Aviation Organization's top technical body has recommended a total ban on shipments of lithium batteries as cargo on passenger aircraft....

Oil & Gas
Oklahoma Company Agrees to Disposal Well Cuts
A recent agreement between Oklahoma's oil and gas regulator and one of the state's largest energy companies to reduce wastewater disposal in response to increased seismicity heads off potential legal action over the company's...

Renewable Energy
California Extends Payments for Rooftop Solar Customers
Rooftop solar customers of California's three investor-owned utilities will continue to get paid full retail rates for the excess energy their systems generate, under a net metering program the California Public Utilities Commission...

Toxic Substances
EU Food Authority Claims Breakthrough in Pesticide Exposure Studies
The European Food Safety Authority (EFSA) said it will use a new risk assessment tool to prepare "breakthrough" exposure studies on the risks to the human thyroid and nervous systems of the combined effects of pesticide residues in...

Vehicle Fuels
Plug Power Expects Higher Sales from Hydrogen Fuel Cells
Plug Power Inc., a maker of fuel-cell systems for forklifts, expects revenue to climb 50 percent in 2016, matching the growth rate set last year....

Water Pollution
GAO Report on WOTUS Lobbying Not Needed in Record: DOJ
A Government Accountability Office report criticizing alleged illegal lobbying by the Environmental Protection Agency to promote its water jurisdiction rule does not belong in the administrative record for litigation on the regulation,...

Water Pollution
Most Coastal Waters in Fair to Poor Health, EPA Says
Water quality in most U.S. coastal waters ranged from fair to poor in 2010, owing mostly to phosphorus pollution, the Environmental Protection Agency said in its most recent ecological assessment....

Water Pollution
N.Y. Takes Steps to Address Water Problem
Gov. Andrew M. Cuomo (D) took the unusual steps Jan. 27 of ordering emergency regulations to

classify the chemical perfluorooctanoic acid (PFOA) as a hazardous substance and to have a plastics plant operated by Saint-Gobain Corp. immediately...

Water Resources
Colorado Lawmakers Debate 2016 Water Policy Goals
Legislative proposals to conserve water, streamline permits and increase storage were the chief items of debate between two state lawmakers at a presentation of the Colorado Water Congress. ...

BNA INSIGHTS

Is Nuisance Litigation Undermining the Clean Air Act's Preemptive Economic Purpose?
The battle over federal Clean Air Act preemption continues to rage in the nation's courts, and until the U.S. Supreme Court decides to review the issue, the answer will probably remain unresolved. A group of slightly older cases held that...

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Comment Deadlines

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Peer Review Summary Report

External Peer Review of EPA's Draft Health Effects Documents for Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS)

November 17, 2014

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TABLE OF CONTENTS

Contents

EXECUTIVE SUMMARY ii
I. INTRODUCTION 1
I.1 Background on Draft Health Effects Documents 1
I.2 Peer Review Process 1
I.3 Peer Review Meeting 3
II. CHARGE TO REVIEWERS 5
III. GENERAL IMPRESSIONS 7
IV. RESPONSE TO CHARGE QUESTIONS 17
Charge Questions 17
Question 1. 17
Question 2. 22
Question 3. 27
Question 5. 34
Question 6. 36
Question 7. 39
Question 8. 43
Question 9. 47
Question 10. 50
Question 11. 53
Question 12 55
V. SPECIFIC OBSERVATIONS 58
APPENDIX A: PEER REVIEWER BIOSKETCHES A-1
APPENDIX B: MEETING AGENDA B-1
APPENDIX C: MEETING ATTENDEE LIST C-1

EXECUTIVE SUMMARY

Versar, Inc. (Versar), a contractor for the Environmental Protection Agency (EPA), coordinated an external peer review of EPA's draft health effects documents for Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS), and organized a two-day public peer review meeting in Arlington, Virginia on August 21 and 22, 2014. The peer review of EPA's draft health effects documents was initiated with a pre-meeting written peer review managed by Versar and conducted by seven independent expert peer reviewers. The role of the peer reviewers was to evaluate the scientific and technical merit of the draft documents and provide their responses to 12 charge questions. Peer reviewers were charged only with evaluating the quality of the science included in EPA's draft health effects documents and were not charged with making any regulatory recommendations or reaching consensus in either their deliberations or written comments. The two-day peer review meeting, which directly followed the written peer review period, was held to discuss the scientific basis supporting EPA's draft health effects documents and to provide members of the public with an opportunity to observe the peer reviewer deliberations.

On the first day of the meeting, Versar began by providing information on the overall peer review process and introducing the peer reviewers. In addition, EPA provided background information on the draft documents and approach used in the development of the documents. Following opening remarks by Versar and EPA, the peer reviewers began their discussions on the PFOA draft document, moderated by the Chair, Dr. Deborah Cory-Slechta. The discussions centered on individual responses to EPA's 12 charge questions. Additionally, some comments submitted to the public docket prior to the meeting were also discussed. The second day of the meeting began with brief remarks from Versar followed by discussions focusing on the PFOS draft document.

The reviewers began the discussion on the first day with the recognition of the significant amount of data that is available for both PFOA and PFOS and thus to be considered by the EPA for incorporation into the document and for the ultimate derivation of reference dose (RfD) values. In general, the reviewers commended EPA in doing a very good job of pulling together this significant and extensive body of information and of condensing it into its most critical pieces for the derivation of RfD values. This was especially the case given that there is significant human and animal data available for both chemicals, as well as inconsistencies in the data.

The reviewers did offer numerous suggestions for improving the documents, many of which are applicable to both PFOA and PFOS. In general, the suggestions relate to the statement of the problem and defining the database that was utilized, clarity and ease of presentation, and transparency of the reports, as well as to issues of modeling, use of human data, and liver weight increases as the most sensitive endpoint. These comments are summarized below, but please note that this does not reflect a consensus or group perspective.

- All reviewers agreed that it would be extremely useful to include an opening section of the document describing in some detail the literature that was reviewed. More specifically, this would include a description of the dates that were included in the

literature review, whether requirements for peer-reviewed materials were imposed, and what criteria were used in eliminating studies from consideration. This would assist in identifying studies that should potentially have been included. Many additional studies of importance were suggested for review at the meeting by the peer reviewers.

- The reviewers also agreed that there are some significant studies that have been published recently on PFOA and PFOS that may be relevant, and these should be included in the documents during the current revisions.
- Reviewers also felt that the Tables in the documents could be made much more useful to readers with the addition of columns that included more study details. This would minimize the amount of back-and-forth to the text that was required to assess conclusions.
- Additionally, there was a strong agreement that the Hazard Identification section of the documents should include a more systematic review of study strengths and limitations. These were noticeably absent in the human studies, where all studies were considered equivalent but also applicable to animal studies.
- Reviewers also felt that each Chapter should include introductory paragraphs, as well as concluding paragraphs, that would provide better integration of the material across the Chapters, as well as summarize the conclusions arrived at from the text in the Chapter. This would also facilitate the ability of readers to follow the presentations provided in Chapter 5 of the derivation of RfDs.
- As pointed out at the peer review meeting, the authors would be well advised to base these documents on the new documents being produced by EPA's National Center for Environmental Assessment (NCEA) for the Integrated Risk Information System (IRIS) program. While the reviewers recognize that the PFOA and PFOA health effect documents will not be used specifically for the same purpose, they will still likely be held to the same standards of presentation, clarity and transparency and thus, the new NCEA formats provide good models to follow.
- With respect to content of the documents, there was initial confusion around some of the modeling outcomes and assumptions based on values that were provided in the documents. However, following explanation from EPA personnel in response to clarifying questions from the reviewers at the peer review meeting, it became clear that there were errors in these values and once these were corrected, concerns about the specific values used in some modeling were allayed.
- Even with the corrections to the modeling, there were residual concerns among reviewers based on toxicokinetic properties of these chemicals, with respect to applying the same candidate RfD values to both short and long-term exposures, which in animal studies ranged from 11 to 182 days with reviewers expressing a need for this to be re-considered.
- There were mixed comments by reviewers with respect to the decision not to use human data in the derivation of RfD values. All reviewers generally agreed that the rationales provided for the exclusion of the human data were not actually appropriate, as it is certainly not the case that such use would be precluded by the fact that there were

multiple and not sole exposures to either PFOA or PFOS. There was discussion as to the possibility of whether human data could be utilized in these assessments, and that ranged from an opinion that these data would not be useful to the view that it might be possible to use these data, despite the absence of information on route of exposure. Collectively, the range of opinions suggests that the issue should be revisited, and that appropriate rationales should be provided for the decision that is reached in the revised version.

- For both PFOA and PFOS, the RfDs were ultimately based on increases in liver weights in animal studies. There was significant discussion among reviewers as to the appropriateness of this endpoint for the derivation of the RfDs. That range of opinion spanned from an interpretation that these did constitute adverse effects in that they are a direct effect of a chemical exposure, whereas other reviewers saw these as adaptive effects. Collectively, the range of opinions suggests that the issue should be revisited, and that appropriate rationales should be provided for the decision that is reached in the revised version.

Following the meeting, peer reviewers were given additional time to complete their individual written reviews, which were submitted to Versar upon completion. These final written comments are contained in Sections III, IV, V of this report and fall into three categories: general impressions, responses to charge questions, and specific observations. Written peer review comments, as well as comments submitted to the docket by members of the public, will be considered by EPA as it revises the draft documents.

I. INTRODUCTION

I.1 Background on Draft Health Effects Documents

On February 28, 2014, the EPA's Office of Water (OW) announced in the Federal Register the release of the draft health effects documents for Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS) for purposes of public comment (scientific views) and peer review (http://www.gpo.gov/fdsys/pkg/FR-2014-02-28/pdf/2014-04455.pdf). The draft documents and charge questions were prepared by the Health and Ecological Criteria Division (HECD), within EPA's OW, in order to support future regulatory evaluations and decisions. EPA will consider the public comments and peer reviewer comments when revising the documents. Once the PFOA and PFOS health effects documents are finalized, they will be utilized to develop lifetime health advisory values for each chemical. PFOA and PFOS are listed on the third Contaminant Candidate List (CCL3)[1] and both chemicals are currently being monitored under the third Unregulated Contaminant Monitoring Rule (UCMR3)[2].

I.2 Peer Review Process

Consistent with guidelines for the peer review of highly influential scientific assessments, Versar, an EPA contractor, was tasked with assembling six to seven scientific experts to evaluate the draft PFOA and PFOS documents. The purpose of the peer review was to provide a documented, independent, and critical review of the draft health effects documents, and identify any necessary improvements to the documents prior to being published. In assembling these peer reviewers and coordinating the peer review, Versar was charged with evaluating the qualifications of peer review candidates, conducting a thorough conflict of interest (COI) screening process, independently selecting the peer reviewers, distributing review materials, maintaining contact with the peer reviewers, organizing and hosting the public peer review meeting, and developing a final peer review report.

The peer review selection process was initiated with a three-week public nomination period that was held from February 28, 2014 to March 21, 2014, as documented in the Federal Register on February 28, 2014 (http://www.gpo.gov/fdsys/pkg/FR-2014-02-28/pdf/2014-04455.pdf). During this period, members of the public were able to nominate scientific experts with knowledge and experience in one or more of the following areas: (1) epidemiology, (2) toxicology (liver effects, immunotoxicity, neurotoxicity, developmental and reproductive toxicology, etc.), (3) membrane transport, (4) human health risk assessment, (5) pharmacokinetic models, and (6) mode-of-action for cancer and noncancer effects. Concurrently, Versar conducted an independent search for qualified scientific experts to augment the list of publically-nominated candidates. In total, Versar evaluated 29 interested and available candidates who were either nominated by the public (n=18) or identified by Versar (n=11).

[1] CCL3 is a list of contaminants that are currently not subject to any proposed or promulgated national primary drinking water regulations, that are known or anticipated to occur in public water systems, and which may require regulation under the Safe Drinking Water Act (SDWA). Additional information about the CCL3 can be found at the following website: http://water.epa.gov/scitech/drinkingwater/dws/ccl/ccl3.cfm.

[2] EPA uses the Unregulated Contaminant Monitoring (UCM) program to collect data for unregulated contaminants suspected to be present in drinking water. Results from UCMR3 can be examined as they become available at the following website: http://water.epa.gov/lawsregs/rulesregs/sdwa/ucmr/ucmr3/.

Versar considered and screened all 29 candidates against the selection criteria described in the Federal Register dated February 28, 2014, which included: (1) having demonstrated expertise in the areas described above, based on information in their submitted resume, biographical sketch and/or current publications, (2) being free of any COI and the appearance of the lack of impartiality, and (3) being available to participate in-person in a two-day peer review meeting in the Washington DC area in the July or August 2014 timeframe. Following the screening process, Versar narrowed the list of potential reviewers to 15 candidates and provided to EPA the names of the candidates selected by Versar to be on the interim list. Additionally, information on the 15 candidates, including their professional affiliations, expertise, education, and professional experience were provided for the interim list and published in the Federal Register on April 30, 2014 (http://www.gpo.gov/fdsys/pkg/FR-2014-04-30/pdf/2014-09888.pdf). The Federal Register also requested the public to submit relevant information or documentation on the interim list of candidates that Versar should consider during the evaluation process of selecting the final six to seven reviewers.

Following the close of the public comment period on the interim list of potential reviewers, Versar re-evaluated each interim candidate's credentials to select the experts who, collectively, provided expertise spanning the multiple subject matter areas covered in the draft documents and provided a balance of perspectives. In addition, Versar evaluated the availability of each candidate to ensure all final peer reviewers were available on the same days for the meeting in the selected timeframe. Once the evaluation process was completed, Versar narrowed the interim list of 15 candidates and selected the seven final peer reviewers. In addition, Versar selected Dr. Deborah Cory-Slechta as Chair of the peer review meeting due to her expertise in toxicology as well as her strong record of chairing and participating in peer review panels, scientific meetings, and workshops. A list of the final seven peer reviewers who participated in this review is provided below. In addition, each reviewer's biographical sketch is included in Appendix A.

Following the selection process, Versar distributed EPA's draft PFOA and PFOS documents and 12 charge questions (see Section II) to the peer reviewers. The peer reviewers were asked to evaluate the scientific and technical merit of the draft documents and provide their responses to the 12 charge questions. This included evaluating the appropriateness of the quality, accuracy, and relevance of the data in the documents. Peer reviewers were not charged with making any regulatory recommendations or reaching consensus in either their written comments or public deliberations. In addition to being provided the draft documents and charge questions, comments submitted to EPA's public docket (Docket ID number EPA–HQ–OW–2014–0138) during each document's 60-day public comment period were provided to the peer reviewers ahead of the meeting for their consideration. Also, a brief summary of the public comments was developed by Versar and provided to the reviewers. However, peer reviewers were not asked to evaluate or respond to comments submitted to the docket.

Versar managed the pre-meeting peer review period, which provided the peer reviewers approximately two months to evaluate the draft health effects document and complete their written reviews. Following receipt of the peer reviewers' draft comments, Versar compiled the comments into a pre-meeting peer review report and distributed them to the peer reviewers and

EPA to prepare for the public peer review meeting. These preliminary responses to the charge questions formed the basis of reviewer discussions on Days 1 and 2 of the public meeting.

Peer Reviewers:

James V. Bruckner, Ph.D.
University of Georgia

Deborah A. Cory-Slechta, Ph.D.
University of Rochester School of Medicine and Dentistry

Jamie C. DeWitt, Ph.D.
East Carolina University

Jeffrey W. Fisher, Ph.D.
U.S. Food & Drug Administration

William L. Hayton, Ph.D.
The Ohio State University

Matthew P. Longnecker, Sc.D, M.D
National Institute of Environmental Health Sciences

Angela L. Slitt, Ph.D.
University of Rhode Island

I.3 Peer Review Meeting

On August 21 and 22, 2014, Versar convened a public peer review meeting in Arlington, Virginia. This meeting was held to discuss the scientific basis supporting EPA's draft health effects documents and to provide members of the public with an opportunity to observe the peer reviewer deliberations. The meeting followed both the documents' public comment period, during which members of the public were able to submit written comments, and the pre-meeting written peer review period, during which the seven selected peer reviewers evaluated EPA's draft health effects documents and provided preliminary comments in response to the charge questions.

Versar managed the pre-meeting registration period, which allowed members of the public to register to attend the meeting in person or remotely via teleconference and/or webinar. Members of the public were able to register online, via Versar's registration website (http://peerreview.versar.com/epa/pfoa/) as well as by telephone, email, or U.S. mail. In advance of the meeting, Versar provided all registered attendees with pre-meeting handouts, which included the agenda and logistics information.

On the first day of the meeting, Versar began by providing information on the overall peer review process and introducing the peer reviewers. In addition, EPA provided background

information on the draft documents and approach used in the development of the documents. Following opening remarks by Versar and EPA, the peer reviewers began their discussions on the PFOA draft document, which centered on individual responses to EPA's 12 charge questions. The remainder of the day was dedicated to discussions on the PFOA document, which were moderated by the Chair, Dr. Deborah Cory-Slechta. The second day of the meeting began with brief remarks from Versar followed by discussions focusing on the PFOS draft document. Approximately 23 public observers attended the peer review meeting in person and 21 observers attended the meeting via teleconference and/or webinar. Please see Appendix B for the meeting agenda and Appendix C for a list of public attendees.

Following the public peer review meeting, peer reviewers were given additional time to complete their individual written reviews. These final written comments are contained in Sections III, IV, and V of this report. Written peer review comments, as well as comments submitted to the EPA docket by members of the public, will be considered by EPA as it revises the draft health effects documents.

II. CHARGE TO REVIEWERS

PFOA and PFOS are environmentally persistent organic fluorocarbons (PFC) that have been identified in ambient waters, ground water, drinking water, and biosolids. They are metabolically inert but have the ability to bind to and interact with a variety of biomolecules leading to responses in living organisms. Both compounds have a substantial database of epidemiological, pharmacokinetic, toxicological and mechanistic studies. The two documents submitted for peer review include health assessment chapters that will be used 1) to provide information to drinking water treatment plant operators regarding the significance of monitoring results with respect to potential health outcomes and 2) to determine whether the perfluorinated compounds currently being monitored at Public Drinking Water Systems require regulation. The health information at that time will be accompanied with chapters on environmental fate, occurrence at public drinking water systems and occurrence in other media. The quantitative aspects of the Health Assessment documents will also be used to develop lifetime Health Advisories for both compounds.

Charge Questions

1. Please comment on the strengths, weaknesses, and characterization of the studies selected as key for quantification.

2. Please provide citations (and, where possible, pdfs or hard copies) for any references you suggest EPA consider adding to the document. Describe where you suggest these references be incorporated.

3. The OW concluded that the human epidemiology data for PFOS/PFOA do not provide adequate quantifiable dose-response information for use as the basis of a candidate RfD because of uncertainty regarding the routes, levels and timing of exposures plus the confounding influences of other PFCs present in serum. Please comment of the OW characterization of the data.

4. Please comment on the transparency and characterization of the epidemiological data.

5. The OW has concluded that the cancer classifications for PFOA and PFOS are most consistent with respective classifications of suggestive evidence for carcinogenicity as described the EPA Guidelines for Carcinogen Risk Assessment (pp. 2-56, 2-57). Please comment on the strengths and weaknesses of this classification.

6. Significant interspecies differences in pharmacokinetics exist for both PFOA and PFOS. Adjusting for interspecies differences was an important step in developing candidate RfDs given the totality of the human and animal data. Please comment on the strengths and weaknesses of the pharmacokinetic model adjustments to accommodate the impact of albumin binding and renal tubule transporters in determining average serum values.

7. Table 5-5 in the PFOA document and Table 5-7 in PFOS document list the parameters used for the ORD pharmacokinetic models that provide the final serum and AUC values for calculating the internal dose point of departure for the RfD calculation. Please comment on the strengths and weaknesses of the selected parameters.

8. The volume of distribution (Vd) and half-life values are critical in the derivation of the interspecies uncertainty factor applied in derivation of candidate RfDs from a NOAEL, LOAEL or a BMDL. The available data for both values are provided in Section 3.5.2 and 3.5.3 of both documents. Please comment the strengths and weaknesses of the values selected.

9. A variety of endpoints and studies were used to compare points of departure and the resultant RfDs for both PFOA and PFOS. In addition, comparisons were provided across RfD outcomes based on the model outputs compared to those for the NOAEL, LOAEL and BMDL points of departure. The range of candidate RfDs derived from the different points of departure is fairly narrow. Please comment on the strengths, weaknesses and transparency of this analysis.

10. The RfDs for PFOS and PFOA are derived from the modeled steady state serum concentrations and their association with effects that include short term and longer term exposures with associated diverse effects. The studies considered included effects due to exposure durations that ranged from 11 to 182 days, and occur at comparable human equivalent dose (HED) levels. The current, draft RfDs do not include an uncertainty factor for study duration because of the apparent concordance HEDs despite duration differences. Given this pattern of response, is it appropriate to conclude that the candidate RfDs are applicable to both short-term and lifetime exposures?

11. In addition to using the average serum values from animal studies to calculate internal doses for humans, the animal to human extrapolation can be accomplished by dividing animal average serum values by the human to animal clearance ratios to project a human average serum point of departure in units of mg/L serum. Please provide recommendations for applying uncertainty factors to the extrapolated average human serum values to determine serum-based thresholds that are protective for humans. A NOAEL expressed in average human serum units would be useful in interpreting NHANES population monitoring data.

12. Please describe any suggestions you have for improving the clarity, organization, and/or transparency of the draft documents.

III. GENERAL IMPRESSIONS

James V. Bruckner

PFOA-specific comments

This is one of the most comprehensive Health Effects Documents I have reviewed. The clarity and accuracy of accounts of pertinent research reports/publications are excellent. It is obvious considerable time and efforts were devoted to its composition. If anything, the amount of detail is so great that it is difficult to distill the mass of information on each topic and capture its "essence". This is likely the result of directions the authors were given for writing the document. Some topics in the Hazard Identification section do have summarizing sentences, in which the key/critical studies and their finding(s) are integrated and conclusions reached. It would be very helpful to devote much more attention to this for more topics, perhaps as an addition to Section 4.4 Hazard Characterization.

I do have a real problem with the scientific basis and soundness of certain conclusions in the document. The primary effect of PFOA in different species is increased absolute and/or relative liver weight. These are quite modest, reversible, non-specific effects that usually are not considered toxicologically significant. Livers of mice and rats dosed with PFOA typically exhibited hypertrophy characterized by increased peroxisomes, numerous mitochondria, reduced rough endoplasmic reticulum (RER), proliferation of smooth endoplasmic reticulum (SER), and increased autophagosomes or lipid-like droplets. Such morphological changes, particularly those in RER and SER, are manifestations of microsomal enzyme induction. This is considered adaptive, rather than adverse. Hall et al. (2012) points out that activation of a battery of genes involved in xenobiotic metabolism and transport serve to maintain homeostasis by enhancing the systemic elimination of the foreign chemical. Although PFOA is very poorly metabolized, it does persistently induce microsomal enzymes and the accompanying hepatocellular morphological changes. Upregulation of genes responsible for biliary excretion may be beneficial, since excretion of bilirubin, bile acids and conjugates of toxic chemicals/metabolites would be enhanced.

There are substantial qualitative and quantitative differences in responses of rodents and humans to PPARα activation. Therefore, many of the PFOA-induced alterations in lipid metabolism/homeostasis and associated biological processes in mice will be absent or an order of magnitude less pronounced at comparable doses in humans. Many of PFOA's effects on the liver of rodents are dependent on PPARα activation, though some effects appear to be PPARα-independent. Studies in PPARα-knockout mice show activation of other nuclear receptors by PFOA, including PXR, CAR, LXRA and FXR. Bjork et al. (2011) observed markedly lower transcriptional responses of PPARα, PXR, CAR and FXR to PFOA in cultured human than in cultured rat hepatocytes. These more subtle effects lead the investigators to conclude the changes in human cells reflected an adaptive metabolic remodeling rather than overt metabolic dysregulation, or disorder occurring in rat cells. The PFOA document's authors should go into detail discussing and summarizing the relative toxicological significance of non-PPARα effects in rodents versus humans.

It is important to recognize that clearly adverse effects of PFOA are seen. Lovelass et al. (2008), Cui et al. (2009) and others have seen focal necrosis and degenerative changes in the liver of mice and rats given relatively high doses of PFOA, as well as modest elevations in serum (hepatic) enzyme activities. Wolf et al. (2008a) observed a variety of degenerative structural changes in the liver of PPARα-null mice dosed with PFOA. Sakr et al. (2007a,b) and Olsen and Zobel (2007) reported associations between serum PFOA levels and slightly elevated serum enzyme activities in some occupationally-exposed populations. The increases in enzymes may have been attributable to factors other than PFOA. In light of the foregoing, it would be preferable to utilize hepatic morphological changes in rodents and/or elevated serum enzymes as the critical effect(s), rather than increased liver weight. These are clearly adverse effects seen in both rodents and humans.

An international panel of experts (Hall et al., 2012) opined that an increase in liver weight of ≤ 150%, at doses of chemical that do not produce structural or biochemical evidence of hepatocellular damage, would not be considered adverse. Absolute and relative liver weights were not increased as much as 50% by most PFOA doses in the majority rodent and monkey studies. Perkins et al. (2004), for example, reported dose-dependent increases in liver/body weight in rats fed 1, 10, 30 and 100 ppm PFOA for 13 weeks of 0, 10, 30, and 41%, respectively. Butenhoff et al. (2002) measured increases of 17, 21 and 37.5% and relative liver weight in monkeys given 3, 10 or 30/20 mg PFOA/kg/day for 26 weeks, respectively. Liver hypertrophy of this magnitude does not warrant such a low RfD. By adhering to EPA policies of calculating a $BMDL_{10}$ and using multiple UFs, regardless of the (lack of) severity of the critical effect and relatively low level of concern about other potential health effects, the end result is a vanishingly low RfD (i.e. 0.00002 mg/kg/day). A great deal of time and effort were spent on the PFOA hazard assessment, toxicokinetic modeling and extrapolations, dose metric and POD considerations, etc. Despite all of these scientifically-credible exercises and deliberations, the end result (RfD) seems to this reviewer to have been preordained-- to be extremely low.

Logic expressed on page 5-6, in support of use of liver weight gain as a critical effect and biomarker of loss of hepatocellular homeostasis seems flawed. As pointed out in the second paragraph, liver weight changes were not observed in PFOA-treated mice with a humanized PPARα receptor. It is noted that changes in gene products that modulate lipid metabolism do occur in these mice. EPA argues that this supports adoption of increased liver weight as a biomarker/critical effect. It has not been established that these changes in gene expression are adverse, or whether they are sufficient in magnitude to significantly alter lipid metabolism. It would be expected that repeated dosing with enough of a molecule (i.e., PFOA) that resembles a fatty acid would affect expression of such genes. Reversible changes in total cholesterol, bile acids, bilirubin, etc. have been observed. It has not been established, however, whether mild fluctuations in these indices are detrimental. No increases in mortality from cerebrevascular disease or ischemic heart disease have been found in PFOA-exposed humans. How then does the concurrence of alteration of expression of such genes and of liver weight gain support the latter as toxicologically-significant effect that should be prevented by setting the RfD low enough?

PFOS-specific comments

This Health Effects Document, like that for PFOA, is quite comprehensive. Its descriptions of the many studies of PFOS are clear, quite complete, and apparently quite accurate. As with the

PFOA document, so much detail is given about many studies in the Hazard Identification section, that is difficult to compare study designs/dosage regimens/species/indices/findings/etc. and to draw conclusions. The summary tables for single and multiple studies, however, are quite helpful in this regard. It would also be very useful to have more summary statements or paragraphs at the end of each topic. These should address the scientific importance of findings, their relevance to humans; and their impact on the weight of evidence on an issue.

The hazard characterization section (4.4) is, for the most part, inclusive and balanced in its presentation and integration of findings of the more important studies in each subject area. This is true for both non-cancer and cancer effects in humans and animals. It concerns me, however, that the document's authors do not focus in the remainder of the document on science (i.e., the candidate critical effects and their relevance to human health), but merely choose the most sensitive end-points and stress how similar the RfDs are after dosimetry modeling estimates and adjustments. I am not sure how this similarity of derived points of departure and other values, calculated from dissimilar endpoints, supports or validates the final RfD.

I recommend that an additional section be written, in which the primary adverse effects of PFOS are discussed-- in terms of their relative toxicological significance, their apparent mechanism(s), their relevance to humans, their likelihood in realistic exposure scenarios, and implications of altered experimental indices to actual organ dysfunction.

I am quite concerned about the increased rat pup mortality in several studies at relatively low maternal doses, but not about reversible liver weight changes or centrilobular hypertrophy. Is the decreased pup survival in several studies at relatively low maternal doses of PFOS relevant to humans?-- Is the dose-response curve steep, as suggested by Luebker et al. (2005a), such that there would be less concern about sub-threshold doses? -- What is the most likely mode of action (pulmonary surfactant or maturation, dietary, hormonal)? -- Is decreased survival PPARα-related? -- Is the mechanism in rats relevant to other species? -- Does pup mortality occur in other species at comparable doses? -- Might there be a dose-dependent alteration of maternal-fetal partitioning of PFOS?

Deborah A. Cory-Slechta

Both documents, although the PFOA document to some degree more than PFOS, overall are more of a tabulation of studies than a critical review of studies from which a rationale is presented for a choice of studies to model and from which to derive associated RfDs. The Executive summaries are too abbreviated and do not include sufficient rationale, description and detail to provide the reader with an understanding of how decisions described in Chapter 5 were made. Since in some cases, this will be the only sections read, they could provide a more informative summary.

It would be very helpful to provide a section up front that describes all of the parameters of the literature search, including the years that are included in the document review, as well as descriptions of criteria for studies that were included vs. those that were excluded. In addition, it should be indicated whether there was a criterion that studies be peer-reviewed. This is particularly important given the voluminous size of the data base that has accumulated for these

two chemicals. Given that revisions will be done and that such documents do not get updated with any frequency, it would be good to attempt to include as much of the new pertinent literature as possible.

The section on Toxicokinetics in the documents present studies in detail, but no real conclusions; this is true of most of the sections in these documents. Chapters 3 and 4 in particular read like tabulations of studies rather than critical reviews and because of that, the documents seem disjointed and Section 5, i.e., derivation of values, tend to be difficult to read through and require constant searching back to the original chapters in which they are described. It is critical to identify the strengths and weaknesses of the various studies, and which were given weight to use in the final determinations. It would be helpful if Sections 3, 4 and 5 included an introductory paragraph describing the goal of the chapter, and that each ends with an overall summary with conclusions. The tables in these chapters also would benefit from the inclusion of additional information that ultimately permits comparisons within the Table and does not require continually returning to the text to recall the species, sample sizes, etc.

In the sections on Hazard Identification, it is useful that studies are summarized by target organ, but there are almost no conclusions and no discussions of strengths or weaknesses of studies and therefore their use or not in future decisions. In fact, one is left with the impression that all studies are equal, especially in the section describing human studies. Within Chapter 4, the sub-sections entitled "evaluative and integrative" are actually neither. Data are presented simply as positive or negative with no real discussion of the strengths and limitations and what was concluded overall. For this reason, Chapter 5 is also lacking. It provides very little in the way of rationale and conclusions. Thus, the transparency of the process is really insufficient.

Jamie C. DeWitt

The information presented throughout the documents appears to be accurate (with one minor exception noted in Table 1 of these comments) and is presented clearly. For PFOA, a reference dose (RfD) of 0.00002 mg/kg/day was determined and evidence of carcinogenicity is considered suggestive with a human equivalent dose (HED) of 0.58 mg/kg/day. The RfD was based on changes in liver weight reported as a common denominator in four rodent (three rat and one mouse) studies and carcinogenicity was based on a limited number of epidemiology studies linking kidney and testicular tumors with exposure and evidence of tumor induction in the liver, testes, and pancreas (the "tumor triad") in rats. For PFOS, a RfD of 0.00003 mg/kg/day was determined and evidence of carcinogenicity is considered suggestive but with insufficient evidence to determine human carcinogenic potential. The RfD was based on developmental neurotoxicity and changes in liver weight.

While the carcinogenicity assessment seems appropriate for the two compounds given the limitations of the data sets, changes in liver weight as a basis of both of the RfDs is questionable in terms of its significance to exposed humans. Exposure to these agents increases liver weight and hepatocellular hypertrophy in rodents (and the definition of these endpoints as "adverse" or "toxic" also is contentious); this has been demonstrated across various rodent strains and under myriad exposure paradigms. However, there is no consensus in the scientific community regarding the mechanism by which exposure to these compounds increases liver weight and

induces hepatocellular hypertrophy in rodents and whether any of the putative mechanisms are sufficient to induce hepatotoxicity in exposed humans. Proposed mechanisms include peroxisome proliferator activated receptor alpha (PPARα) activation, activation of other nuclear receptors, peroxisome proliferation (which may or may not be dependent on PPARα activation), and oxidative stress. Humans can certainly respond to PPARα agonists (i.e., fibrate drugs are used as hypolipidemic agents) and a handful of epidemiological studies of highly exposed human populations have reported associations between PFOA/PFOS and alterations in liver enzymes, but the clinical relevance of the changes to the liver enzymes reported for these studies is uncertain. These liver-related changes in humans generally occur at higher doses than required to induce changes in the livers of rodents, which occurs at relatively lower doses than other observed effects. Therefore, a critical endpoint that occurs at very low doses in rodents, has no agreed upon mechanism that may or may not be relevant in humans at relatively high doses, may not be the best choice for the basis of a RfD. Liver weight change has been reported to occur in several species, including non-human primates, and at low doses, it may be an adaptive response and not a toxicological response. While this response may be protective of human health because it is common following low dose exposure to PFOA or PFOS, other endpoints may be more relevant to humans, especially endocrine system effects, including changes to thyroid hormones and mammary gland development, and immune system effects. Endocrine and immune system effects have been reported in exposed humans, suggesting that such endpoints may operate via a mechanism that is more relevant to humans than mechanisms related to changes in liver weights.

In addition, the one developmental neurotoxicity study used, in part, for the PFOS RfD is only weakly supported by additional studies in rodents or other species and is based on behavioral responses that could be influenced by factors other than direct effects on the nervous system. Additional confirmatory studies are necessary for this observation to be considered a critical effect of PFOS exposure.

Finally, while well-written overall, the documents lacked an overall critical analysis or depth required of a risk assessment. Why specific studies were included or not should be better explicated in the text.

Jeffrey W. Fisher

The document was well written in terms of balance and presenting information. Summary statements are needed for chapters; a synthesis/analyses of the data are needed in some cases. A more critical evaluation of the human and non-human responses to PFOA/PFOS is required to justify not using human or non-human primate data. A rationale for the modeling approaches is needed given the more recent PBPK models that are available.

William L. Hayton

The literature that pertains to the health effects of PFOA and PFOS is large and presents a major challenge to accurately summarize and analyze it and develop an RfD for PFOA and PFOS. Reported health effects in animals and humans, sometimes contradictory, include exposure-associated changes in serum cholesterol, lipids, uric acid, and thyroid hormones, obesity-related

metabolism, immune system function, and effects on reproduction, development of the mammary gland, the nervous system, and behavior. Target organ effects (e.g., liver, kidney) have been reported, as well as associations of PFOA and PFOS exposure with testicular, prostate and kidney cancer. Studies in several laboratory animal species have added the complications of interspecies comparisons and extrapolation of findings to humans. In humans, there have been a Phase I clinical trial of PFOA, and epidemiological studies of populations exposed to PFOA and PFOS occupationally and in communities with and without water supplies contaminated with PFOA and PFOS. The draft documents have accurately presented in summary form the results of many animal and human studies and used pharmacokinetic methods to link PFOA and PFOS exposure rates to internal dose metrics such as serum concentration. While the overall effort is commendable, there are two issues that the draft documents raise: 1) the literature cited does not include many apparently relevant published works. The cut-off date for cited literature was early 2013 (this should be indicated in the documents), but commenter's noted a number of pertinent publications in 2011 and 2012 that were not cited, and there have appeared several highly pertinent papers since the cut-off date, and 2) while the descriptions of individual studies are generally clear and accurate, there is a lack of independent, critical analysis of the studies and a lack of synthesis of results from multiple studies common to a particular health effect.

Matthew P. Longnecker

The PFOA and PFOS documents achieve the goal of identifying RfDs that are well founded. My main criticism is that the rationale for not using the human data to provide a POD needs to be strengthened.

For example, in the PFOA document, on page 5-19, first paragraph below the table, it says "human data … lack the exposure information for dose-response modeling." This statement is logically inconsistent with techniques that were used to estimate HED on the basis of serum concentration, as given on page 5-17, near the bottom. Or, in some cases, such as in the C8 study, the exposure estimates that were calculated based on water district were sufficiently good that a dose-response analysis would be possible. In other words, because many human studies have serum concentration of PFOA or reasonable estimated exposure values, the corresponding HED could be estimated, and hence the dose-response could be modeled. Granted, some assumptions would be needed, but the methods could be serviceable (see response to item 3 below). (Some of the above also applies to pages 5-1 and 5-2). More compelling arguments for not basing the POD on human data are, e.g., that: 1) the low probability that humans are 1,000 times more sensitive to PFOA than other species (the number is based on the last column in table 5-9 compared with PFOA values in the C8 study and background exposed populations), especially given the relatively tight agreement between LOAEL (average serum concentration basis) among other species, 2) the possibility that the observed associations in humans were due to unmeasured confounding factors or reverse causality, and 3) other weaknesses in the epidemiologic data such as inconsistent results across studies (selected outcomes), unreplicated findings, or associations with clinical chemistry results for which corresponding adverse clinical correlates (i.e., morbidity) are not clearly established.

Angela L Slitt

The documents provide a very thorough evaluation of PFOA and PFOS studies. It is logically organized, presenting findings in a way that the reader can understand the findings related to human, monkey, and rodents. The organization of the document allows the reader to easily find information about each species within the subchapters and summarizes key points in table form. PFOA is a well-studied compound, with a substantial amount of toxicokinetic and endpoint studies in rodents. Mechanistic data describing the role of membrane transporters to understand gender differences in PFOA elimination in rats is fairly well written. Little data exist regarding contribution of membrane transporters to PFOS disposition and elimination. The documents thoroughly describe species differences in PPAR-alpha signaling that might contribute to observed endpoints in rats, but not humans or monkeys. Overall, both documents are very thorough and provide a reliable basis for PFOS and PFOA evaluation.

For PFOA toxicokinetics, mechanisms of PFOA transport are important for understanding species differences in response to PFOA exposure, with focus placed on kidney. Figure 3-2 in the PFOA document does not adequately present the localization of renal transporters with relationship to their contribution to the urine compartment or renal reabsorption. A very nice diagram showing the subcellular localization of renal transporters presented by Klaassen and Aleksunes (Pharmacol Rev. 2010 Mar;62(1):1-96) clearly depicts the contribution of various transporters to filtrate or blood. This is an easier diagram to put PFOA elimination into context than the one presented. Contribution of membrane transporters to species differences in PFOA excretion Section 3 (specifically 3.4.1) would be put in better context if a table could be generated to compare Km and Vmax values for PFOA for various transporters, with specific focus on species information for OATPs and OATs. Data regarding information on contribution for OATps in liver accumulation of PFOS and PFOA is lacking, with specific regard to species differences. As PFOS is a likely candidate for hepatic uptake transport, understanding a mechanism to explain species differences in hepatic effects possibly due to difference in hepatic exposure is critical. Understanding impact species specific regulation of OATp expression in liver (e.g. whether species difference in PPAR-alpha signaling contributes) is also important in putting rodent distribution data into context.

Increased liver weight is considered to be a critical effect, but how increased liver weight relates to the observed human and monkey health effects needs to be further explained. First, use of liver weight alone might not be substantiate of an effect for point of departure compared to other liver effects observed at higher concentrations, such as increased serum ALT or AST.
The studies that have evaluated these endpoints are well conducted. In layman terms, if someone is walking around with an increased liver weight, is he or she at risk for disease? Will his/her life span be shortened? To increase transparency of the document, a more comprehensive explanation is needed to justify why increased liver weight should be considered as a critical endpoint for human health.

However, it should be noted for future consideration that there are a few publications in mice that do also ascribe liver weight changes in increased lipid accumulation along with increased expression of genes that contribute to fatty liver disease. This is considered to be a gap in knowledge for the field. Will relatively low dose PFOS exposures associated with hepatic

steatosis have other endocrine related effects known to be associated with NAFLD (insulin resistance, impaired glucose tolerance)? Emerging studies are evaluating whether PFOS induces hepatic steaosis and whether it is a PPAR-α mediated effect. For example a study performed by Wan et al. (2012) administered 0, 1, 5 or 10 mg/kg/day PFOS to adult male CD-1 mice for 3, 7, 14 or 21 days. Histological analysis of liver sections, and biochemical/molecular analysis of biomarkers for hepatic lipid metabolism were assessed. Overall, the study reported that PFOS-administration induced hepatic steatosis in a time- and dose-dependent manner. The study also shows a high correlation between liver weight and lipid content. Increased expression of a lipogenic target (CD36/FAT) was observed at 5 and 10 mg/kg PFOS. A second study by Bijland et al. (2011) illustrated that PFOS administration increased liver weight (+ 107%, $p < 0.0001$), which was accompanied by an increased hepatic TG content (+ 192%, $p < 0.0001$, respectively) in E3L.CETP mice on a C57Bl/6 background. At the age of 8–10 weeks, mice were fed a semisynthetic Western-type diet, containing 0.25% (wt/wt) cholesterol, 1% (wt/wt) corn oil, and 14% (wt/wt) bovine fat for 4 weeks in three independent experiments. Upon randomization according to body weight, plasma TC, and TG levels, mice were fed a Western-type diet without or with PFOS (0.003%, ~3 mg/kg/day) for 4–6 weeks. In summary, there is evidence that administration of relatively low PFOS doses to mice can result in hepatic lipid accumulation in the absence of overt "wasting."

Data is lacking as to whether higher species, such as monkeys or humans will also develop PFOS-induced steatosis, which is confounding. Studies have profiled gene expression in wild-type and PPARα-null mice administered PFOS, finding that there is pathology and gene expression consistent with lipid-promoting effects in liver that are independent of PPARα, as they are observed in PPARα-null mice (Rosen et al., 2010). Limitations to the studies are that they did not specifically quantify hepatic lipid content, but inferred that the PFOS-induced vacuolization in liver pathology observed was potentially related to triglycerides. Studies by Bjork et al. (2011) comparing rat and human primary hepatocytes treated with PFOS (25 μM) demonstrated that human hepatocytes were slightly less responsive to the induction of lipid oxidation and synthesis genes, as well as induction of carbohydrate metabolism. It should be noted that the hepatocytes from the study are from a single human donor, the hepatocyte lipid content was not determine, and hepatocyte culture conditions were standard and not optimized to induce steatosis. In summary, the current literature is lacking robust information regarding whether PFOS, which highly concentrates in liver, has a steatotic-inducing effect in human or monkey liver. The evidence in PPARα-null mice indicates that it might have some PPARα independent effects related to hepatic fat accumulation. Because evidence for hepatic lipid content in PPARα-null mice after PFOA or PFOS has been described only by pathology, more robust studies are needed to conclude whether the effects can occur independent of PPARα and the observed increased liver weight is due not only to hypertrophy due to nuclear receptor activation, but lipid accumulation. However, this reviewer is noting this as a concern for the future and area where a gap in knowledge exists.

Moreover, because the reviewer is noting a concern for hepatic fat accumulation that exists in the absence of PPARα, it should be appreciated that traditional markers, such as AST and ALT have poor prognosis for NAFLD or toxicant associated steatohepatitis. Most patients with NAFLD are asymptomatic and the disease is often diagnosed following findings of elevated aminotransferases, especially when combined with other features of metabolic syndrome. These

abnormal liver function tests usually require the physician to distinguish between NAFLD and alcoholic fatty liver disease (AFLD). Some have suggested that a serum AST/ALT ratio >1 are indicative of AFLD. Other possible signs of AFLD include elevated HDL-cholesterol with hypertriglyceridemia. While most diagnoses of NAFLD may result from altered liver function tests, normal serum aminotransferase tests can be seen in patients with both steatosis and NASH (Ipekci, et al., 2003; Mofrad, et al., 2003). Indeed, it is reported that two-thirds of NASH patients may have normal aminotransferase levels at any given time (Oh, et al., 2008; Delgado, 2008; Wieckowska and Feldstein, 2008). Kunde et al. investigated the accuracy of NASH diagnosis by serum ALT in women undergoing gastric bypass surgery (Kunde, et al., 2005). They compared two different reference laboratory cutoffs for "normal" ALT levels, the previous guideline of 30U/L, and new lower level of 19U/L that was suggested to aid in the diagnosis of NAFLD. Importantly, the authors reported that the diagnostic utility of serum ALT remained poor even at the new lower cutoff. Sensitivity and specificity of serum ALT levels were found to be 42% and 80% (ALT > 30U/L) versus 74% and 42% (ALT > 19U/L). These and other studies (Lizardi-Cervera, et al., 2006; Amarapurkar and Patel, 2004; Amarapurka, et al., 2006; Chen, et al., 2006; Fracanzani, et al., 2008; Sorrentino, et al., 2004; Mofrad, et al., 2003; Uslusoy, et al., 2009) illustrate the need for a more effective diagnostic measure for NAFLD, especially the NASH stage. In sum, use of ALT and AST elevation to base the point of departure must be taken into context because they are poor prognostic markers for increased liver accumulation, NAFLD, or even NASH. Use of ALT and AST might not be an appropriate biomarker for measurement of PFOA or PFOS-induced adverse effects on liver and should be considered as a gap in our knowledge for future work.

In the review panel discussion, there was discussion regarding a publication by Hall et al. (2012), which summarized the outcome of a workshop regarding liver hypertrophy and hepatocarcinogenesis. Overall, the workshop concluded that "hepatomegaly as a consequence of hepatocellular hypertrophy without histologic or clinical pathology alterations indicative of liver toxicity was considered an adaptive and a non-adverse reaction". This conclusion is taken in the context of hepatic hypertrophy caused by nuclear receptor activation. This differs from others that have concluded in mouse NTP studies where correlations between liver weight increases and histological parameters and carcinogenesis were assessed, the authors concluded that ''the best single predictor of liver cancer in mice was hepatocellular hypertrophy'' (Allen et al. 2004). Based on the conclusions presented in Hall et al. (2012), increased liver weight might not be considered an appropriate POD because of lack of overt toxicity and hepatomegaly being considered an adverse effect. It should be noted that this reviewer still has concerns regarding this conclusion for PFOS and PFOA because the pathology described in PPARα-null mice reflect increased hepatic lipids and not hepatomegaly due to nuclear receptor activation that is described in this opinion publication. If one considers the pathology examples of hypertrophy presented in Hall et al. (2012), it is quite different from the pathology described for PFOS and PFOA. For clarity, the document should try to delineate the cellular components that are contributing to increased liver weight caused by PFOA and PFOS administration, if such a publication exists (e.g. how much of the liver is associated with protein/peroxisome proliferation increase versus lipid increase). Because the literature is not clear regarding what exactly in the liver is causing increased liver weight, studies documenting hepatic lipid accumulation should not be currently considered for POD. Given the recent opinion cited by Hall et al. (2012), it is

EPA-HQ-2016-005679 02/14/2017

recommended that studies documenting liver damage, such as ALT and AST elevation be currently used as the POD.

The documents often have redundancy in information, especially in regard to hormone effects (there are very similar write ups in sections about effects on thyroid hormone) and metabolic/cardiovascular disease risk factors (e.g. lipid endpoints).

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IV. RESPONSE TO CHARGE QUESTIONS

Charge Questions

Question 1. Please comment on the strengths, weaknesses, and characterization of the studies selected as key for quantification.

James V. Bruckner

PFOA-specific comments
The document's authors have done a good job describing and integrating the findings of the numerous studies in which liver weight gain was observed. Although there is a consensus about the effect and the dosage required to elicit it in different species, this reviewer does not believe it should be utilized, as described above. There are several clearly adverse effects such as elevated serum (hepatic) enzyme activities, focal hepatocellular necrosis, bile duct degeneration and fibrosis, etc. These effects are generally seen in response to relatively high PFOA doses, so the PODs will be higher than with liver weight increase. Alternatively, a human endpoint such as elevated serum cholesterol could be considered. See responses to Charge Question 3.

PFOS-specific comments
There have been a substantial number of well-conducted toxicological studies of PFOS. My major concern, as expressed above, is its potential to cause adverse effects in children. Other than that, PFOS doesn't appear to produce effects other than those anticipated from a repetitive, cumulative dose of an 8-carbon fatty acid.

Deborah A. Cory-Slechta

In general, it appears that, at least with respect to the animal studies, the choices are appropriate both in the case of PFOA and PFOS. The derivation of the RfDs/RfCs are based on studies of sufficient strength, duration and represent the most sensitive endpoints.

Having said that, in both documents, the reader is forced to that conclusion with no real assistance from the text itself. There is virtually no discussion of the strengths and weaknesses of the studies overall. Human study outcomes for the most part are simply enumerated, although an occasional statement will be made about a limitation (usually) of one of those studies. There is no discussion in the human studies of the power to detect effects, the sample sizes, etc. Much weight seems to be given to occupational studies in some cases, being used to essentially dismiss effects in a community cohort as the same effect was not seen in occupationally exposed workers, when in fact finding effects in a population with seemingly longer, albeit lower exposure levels actually makes the outcome more robust. Also, population studies with smaller sample sizes that nevertheless find significant effects are in fact more compelling and suggest robust effects which can be detected even with a small sample size. This deficiency is manifest in statements such as those in the PFOS document (p. 5-1) that 'in most cases the findings are suggestive and not conclusive of an effect'.

There is a bit more discussion of the animal studies in both documents, at least with respect to methods, but as with the human studies, there is little text addressing which studies represent stronger studies or what the weaknesses are. From these increase liver weight has been chosen as the endpoint from which to derive RfDs. This reviewer does not have an issue with that choice, as while it has been described as adaptive by some, it represents a response to an involuntary exposure with a direction of effect that is potentially associated with adverse consequences. The fact that it is reversible when exposure ends seems irrelevant as reversal of exposure is not happening in the human environment.

Jamie C. DeWitt

Strengths: The studies selected as key for quantification were generally well-conducted studies, employing a range of doses and sample sizes large enough for detecting statistical differences. Additionally, the doses associated with LOAELs for the identified critical endpoints were not associated with signs of overt or systemic toxicity in the animal models and nearly all of the studies measured serum and/or tissue concentrations of the parent compounds.

Weaknesses: No obvious experimental design weaknesses were noted in any of the studies selected as key for quantification.

Characterization: The studies selected as key for quantification for PFOA are all rodent studies while at least one study selected for PFOS quantification includes a non-human primate study. It is therefore surprising that the PFOA database does not include, as a study key for quantification, the Butenhoff et al. 2002 study of non-human primates. Additionally variability in putative mechanisms among species was not adequately addressed in the characterization of the selective studies, although all of the selective studies were descriptive and not mechanistic.

Jeffrey W. Fisher

PFOA and PFOS: Data bases are massive and both need to be updated. Several human studies and a few non-human primate toxicity studies are available. The authors need to explain why these studies are not adequate for causality (dose-response).

William L. Hayton

An advantage to assessment of health effects for both PFOA and PFOS is the large amount of published work that informs the topic. While the draft health-effects documents have summarized the results of many pertinent studies, the literature reviewed was not comprehensive, which projects an appearance of weakness. The documents do not state whether the intention was to include all relevant health-effects literature, or to be selective and summarize those studies judged to be most relevant. Such a statement at the beginning of the documents would be helpful; a cut-off date for the literature review would also frame expectations of readers. If the intention was to be selective, a description of selection criteria would help allay concerns of readers about papers that were not included. If the intention was to comprehensively review all the PFOA and PFOS health-effects literature, then it appears that

more work should be done to include omitted works. Public comments list a number of works to consider for inclusion.

A general, albeit minor weakness of the literature is that PFOA and PFOS serum concentrations in control animals were not measured for many studies – they were likely non-zero and, since there is no information on how high they were, it is possible that baseline health-effects metrics were affected and that dose-response relationships were affected, especially in the low dose range. It is perhaps worthwhile to mention this shortcoming somewhere in the health effects documents.

Matthew P. Longnecker

EPA may want to consider the article by AP Hall et al. 2012, about liver hypertrophy. The defense of increased liver weight as the POD (or a POD) could be strengthened by evaluating the evidence in the context of Hall's Figure 9, where evidence regarding hepatoxicity and toxic mechanisms are also considered. In this case, the possibility of an unknown mechanism exists that could be relevant to humans, and long-term exposure could have effects that have not yet been detected. See Hall page 986, where it defines adverse as: "…affects [response] to an additional environmental challenge". Thus, an adverse effect, via an unknown mechanism, by this definition is possible and has not been studied in animals or humans.

While AP Hall's article is not all that supportive of using increased liver weight as a point of departure (unless certain criteria are met), they are focused on animal studies, especially those done in rodents. If increased relative liver weight were to occur in a human population, I suspect that it would be considered an adverse outcome, whether or not there was evidence of hepatotoxicity or a specific mechanism. Note also that for PFOA, in monkeys, there was an increase in relative liver weight with chronic exposure (PFOA document, page 4-66), so increase in liver weight in the animal experiments may be relevant to humans.

An additional comment of relevance here pertains to whether the human data support hepatoxicity. While there are studies that report elevated liver function tests in subjects with higher serum concentrations of perfluorakyl substances, these elevations do not clearly support the presence of toxicity. Again, AP Hall's discussion of what constitutes evidence of hepatoxicity is relevant here, and takes into account the number of LFTs elevated, the specific LFTs involved, and the magnitude of their elevation.

Finally, as discussed at the meeting, for the PFOA document on page 5-23 ("RfD Selection"), and the PFOS document on page 5-26 ("RfD selection"), I suggest minor editorial changes to deemphasize the "consistency of response" point and instead focus a little more on how the RfD is robust to choice of POD endpoints. If the selection of RfD does not hinge on increased liver weight as a POD, it will be more defendable.

Transparency might be increased by saying why (more clearly, or more clearly by implicit reasoning) the Macon et al. 2011 study, in which the LOAEL was 0.01 mg PFOA/kg from GD10 to GD17, based on delayed mammary gland development, was not considered as a POD, and why the Hines et al. 2009 study, in which the LOAEL was 0.01 mg PFOA/kg from GD1 to

GD17, based on various outcomes, was not considered as a POD. The PFOS studies with low LOAELs were considered in the dose-response assessment (no suggestions for improvement there).

Angela L Slitt

My response is basically the same as my General Impressions above.

The documents provide a very thorough evaluation of PFOA and PFOS studies. It is logically organized, presenting findings in a way that the reader can understand the findings related to human, monkey, and rodents. The organization of the document makes allows the reader to easily find information about each species within the subchapters and summarizes key points in table form. PFOA is a well-studied compound, with a substantial amount of toxicokinetic and endpoint studies in rodents. Mechanistic data describing the role of membrane transporters to understand gender differences in PFOA elimination in rats is fairly well written. Little data exist regarding contribution of membrane transporters to PFOS disposition and elimination. The documents thoroughly describe species differences in PPAR-alpha signaling that might contribute to observed endpoints in rats, but not humans or monkeys. Overall, both documents are very thorough are provide a reliable basis for PFOS and PFOA evaluation.

For PFOA toxicokinetics, mechanisms of PFOA transport are important for understanding species differences in response to PFOA exposure, with focus placed on kidney. Figure 3-2 in the PFOA document does not adequately present the localization of renal transporters with relationship to their contribution to the urine compartment or renal reabsorption. A very nice diagram showing the subcellular localization of renal transporters presented by Klaassen and Aleksunes (Pharmacol Rev. 2010 Mar;62(1):1-96) clearly depicts the contribution of various transporters to filtrate or blood. This is an easier diagram to put PFOA elimination into context than the one presented. Contribution of membrane transporters to species differences in PFOA excretion Section 3 (specifically 3.4.1) would be put in better context if a table could be generated to compare Km and Vmax values for PFOA for various transporters, with specific focus on species information for OATps and OATs. Data regarding information on contribution for OATps in liver accumulation of PFOS and PFOA is lacking, with specific regard to species differences. As PFOS is a likely candidate for hepatic uptake transport, understanding a mechanism to explain species differences in hepatic effects possibly due to difference in hepatic exposure is critical. Understanding impact species specific regulation of OATp expression in liver (e.g. whether species difference in PPAR-alpha signaling contributes) is also important in putting rodent distribution data into context.

Increased liver weight is considered to be a critical effect, but how increased liver weight relates to the observed human and monkey health effects needs to be further explained. In layman terms, if someone is walking around with an increased liver weight, is he or she at risk for disease? Will his/her life span be shortened? To increase transparency of the document, a more comprehensive explanation is needed to justify why increased liver weight should be considered as a critical endpoint for human health.

Use of humanized PPARα mice are a sexy tool to delineate species differences in effects associated with peroxisome proliferation. For transparency, the document should acknowledge the limitations of that model. Specifically, lack of response may not necessarily correlate to a lack of response for human PPARα because of species differences in binding to cogate DNA elements (e.g. a human receptor may have lower binding capacity to mouse DNA due to structural differences and species differences in co-activator/co-repressor interactions). Wording in the documents using these mice should acknowledge this limitation.

The documents often have redundancy in information, especially in regard to hormone effects (there are very similar write ups in sections about effects on thyroid hormone) and metabolic/cardiovascular disease risk factors (e.g. lipid endpoints).

Question 2. Please provide citations (and, where possible, pdfs or hard copies) for any references you suggest EPA consider adding to the document. Describe where you suggest these references be incorporated.

James V. Bruckner

PFOA-specific comments
Fabrega, F. et al. (2014). PBPK modeling for PFOS and PFOA: Validation with human experimental data. Toxicol. Lett. On line. (Hard copy available)

Stahl, T., Mattern D and Brunn, H. (2011). Toxicology of perfluorinated compounds. Environ. Sci. Europe 23: 38-60.

Hall, A. P., et al. 2012. Liver hypertrophy: A review of adaptive (adverse and non-adverse) changes- Conclusions from the 3rd International ESTP Expert Workshop. Toxicol. Pathol. 40: 971-994.

Bjork, J. A., Butenhoff, J. L., and Wallace, K. B. 2011. Multiplicity of nuclear receptor activation by PFOA and PFOS in primary human and rat hepatocytes. Toxicology 228: 8-17.

PFOS-specific comments
No additional references were located.

Deborah A. Cory-Slechta

For both PFOA and PFOS, the document should include a description of the process through which studies were identified and how they were processed for inclusion or not. It is not clear what the exact dates of the studies examined included, i.e., what the cut-off date was for these studies. This makes it difficult to evaluate whether there are missing studies. That said, this reviewer is not aware of any specific omissions in the peer-reviewed literature other than those that were discussed at the face-to-face meeting.

Jamie C. DeWitt

Granum, B., Haug, L.S., Namork, E., et al. 2013. Pre-natal exposure to perfluoroalkyl substances may be associated with altered vaccine antibody levels and immune-related health outcomes in early childhood. J Immunotoxicol. 10:373–379; Looker, C., Luster, M.I., Calafat, A.M., et al. 2014. Influenza vaccine response in adults exposed to perfluorooctanoate and perfluorooctanesulfonate. Toxicol. Sci. 138:76–88.

Any time the Grandjean et al. (2012) findings related to PFAS and vaccine responses are discussed, these references could/should be discussed as well as they report related findings in human populations. Although they also are confounded by multiple PFAS (as was the Grandjean et al. study), they lend additional support to immunotoxicity as an endpoint worthy of consideration. However, it is noted that these references were published after the cutoff date for consideration for inclusion in the document.

Lopez-Espinosa, M.J., et al. 2012. Thyroid function and perfluoroalkyl acids in children living near a chemical plant. Environ.Health Perspect. 120:1036–1041. This study is missing from the discussion of thyroid hormone disruption. It reports a positive correlation between hypothyroidism and PFOA in children from the C8 population aged 1-17.

Corsini E., et al. 2011. In vitro evaluation of the immunotoxic potential of perfluorinated compounds (PFCs). Toxicol. Appl. Pharmacol. 250:108-116. Corsini E. et al. 2012. In vitro characterization of the immunotoxic potential of several perfluorinated compounds (PFCs). Toxicol. Appl. Pharmacol. 258:248-255.These studies are in vitro/ex vivo studies of human-derived cells that provide evidence that in vitro measures of immunocompetence in mice may be relevant to the human experience.

Jeffrey W. Fisher

For completeness sake, at least, please update lab animal studies conducted since 2012.

William L. Hayton

A review of PFOA health-effects literature (GB Post et al. (2012) Environ. Res. 116: 93-117) provides an excellent, in-depth discussion of many issues covered in the PFOA health effects document. Consider citing this review in the document.

The literature on PFOA and PFOS toxicokinetics (Section 3) has been comprehensively covered in the health effects documents, with the notable omission of Wambaugh et al., Dosimetric Anchoring of *In Vivo* and *In Vitro* Studies for Perfluorooctanoate and Perfluorooctanesulfonate. Toxicol. Sci. 136:308-327, 2013. This paper informed a significant part of the health effects documents.

Commenter's have suggested a number of references to consider with regard to Section 4 Hazard Identification. Many recent publications report on toxicity associated with PFOA/PFOS exposure. For the Dose-Response Assessment (Section 5) it is desirable to focus on those toxicities that have occurred at the lowest PFOA/PFOS exposures. For PFOA, the literature that is used in Section 5 to determine an RfD was published prior to 2009 (Tables 5-8 – 5-11). The benchmark response chosen based on the Section 4 literature was a 10% increase in liver weight, which was the biological response that occurred at the lowest PFOA exposure; it was acknowledged that this response "… is a biomarker for systemic exposure in rodents, rather than a biomarker of adversity …" (p. 5-6). More recent studies of hazard have identified potential adverse effects that result from, or are associated with, PFOA exposures that are lower than the LOAEL for a 10% increase in liver weight. For example, adverse effects on fetal, neonatal and early childhood stages of development may occur at lower exposures than does liver weight gain, which suffers in addition from not being a biomarker of adversity, and which therefore raises a question about the validity of any RfD based upon it. Macon et al. 2011 reported an LOAEL for delayed mammary gland development of 0.01 mg/kg administered to pregnant CD-1 mice during GD10 – GD17. As this relatively brief exposure was well below that required for steady state, it is possible that had the dams been at steady state at the time of conception (about 9 weeks of exposure) a much lower LOAEL may have been observed; i.e., a much lower dose

rate at steady state would have produced the same exposure to the fetal pups as did the 0.01 mg/kg administered to the dams during GD10 – GD17. The steady state situation is more relevant to adverse effects in humans than is a brief exposure.

Matthew P. Longnecker

I suggest you include the following citation and include a discussion of the evidence presented: Paula I. Johnson, Patrice Sutton, Dylan S. Atchley, Erica Koustas, Juleen Lam, Saunak Sen, Karen A. Robinson, Daniel A. Axelrad, and Tracey J. Woodruff. The Navigation Guide—Evidence-Based Medicine Meets Environmental Health: Systematic Review of Human Evidence for PFOA Effects on Fetal Growth. Environ Health Perspect; DOI:10.1289/ehp.1307893 (in press and available through the journal's website).

Based on the meta-analysis in this paper, the evidence that PFOA is associated with lower birthweight is consistent. Thus, the rationale for not basing the POD on the human data needs to be strengthened, as noted above. The Johnson et al. report could be discussed in the section on anthropometric endpoints that begins on p 4-22.

The relationship between birthweight and PFOA or PFOS may be confounded because glomerular filtration (and hence excretion of the compounds) is proportional to birthweight, as discussed in:

Morken NH, Travlos GS, Wilson RE, Eggesbø M, Longnecker MP. Maternal glomerular filtration rate in pregnancy and fetal size. PLoS One. 2014 Jul 8;9(7):e101897

In the PFOA document, on page 4-18, you might want to also cite:

Taylor KW, Hoffman K, Thayer KA, Daniels JL. Polyfluoroalkyl chemicals and menopause among women 20-65 years of age (NHANES). Environ Health Perspect. 2014 Feb;122(2): 145-50.

The Taylor et al., like the Knox et al. report (already cited in the PFOA document) is from a large-cross sectional study. Both studies, in their discussion sections, note that the association of PFOA or PFOS concentration in serum with age at menopause could be expected because postmenopausal women have lost a route of excretion for the compound and will have higher serum concentrations on that basis. It would be worth noting this possible explanation in the PFOA document on page 4-18, and in the PFOS document on page 4-8.

Additional data are available on the potential carcinogenicity of PFOA:

- Steenland K, Woskie S. Cohort mortality study of workers exposed to perfluorooctanoic acid. Am J Epidemiol. 2012;176(10):909-17.
- Barry V, Winquist A, Steenland K. Perfluorooctanoic acid (PFOA) exposures and incident cancers among adults living near a chemical plant. Environ Health Perspect. 2013;121(11-12):1313-8
- Hall AP et al. Toxicol Pathol 2012:40:971-94. (About liver hypertrophy.)

The Steenland and Barry reports could be discussed in Section 4.1.2, on pages 4-28 and 4-29, respectively.

Angela L. Slitt

1) Evidence is presented for PFOA and PFOS as substrates for the related OATp1d1 in zebra fish. Establishing whether PFOS is an OATp transporter substrate is needed to better understand PFOS accumulation in liver. This study suggests that it might be. The following finding should be included in the PFOS document in Section 3.2.3 and the PFOA document in Section 3.0:
    a. Popovic M, Zaja R, Fent K, Smital T. Toxicol Appl Pharmacol. 2014 Interaction of environmental contaminants with zebrafish organic anion transporting polypeptide, OATp1d1 (Slco1d1).

2) This publication presents the finding that PFOS inhibits Pgp, Mrp1, and Mrp4 activity. The following finding should be included in the PFOS document in Section 3.2.3 and the PFOA document in Section 3.0:
    a. Dankers AC1, Roelofs MJ, Piersma AH, Sweep FC, Russel FG, van den Berg M, van Duursen MB, Masereeuw R. Toxicol Sci. 2013 Dec;136(2):382-91. Endocrine disruptors differentially target ATP-binding cassette transporters in the blood-testis barrier and affect Leydig cell testosterone secretion in vitro.

3) PFOS induced ABC transporters in grey mullets.
    a. de Cerio OD1, Bilbao E, Cajaraville MP, Cancio I. Gene. 2012 Apr 25;498(1):50-8. Regulation of xenobiotic transporter genes in liver and brain of juvenile thicklip grey mullets (Chelon labrosus) after exposure to Prestige-like fuel oil and to perfluorooctane sulfonate.

4) These are new publications regarding epidemiology findings for PFOS and PFOA exposure and serum lipids:
    a. Fitz-Simon N, Fletcher T, Luster MI, Steenland K, Calafat AM, Kato K, Armstrong B. Epidemiology. 2013 Jul;24(4):569-76. doi: 10.1097/EDE.0b013e31829443ee. Erratum in: Epidemiology. 2013 Nov;24(6):941.
    b. Starling AP, Engel SM, Whitworth KW, Richardson DB, Stuebe AM, Daniels JL, Haug LS, Eggesbø M, Becher G, Sabaredzovic A, Thomsen C, Wilson RE, Travlos GS, Hoppin JA, Baird DD, Longnecker MP. Perfluoroalkyl substances and lipid concentrations in plasma during pregnancy among women in the Norwegian Mother and Child Cohort Study. Environ Int. 2014 Jan;62:104-12.
    c. Fu Y, Wang T, Fu Q, Wang P, Lu Y. Associations between serum concentrations of perfluoroalkyl acids and serum lipid levels in a Chinese population. Ecotoxicol Environ Saf. 2014 Aug;106:246-52.
5) These are publications regarding PFOS exposure and hepatic steatosis:
    a. Lv Z, Li G, Li Y, Ying C, Chen J, Chen T, Wei J, Lin Y, Jiang Y, Wang Y, Shu B, Xu B, Xu S. Glucose and lipid homeostasis in adult rat is impaired by early-life exposure to perfluorooctane sulfonate. Environ Toxicol. 2013 Sep;28(9):532-42. doi: 10.1002/tox.20747. Epub 2011 Aug 24. PMID: 23983163 Select item 22484034

b. Wan HT, Zhao YG, Wei X, Hui KY, Giesy JP, Wong CK. PFOS-induced hepatic steatosis, the mechanistic actions on β-oxidation and lipid transport. Biochim Biophys Acta. 2012 Jul;1820(7):1092-101. doi: 10.1016/j.bbagen.2012.03.010. Epub 2012 Mar 28. PMID: 22484034 [PubMed - indexed for MEDLINE] Free Article
c. Bijland S, Rensen PC, Pieterman EJ, Maas AC, van der Hoorn JW, van Erk MJ, Havekes LM, Willems van Dijk K, Chang SC, Ehresman DJ, Butenhoff JL, Princen HM. Perfluoroalkyl sulfonates cause alkyl chain length-dependent hepatic steatosis and hypolipidemia mainly by impairing lipoprotein production in APOE*3-Leiden CETP mice. Toxicol Sci. 2011 Sep;123(1):290-303. doi: 10.1093/toxsci/kfr142. Epub 2011 Jun 24.

Question 3. The OW concluded that the human epidemiology data for PFOS/PFOA do not provide adequate quantifiable dose-response information for use as the basis of a candidate RfD because of uncertainty regarding the routes, levels and timing of exposures plus the confounding influences of other PFCs present in serum. Please comment of the OW characterization of the data.

James V. Bruckner

PFOA-specific comments
The document's authors have done a good job summarizing and accurately characterizing the epidemiology literature for various endpoints in Section 4.4 - Hazard Characterization. It is true there are a number of confounding factors that make estimation of PFOA exposures difficult. The EPA might consider, however, utilization of reverse dosimetry modeling. There is a reasonable body of data on serum PFOA levels, which could be used to estimate a range of PFOA exposures that would result in such internal doses.

PFOS-specific comments
I agree that human epidemiology data do not provide an adequate basis for calculation of a RfD or RfC. A reverse dosimetry modeling approach, however, could be used to estimate a range of PFOS exposures that could have resulted in measured body burdens. The human data might then be utilized in the risk assessment.

Deborah A. Cory-Slechta

It is not clear that such an assertion should be used in the construction of this document. It is not clear why the route of exposure should be raised to a concern in the calculations, in fact in the human environment, there are exposures from multiple routes, no doubt and thus this is consistent with human environmental exposures. Further, if there is data on serum levels, it should reflect that cumulative exposure across exposure routes. Indeed, at the end, the goal is to arrive at an RfD based on serum levels. There is, moreover, no guarantee that there is no contamination in studies in animals from food, glassware etc.

Furthermore, in many epidemiological studies in which mixed exposures are the norm, controlling for other exposures is utilized to address this concern and to therefore make conclusions about individual exposures. In point of fact, in every single human study, there will invariably be other exposures and not a single exposure, and thus this strategy essentially says that no human studies can ever be used for any risk assessments. The stated rationales for not using human data based on these statements is not adequate. This is why it is important as well to evaluate the strengths and weaknesses of each of the studies in terms of whether appropriate controlling for other known exposures was carried out and sample sizes sufficient etc. to arrive at some conclusions with respect to their ultimate usability in constructing RfDs.

Jamie C. DeWitt, Ph.D..

While the OW characterization of the epidemiological data for PFOA/PFOS is, technically, true, it also is somewhat misguided. Almost any epidemiological database will contain uncertainty regarding the routes, levels, and timing of exposures and will have confounding influences of other compounds. Very few epidemiological studies are free from these uncertainties, but when similar observations and conclusions are reached from multiple studies with these types of uncertainties, the database becomes useful for determining a candidate RfD or other value relevant to human health. What is particularly valuable about the PFOA/PFOS database is that it is relatively extensive in that it includes data not only from occupationally-exposed humans, but from people highly exposed to environmental concentrations of PFOA/PFOS and from people in the general population who have detectable concentrations of these compounds. Additionally, for establishing an RfD, do all of these uncertainties need to be absent? In other words, do animal studies used to derive RfDs lack these uncertainties?

What is missing from the OW characterization of the epidemiological data is a thorough evaluation concerning hepatotoxicity and developmental toxicity reported in human populations and how these endpoints are relevant to or related to animal studies.

Jeffrey W. Fisher

The use of non-human and human data is very important for interpreting exposure extrapolations from rats. I am not an epidemiologist so I cannot comment with authority on the epidemiology data for dose-response. Justify why human data are not suitable for use in the analysis of the health hazards of PFOA and PFOS.

William L. Hayton

There are a number of epidemiological studies that have been based on large numbers of subjects chronically exposed (over decades in some studies) to the subject compounds over a broad range of intakes. Steady state serum concentrations have also been available for quantification of the systemic exposure. While the route, levels and timing of the exposures may have been uncertain, the long half-lives of PFOA and PFOS in humans and the long periods of exposure to them indicate that 1) subject serum concentrations were generally at steady state, and 2) daily fluctuations in the amount and timing of the exposure would not produce much day-to-day fluctuation in the serum concentration of PFOA/PFOS. These consequences of the long exposure period and long half-life indicate that variability in the route and level of exposure would not have led to a measured serum concentration that was unrepresentative of the subjects' long-term average serum concentration. The serum concentration then should be relatively stable over time and it should reflect an integrated measure of the individual's exposure to PFOA and PFOS.

The serum concentration is a quantitative measure of systemic exposure to the subject chemicals, and is arguably a better metric of exposure than are intake rate. The over-all rate of intake (R) that produces a particular steady state serum concentration (Css) can readily be

calculated from the clearance (CL) of the chemicals, which is about 0.08 mL/d/kg body weight: R = Css x CL. The calculated rate of intake would represent all intake routes.

Confounding influences of other PFCs and indeed other chemicals and life-style factors such as smoking, diet, alcohol use, etc. would have to be considered, as is generally the case with epidemiological studies. Methodology exists for dealing with such influences.

Thus it appears that the epidemiological results should be used in the RfD determination. Their strength is that uncertainties associated with extrapolation from laboratory animal studies are avoided. Health effects that are positively associated with serum PFOA/PFOS concentration and that are observed in large populations of subjects should seriously be considered as potentially arising from PFOA/PFOS exposure. If mode of action studies in lab animals or in vitro studies support a cause-effect relationship, then the threshold serum concentration could inform the calculation of the RfD.

Matthew P. Longnecker

As noted in the General Impressions section above, the human studies with data on plasma or serum concentrations of PFOA and PFOS, especially for several categories of such levels, could be used to estimate dose-response information. However, there are other reasons why the human data may not be useful for setting the RfD (see above). Either PK or PBPK models might be useful for estimating the dose that human are exposed to; an advantage of a PBPK model is that it could incorporate information about routes and timing of exposure. Estimates of the contribution of various routes are available (e.g., Haug et al. 2011; Lorber & Egeghy 2011), and exposure trends could be assumed and evaluated in sensitivity analyses. Some occupational studies had data that allowed an estimate of serum levels, or measured them directly. Several reports show estimated exposure based on serum concentrations of PFOA or PFOS (Locissano et al. 2013; Lorber & Egeghy 2011; Thompson et al. 2010). With respect to confounding, the assessment of how likely this is could be informed by: 1) the correlation of serum concentration of PFOA, PFOS, and other compounds of this type in a particular study population (or in a series of studies), and 2) whether the other compound(s) has been associated with the particular outcome being considered. If the correlation is low or the other compound has not been associated with the outcome, concern about confounding may not be strongly justified. Without additional consideration of data that address these points, it may be premature to assume confounding would be a problem.

Angela L. Slitt

Strengths of the studies: Several studies, which all demonstrate a positive association between serum PFOA and/or PFOS and cholesterol or LDL levels are based on drinking water as a route of exposure. These studies are in agreement with Nelson et al., 2010, which was analyzing data from the 2003-4 NHANES study. Steenland et al., 2009 (*Environ Health Perspect*. Jul 2009; 117(7): 1083–1088) as part of the C8 Health Project collected data on 69,030 subjects with findings that serum PFOA was higher for males, those consuming local vegetables, and those using well water rather than public water, and *lower for those using bottled water.* The estimated response rate for participants >20 years of age was 81% and mean serum PFOA

concentration was 83 ng/l. Subjects were eligible to participate in the C8 Health Project if they had consumed drinking water for at least one year before 3 December 2004 supplied by Little Hocking Water Association (Ohio), City of Belpre (Ohio), Tuppers Plains Chester Water District (Ohio), Village of Pomeroy (Ohio), Lubeck Public Service District (West Virginia), Mason County Public Service District (West Virginia), or private water sources within these areas that were contaminated with PFOA. Subjects were also eligible if they could document that they had either worked in a contaminated water district or went to school there for at least one year. From this population, which the route of exposure is considered to be primarily via drinking water, serum lipids were analyzed with regard to PFOA levels and a positive correlation was observed for all serum lipids except HDL. Frisbee further characterized this cohort, analyzing 12, 476 children and adolescents included in the C8 Health Project, finding an increase in total cholesterol.

A recent epidemiology study (Fitz-Simon et al., 2013), not included in the current documents, described positive associations between PFOA and PFOS in serum LDL cholesterol. This study examined a study population that consisted of 560 adults living in parts of Ohio and West Virginia where public drinking water had been contaminated with PFOA. They had participated in a cross-sectional study in 2005-2006, and were followed up in 2010, by which time exposure to PFOA had been substantially reduced. Overall, the findings demonstrate a positive association between serum PFOA and PFOS levels and serum and LDL cholesterol.

Weaknesses: The studies did not appear to analyze PFOS or PFOA levels in drinking water from the participants analyzed and did not analyze data based on the length of exposure.

Question 4. Please comment on the transparency and characterization of the epidemiological data.

James V. Bruckner

PFOA-specific comments
See comments above.

PFOS-specific comments
The document's authors have done a good job describing and summarizing the designs and findings of the epidemiology studies.

Deborah A. Cory-Slechta

The PFOA document in particular and to some extent the PFOS document present all of the epidemiological studies but do not actually evaluate them; there is not a consistent indication of individual strengths and limitations of the studies, failures or not to adequately control potential confounding variables. Furthermore, there is no 'power analysis' type of evaluation, i.e., some of these studies included very small sample sizes and thus their power to actually detect effects may be limited, and yet they all appear to be weighted basically the same, i.e., studies with very small sample sizes with obviously extremely limited power to detect any effects appear to be considered the same as those with extremely large sample sizes. Studies with small sample sizes that nevertheless do find an effect of PFOA or PFOS actually suggest a robust type of effect.

The discarding of positive associations in human epidemiological studies because they do not produce frank clinical disease seems inappropriate and inconsistent with other EPA documents. For example, p. 4-3 in the PFOS document states that only a small number of ALT values were outside the normal range making the results difficult to interpret in terms of health. Physiological changes that are moving in the wrong direction, even if sub-clinical at the time, are still adverse effects. Are actual clinical diagnoses required for an adverse effect? This is especially the case given that the ranges of normal across populations are extremely broad.

The latter also raises the question of the cumulative toxicity of PFOA and PFOS and whether any consideration is being given to this.

Another such example is in the PFOS document, where it actually refers to a statistically significant, but not toxicologically significant effect (p.4-38); what does that mean? Also, p. 5-4 appears to dismiss any changes in thyroid function since no evidence of clinical hypothyroidism actually occurred. This whole approach with the human studies seems quite inconsistent with the reliance on increased liver weight in the absence of clinical pathology as the endpoint in the human studies.

Jamie C. DeWitt

It is not obviously or abundantly clear how the OW characterized the epidemiological data for either PFOA or PFOS. The studies were well-described, but the contribution of particular studies

to the overall assessment was not. The results of studies described in the hazard characterization section (4.4) need to be better characterized. For example, in the PFOA risk assessment:

- An increase in serum lipids associated with PFOA/PFOS exposure in humans is discussed as a risk factor for cardiovascular disease in humans; however, no evidence of increased cardiovascular disease has been observed in human populations. Additionally, serum lipids typically are decreased in animal models after PFOA/PFOS exposure, which is thought to be associated with/typical of exposure to agents that activate PPARα. If humans are known to respond to PPARα activators (i.e., fibrate drugs), why would the results between humans and animal models be discordant? This should be discussed.
- Several epidemiological studies reporting changes in liver enzymes clearly state that the clinical relevance of the changes in enzymes is unknown. Therefore, stating that the human studies "suggest effects on the liver as indicated by increases in liver enzymes" amounts to a mischaracterization of the data.
- No direct evidence of hepatotoxicity has been reported in epidemiological studies. This should be discussed.
- More in-depth characterizations are needed for the additional sections of the hazard characterization, with the exception of the thyroid section, which was well-described.

For example, in the PFOS risk assessment: Similarly to the PFOA risk assessment, the hazard characterization section needs to better discussion differences and similarities between effects reported in humans and effects reported in animal models.

Jeffrey W. Fisher

I am not an epidemiologist, but it appears to be adequate. Better characterization of the pros and cons of the human analyses and interpretation of the outcomes would be helpful.

William L. Hayton

The characterization of the individual epidemiological studies presented seems to be adequate. Public comments have identified the need to distinguish positive and negative associations with statistical significance, which seems to be a fair criticism. As noted in the response to Question 2, there are relevant studies that have not been described in the health-effects documents that ought to be considered and this includes some epidemiological studies. Most of the cited epidemiological studies have focused on healthy adults – workers exposed occupationally, residents of communities with or without contaminated water. These populations might be expected to be less sensitive to adverse effects than would early life stages and particular disease populations. Studies of potentially more sensitive populations would be desirable. The Frisbee et al. (2010) study of children 1-11.9 years and adolescents 12-17.9 years showed significant positive associations with serum lipid levels. Studies such as this one would be informative.

Matthew P. Longnecker

Please see the long paragraph above, under General Impressions, and some of the comments in response to item #2 above. Another point that the authors may want to consider is that studies that examine external exposure in relation to health outcomes may have special advantages in the case of PFOA and PFOS. While in general it is considered best to have a measure of exposure that is based on a biomarker of internal exposure, this may be problematic for several outcomes for PFOA and PFOS, because of the possibility of confounding or reverse causality that would not be an issue if an external estimate of exposure were used. For example, in Steenland K, Zhao L, Winquist A. A cohort study of workers exposed to PFOA. Occup Environ Med. 2014 Jun;71 Suppl 1:A55, when an external estimate of exposure was used for the Washington Works employees, no association with elevated cholesterol was found. The Viera et al. (2013) results are based on external estimates of exposure, whereas the similar study by Barry et al. (2013) are based on serum levels or estimates based on serum levels. The fact that association with kidney cancer is present in the Viera study decreases concern that the association was due to reverse causality. Steenland et al. 2012 used an external estimate of exposure to study cancer mortality and also found an association with kidney cancer. Lundin et al. (external estimate of exposure) had no cases of kidney cancer, though their study was also small.

Angela L. Slitt

The epidemiology data is well described and a thorough read. The data would be put in better context for the reader if there are average serum concentrations or ranges for the studies summarized in tables in addition to other key pieces of information.

A recent publication should be included in the document for consideration. Simon N, Fletcher T, Luster MI, Steenland K, Calafat AM, Kato K, Armstrong B. Epidemiology. 2013 Jul;24(4):569-76. doi: 10.1097/EDE.0b013e31829443ee.

Question 5. The OW has concluded that the cancer classifications for PFOA and PFOS are most consistent with respective classifications of suggestive evidence for carcinogenicity as described the EPA Guidelines for Carcinogen Risk Assessment (pp. 2-56, 2-57). Please comment on the strengths and weaknesses of this classification.

James V. Bruckner

PFOA-specific comments
I agree with EPA's choice of "Suggestive Evidence for Carcinogenicity." Epidemiological findings in occupationally-exposed and general populations to date are equivocal. Increases in Leydig cell tumors and liver adenomas have been reported in high-dose male rats. Increased incidences of pancreatic cell hyperplasia/adenomas and ovarian stromal hyperplasia/adenoma have been observed in female rats. More studies are necessary to confirm/expand these findings, and to assess carcinogenic potential in other species. Most mutagenicity and genotoxicity assays have been negative. Thus, there is some, but not undue cause for concern about the human carcinogenic potential of PFOA.

PFOS-specific comments
The document's authors have adequately and convincingly presented evidence for classifying PFOS as "suggestive of carcinogenicity."

Deborah A. Cory-Slechta

The classification of both PFOA and PFOS evidence for carcinogenicity as suggestive seems consistent with the clear limitations in the available data bases. In addition, the animal studies are limited to one species and mutagenicity does not occur in response to PFOA.

Jamie C. DeWitt

This classification is appropriate for both PFOA and PFOS given the epidemiological evidence, which is somewhat limited for PFOA and quite limited for PFOS. For PFOA, there is an association between kidney and testicular cancer, but there are limited data in animal models for these cancers and there is uncertainty that the mechanism of PFOA-induced carcinogenicity in animal models is applicable to humans. Studies of PFOS have the same limitations, but epidemiological studies have failed to find an association between PFOS exposure and cancer.

Jeffrey W. Fisher

I did not review the cancer studies for PFOA and PFOS.

William L. Hayton

The classification of "suggestive" is not unreasonable. The epidemiological studies, while showing apparent associations between PFOA exposure and cancer incidence in testicle and kidney as well as other tissues, do not provide a cause-effect relationship. However, they certainly do raise a concern about the carcinogenicity of the subject substances. Studies in animals have demonstrated conclusively that PFOA causes liver cancer in rats but the MOA that involves PPAR activation is absent in humans and it has been concluded that PFOA and PFOS cannot be carcinogenic in humans via this mechanism.

An EPA SAB panel (2006) consideration of this question resulted in a majority of the panel members favoring a classification of "likely to be carcinogenic" for PFOA. Board members acknowledged the PPAR MOA argument against causation of cancer in humans, but also found evidence that liver cancer in rats administered PFOA may also have had a MOA independent of PPAR activation. Recent epidemiological studies have added to the weight of evidence for an association between PFOA/PFOS exposure and cancer. Therefore a classification of "likely" is also not unreasonable to this reviewer. Lacking expertise in the nuances of applying the EPA's classification scheme, it is difficult for this reviewer to argue in favor of either "suggestive" or "likely".

Matthew P. Longnecker

The classification as "suggestive evidence for carcinogenicity" for both PFOA and PFOS is consistent with the guidelines put forth in the EPA Guidelines for Carcinogen Risk Assessment (2005). There are few pertinent data, including some suggestive but weak human evidence. There is clearly not enough evidence to classify these agents as likely human carcinogens.

Angela L. Slitt

Overall, the assessments for each PFOS and PFOA appear to be consistent with the EPA Guidelines for Carcinogen Risk Assessment (U.S. EPA, 2005a).

Strengths: Both classifications use evidence from human studies as guidance.

PFOS: The limited data that exist regarding PFOS and cancer were presented, the classification for PFOS under the EPA Guidelines for Carcinogen Risk Assessment (U.S. EPA, 2005a) is currently consistent with the suggestive evidence of carcinogenic potential descriptor. This assessment is reasonable, given that it is based on two studies that show a slight increase in adenomas that occurred in males and females.

PFOA: There is conflicting evidence regarding PFOA exposure and cancer risk. However, several human studies have found associations between PFOA exposure and elevation of cancer of the bladder and kidney. This is also supported by a chronic bioassay in rats, which demonstrated that PFOA was tumorigenic.

Question 6. Significant interspecies differences in pharmacokinetics exist for both PFOA and PFOS. Adjusting for interspecies differences was an important step in developing candidate RfDs given the totality of the human and animal data. Please comment on the strengths and weaknesses of the pharmacokinetic model adjustments to accommodate the impact of albumin binding and renal tubule transporters in determining average serum values.

James V. Bruckner

PFOA-specific comments
The adjustments made to accommodate the influence of albumin binding and saturable renal tubular resorption of PFOA seem reasonable. I would defer, however, to someone with more experience in providing for these processes in PBPK models.

PFOS-specific comments
The PBPK model adjustments to estimate human equivalent doses appear to be appropriate. I defer to someone more qualified on the subject.

Deborah A. Cory-Slechta

This falls outside my area of expertise and therefore no significant comments are provided. However, at the face-to-face meeting there was significant discussion regarding the modeling, including clarifications from Dr. Wambaugh that were found by those panel members with expertise to clarify these issues and these particular issues were considered adequately addressed.

Jamie C. DeWitt

Several PK models have been reported in the literature for these compounds and are relatively well described in the documents. The documents assert that the existing PK models do not consider the impact of renal tubule transporters and albumin binding; while, many of the existing models appropriately predict serum concentrations in humans and other species, but they are mostly based on empirical models. Please explain the weaknesses of such empirical models.

Additionally, numerous studies for both compounds report serum and tissue concentrations in humans and other species, which can be compared to existing models. Both documents present a revised model that amounts to a reanalysis of data from studies that report serum concentrations. A more thorough discussion of the improvements made by the reanalysis is needed to better understand if the improved model adequately estimates or predicts the clearance rate and other parameters for which confidence is low. Alternatively, the publication (Wambaugh et al., 2013) that thoroughly describes the reanalysis could be referenced.

Jeffrey W. Fisher

Serum protein binding: Both PFOA and PFOS are highly bound in serum proteins across species, thus model adjustments seem trivial for interspecies extrapolation. Steady-state conditions can be assumed to estimate the free fraction (e.g., 2% based on paper by Han et al., 2003 for humans). I did not find a discussion about the half-life of serum proteins, which may have some influence on the 'apparent' serum half-life of PFOA and PFOS. The estimated fraction of free PFOA or PFOS is important for describing urinary and fecal elimination in rats (and other species) and the plasma concentrations of total PFOA and PFOS. Thus, the model predicts total PFOA and PFOS in serum or plasma, but the free fraction estimates drive the gradual clearance of total BPA from plasma or serum by describing clearance of free.

Renal reabsorption: The renal reabsorption hypothesis involving species specific and sometimes gender specific transporters to describe the pharmacokinetic data represents sound judgment. This departure from normal allometric scaling is suggestive of active transport processes. Few PBPK models explicitly describe transporters with drugs or chemicals, although the field is moving in this direction. Thus, the approach used for PFOA and PFOS is adequate, that is, a hypothesis was evaluated by employing empirical PK-based kinetic analyses. Because the mechanistic details are missing for each species/gender, scaling of this biological phenomenon is not possible at this time. This is not a weakness, but represents the state of the science.

William L. Hayton

A very important strength of the documents is the attempt to deal with the interspecies differences in pharmacokinetics so that adverse effects across species are compared on the basis of internal, systemic exposure to PFOA and PFOS, instead of basing comparisons on the administered mg/kg dosages. PFOA and PFOS have complicated pharmacokinetics that have proven difficult to model. While a relatively simple one-compartment model appears adequate to analyze single, low doses, this model fails when it is extended to higher doses and repeated doses. Nonlinearities appear associated with saturable plasma protein binding and with saturation of transporters thought to be involved in the reabsorption of the compounds from renal filtrate.

A weakness of the pharmacokinetic model adjustments is the lack of robustness of the models. Despite the extensive efforts of talented pharmacokineticists, development of a model that scales across species and handles a range of dosages and a variety of administration routes has proven elusive. The two compartment model of Andersen et al. (2006) has formed the basis of the model used in the draft documents. The model incorporates saturable resorption of PFOA and PFOS from renal tubular filtrate. While protein binding is known to be saturable (fraction free increases with concentration), the model uses a species-specific but constant free fraction. Model parameter values for mouse, rat and monkey were used to predict reasonably well measured serum concentrations after a fixed daily dosing regimen, Tables 5-6 – 5-8 for PFOA and 5-8 and 5-10 for PFOS. This agreement between predicted and measured serum concentrations gives confidence that the model-calculated AUC values and final serum concentrations associated with adverse health effects (or in the case of liver weight, biological marker of exposure) are realistic and a basis for estimation of RfD. While the model used

appears adequate for the purpose, the model parameters that were used have some markedly non-physiological values. (Information subsequently provided at the reviewers meeting explained some of the departure from expected physiological values, as discussed in a following section.)

Matthew P. Longnecker

For PFOA and PFOA, the MCMC model results (predicted final serum value) were compared to the measured final serum values, and the agreement was fairly good. For PFOS, the MCMC model results were compared to those from Loccisano et al. (2012b) and were found to be similar, which is also reassuring. Because the PBPK models of PFOA and PFOS are empirical, and have been shown to give results that agree reasonably well with observed data, the adjustments to accommodate the impact of albumin binding and renal tubule transporters are not critical. More data on albumin binding and renal tubule transporters might allow improved understanding of the pharmacokinetics of these compounds, but may not necessarily cause substantial improvements in the empirical predictions from current models.

Angela L. Slitt

The current weakness of the models is that data on species differences in PFOA and PFOA for various key transporters is limited and the document is also using mRNA data for various transport proteins to explain gender differences in urinary elimination. First, with regard to PFOS accumulation in the liver compartment, it is necessary to compare affinity of human versus rat for OATp mediated transport. This alone is tricky because of species differences in OATps. If PFOS-induced liver effects are related to PFOS accumulation in liver, it is would be helpful to understand whether a lower affinity of human OATp1b1 and 1b3 compared to rat OATp1a1 predicts lower hepatic PFOS accumulation. More is known about PFOA, but a similar argument can be made for PFOA. In addition, more comprehensive, controlled assessment of renal transporter affinity for PFOA and PFOS is needed to better model the species difference in urinary elimination.

The document often speculates about PFOA or PFOS regulation of transporter expression, but some papers cited (Cheng and Klaasen) do not have enough data at the protein level to support whether these differences in transporter expression are the drivers of toxicokinetic differences between males and females.

Question 7. Table 5-5 in the PFOA document and Table 5-7 in PFOS document list the parameters used for the ORD pharmacokinetic models that provide the final serum and AUC values for calculating the internal dose point of departure for the RfD calculation. Please comment on the strengths and weaknesses of the selected parameters.

James V. Bruckner

PFOA-specific comments
Despite the complexities and unknowns involved in plasma protein binding and renal tubular functions (i.e., glomerular filtration, basolateral tubular excretion and resorption, and apical tubular excretion and resorption), it is necessary to: (a) simply model only for saturable tubular resorption; and (b) use a range, or distribution of parameter values consistent with existing kinetic data. Unfortunately, optimization sometimes results in selection of physiological parameters that are not biologically-realistic, or plausible.

PFOS-specific comments
The parameters used in the modeling are biologically plausible.

Deborah A. Cory-Slechta

This falls outside my area of expertise and therefore no significant comments are provided. However, at the face-to-face meeting there was significant discussion regarding the modeling, including clarifications from Dr. Wambaugh that were found by those panel members with expertise to clarify these issues and these particular issues were considered adequately addressed.

One unclear component of Table 5-7 in the PFOA document is the column labeled Species/Strain Used for prediction, which in every case is the same as the column labeled Species/Strain and is not otherwise adequately explained.

Jamie C. DeWitt

It is not clear that the parameters in Table 5-5 in the PFOA document and Table 5-7 in the PFOS document are from the Andersen et al. 2006 PK model or if they are parameters used in the reanalysis of the data. This needs to be better explained in both documents. Additionally, all of the units in the tables need to be explained and re-checked for accuracy.

Jeffrey W. Fisher

The authors should entertain the calculation of data derived AUC (e.g. Table 5-6) to compare to the model derived AUC, just as was done with measurement of total PFOA in serum. This works for the animal studies. The choice of using the empirical model over the more recent physiological models may be a weakness and our understanding of transporters advance.
The evolution of chemical-specific PBPK models for use in risk assessment and regulatory applications has repeated itself several times. This is, the first empirical non-physiological model(s) or PBPK models contain hypotheses generating ideas and later models test some of

these hypotheses, especially if additional experimental data become available. In the case of PFOA and PFOS, the EPA selected not to use the most recent PBPK models for PFOA and PFOS, but instead use a computational empirical based model (Andersen et al. 2006) that was the first attempt to quantitatively interpret the kinetics of PFOA and PFOS across species of laboratory animals. The authors did publish their model (Wambaugh et al., 2013). The authors chose not to use a human model because a lack of information for Bayesian analyses. The justification for their extrapolation methods should be stated and the published reference for the model should be cited.

Model parameter distributions (Bayesian analyses) appear to be biologically implausible in some cases, covering many orders of magnitude. The authors should discuss this issue and check the units of model parameters in Tables.

Both model parameters tables need to include a description of what the parameter represents and cite a figure. The figures showing the Andersen et al. 2006 model do not show all the model parameters and have different nomenclature.

The Andersen et al. 2006 paper is a critical paper offering a quantitative explanation for the PFOA and PFOS kinetic data sets.

William L. Hayton

In the "Pharmacokinetic Model Approach" sections of the documents, it is not made sufficiently clear that the parameter values in Table 5-5 (PFOA) and Table 5-7 (PFOS) were from re-fitting the published data, rather than using parameter values from the original literature reports.

PFOA Table 5-5, p. 5-12

Body Weight and Cardiac Output values are reasonable and typical.

k_a values for mouse and monkey seem extremely large; absorption half-lives would be on the order of 10 seconds, which is physiologically unrealistic. All of an oral dose would be absorbed within a minute, mimicking a rapid i.v. bolus dose. Serum concentration-time profiles may not be sensitive to these values, however so they are not disconcerting for the intended use of the models. The rat values appear reasonable.

V_{cc} values appear reasonable. The total steady-state volume of distribution value [$V_{ss} = V_{cc} \times (1 + R_{v2:v1})$] compares favorably with one-compartment V_d values for CD1 mouse, but V_{ss} values for the other columns (species) appear too large, due to the large $R_{v2:v1}$ values.

k_{12} values vary a lot across the columns, suggesting that k_{12} may be highly correlated with another parameter (e.g., $R_{v2:v1}$).

$R_{v2:v1}$ values also vary a lot across the columns.

T_{maxc} values are consistent across the columns; expressed in Gm/hr, they seem very large. For example, 2032 Gm/hr (4.91 moles x 414 Gm/mole) for the CD1 mouse. Even on a kg body weight basis could mouse renal tubules resorb 2 kg PFOA per hour? This maximum rate of resorption must far exceed the rate of filtration of PFOA at the glomerulus. (Clarification at the reviewers meeting explained this apparent departure from physiological reality. The units had been mis-specified in Tables 5-5 and 5-7. They were in fact micromole per hour

and micromolar for T_{maxc} and k_T instead of molar based. Thus T_{maxc} mouse value was 2 mg/hr, which is physiologically plausible.)

k_T values are the concentration in glomerular filtrate that half saturates the resorption transporters. Expressed in mg/mL, they seem large, much larger than the urine concentration that would be expected; e.g., for CD1 mouse, k_T is 15 mg/mL where free serum concentrations (Free x C_{serum}) would be about 0.3 µg/mL with 10 mg/kg in the mouse. So the transporter would not become saturated except at extreme doses. The value used by Andersen et al. (2006) for monkey was 0.00001 mg/mL. Unit specified in Tables 5-5 and 5-7 should be µM, not M.

Free fraction values measured in vitro are 0.01 or less at low PFOA serum concentrations (Table 3-1). The Free values for rat seem much higher than the measured values.

Q_{filc} is defined as a fraction of blood flow (renal or cardiac output?) to the filtrate (bottom of p. 5-11) but has units of flow in Table 5-5.

V_{filc} values are much smaller than the 0.01 L value used by Andersen et al. (2006), although Andersen et al. state that the model output is insensitive to this parameter and that their value was assumed.

PFOS Table 5-7, p. 5-15

Body Weight and Cardiac Output values are reasonable and typical.

k_a values for female mouse and monkey seem extremely large – see comment above for PFOA.

V_{cc} values appear reasonable. See comment above for PFOA.

k_{12} values vary a lot across the columns, suggesting that k_{12} may be highly correlated with another parameter.

$R_{v2:v1}$ values appear reasonable and consistent with other reports of V_{ss} values for PFOS.

T_{maxc} values are highly variable across the columns and seem much higher than physiological reality would allow. See comment above for PFOA.

k_T values are physiologically unrealistic and highly variable across columns. See comment above for PFOA.

Free fraction values have been measured in vitro and are 0.01 or less at low PFOS serum concentrations (Table 3-1, p. 3-3). The Free values in Table 5-7 are consistent with the measured values.

Q_{filc} is defined as a fraction of blood flow (renal or cardiac output?) to the filtrate (bottom of p. 5-14) but has units of flow in Table 5-7.

V_{filc} values are much smaller than the 0.01 L value used by Andersen et al. (2006), although Andersen et al. state that the model output is insensitive to this parameter and that their value was assumed.

While the parameter values for the pharmacokinetic models predict reasonable serum concentrations that generally agree with measured values (Tables 5-6 – 5-8 for PFOA and Tables 5-8 and 5-10 for PFOS), their high interspecies variability suggest that the models may be unreliable for prediction of internal exposures after other intake regimens and during a depuration phase.

Matthew P. Longnecker

Please see the answer to the previous question.

EPA-HQ-2016-005679 02/14/2017

Angela L. Slitt

The parameters included appear to be appropriate, but this lies outside of my area of expertise.

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Question 8. The volume of distribution (Vd) and half-life values are critical in the derivation of the interspecies uncertainty factor applied in derivation of candidate RfDs from a NOAEL, LOAEL or a BMDL. The available data for both values are provided in Section 3.5.2 and 3.5.3 of both documents. Please comment the strengths and weaknesses of the values selected.

James V. Bruckner

PFOA-specific comments
The adult male rat data of Kemper (2003), from which the rat half-life and clearance (CL) were obtained, appear to be solid. It is reasonable to select the human half-life of 2.3 years reported by Bartell et al. (2010), as their study population included equal numbers of males and females. Division of the rat CL by the human CL to yield a value of 219 is fine. I did not examine the publication of Bartell et al. (2010) to evaluate their data or methodology used to derive a human half-life of 2.3 years. Therefore, I am uncertain about its accuracy.

PFOS-specific comments
I would again defer to someone with more expertise.

Deborah A. Cory-Slechta

This falls outside my area of expertise and therefore no significant comments are provided. However, at the face-to-face meeting there was significant discussion regarding the modeling, including clarifications from Dr. Wambaugh that were found by those panel members with expertise to clarify these issues and these particular issues were considered adequately addressed.

Jamie C. DeWitt

While the overview of the individual studies that calculated Vd and half-life for each compound was detailed and complete, the rationale and analysis concerning *why* particular values were selected were insufficient. Additionally, as addressed in Charge Question 6, the rate of clearance/elimination likely contributes to the differences in half-life that are not associated with differences in the Vd. Therefore, a 3-fold uncertainty factor for species differences in pharmacodynamics (UF_A) was utilized for both compounds. What was the justification for using a UF_A of 3? The section on UF application needs a more thorough discussion regarding the choice of this value given differences in clearance. If the section on model adjustment (a suggestion in Charge Question 6) is better described, this comment may no longer be applicable.

Jeffrey W. Fisher

The use of this non-compartmental method should be justified. Why not use a PBPK model? Assuming steady state in the humans does allow for calculation a human equivalent serum concentration associated with a laboratory animal concentration. In what region of the exposure-dose range would nonlinearity occur in humans? Some type of discussion is needed

about the assumptions of this methodology and why it was used. I would like to see statements about if the NOAEL, LOAEL, and a BMDL doses are in the linear range for kinetics.

The authors should use the Bayesian analysis for animal studies to inform the UF. Use percentiles to explore Vd and half-life to support UF values. I did not see any attempt to use distribution information generated from the model beyond the central tendency or mean values. Please state why this is the case. It seems that the distribution information generated from the Bayesian analysis could be used to support UF development.

William L. Hayton

PFOA-specific comments

For male rat, the Kemper (2003) study appears to be the best source of pharmacokinetic parameter values, which were obtained by a model-independent analysis of serum concentration-time data from rats that were dosed by oral gavage at dosages of 0.1, 1.0, 5.0 and 25 mg/kg. In addition, there was a 1.0 mg/kg dosage administered intravenously, and a 0.1 mg/kg oral gavage dose with an extended sampling time. Each treatment used four animals. The CL and $t_{1/2}$ values appeared to be independent of dosage and route of administration. It would therefore be reasonable to average all 6 mean values for each parameter to give an over-all mean of 24 determinations. The average (n=24) values for male rat were CL = 0.0209 L/kg/d and $t_{1/2}$ = 7.83 d. These values can be used to calculate a V_d value ($t_{1/2}$ x CL / ln 2), which is 0.236 L/kg.

It is not apparent on p. 5-20 why a V_d value of 0.17 was used with half-life to calculate CL_{rat} when Kemper (2003) reported CL values and not $t_{1/2}$ values. (At the peer review meeting, it was clarified that the data of Kemper (2003) were re-analyzed and as a result the parameter values in the health effects documents differ somewhat from those published with the data in the original reports.)

The CL_{human} value was taken to be 0.00014 L/kg/d. There are no direct measurements of this parameter. Thompson et al. (2010) assumed that the intake rate of PFOA for subjects using PFOA-contaminated water was 91% of the PFOA in 1.4 L/d of water. This intake rate was used along with a PFOA half-life of 2.3 years to calculate a V_d value of 0.17 L/kg. This is the same value that was used in the health effects document for the rat (p. 5-20). The V_d values available in mouse, rat and monkey are about 0.2 L/kg, so the $V_{d,human}$ set at 0.17 L/kg is not unreasonable but it lacks the certainty of the rat V_d value.

The health effects document used a $t_{1/2}$ for PFOA in human of 839.5 d (2.3 years), which seems to be toward the low end of the range of values that have been reported. Along with Vd = 0.17 L/kg one arrives at CLhuman = ln 2 x 0.17 / 839.5 = 0.00014 L/kg/d.

The ratio CL_{rat} / CL_{human} calculated using the mean CL_{rat} from Kemper (2003) would be 0.0209 / 0.00014 = 149, which is about twice the value calculated on p. 5-21. This difference arises from the calculation of CL_{rat} using the $V_{d,human}$ and a half life of 11.5 d instead of using the CL_{rat} directly from Kemper (2003). The mean half life from Kemper (2003) was 7.8 d.

The CL_{mouse} / CL_{human} ratio is accurate, using Lou et al. (2009) data. A calculation for monkey is not shown.

PFOS-specific comments
Chang et al. (2012) appears to be the best source of pharmacokinetic parameter values for mouse, rat and monkey. Butenhoff and Chang (2007) is given as the reference for a 48-day half-life in rat; this is a final report, internal to 3M. The Chang paper gives half-life values for male and female Sprague-Dawley rat at 2 mg/kg and 15 mg/kg. The average V_d for the four groups of three/group was 0.71 L/kg. This is higher than the 0.23 L/kg value used in the draft document (p. 5-23). The 0.71 L/kg value is also higher than values for mouse, monkey and human, which are closer to the 0.23 L/kg value used in the draft document. The draft document ought to acknowledge this difference; it may be that the value in the 3M report is lower than the published value; Chang was a co-author for both sources. The Chang et al. (2012) paper gives CL_{rat} values that are 0.0051 L/h/kg for female (similar for 2 and 15 mg/kg doses) and for males, 0.022 and 0.011 L/h/kg for the 2 and 15 mg/kg doses. A single average value for CL_{rat} would be 0.011 L/h/kg, about 3 times the value used for the UF_A calculation in the draft document. The male value is about 2-3 times the female value and it may be appropriate to calculate a different UF_A value for each sex. Using the single CL_{rat} averaged across two doses and both sexes (0.011 L/H/kg) would give a CL_{rat} / CL_{human} ratio of 0.011 / 0.000081 = 135 and a UF_A = 407, substantially higher than the value of 123 in Table 5-15.

The UF_A values calculated for mouse and monkey appear to be in line with the literature values for PFOS CL values in these species.

Matthew P. Longnecker

The PBPK model of Loccisano et al. 2013 (for humans) can be used to calculate a volume of distribution for PFOA of 177 ml/kg, which is very close to the value of 170 ml/kg based on Thompson et al.'s 2010 one-compartment model . For PFOS, the corresponding value from the PBPK is 280 ml/kg, compared with the value of 230 ml/kg used in the Health Effects Document. This 22% difference could have an impact on some calculations. (Note: the PBPK model-based volumes of distribution were calculated by Marc-Andre Verner of the University of Montreal. He had calculated these values in the course of a separate project.)

For humans, the half-life data all depend on the assumption that ongoing exposure is negligible compared to baseline exposure, a reasonable assumption in most of the populations used to estimate half-life. While the Seals et al. (2011) gave estimates that were slightly different for PFOA in some cases, the methods employed in this study were not as strong as for Bartell et al. (2010) or the Burris et al. studies (2000; 2002). The agreement within species for the half-life estimates for PFOS are reassuring. The animal data on the half-life of PFOA are relatively sparse (2 rat studies that agreed reasonably well, 1 mouse study, 1 monkey study). For PFOA, the UF_As and RfD that were calculated based on the half-lives (expressed as clearance) would not have been substantially altered by alternate choices for specific values. The same is true for PFOS.

Angela L. Slitt

Strengths of the available data is that for the several species thorough evaluated, the half-life values are very consistent. For example, the several human studies cited report a range in calculated PFOS half-life in humans to be 4.1-8.67 years, two studies putting monkeys at 110-132 days, and rat generally has a narrow range with 3 out of 4 values provided ranging from 39.8-48.2 days for PFOS. An inconsistency is the Chang et al., 2012 describing a half-life of females of 66.7 days when in general female rodents may have faster elimination of PFOS.

Question 9. A variety of endpoints and studies were used to compare points of departure and the resultant RfDs for both PFOA and PFOS. In addition, comparisons were provided across RfD outcomes based on the model outputs compared to those for the NOAEL, LOAEL and BMDL points of departure. The range of candidate RfDs derived from the different points of departure is fairly narrow. Please comment on the strengths, weaknesses and transparency of this analysis.

James V. Bruckner

PFOA-specific comments
The procedure used to calculate PODs adheres to standard EPA guidelines and policy. The presentation of their derivation is clear, concise and transparent. It is certainly interesting that the range of PODs and resulting candidate RfDs is so narrow. Nevertheless, as discussed previously, I do not agree with their selection.

PFOS-specific comments
See my comments under General Impressions.

Deborah A. Cory-Slechta

While it is the case for both PFOA and PFOS that values from different points of departure are fairly narrow, the transparency of the analyses in neither case is clear. There is no rationale described even as to why these analyses were done on all of the studies, what was the primary study and how others related to that etc., i.e., this presentation does not follow the typical presentation format of IRIS documents in either its presentation of rationales and strategies, nor in the conclusions that it reaches. In both cases, it is only the single sentence indicating that modeling from one particular study will be protective of effects at other studies using higher exposures. This section in both documents needs introductory paragraphs that describe the specific strategy, choices of studies and the rationales for those choices.

As noted in response to Charge Question 3, the rationale for discarding the human epidemiological studies is not sufficient and requires rationale other than that stated and therefore, the question of using the human data remains open. As noted in response to Charge Question 1, in this reviewer's opinion, the increased liver weight can be justified as a departure point for assessment of RfDs, but as discussed at the face-to-face meeting, additional text supporting this choice is needed.

Jamie C. DeWitt

This particular section contained inadequate detail on why particular studies *were* or *were not* chosen. For example, immunotoxicity as an endpoint was not chosen for PFOS, based on "*in vitro* measures of immunocompetence on mice may not be relevant to the human experience and limited human data from epidemiology studies are inconclusive regarding the immunotoxicity of PFOS in humans"; however, the breadth of data from in vitro/ex vivo immunotoxicity studies for PFOS were not thoroughly discussed (please see Charge Question #2 for two additional in vitro studies).

For both compounds, an increase in absolute liver weight was selected as an endpoint as it was a common *effect* [sic] in both short and long term studies. However, the toxicological relevance of an increase in absolute liver weight was not discussed other than to indicate that it was a sign of altered homeostasis. Further, the co-occurrence of increases in absolute liver weight with other toxicologically-relevant endpoints (i.e., immunotoxicity and/or reproductive/developmental toxicity) is not a toxicologically valid justification for the use of liver weight as an endpoint for an RfD. Therefore, the analysis was not sufficiently transparent to deduce its relative strengths and weaknesses. Certainly, choosing an endpoint that occurs across species and occurs at relatively low doses will likely be protective of exposed humans; however, will it be a defensible endpoint? As currently written, the choice of this endpoint for an RfD is not adequately defended.

Jeffrey W. Fisher

I did not review the toxicity data.

William L. Hayton

For PFOA, a 10% increase in liver weight was selected as the metric for effect, which was "… not made based on toxicity but on the desire to find a common denominator against which to evaluate dose-response across studies and justified by the fact that other adverse effects accompanied the LOAEL for increased liver weight in some cases." The lowest serum concentration associated with an increase in liver weight was calculated for female mouse to be 20.33 mg/L (p. 5-16, PFOA document). These data are referenced to DeWitt (2008); this paper has only summary information on liver weights, all of which exceeded 20% weight gain, going as high as 70%; and it is not apparent in PFOA document how these liver weight gains were used to estimate an LOAEL for 10% liver weight gain.

Many of the animal studies of hazard assessment were conducted under conditions where the duration of the exposure was relatively short compared with the half-life, and steady state had not been achieved. It is not apparent how the NOAEL and LOAEL values from such studies were adjusted to account for the non-steady state situation. For example, the 20.33 mg/L PFOA concentration associated with a 10% increase in liver weight (Table 5-9) emanated from a 15 day drinking water exposure to 0.94 mg/kg/day that resulted in an average serum exposure of 20.33 mg/L (0 – 29.7 mg/L over 15 d, Tables 5-7, 5-9). For a fixed daily dose, the time to 90% steady state for mouse would be about 63 days (3.3 x half life, which was 19 days), and after 15 days the serum concentration would only be about 15% of its steady-state value. This seems to suggest that the RfD would have been over-estimated by a factor of 7, since the 0.94 mg/kg/day at steady state would have produced a serum concentration of about 150 mg/L, not 20.33 mg/L. This analysis is based upon the behavior expected from one-compartment model pharmacokinetics. As discussed on p.5-9 of the PFOA document, the steady-state serum concentration of PFOA is achieved in a much shorter time than one-compartment model kinetics would predict. Whether the target-site steady-state concentration of PFOA also occurs in a much shorter time than one-compartment model kinetics would predict (3.3 x half life) is apparently unknown.

EPA-HQ-2016-005679 02/14/2017

Matthew P. Longnecker

This part of the document seemed especially strong and transparent. The agreement between methods was reassuring. The weaknesses and assumptions were well discussed. Please see the minor editorial comment on this issue given for Charge Question 1, above.

Angela L. Slitt

The RfD Point of Departure was based on animal studies that include monkey and rat.

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Question 10. The RfDs for PFOS and PFOA are derived from the modeled steady state serum concentrations and their association with effects that include short term and longer term exposures with associated diverse effects. The studies considered included effects due to exposure durations that ranged from 11 to 182 days, and occur at comparable human equivalent dose (HED) levels. The current, draft RfDs do not include an uncertainty factor for study duration because of the apparent concordance HEDs despite duration differences. Given this pattern of response, is it appropriate to conclude that the candidate RfDs are applicable to both short-term and lifetime exposures?

James V. Bruckner

PFOA-specific comments
I do not believe it is appropriate to conclude that the candidate RfDs are applicable to both short-term and lifetime exposures. Steady-state is apparently achieved in monkeys within 4 - 6 weeks (Butenhoff et al., 2002). Steady-state likely takes considerably longer in humans. Thus, RfDs for shorter periods of exposure should be based upon results of studies of similar duration.

PFOS-specific comments
I do not believe the candidate RfDs, as calculated, are applicable to different durations of exposure.

Deborah A. Cory-Slechta

While initially believing that it was appropriate conclusion for PFOA and PFOS, based on the correspondences in RfDs across short and longer term exposure, discussion at the face-to-face meeting made clear that this approach is not reasonable and requires additional consideration.

Jamie C. DeWitt

This approach may be appropriate given the relative similarity of serum concentrations attained regardless of study duration, i.e., steady state in serum is attained after a relatively short period of exposure. This appears to be consistent across studies with various species of animal models. However, the document authors might need to reconsider given what we may or may not know about liver hypertrophy. In the Hall et al. (2012) paper on liver hypertrophy (discussed during the public meeting), increase in liver weight is an adaptive response that may not be adverse UNLESS weight increases >150% over a three month or longer period may. Following this large and prolonged increase in weight, the end result may be a hepatocarcinogenic response. However, none of the studies contained in the documents indicate that longer term exposures increase liver weight to this degree.

Jeffrey W. Fisher

The departure from K=CXT (Haber's law) should be based on the toxicity endpoints of concern and what is known about dose-exposure kinetics/responses for these chemicals and other chemicals that target the same endpoint, not that the HED values are comparable. The NAS

AEGL committee only considered primary irritation for inhaled chemicals as an endpoint that was independent of duration of exposure. There is an SOP if needed for reference.
The time to steady state should be included in a table for the lab animals. Toxicity studies conducted for less than 30 days (perhaps?) are not at steady state for the pharmacokinetics of PFOA. Thus the measured serum levels would be different than at steady state. The shorter the duration of the toxicity test, the more impact this could have on extrapolation to chronic exposures in humans. My personal preference would be to use PBPK models for all species and consider only long term exposures for extrapolation to humans.

William L. Hayton

This depends in part on how quickly the PFOA/PFOS concentrations at sites of toxicity come to steady state. Since the Vd for these chemicals is small (~ 0.3 L/kg) it seems likely that the concentrations in tissues rise in pseudo equilibrium with the rise in serum concentration. That said, the half lives are relatively long due to the very small clearance ($t_{1/2} = \ln 2 \times V_d / CL$). If one-compartment kinetics apply, then a guideline for time to 90% steady state is 3.3 $t_{1/2}$. For studies that expose animals for a period of time shorter than 3.3 $t_{1/2}$, the serum concentration would not be at steady state and the internal systemic exposure (serum concentration) would be less than what it would be if the exposure were longer than 3.3 $t_{1/2}$. This effect would seem to lead to overestimation of the intake rate that was associated with a particular internal exposure and associated biological endpoint. For example, the $t_{1/2}$ of PFOS in mouse is about 36 days and 3.3 $t_{1/2}$ is 120 days. Consider a 28-day exposure using a fixed daily dose that produced an LOAEL of "X" mg/kg/day. On Day 28, the body level would only be 42% of the steady state level, and the average body level over the 28-day period would be about 21% (approximating the increase as linear and not exponential). The true LOAEL would be 0.21 "X" mg/kg/day; i.e., intake of 0.21 "X" mg/kg/day would produce a body level at steady state that was the same as the average body level produced by X mg/kg/day administered over 28 days. The time to 90% steady state for a fixed intake rate is quite long; from the literature in the health effects documents, the times in the following table were calculated. From this line of reasoning, exposure times less than two half-lives begin to significantly overestimate intake rates associated with particular endpoints. This analysis is based upon the behavior expected from one-compartment model pharmacokinetics. As discussed on p.5-9 of the PFOA document, the steady-state serum concentration of PFOA is achieved in a much shorter time than one-compartment model kinetics would predict. Whether the target-site steady-state concentration of PFOA also occurs in a much shorter time than one-compartment model kinetics would predict (3.3 x half life) is apparently unknown.

	CL		Vd		$t_{1/2}$		Time to 90% steady state	
	[mL/d/kg]		[mL/kg]		[d]		[d]	
Species	PFOA	PFOS	PFOA	PFOS	PFOA	PFOS	PFOA	PFOS
Mouse	6.6	5	180	265	19	36	63	120
Rat - Male	23	16	273	947	8.4	40	28	92
Rat - Female	776	5.2	150	476	0.13	66	0.43	218
Monkey	6.3	1.4	190	238	27	121	89	400
Human	0.085	0.08	170	230	1378	2000	12.5 yr	18 yr

In addition, this line of reasoning may be incorrect if the assumption of one-compartment kinetics is incorrect. For multi-compartment models the serum concentration and target organ/tissue could come to their pseudo steady state levels relatively quickly while slowly equilibrating (deep) sites slowly approached steady state. Simulation with PBPK models for PFOS and PFOA may help answer this question.

Associated with the uncertainty introduced by exposures that were shorter than the time to achieve steady-state concentration at the target site is the exposure time required for the adverse effect to be expressed. While some adverse effects may occur immediately and directly in proportion to the concentration of PFOA or PFOS at the target site, other adverse effects may be slow to become manifest. These "indirect adverse response" behaviors are well known in the drug action arena; e.g., certain antidepressant drugs require several weeks exposure to the target site before the effect of the drug appears. This lag time is not associated with pharmacokinetics (time to steady state) but with indirect-response pharmacodynamics. It could be argued that uncertainty factors are needed for both pharmacokinetics (pre-steady state condition) and pharmacodynamics (or toxicodynamics) to account for possible indirect response behavior.

Matthew P. Longnecker

EPA might want to consider using an uncertainty factor for duration, for two reasons. First, the monkey data for PFOS used for the point of departure were from a study where the duration of exposure was relatively short-term relative to the half-life, and it appeared that duration of dose affected liver and other adverse outcomes detected at higher doses, and no monkey data were used in the POD for PFOA. Second, questions raised by Drs. Hayton and Fisher at the peer-review meeting made me less comfortable with the calculations that used average serum concentration derived from the AUC and duration of dosing to compare with humans, who are more likely to be near steady-state.

Angela L. Slitt

Yes, but this lies outside of my area of expertise.

Question 11. In addition to using the average serum values from animal studies to calculate internal doses for humans, the animal to human extrapolation can be accomplished by dividing animal average serum values by the human to animal clearance ratios to project a human average serum point of departure in units of mg/L serum. Please provide recommendations for applying uncertainty factors to the extrapolated average human serum values to determine serum-based thresholds that are protective for humans. A NOAEL expressed in average human serum units would be useful in interpreting NHANES population monitoring data.

James V. Bruckner

PFOA-specific comments
No comment.

PFOS-specific comments
No comment.

Deborah A. Cory-Slechta

In initial response to charge questions, I found it difficult to understand specifically what this charge question was asking for a response to: Does this refer to the data in Table 5-10 for PFOA? Wouldn't you include animal to human UF values at the least. Since the data for the studies listed in the Table is not clear as to their duration (columns are needed for this information, or add to the Study box), it is not clear whether a UF for study duration is warranted. It is not clear how sex differences are being accommodated in any of these.

At the face-to-face meeting, however, with some additional input from EPA, it was clear to all that there was no need to do such derivations from animal to human, which could instead be derived directly from the human data and thus presumably this is no longer an issue.

Jamie C. DeWitt

Would this approach take into account differences between animal studies that have a defined exposure duration and data from NHANES, where exposure duration is assumed to be continuous (although it may not be), if exposure duration does not appear significantly impact serum concentrations? Additionally, how would the half-life estimations from the Seals et al. (2011) study, which contained two half-life estimations based on concentration and time, impact this approach?

Jeffrey W. Fisher

Again, is the system linear in the exposure/dose ranges of interest? I would try to determine an UF by exploring a range of predicted human serum levels. Attempt to use 5,50, and 95% for animal serum concentrations with a 5,50, and 95% CL values in the animals and for the human perhaps use two CL values representing a high and low. The idea is to use as much information

as you can to determine the possible range of values. This will help guide the selection of uncertainty values.

William L. Hayton

This calculation is equivalent to dividing the animal dosage by the CLhuman, assuming that the animal serum concentration is at steady state ($C_{ss,animal}$) maintained by a constant dose rate (DR).

$$C_{ss,animal} / CL_{human} \div CL_{animal} = CL_{animal} * C_{ss,animal} / CL_{human} = DR / CL_{human}$$

This calculation would give the steady-state serum concentration in human that would be produced by the animal dose rate. (I will have to study this to understand the question; the calculation does not make sense to me.)

At the peer review meeting, the aim of this calculation was clarified. Authors desired a way to calculate a steady-state serum concentration ($C_{ss,human}$) that would result from the human equivalent dose rate (HED) administered until steady state. The appropriate calculation would be:

$$C_{ss,human} = HED / CL_{human}$$

Matthew P. Longnecker

The proposed division by animal clearance ratios does not make sense to me. The average serum values from animal studies is already taking pharmacokinetic variability in blood levels during the observation period into account, and human blood levels will be relatively constant. Thus, it would make sense to directly compare the POD estimated average serum concentrations from animal models to the blood levels in NHANES. With respect to uncertainty factors that would be need consideration for this approach, it seems that UF_H, UF_L (For LOAEL and HED_{LOAEL}), UF_D, and the component of UF_A that takes pharmacodynamics into account would all still be applicable.

Angela L. Slitt

This is outside of my area of expertise.

Question 12. Please describe any suggestions you have for improving the clarity, organization, and/or transparency of the draft documents.

James V. Bruckner

PFOA and PFOS-specific questions
See specific observations.

Deborah A. Cory-Slechta

While the EPA authors are aware of updates in the IRIS process, it might be very helpful to look at some of the new documents coming through that process for guidance as to the levels of critique and evaluation that are now included in these documents. They also include an introductory chapter focused specifically on the literature searches and literature that is included vs. excluded.

The Executive summary does not provide sufficient rationale and descriptions to lead a reader through the steps to what is concluded and reads more like an abstract than an Executive Summary. Since this may be the only section read by many reviewers, it is important that it provide a succinct journey through the process. Here again, the new IRIS documents (e.g., trimethylbenzene) could provide a useful template.

Chapters 3, 4 and 5 could each benefit from an opening paragraph describing what the section's goals are, and integration and conclusion sections at the end that establish the basis for the presentation in Chapter 5. Currently the Hazard Identification studies generally treat all studies as of equal strength/power, which is certainly not the case. These chapters should present that kind of critical and transparent assessment as it ultimately serves as the basis for decisions that are made.

The inclusion of sections on in Vitro data did not ultimately seem particularly relevant in the outcome for these compounds and could be significantly shortened to add more to Chapter 4 on study strengths and weaknesses. However, where pertinent, it would probably be more useful to break that section up and insert test where it follows an in vivo discussion.

Tables could be considerably improved and made far more useful to the reader for comparative assessments. As of now, they require going back and forth to the text to capture additional details of the studies, e.g., sample sizes, species etc. and could benefit the reader significantly with those additions. For the human assessments, it is equally important to include these details in the chapter as well as a column of study strengths and limitations.

While charge questions ask whether the appropriate studies were chosen as key studies, this reviewer does not remember that that term was even used in the documents, certainly no explicit mention was made as to which studies were considered key studies. This would seem to be a section that should be included in Chapter 4 more explicitly. Chapter 5 of both documents, more so PFOA, are confusing as almost all studies are subjected to modeling, for reasons that are

never presented in sufficient detail and simply followed by statements that a selected study (not really well presented in Chapter 4 as a selected study) will protect against other adverse effects.

Jamie C. DeWitt

The documents lack a critical analysis of differences between findings of epidemiological studies and findings of animal models. As stated in the comments to Charge Question #3, what is particularly valuable about the PFOA/PFOS database is that it is relatively extensive in that it includes data not only from occupationally-exposed humans, but from people highly exposed to environmental concentrations of PFOA/PFOS, and from people in the general population who have detectable concentrations of these compounds.

Critical to this analysis is a discussion of concordance and lack of concordance between human data and animal model data. For example, immunotoxicological findings appear to be consistent between humans and rodent models whereas serum lipids are not. How do these differences impact the overall confidence in the database and derivation of the RfD?

All of the sections related to the PK models developed by ORD need additional information for clarity and transparency. As written, it is not clear that the PK values presented throughout the document actually represent a reanalysis of existing data from studies that reported serum concentrations. The Wambaugh et al. (2013) study could be referenced to shorten this exercise as this publication provides details on the reanalysis of existing data.

Justifications for choosing or not choosing particular values or endpoints need to be more thoroughly detailed throughout both documents, especially for endpoints that appear to occur in both experimental animal models and exposed humans (i.e., thyroid hormone disruption and immunotoxicity).

Jeffrey W. Fisher

These documents represent an enormous undertaking to describe studies with PFOA and PFOS. Keep the same writing style for reporting studies. This was very good. A synthesis of the most important studies is needed and some statements about why other studies are not used by EPA. It is easy to get lost in the document because of its size, but if there was an analysis or synthesis section for the key toxicity studies and another for PK modeling rationale, it would help readers.

William L. Hayton

It would be helpful to use one set of units for test article amount and concentration. The draft documents use ng/mL, μg/mL, μg/L, ppb, ppm, and μM for PFOA/PFOS concentration in water, diet, and serum. It would be more straightforward to use one concentration term, preferably ng/mL, and perhaps μg/mL in addition as necessary. But making comparisons among ng/mL, ppm, and μM is a distraction.

In Section 3 of both documents, it would be helpful to include a summary table of primary pharmacokinetic parameter values for the species included in this section. Tables 3-17 – 3-20 in

the PFOS draft document are a good start. In the PFOA document, Table 3-23 lacks CL values, and Tables 3-24 and 3-25 lack V_d values. For the pharmacokinetic model analyses presented, primary parameters values could be limited to CL, Vd_{ss}, and half life (see table in response to question 10). The CL and V_{dss} values should be normalized to body weight. Where there are multiple models for a species, there should be separate entries for each study. Where there are multiple dosages for a species, there should be separate entries for each dosage. For the PBPK models, V_{dss} values are not available and therefore should not be included. Such a table would be helpful to show consistency or lack thereof among studies and would facilitate selection of the best available values for CL and V_{dss} for use in a human PK model that would predict steady-state serum concentration from intake (dosing) rate and, conversely, predict intake rate from steady-state serum concentration. These predictions are probably the primary reason to include a pharmacokinetics section in the documents.

The pharmacokinetic sections of both documents lack example graphs of serum concentration-time data on semilog coordinates for PFOA and PFOS. Inclusion of a few representative graphs would help the reader evaluate the consistency of the data used to generate the pharmacokinetic parameter values, and where model-based equations have been fitted to the data, the scatter of the measured concentrations around the model-predicted line would be informative as to the goodness of fit and the validity of the model and its parameters.

Matthew P. Longnecker

I can see advantages to treating this more like a systematic review of the literature, where the specific search algorithm for included articles is laid out, as are the range of dates of publication to be considered, and any other selection criteria applied for articles considered. In these documents, while the review of earlier literature appears to be comprehensive, after some point there must have been some decision making about which of the more recent articles to include.

The EPA has many guidelines about how data like these are to be evaluated, yet in the document few, if any, references to these guidelines were cited. Because so many guidelines exist, it could help readers if the authors cited specific places in critical documents that provide guidance for specific decisions.

Angela L. Slitt

The document reads very well. Although not included in the RfD determination, including a table of the observed human effects along with serum concentrations in Section 5.0 would put Tables 5-2 and 5-3 into context. Some sort of layman explanation to help understand why only non-human exposures are being included would be helpful to the general public.

V. SPECIFIC OBSERVATIONS

James V. Bruckner

Specific Observations for *Health Effects Document for Perfluorooctanoic Acid*		
Page	**Paragraph**	**Comment or Question**
3-11	5, lines 7-11	It is stated that the PFOA concentration in bile increased by a factor of 12.5 with the increase in PFOA dose from 12.5 to 25 umol/kg in wild-type mice and 19.5 in PPARα-null mice. These factors should be 2.8 for wild-type and 6.1 for PPARα-null mice. The document's authors may want to rethink their interpretation of the data. The results for the wild-type mice do suggest saturation of transport from liver to bile ducts, but the PPARα-null results do not, indicating a role for PPARα in this process. In contrast to the foregoing, the findings of Lou et al. (2009) (p. 3-11, pgr. 2) indicate their highest dose of PFOA is cleared from the blood of mice more rapidly than lower doses, suggesting saturation of hepatic and/or renal reuptake transporters. What is the relative importance of biliary and renal elimination of PFOA?
3-12	3, lines 2-4	It should also be stated that upregulation of MRP3&4 and the OATs may be <u>beneficial</u>, due to increased biliary excretion of bile acids, bilirubin, conjugated metabolites of toxic chemicals, etc.
3-14	1 & 2	It might be stated that the findings of Hinderliter (2004) support those of Han (2003), in regards to development of female rats.
3-14& 3-15		It is problematic to try to compare values in Table 3-14 with values referred to at the end of the second paragraph on p. 3-17. Whole pup and pup serum PFOA levels decrease between PND 1&18 for each dosage in the table. It would be preferable to include another table showing the PFOA levels with body weight taken into account. Table 3 – 14 and other tables should include the species in the title. It would also be helpful to include some details of the experimental protocol in the footnotes.
3-20		It would be useful at the end of this section (Distribution During Pregnancy and Lactation) to summarize the primary findings, or conclusions that can be drawn from the data that were presented.
3-23	4, line 2	It should be emphasized that urinary excretion of PFOA was substantially higher in female than male rats.
3-28	2, line 4	Replace "receptors" with "transporters".
3-28	6	Did 10 uM PFOA inhbit PAH and estrone uptake to a greater extent than 100 uM PFOA?
3-29	3 & 4	It is not clear what Yang et al. (2009) concluded about the role of OATp1a1 in the uptake of PFOA from glomerular filtrate.
3-32	3 & 4	These two summary paragraphs are very helpful.
3-37	1, line 1	Should "adsorption" be "absorption"?

Specific Observations for *Health Effects Document for Perfluorooctanoic Acid*		
Page	**Paragraph**	**Comment or Question**
4-7 & 4-9	Tables 4-1 & 4-2	Tables 4-1 and 4-2 are quite helpful in integrating the results of studies of occupationally-exposed populations.
4-13		A concluding sentence should be added to summarize the findings of a lack of association of PFOA with diabetes, metabolic syndrome, etc.
4-32		The NOAEL and/or LOAEL for this study should be stated at the end of the paragraph.
4-34	2	Is the LOAEL for liver effects 1 ppm in the study of Loveless et al. (2008)?
4-38	1	Include the meaning of the abbreviation "mPPARα".
4-39		Inclusion of the table for Minata et al. (2010) would be useful to help readers better comprehend the study findings.
4-40		A table of short-term LOAELs and NOAELs should be added here or in Section 5.
4-47	2	It is hard to believe, judging from the slight difference in mean values and their standard deviations, that absolute and relative liver weights are significantly higher than controls in the 1 mg/kg/day group.
4-67	2, line 5	Insert "absolute" before "liver weight".
4-69	1, lines 1 & 2	It might be worthwhile to point out that the actual study by Butenhoff et al. was conducted prior to 2004.
4-73		A summary sentence (or two) should be added at the end of the Mutagenicity and Genotoxicity section.
4-83		A summary paragraph should be included at the end of the Immunotoxicity section.
4-101	1, line 14	Insert the word "some" before "occupational studies". In order to present a more balanced perspective of findings in occupational studies, the following sentences could be added at the end of the paragraph: "Olson and Zobel (2007) examined groups of male workers at 3 fluorochemical production facilities. Serum PFOA concentrations were not associated with total cholesterol, LDL or HDL in workers at these facilities."
4-102	4	It should be stated that the increases in serum enzyme activity in workers were quite modest/small. The following sentence should be added at the end of the paragraph: "Emmett et al. (2006), however, found no association between serum PFOA and liver or renal enzymes".
4-103	2, line 2	Change "apoptotic or necrotic damage of" to "apoptosis or necrosis of". Apoptosis and necrosis are types of cell death, not damage/injury.
4-103	3, line 1	It is true that PFOA may interfere with the biliary excretion of other compounds that are transported by the same transporters. Upregulation of the genes for these transporters, however, may be beneficial in that the excretion of bile acids, bilirubin and conjugates

Specific Observations for *Health Effects Document for Perfluorooctanoic Acid*		
Page	**Paragraph**	**Comment or Question**
		of toxic chemicals/metabolites may be hastened.
4-103	4, line 2	I would avoid the word "critical" until the section on Dose-Response Assessment.
4-103	4	Increases in absolute and relative liver weights were dose-dependent (Cui et al., 2009; Elcombe et al., 2010; Wolf et al., 2008a)
4-103	5	It is important to distinguish between effects of PFOA on rough and smooth endoplasmic reticulum (RER and SER). RER content was diminished, but there was a proliferation of SER.
4-104	2, line 5	This last line should be amended to read "that PFOA has some effects of unknown toxicological significance that appear to be independent of PPARα activation.
4-104	4, line 3-5	The meaning of the sentence is not clear. Has something been omitted?
4-105	3, line 3	Add "of offspring" between "abilities" and "at 6 and 18". Include Fei and Olsen's (2011) finding of no association between prenatal PFOA exposure and behavioral or coordination problems in children at age 7.
4-109	3	The species (i.e., mice) studied by White et al. (2009) and by Wolf et al. (2007) should be stated.
4-111	4, line 2	Replace "examine" with "determine whether there was".
4-112	2, lines 1 & 2	The first sentence is misleading and should be rewritten. Butenhoff et al. (2012) did not see a significant increase in liver adenomas or carcinomas. Biegel et al. (2001) reported an increased incidence of hepatic adenoma but not carcinoma.
4-112	2, line 13	What is hepatic cystoid degeneration?
4-114	2, line 3	Insert "decreased" before "apoptosis".
4-115	5, line 2	What is meant by "PRAR exposures"?
4-116		There is no mention of PFOA-induced changes in expression of genes (e.g., cell cycle control, peroxisomes biogenesis, inflammation, etc.) that are PRARα-dependent. There is no mention of the role of PRARα or peroxisomes in oxidative injury and carcinogenesis.
4-120	1, lines 11 & 12	Insert "these" between "that" and "hormones".
4-121	3	It would be helpful to give the PFOA dosages of White et al. (2007) and one or two other studies, so the reader will have some idea of the magnitude of PFOA exposure required to alter mammary gland development.
5-1		RfD: Omit the word "wealth" from the bullet pertaining to epidemiology studies. There have been relatively few epidemiology studies of PFOA-exposed populations.

Specific Observations for *Health Effects Document for Perfluorooctanoic Acid*		
Page	**Paragraph**	**Comment or Question**
5-2	1, lines 2-6	Another obvious point should be made here, mainly that occupational exposures result in much higher plasma PFOA levels and body burdens than do environmental exposures. Thus, it would be anticipated that adverse effects would be more apparent in PFOA facility workers.
5-2	1, line 5	Include the words "in some instances" between the words "shown" and "between". Otherwise, it appears from this paragraph the serum PFOA concentrations are consistently/usually associated with the various maladies.
5-2	3, line 8	Insert "failure to attain" between the words "with" and "developmental".
5-7	2, line 4	Insert the word "rodent" between "between" and "species"
5-19	1, line 1	Insert "from some studies" between "data" and "have".

Specific Observations for *Health Effects Document for Perfluorooctane Sulfonate*		
Page	**Paragraph**	**Comment or Question**
3-2	5, lines 2 & 3	It is stated here that "the ratio of PFOS identified in serum and liver tissue are similar". Do the authors mean that PFOS concentrations in the serum and liver are similar?
3-2	6	How does PFOS distribute between plasma lipoproteins and proteins/albumin?
3-5	1, lines 9& 10	How much lower were milk PFOS levels than serum levels?
3-7	1, line 2	Oral and gavage are redundant.
3-16	Figure 3-1	This figure nicely illustrates relative PFOS levels in dams and fetuses/pups over time.
3-19	1,line 3	Insert "groups" between "day" and "on".
3-21	1, line 10	Substitute "longer" for "slower".
3-23	2	It is not clear who conducted the human PBPK modeling nor which model they used.
4-21	2, lines 1-3	What did the 2nd monkey die from?
4-26	3, line 3	The word "concentrations" should be replaced by "doses".
4-39	1	Does an increase in motor activity on PND 17, but no such effect on PND 13, 21 or 61, constitute a toxicologically-significant effect?
4-56	2, lines 1 & 2	It is stated that "taken together, these studies suggest a PPARα-independent mechanism…" Of the studies reviewed to this point in the document, only that of Abbott et al. (2009) supports this premise. Qazi et al. (2009), Rosen et al. (2010) and other groups of investigators have reported other PPARα-independent effects of PFOS.
4-60	2, lines 15-17	Is oxidative damage likely to be operative to a significant extent at lower PFOS doses?

Specific Observations for *Health Effects Document for Perfluorooctane Sulfonate*		
Page	**Paragraph**	**Comment or Question**
4-61	2, line 4	What is meant by "The concentration…"?
4-61	4, line 2	Change "dose of exposure is" to "levels of exposure are".
4-62	1, lines 2 & 3	What did Olsen et al. (2003) find correlation between?
4-62	3, lines 4 & 5	Identify the species (i.e., rat) studied by Chang et al. (2009) and Stein et al. (2012).
4-62	5	The liver of rats and monkeys was examined for histopathological changes, but the histological changes should not be considered lesions nor pathological.
4-68	4, lines 5 & 6	The elevated incidence of hepatocellular adenomas/ carcinomas was almost entirely due to adenomas. Only 1 of 60 high-dose female rats exhibited carcinoma.
4-69	5, lines 3 & 4	It is stated here that there was no increase in hepatocellular proliferation detected in the subchronic study of Seacat et al. (2003). It is stated previously on page 4-69 that "the data for PFOS are adequate to support some but not all key events…" I assume that cell proliferation is thought to be a missing event. Seacat et al. (2003) reported that the average hepatocyte proliferation index was not increased, but that some animals exhibited mild increases. It is clear in the current document that PFOS is not as potent a PPARα inducer as PFOA.
5-4	2 & 5, line 7	Again the terms "histopathological" and "lesions" are misnomers.
5-4	3, line 9	What is meant by a "biologically significant decrease in survival" at 0.8 mg/kg?

Deborah A. Cory-Slechta

Specific Observations for *Health Effects Document for Perfluorooctanoic Acid*		
Page	**Paragraph**	**Comment or Question**
Chapter 5		The text of Chapter 5 in the PFOA document (and other places) continues to state that a 10% increase in liver weight would not be an adverse effect, but merely a denominator for loss of homeostasis. On what basis was this conclusion derived? What is the support for this statement? It appears that benchmark dosing was applied to studies that had liver weight as the common denominator, but does this accommodate the lowest NOAELS and LOAELS observed for any endpoint in the long duration studies? Use of just studies with the common denominator because they provide replication ignores the fact that some other effect may occur at lower levels but simply hasn't been evaluated in as many studies as focused on PPARa-based targets. If this isn't the case, then the text should clearly address this.
5-7	2	States that the $BMDL_{10}$ values all fall below the experimental LOAELs. So, what does that mean, is there some conclusion that is supposed to be reached from this? IF so, please state it.
5-13	1	States "Generally these values were similar." What does similar mean? What is acceptable in this context?
5-16	3	States that the half-life value Bartell et al. (2010) was sued for half-life because it seemed more relevant to scenarios where exposure result from ingestion of contaminated drinking water by members of the general population. This rationale does not appear to consider the potential different strengths and weaknesses of the other potential studies. Is it necessarily the case that general population is more important than occupational studies? The rationale needs to be described in greater detail. Virtually no rationale is provided for the choice of the Thompson et al. (2010) study for a volume of distribution value.

Specific Observations for *Health Effects Document for Perfluorooctane Sulfonate*		
Page	**Paragraph**	**Comment or Question**
1-1	4	There are similar concerns for the PFOS document. Loose terminology should be eliminated, e.g., what is a 'finding of note' as used in the executive summary for PFOS.
Executive summary		The PFOS executive summary is of limited utility; for many readers this may be as much of the document as they read; as currently written it is not clear or transparent nor does it sufficiently explain how it arrived at an RfD.
3-3	Table 3-1	Couldn't a sentence essentially substitute for Table 3-1; it really isn't useful.
3-25	1	Loose terminology should be eliminated, e.g., what is "generally good"
3-26	Figure 3-7	Figure 3-7 has no explanation of what is the black vs. gray line.
All tables		There is a need to improve all of the tables; they should always include study name/year, sample size and exposure duration information on them; this would make all of the comparisons easier to evaluate and not require the reader to continue to go back and forth to the text.
4-4	Table 4-1	For example, table 4-1 has only study name and year, but what really matters is also exposure duration and sample sizes, because the comparisons of outcomes in the Table depend upon the power of the study to detect effects at the very least.
4-9	Table 4-2	The same comment applies to Table 4-2 and any others with this intended purpose.
4-11	Table 4-3	Table 4-3 needs sample sizes, exposure duration etc.
4-24	Table 4-7	Tables that summarize a significant amount of data from a single study (e.g., 4-7) should include the study authors and year in the Table title so it doesn't have to be searched for.
		In several instances in the PFOS document, adverse effects early that appear to be reversed at a later age are discounted with the suggestion that they therefore do not matter; given our increasing understanding of the importance of early changes in terms of epigenetic changes, this is no longer appropriate and in fact, misleading.
5-16	Table 5-8	What do the parentheses signify?
5-17	Table 5-9	What do the parentheses signify?

Jamie C. DeWitt

Specific Observations for *Health Effects Document for Perfluorooctanoic Acid*		
Page	**Paragraph**	**Comment or Question**
4-102	2 & 3	DeWitt et al. 2009 also included data on triglyceride levels in C57BL/6 mice exposed to PFOA for 15 days; triglyceride levels were dose-responsively decreased.

Jeffrey W. Fisher

No specific observations.

William L. Hayton

Specific Observations for *Health Effects Document for Perfluorooctanoic acid*		
Page	**Paragraph**	**Comment or Question**
1-2	Last, line 5	Delete "in"; should read "… in rats was analyzed …"
3-2	1, lines 6-8	Assumption that fecal excretion represented unabsorbed PFOA is problematic; suggest rephrasing this sentence.
3-3	Table 3-1	Protein binding is important for PK modeling, where the fraction unbound (fup) is the important parameter, not the fraction bound. Suggest listing fup values rather than percent bound.
3-6	Last, line 3	"concentration" should be "dose rate"
3-8	2, line 4	In addition to liver, kidney, and blood, other tissues are prominent. E.G., Table 42 of Kemper shows that in male at 1 mg/kg, t=Tmax, GI tract, GI contents, muscle, bone and skin contained a greater percentage of dose than did the kidney.
3-8	2, line 8	"Blood to kidney" should be "kidney to blood"
3-8	2, line 10-11	In Kemper, Tables 44-45, blood to kidney ratios are not 10 or higher in males.
3-8	2	This paragraph reports both percent of dose found in tissues, and concentrations found in tissues. But Tables 3-4 and 3-5 present only the former. When presenting tissue concentrations, please make it clear that those data are not shown.
3-18	Last, line 3	"were" is repeated.
3-19	1, line 1	Technically incorrect to say that the level peaked at PND7; that was the earliest sample time. The peak may have occurred before PND7.
3-19	Table 3-15	The last dose was on GD17; strange that at 1 and 3 mg/kg the serum concentration increases from PND7 to PND14.
3-22	4	Last sentence is garbled.
3-22	4, 5	Agree that biliary elimination is possible, but it could be that chloestyramine binds PFOA and PFOS in the GI tract lumen after they passively diffuse from the blood to the gut. There seems to be no direct evidence of biliary elimination, e.g., bile collected from treated animals.
3-23	Last, line 4	Should be Table 3-18.
3-34	Last, line 9	Should be "nonlinear least squares"
3-35	Table 3-23	Column 2, "Adsorption" should be "Absorption"
3-38 3-38	2 Figure 3-7	The arrow from Gut to Liver appears to point in the wrong direction; it should represent biliary excretion of PFOA from Liver to Gut.
3-43	Last line	"… indicating the absence of active excretion in human kidneys." This does not follow from the observation of renal clearance being about 0.001% of GFR. A plasma free fraction of 0.001 would

Specific Observations for *Health Effects Document for Perfluorooctanoic acid*		
Page	**Paragraph**	**Comment or Question**
		account for the CLr being 0.1% of GFR, and passive tubular reabsorption would make it 0.001% of GFR since urine flow is about 1% of GFR. Other scenarios are possible that do not invoke the absence or presence of active excretion.
3-44	Table 3-24	Should report all data values with three significant figures. For example, Lambda z values have only one sig. fig., while $T_{1/2}$ values have 5-6.
3-46	2	This reviewer does not follow the derivation and use of a value for volume of distribution with regard to intake rate and serum concentration of PFOA. If the subjects were at steady state, the body burden would have to be known. At steady state, the serum concentration would be independent of the volume of distribution, so any V value ought to match the intake rate to the steady state serum concentration.
4-9	1	Log transformed concentration was 1.51 and 1.48 ng/mL – are these the logarithms? IE, are the actual concentrations 10^1.51 = 32 and 10^1.48 = 30 ng/mL?
4-20	2, line 8	Anderson here is spelled Andersen in the reference list.
4-30	1, line 9	prostrate should be prostate.
4-31	4, line 10	decreased should be decrease.
4-112	1	It would be helpful to restate the serum concentrations for the Eriksen and Vieira studies, or refer reader to p. 4-29 where they are provided.
4-112	1, line 9	Delete "for".
4-112	2, line 12	Delete "were".
4-118	4	Delete "of actions" after MOAs
4-120	3	The broad range of half lives could also be due to person-to-person variability in the free fraction of PFOA in serum (fup). This is the case for highly bound drugs; e.g., warfarin.
5-1	3	Pharmacokinetic is misspelled.
5-1	5	Disagree – exposure assessment based on the human data is feasible. In fact, the serum concentrations are a better measure of exposure than are intake measures as they reflect all intake pathways and eliminate bioavailability and pharmacokinetic influences on internal exposure.
5-12	Last	Table numbers should be 5-6, 5-7, and 5-8.

Specific Observations for *Health Effects Document for Perfluorooctane Sulfonate*		
Page	**Paragraph**	**Comment or Question**
3-5	3	The low CSF : serum concentration ratio could also be due to an export transporter that pumps PFOS out of the CSF and/or to extensive serum protein binding, where only the free serum concentration of PFOS is in equilibrium with the free PFOS concentration in the CSF.
3-22	2	The free fraction used for the model is much larger than that determined experimentally, Table 3-1; this should be pointed out in the text.
3-22 3-23	2 Figure 3-5	The arrow from Gut to Liver appears to point in the wrong direction; it should represent biliary excretion of PFOS from Liver to Gut.
3-24	4	Anderson should be Andersen.
4-26	4	"concentrations" should be "dosages".
5.2	3	Should note for many of these studies, that steady state may not have been achieved due to the long half-life of PFOS. Half-life values from Section 3 are: mouse, 37 days; rat male, 40 days and female 64 days; monkey, 120 days. Using a one-compartment PK model, the time to 90% steady state is 3.3 half lives.
5-5	3	The NOAEL for liver effects in rats of 0.072 mg/kg/day is not consistent with p. 5.4, para. 2, which states that lesions of the liver <u>were</u> observed in male rats after 104 weeks at this dosage.
5-7	2	For female rat, the PFOS half life is about 60 d and the period of gestation is about 20 d or one-third of a half life. If PFOS is administered to the dam only during gestation at a fixed daily dose, the serum concentration of PFOS would rise from 0 to 21% of the steady-state serum concentration that the fixed dose rate would produce at steady state. The exposure of the fetus during gestation would average only about 10% of the exposure that would have occurred if the dam had received PFOS for 4 half-lives (240 days) prior to mating. BMDs based on such a fixed dose could be elevated by as much as a factor of 10 compared with the steady state situation. Steady state would be the relevant situation for humans. For the Luebker study (Table 5-3) the serum concentration during gestation would have increased from about 38% to 50% of the eventual steady state concentration.

Matthew P. Longnecker

Specific Observations for *Health Effects Document for Perfluorooctanoic*		
Page	**Paragraph**	**Comment or Question**
3-28	1st complete	Should the end of the sentence be "increase the transporters" rather than "increase the receptors"?
3-30	2nd complete	L 3, would insert "transfected" between "OAT3" and "cells"
3-39	1st complete	Next to last sentence: I doubt that Olsen assumed the major source of exposure was drinking water in the occupational study
3-41	4th complete	In the first formula listed, the plus sign should be an equal sign
4-9	1st complete	L 3 from bottom: the values of 1.51 and 1.48 given are probably better described as geometric means.
4-16	2nd complete	L 3 from bottom: would insert "draw" after "blood"
4-21	2nd complete	L 5: the value of 6.78 ug/L is a water level, not a serum level; this issue recurs on P 4-23, paragraph at bottom
4-30	1st complete	L 8: should read "exposure categories" rather than "cancer categories"?
4-37	Table	Would note dose of PFOA somewhere in table or footnote
4-55	Last para	L 3: should the ">" be a "<"?
4-79	Last para	Last sentence: should "50 and 25" be "50 and 250"?
4-80	1st complete	The last sentence does not accurately describe the table. E.G., the CD4+CD8+ cells decreased at the 47.21 mg/kg/d dose
4-82	Next to last para	Last sentence: the 37.5 mg/kg/dose is not mentioned earlier, so this is a little confusing.
4-85	Last para	L 2: should "0.5" be "0.05"?; Same issue for L 5.
4-89	4th para	How long were the animals dosed?
4-110	3rd complete para	L 5: should "serum" be "blood"?
4-113	3rd complete para	L 1: insert "in" before "liver cells"
5-4	Last para	Were the criteria for inclusion in Table 5.2 the same as for Table 5.1?
5-12	Para below table	8-6, 8-7, and 8-8 should be 5-6, 5-7, and 5-8
5-16	Last line	I do not see in the Thompson et al. (2010) study any mention of using exposure data from NHANES to calibrate the volume of distribution. Other sources of data were used, where the water had been contaminated.
5-17	1st formula	"/day" should be deleted from "0.17 $L/kg_{bw}/day$"
5-20	Table 5-12	The first three values in the UF_{total} column need to be corrected; they should be 21900, 219000, and 21900
5-21	Paragraph above table	Last sentence: UF_L should be UF_H
5-21	Last sentence	UD_S should be UF_S

Specific Observations for *Health Effects Document for Perfluorooctanoic*		
Page	**Paragraph**	**Comment or Question**
5-27	Calculations	The text says the body weight conversions should be based on the ¾ power. If so, the HED formulas are incorrect, and the HED should be 1.99 x 0.0254 = 0.0506, the dosimetric adjustment factor should be 0.0254, and the CSF should be 1.57. All the figures here should be checked as should the paragraph on P 5-28. The HED is 2,530-fold greater than the RfD, not 29,000.

Specific Observations for *Health Effects Document for Perfluorooctane Sulfonate*		
Page	**Paragraph**	**Comment or Question**
1-1	2nd	1st sentence: would revise for clarity. Do you mean uncertainties exist about whether PFOS-induced peroxisome proliferation is involved in causing PFOS-induced hepatic lesions?
1-1	3rd	1st sentence: would revise for clarity; the occupational studies were done at PFOS production plants, but to my knowledge there are no residential populations that have been studied for health effects who lived near PFOS production plants. (Mid-Ohio valley factory was a source of PFOA.) In the 2nd sentence, I do not believe that exposure was mainly through contaminated drinking water in any of these studies.
4-66	2nd	The earlier summary of the Bloom et al. study (P 4-10) said the results were not statistically significant, whereas here the interpretation appears to be that the study found an association. The interpretation does not seem consistent across the two sections.
5-17	Below table	L 3: the word "terminal" should be deleted from this sentence
5-20	1st formula	The "/day" should come out of "0.23 L/kg bw/day"
5-26	L 2 from bottom	This should be 35 ug/L not 35 mg/L

Angela L. Slitt

No specific observations.

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APPENDIX A: PEER REVIEWER BIOSKETCHES

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External Peer Review Meeting on EPA's Draft Health Effects Documents for Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS)

Crystal City Marriott at Reagan National Airport
1999 Jefferson Davis Highway
Arlington, VA, 22202-3526

August 21 and 22, 2014

LIST OF PEER REVIEWERS

James Bruckner, Ph.D.
University of Georgia

Deborah Cory-Slechta, Ph.D. (chair)
University of Rochester School of Medicine and Dentistry

Jamie DeWitt, Ph.D.
East Carolina University

Jeffrey Fisher, Ph.D.
U.S. Food & Drug Administration

William Hayton, Ph.D.
The Ohio State University (Emeritus)

Matthew Longnecker, Sc.D., M.D
National Institute of Environmental Health Sciences

Angela Slitt, Ph.D.
University of Rhode Island

BIOGRAPHICAL SKETCHES OF PEER REVIEWERS

James Bruckner, Ph.D.
University of Georgia

Dr. Bruckner is Professor of Pharmacology and Toxicology at the University of Georgia College of Pharmacy. He is also Professor in the Department of Physiology and Pharmacology at the University of Georgia College of Veterinary Medicine. He received his Ph.D. in Toxicology from the University of Michigan in 1974. He has previously held faculty positions at the University of Kansas and the University of Texas Medical School at Houston. He is actively engaged in graduate education and in federally-funded research projects. Dr. Bruckner's research focus is on the toxicology and toxicokinetics of solvents, drug-solvent interactions at occupational exposure levels, and toxicokinetic bases for susceptibility of children to insecticides and other chemicals. Dr. Bruckner has published more than 200 journal articles, book chapters, and abstracts. He has also served on a variety of expert panels and committees for the EPA, National Institutes of Health, National Aeronautics and Space Administration, Agency for Toxic Substances and Disease Registry, Food and Drug Administration, and the National Academy of Sciences.

Deborah Cory-Slechta, Ph.D. (chair)
University of Rochester School of Medicine and Dentistry

Dr. Cory-Slechta is a Professor in the Department of Environmental Medicine and the Department of Pediatrics at the University of Rochester School of Medicine and Dentistry, where she also serves as co-director of the Behavioral Sciences Facility Core and director of the Animal Behavior Core. Dr. Cory-Slechta received her Ph.D. from the University of Minnesota in 1977 and worked as a junior staff fellow of the National Center for Toxicological Research beginning in 1979. She was appointed to the faculty of the University of Rochester Medical School in 1982 and was appointed Chair of the Department of Environmental Medicine and Director of the NIEHS Environmental Health Sciences Center at the University of Rochester in 1998. From 2000 to 2002, she was the Dean for Research and Director of the AAB Institute for Biomedical Sciences. Following her appointment as Dean, she served from 2003 to 2007 as the Chair of the Department of Environmental and Occupational Medicine at Robert Wood Johnson Medical School and as Director of the Environmental and Occupational Health Sciences Institute, a joint Institute of the Robert Wood Johnson Medical School and Rutgers. Dr. Cory-Slechta's research has focused largely on environmental neurotoxicants as risk factors for behavioral disorders and neurodegenerative disease. These research efforts have resulted in over 170 papers and book chapters to date. Dr. Cory-Slechta has served on numerous national research review and advisory panels, including committees of the National Institutes of Health, the National Institute of Environmental Health Sciences, the Food and Drug Administration, the National Center for Toxicological Research, the Environmental Protection Agency, the National Academy of Sciences, the Institute of Medicine, and the Agency for Toxic Substances and Disease Registry, Centers for Disease Control. In addition, Dr. Cory-Slechta has served on the editorial boards of several journals including *Environmental Health Perspectives*, *Neurotoxicology*, *Toxicology*, *Toxicological Sciences*, *Fundamental and Applied Toxicology*, *Neurotoxicology* and *Teratology*, and *American Journal of Mental Retardation*. She has held the elected positions of President of the Neurotoxicology Specialty Section of the Society of Toxicology, President of the Behavioral Toxicology Society, and been named a Fellow of the American Psychological Association. She also previously served on the EPA Science Advisory Board Perfluorooctanoic Acid Risk Assessment Review Panel.

Jamie DeWitt, Ph.D.
East Carolina University

Dr. DeWitt is an Assistant Professor in the Department of Pharmacology and Toxicology at the Brody School of Medicine at East Carolina University (ECU). She is affiliated with The Harriet and John Wooten Laboratory for Alzheimer's and Neurodegenerative Diseases Research and holds an adjunct appointment in the ECU Department of Public Health. Dr. DeWitt received her Ph.D. in Environmental Science and Neural Science from the School of Public and Environmental Affairs and Program in Neural Science at Indiana University in 2004. She also completed postdoctoral training in Developmental Cardiotoxicity at Indiana University-Bloomington and in Immunotoxicology at EPA through a cooperative training agreement with the University of North Carolina at Chapel Hill. Dr. DeWitt's main research focus is on how toxicants found in the environment can lead to neurodevelopmental and neurodegenerative disorders via disruption of the developing immune system. Much of her past research has

involved the immunotoxicity of perfluorooctanoic acid (PFOA) and related polyfluroalkyl substances (PFASs). Dr. DeWitt has published seven peer reviewed research articles, three review papers and two book chapters that address the biological effects of PFOA, as well as one paper on the effects of PFOS on immune function. Her publications describe effects as well as underlying mechanisms following adult and developmental exposure. Her research experience and publication record (more than 25 peer reviewed manuscripts, 6 review articles, 9 book chapters) extend beyond the effects of PFAAs and working with rodent models. She is currently editing a book on the general toxicity of PFASs and is a current member of the mechanistic working group for Monograph 110 of the International Agency for Research on Cancer, which will include an assessment of PFOA. She is on the editorial boards of the *Journal of Immunotoxicology* and the *Journal of Environmental Toxicology and Health* and has reviewed grants for the Department of Defense and the National Institute of Occupational Safety and Health. She has also been manuscript reviewer for more than 20 journals. Dr. DeWitt is the current president of the North Carolina chapter of the Society of Toxicology and the Junior Councilor for the Immunotoxicology Specialty Section of the Society of Toxicology. She also was awarded the Outstanding Young Investigator Award from the Immunotoxicology Specialty Section in 2013.

Jeffrey Fisher, Ph.D.
U.S. Food & Drug Administration

Dr. Fisher is a Research Toxicologist at the U.S. Food and Drug Administration (FDA), National Center for Toxicological Research. He was formerly a Professor in the Department of Environmental Health Science, College of Public Health at the University of Georgia (UGA). He joined UGA in 2000 and served as Department Head of the Department of Environmental Health Sciences from 2000 to 2006 and Director of the Interdisciplinary Toxicology Program from 2006-2010. Prior to joining UGA, he spent most of his career at Wright Patterson Air Force Base (AFB), where he was Principal Investigator and Senior Scientist in the Toxics Hazards Division and Technical Advisor for the Operational Toxicology Branch. Dr. Fisher's research interests are in the development and application of biologically based mathematical models to ascertain health risks from environmental, food-borne and occupational chemical exposures. Dr. Fisher's modeling experience includes working with chlorinated and non-chlorinated solvents, fuels, pesticides, perchlorate and bisphenol A. He has developed physiologically-based pharmacokinetic (PBPK) models for use in cancer risk assessment, estimating lactational transfer of solvents, understanding in utero and neonatal dosimetry, quantifying metabolism of solvent mixtures and developing biologically motivated models for the hypothalamic-pituitary-thyroid axis in rodents and humans. Dr. Fisher has published over 140 papers on pharmacokinetics and PBPK modeling in laboratory animals and humans. He has served on several national panels and advisory boards for the DoD, ATSDR, USEPA and non-profit organizations. He was a U.S. delegate for the North Atlantic Treaty Organization. Dr. Fisher served on the International Life Sciences Institute Steering Committee, which evaluated chloroform and dichloroacetic acid using EPA-proposed Carcinogen Risk Guidelines. He is Past President of the Biological Modeling Specialty Section of the Society of Toxicology, reviewer for several toxicology journals, and was Co-Principal Investigator on a National Institutes of Health (NIH)-supported workshop on Mathematical Modeling at the University of Georgia in the fall of 2003. Dr. Fisher was also a member of the National Academy of Sciences subcommittee on Acute Exposure

Guideline Levels (AEGLs) from 2004-2010 and Science Advisory Board (SAB) for the US EPA (2007-2010). He is an ad hoc EPA SAB member for dioxin and perchlorate. Dr. Fisher is a Fellow of the Academy of Toxicological Sciences, an associate editor for Toxicological Sciences, and on the editorial board of *Journal of Environmental Science and Health Part C Environmental Carcinogenesis & Ecotoxicology Reviews*.

William Hayton, Ph.D.
The Ohio State University (Emeritus)

Dr. Hayton is a Professor Emeritus in the College of Pharmacy at The Ohio State University. Dr. Hayton received a Ph.D. in Pharmaceutics from the State University of New York at Buffalo in 1967. He was a member of the Washington State University College of Pharmacy faculty for 19 years, rising to Chair of the Pharmacology/Toxicology Graduate Program in 1982 and Acting Dean at the College of Pharmacy in 1987. In 1990, he transferred to the Ohio State University as Chair of the Division of Pharmaceutics, where he later served as Associate Dean for the Graduate Programs and Research until his retirement in 2010. Dr. Hayton's expertise is pharmacokinetics, particularly construction and validation of mathematical models that describe or explain the kinetics of complex biological systems. One recent research interest is characterization of the Fc receptor-mediated transport and catabolism of albumin and IgG in wild type and FcR knockout mice. A second recent project is the quantitative modeling of the female hypothalamus-pituitary-gonad (HPG) axis in the female rainbow trout (Oncorhynchus mykiss). The model is based on and integrates the biology of gonadotropin, estrogen, androgen and maturational hormone signaling systems, and it includes key intermediate steps in the signaling pathways; viz., gonadotropin and sex steroid synthesis, hormone receptors and their corresponding mRNA levels. Dr. Hayton's expertise extends to interspecies scaling of pharmacokinetic model parameter values and xenobiotic metabolism. Dr. Hayton is author or co-author of over 100 peer-reviewed scientific publications and has held peer-reviewed grant support from the National Institutes of Health, EPA, Air Force Office of Scientific Research, Food and Drug Administration, and U.S. Fish and Wildlife Service. He previously served on the EPA Science Advisory Board Perfluorooctanoic Acid Risk Assessment Review Panel.

Matthew Longnecker, Sc.D., M.D
National Institute of Environmental Health Sciences

Dr. Longnecker, M.D., Sc.D., is the head of the Biomarker-based Epidemiology Group at the National Institute of Environmental Health Sciences (NIEHS). Dr. Longnecker received an M.D. from Dartmouth Medical School and completed a residency in internal medicine at Temple University Hospital in Philadelphia. After receiving a Sc.D. in Epidemiology from Harvard School of Public Health in 1989, he served as an Assistant Professor in the Department of Epidemiology at the University of California, Los Angeles, School Of Public Health. Since 1996, Dr. Longnecker has served as Adjunct Professor/ Associate Professor in the Department of Epidemiology, School of Public Health, University of North Carolina at Chapel Hill. He came to the NIEHS Epidemiology Branch in 1995, as a tenure-track investigator. Dr. Longnecker's research program is focused on the health effects of persistent organic pollutants (e.g., the DDT metabolite p,p'-DDE, and polychlorinated biphenyls). He is particularly interested in the effects of intrauterine exposure to persistent organic pollutants in relation to intrauterine growth,

preterm birth, birth defects, neurologic findings at birth, growth, neurodevelopment, intelligence, and hearing. Recently, Dr. Longnecker has completed and has ongoing a series of studies on perfluorinated alkyl substances in relation to reproductive and pediatric outcomes. In addition, he has begun studying the effects of early, low-level exposure to the nonpersistent pollutants, bisphenol A and organophosphate pesticides. Dr. Longnecker's research efforts have resulted in over 180 papers and book chapters to date. He has served as a leader for numerous national and international committees, such as for the Society for Epidemiologic Research and the International Society for Environmental Epidemiology, and has been on numerous national and international scientific advisory boards, including the EPA Science Advisory Board for the Perfluorooctanoic Acid Risk Assessment Review.

Angela Slitt, Ph.D.
University of Rhode Island

Dr. Slitt is an Associate Professor in the Department of Biomedical and Pharmaceutical Sciences at the University of Rhode Island. Dr. Slitt received her Ph.D. in Pharmacology and Toxicology from the University of Connecticut in 2000, and then served until 2004 as a postdoctoral fellow at the University of Kansas Medical Center. Dr. Slitt has been a faculty member at the University of Rhode Island since 2006. Dr. Slitt's graduate and postdoctoral training was heavily focused on liver biology and health, with a focus in the area of toxicology, and included research in nuclear receptors, biotransformation, and transporter expression. Her current research interests focus on how 1) expression of drug transporters affects chemical disposition and toxicity, 2) nutrition and intake of dietary antioxidants affects the expression of drug transporters, 3) liver disease (i.e., diabetes, cholestasis, and ethanol cirrhosis) affects transporter expression and chemical disposition, and 4) transporter expression affects cholesterol transport and susceptibility to gallstone formation. She has also recently investigated the effect of PFOS on caloric restriction in mice. Dr. Slitt is presently on the Editorial Board of *BMC Pharmacology and Toxicology, Journal of Biochemical and Molecular Toxicology*, and *Toxicology Methods and Mechanism*, and is an ad-hoc reviewer for numerous other journals. She is author or co-author of over 50 peer-reviewed scientific publications, and was recently awarded the University of Rhode Island Early Career Faculty Research Excellence Award.

EPA-HQ-2016-005679 02/14/2017

APPENDIX B: MEETING AGENDA

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AGENDA

External Peer Review Meeting on EPA's Draft Health Effects Documents for Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS)

Crystal City Marriott at Reagan National Airport
1999 Jefferson Davis Highway
Arlington, VA, 22202-3526

August 21, 2014

Day 1 – PFOA Health Effects Document

8:30 AM	**Meeting Sign-In Begins**
9:00 AM	**Welcome, Goals of Meeting, and Introductions** David Bottimore, Versar, Inc.
9:15 AM	**Welcome by EPA and Overview of PFOA/PFOS Health Effects Documents** Elizabeth Doyle, Chief, EPA/OST/OW/HECD
9:30 AM	**Chair's Introduction and Review of Charge** Deborah Cory-Slechta, *Chair*
10:00 AM	**Discussion Session – "Round Table" General Overview Comments**
10:45 AM	*Break**
11:00 AM	**PFOA Discussion Session – Responses to Charge Questions:** Question 1: Strengths, Weaknesses, and Characterization of the Studies Selected Question 2: Additional References for Consideration Question 3: Conclusions on Human Epidemiology Data Question 4: Transparency and Characterization of Epidemiologic Data
12:15 PM	*Lunch**
1:15 PM	**PFOA Discussion Session – Response to Charge Questions:** Question 5: Cancer Classification Question 6: Pharmacokinetic Model Adjustments Question 7: Selected Parameters for Pharmacokinetic Model Question 8: Volume of Distribution (Vd) and Half-life Values
2:45 PM	*Break**
3:00 PM	**PFOA Discussion Session – Response to Charge Questions:** Question 9: Points of Departure and RfDs Question 10: RfDs and Applicability to Short-and Long-term Exposures Question 11: Uncertainty Factors Question 12: Other Suggestions for Improving the Document
5:00 PM	**Adjourn**

**Time for breaks and lunch are approximate and at the Chair's discretion.*

AGENDA

External Peer Review Meeting on EPA's Draft Health Effects Documents for Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS)

Crystal City Marriott at Reagan National Airport
1999 Jefferson Davis Highway
Arlington, VA, 22202-3526

August 22, 2014

Day 2 – PFOS Health Effects Document

9:00 AM	**Recap of Day 1 and Agenda for Day 2** David Bottimore, Versar, Inc.
9:15 AM	**Chair's Review of Charge for Day 2** Deborah Cory-Slechta, *Chair*
9:30 AM	**Discussion Session – "Round Table" General Overview Comments**
10:00 AM	*Break**
10:15 AM	**PFOS Discussion Session – Responses to Charge Questions:** Question 1: Strengths, Weaknesses, and Characterization of the Studies Selected Question 2: Additional References for Consideration Question 3: Conclusions on Human Epidemiology Data Question 4: Transparency and Characterization of Epidemiologic Data
11:30 PM	*Lunch Break**
12:30 PM	**PFOS Discussion Session – Response to Charge Questions:** Question 5: Cancer Classification Question 6: Pharmacokinetic Model Adjustments Question 7: Selected Parameters for Pharmacokinetic Model Question 8: Volume of Distribution (Vd) and Half-life Values
2:15 PM	*Break**
2:30 PM	**PFOS Discussion Session – Response to Charge Questions:** Question 9: Points of Departure and RfDs Question 10: RfDs and Applicability to Short-and Long-term Exposures Question 11: Uncertainty Factors Question 12: Other Suggestions for Improving the Document
5:00 PM	**Adjourn**

**Time for breaks and lunch are approximate and at the Chair's discretion.*

EPA-HQ-2016-005679 02/14/2017

APPENDIX C: MEETING ATTENDEE LIST

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LIST OF OBSERVERS ATTENDING MEETING IN PERSON
August 21 and 22, 2014

External Peer Review Meeting on
EPA's Draft Health Effects Documents for
Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS)

Name	Affiliation
Janet Anderson, Ph.D.	U.S. Air Force
Robert Bilott	Taft Stettinius & Hollister LLP
Norman Birchfield	U.S. EPA
John Butenhoff, Ph.D.	3M Company
Sue Chang, Ph.D.	3M Company
C-H Selene Chou, Ph.D.	Agency for Toxic Substances and Registry
Steven Chranowski	Chemistry Council of New Jersey
Joyce Donohue	U.S. EPA
Elizabeth Doyle	U.S. EPA
Colleen Flaherty	U.S. EPA
Maria Hegstad	Inside EPA
Gerald Kennedy	DuPont
La Rae Landers	Dept of the Navy BRAC PMO
Willington Lin	Public
Angela Lynch, Ph.D.	American Chemistry Council
Geary Olsen, Ph.D.	3M Company
Gloria Post, Ph.D.	New Jersey Department of Environmental Protection
Resha Putzrath, Ph.D.	Navy and Marine Corps Public Health Center
Jennifer Seed	U.S. EPA
Shalene Thomas	AMEC Environment & Infrastructure
Steve Via	American Water Works Association
Anthony Walters	United Science, LLC
Carol Wood	Oak Ridge National Laboratory

LIST OF OBSERVERS ATTENDING VIA PHONE
August 21 and 22, 2014

External Peer Review Meeting on
EPA's Draft Health Effects Documents for
Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS)

Name	Affiliation
Matthew Bailor	Gilmore & Associates, Inc.
Victoria Binetti	U.S. EPA
Stuart Cagen, Ph.D.	Shell Health
Andrea Candara	New York State Department of Health
Tom Cleveland	Decatur Utilities
Jason Conder	ENVIRON International Corporation
Michelle Deveau	Health Canada
Stiven Foster	U.S. EPA
Helen Goeden	Minnesota Department of Health
Christopher Lau, Ph.D.	U.S. EPA
Anita Meyer, DABT	U.S. Army Corps of Engineers
Jonathan Naile, Ph.D.	Shell Health
Jessica Nelson	Minnesota Department of Health
Bridget O'Brien	U.S. EPA
Ramasamy Santhini	U.S. EPA
Robert Rickard	U.S. EPA
John Wambaugh	U.S. EPA
Carol Rowan West, Ph.D.	Massachusetts Department of Environmental Protection
Linda Wilson	New York State Office of the Attorney General
Virginia Yingling	Minnesota Department of Health
Tsedash Zewdie	Massachusetts Department of Environmental Protection

Peer Review Summary Report

External Peer Review of EPA's Draft Health Effects Documents for Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS)

November 17, 2014

Peer Reviewers:

James V. Bruckner, Ph.D.
Deborah A. Cory-Slechta, Ph.D.
Jamie C. DeWitt, Ph.D.
Jeffrey W. Fisher, Ph.D.
William L. Hayton, Ph.D.
Matthew P. Longnecker, Sc.D, M.D
Angela L. Slitt, Ph.D.

Contract No. EP-C-13-010
Task Order 2013-06

Prepared for:
U.S. Environmental Protection Agency
Office of Science and Technology/Office of Water
Health & Ecological Criteria Division
1200 Pennsylvania Avenue, NW (MC4304T)
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Prepared by:
Versar, Inc.
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TABLE OF CONTENTS

Contents

EXECUTIVE SUMMARY ii
I. INTRODUCTION 1
I.1 Background on Draft Health Effects Documents 1
I.2 Peer Review Process 1
I.3 Peer Review Meeting 3
II. CHARGE TO REVIEWERS 5
III. GENERAL IMPRESSIONS 7
IV. RESPONSE TO CHARGE QUESTIONS 17
Charge Questions 17
Question 1. 17
Question 2. 22
Question 3. 27
Question 5. 34
Question 6. 36
Question 7. 39
Question 8. 43
Question 9. 47
Question 10. 50
Question 11. 53
Question 12 55
V. SPECIFIC OBSERVATIONS 58
APPENDIX A: PEER REVIEWER BIOSKETCHES A-1
APPENDIX B: MEETING AGENDA B-1
APPENDIX C: MEETING ATTENDEE LIST C-1

EXECUTIVE SUMMARY

Versar, Inc. (Versar), a contractor for the Environmental Protection Agency (EPA), coordinated an external peer review of EPA's draft health effects documents for Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS), and organized a two-day public peer review meeting in Arlington, Virginia on August 21 and 22, 2014. The peer review of EPA's draft health effects documents was initiated with a pre-meeting written peer review managed by Versar and conducted by seven independent expert peer reviewers. The role of the peer reviewers was to evaluate the scientific and technical merit of the draft documents and provide their responses to 12 charge questions. Peer reviewers were charged only with evaluating the quality of the science included in EPA's draft health effects documents and were not charged with making any regulatory recommendations or reaching consensus in either their deliberations or written comments. The two-day peer review meeting, which directly followed the written peer review period, was held to discuss the scientific basis supporting EPA's draft health effects documents and to provide members of the public with an opportunity to observe the peer reviewer deliberations.

On the first day of the meeting, Versar began by providing information on the overall peer review process and introducing the peer reviewers. In addition, EPA provided background information on the draft documents and approach used in the development of the documents. Following opening remarks by Versar and EPA, the peer reviewers began their discussions on the PFOA draft document, moderated by the Chair, Dr. Deborah Cory-Slechta. The discussions centered on individual responses to EPA's 12 charge questions. Additionally, some comments submitted to the public docket prior to the meeting were also discussed. The second day of the meeting began with brief remarks from Versar followed by discussions focusing on the PFOS draft document.

The reviewers began the discussion on the first day with the recognition of the significant amount of data that is available for both PFOA and PFOS and thus to be considered by the EPA for incorporation into the document and for the ultimate derivation of reference dose (RfD) values. In general, the reviewers commended EPA in doing a very good job of pulling together this significant and extensive body of information and of condensing it into its most critical pieces for the derivation of RfD values. This was especially the case given that there is significant human and animal data available for both chemicals, as well as inconsistencies in the data.

The reviewers did offer numerous suggestions for improving the documents, many of which are applicable to both PFOA and PFOS. In general, the suggestions relate to the statement of the problem and defining the database that was utilized, clarity and ease of presentation, and transparency of the reports, as well as to issues of modeling, use of human data, and liver weight increases as the most sensitive endpoint. These comments are summarized below, but please note that this does not reflect a consensus or group perspective.

- All reviewers agreed that it would be extremely useful to include an opening section of the document describing in some detail the literature that was reviewed. More specifically, this would include a description of the dates that were included in the

literature review, whether requirements for peer-reviewed materials were imposed, and what criteria were used in eliminating studies from consideration. This would assist in identifying studies that should potentially have been included. Many additional studies of importance were suggested for review at the meeting by the peer reviewers.

- The reviewers also agreed that there are some significant studies that have been published recently on PFOA and PFOS that may be relevant, and these should be included in the documents during the current revisions.
- Reviewers also felt that the Tables in the documents could be made much more useful to readers with the addition of columns that included more study details. This would minimize the amount of back-and-forth to the text that was required to assess conclusions.
- Additionally, there was a strong agreement that the Hazard Identification section of the documents should include a more systematic review of study strengths and limitations. These were noticeably absent in the human studies, where all studies were considered equivalent but also applicable to animal studies.
- Reviewers also felt that each Chapter should include introductory paragraphs, as well as concluding paragraphs, that would provide better integration of the material across the Chapters, as well as summarize the conclusions arrived at from the text in the Chapter. This would also facilitate the ability of readers to follow the presentations provided in Chapter 5 of the derivation of RfDs.
- As pointed out at the peer review meeting, the authors would be well advised to base these documents on the new documents being produced by EPA's National Center for Environmental Assessment (NCEA) for the Integrated Risk Information System (IRIS) program. While the reviewers recognize that the PFOA and PFOA health effect documents will not be used specifically for the same purpose, they will still likely be held to the same standards of presentation, clarity and transparency and thus, the new NCEA formats provide good models to follow.
- With respect to content of the documents, there was initial confusion around some of the modeling outcomes and assumptions based on values that were provided in the documents. However, following explanation from EPA personnel in response to clarifying questions from the reviewers at the peer review meeting, it became clear that there were errors in these values and once these were corrected, concerns about the specific values used in some modeling were allayed.
- Even with the corrections to the modeling, there were residual concerns among reviewers based on toxicokinetic properties of these chemicals, with respect to applying the same candidate RfD values to both short and long-term exposures, which in animal studies ranged from 11 to 182 days with reviewers expressing a need for this to be re-considered.
- There were mixed comments by reviewers with respect to the decision not to use human data in the derivation of RfD values. All reviewers generally agreed that the rationales provided for the exclusion of the human data were not actually appropriate, as it is certainly not the case that such use would be precluded by the fact that there were

multiple and not sole exposures to either PFOA or PFOS. There was discussion as to the possibility of whether human data could be utilized in these assessments, and that ranged from an opinion that these data would not be useful to the view that it might be possible to use these data, despite the absence of information on route of exposure. Collectively, the range of opinions suggests that the issue should be revisited, and that appropriate rationales should be provided for the decision that is reached in the revised version.

- For both PFOA and PFOS, the RfDs were ultimately based on increases in liver weights in animal studies. There was significant discussion among reviewers as to the appropriateness of this endpoint for the derivation of the RfDs. That range of opinion spanned from an interpretation that these did constitute adverse effects in that they are a direct effect of a chemical exposure, whereas other reviewers saw these as adaptive effects. Collectively, the range of opinions suggests that the issue should be revisited, and that appropriate rationales should be provided for the decision that is reached in the revised version.

Following the meeting, peer reviewers were given additional time to complete their individual written reviews, which were submitted to Versar upon completion. These final written comments are contained in Sections III, IV, V of this report and fall into three categories: general impressions, responses to charge questions, and specific observations. Written peer review comments, as well as comments submitted to the docket by members of the public, will be considered by EPA as it revises the draft documents.

I. INTRODUCTION

I.1 Background on Draft Health Effects Documents

On February 28, 2014, the EPA's Office of Water (OW) announced in the Federal Register the release of the draft health effects documents for Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS) for purposes of public comment (scientific views) and peer review (http://www.gpo.gov/fdsys/pkg/FR-2014-02-28/pdf/2014-04455.pdf). The draft documents and charge questions were prepared by the Health and Ecological Criteria Division (HECD), within EPA's OW, in order to support future regulatory evaluations and decisions. EPA will consider the public comments and peer reviewer comments when revising the documents. Once the PFOA and PFOS health effects documents are finalized, they will be utilized to develop lifetime health advisory values for each chemical. PFOA and PFOS are listed on the third Contaminant Candidate List (CCL3)[1] and both chemicals are currently being monitored under the third Unregulated Contaminant Monitoring Rule (UCMR3)[2].

I.2 Peer Review Process

Consistent with guidelines for the peer review of highly influential scientific assessments, Versar, an EPA contractor, was tasked with assembling six to seven scientific experts to evaluate the draft PFOA and PFOS documents. The purpose of the peer review was to provide a documented, independent, and critical review of the draft health effects documents, and identify any necessary improvements to the documents prior to being published. In assembling these peer reviewers and coordinating the peer review, Versar was charged with evaluating the qualifications of peer review candidates, conducting a thorough conflict of interest (COI) screening process, independently selecting the peer reviewers, distributing review materials, maintaining contact with the peer reviewers, organizing and hosting the public peer review meeting, and developing a final peer review report.

The peer review selection process was initiated with a three-week public nomination period that was held from February 28, 2014 to March 21, 2014, as documented in the Federal Register on February 28, 2014 (http://www.gpo.gov/fdsys/pkg/FR-2014-02-28/pdf/2014-04455.pdf). During this period, members of the public were able to nominate scientific experts with knowledge and experience in one or more of the following areas: (1) epidemiology, (2) toxicology (liver effects, immunotoxicity, neurotoxicity, developmental and reproductive toxicology, etc.), (3) membrane transport, (4) human health risk assessment, (5) pharmacokinetic models, and (6) mode-of-action for cancer and noncancer effects. Concurrently, Versar conducted an independent search for qualified scientific experts to augment the list of publically-nominated candidates. In total, Versar evaluated 29 interested and available candidates who were either nominated by the public (n=18) or identified by Versar (n=11).

[1] CCL3 is a list of contaminants that are currently not subject to any proposed or promulgated national primary drinking water regulations, that are known or anticipated to occur in public water systems, and which may require regulation under the Safe Drinking Water Act (SDWA). Additional information about the CCL3 can be found at the following website: http://water.epa.gov/scitech/drinkingwater/dws/ccl/ccl3.cfm.

[2] EPA uses the Unregulated Contaminant Monitoring (UCM) program to collect data for unregulated contaminants suspected to be present in drinking water. Results from UCMR3 can be examined as they become available at the following website: http://water.epa.gov/lawsregs/rulesregs/sdwa/ucmr/ucmr3/.

Versar considered and screened all 29 candidates against the selection criteria described in the Federal Register dated February 28, 2014, which included: (1) having demonstrated expertise in the areas described above, based on information in their submitted resume, biographical sketch and/or current publications, (2) being free of any COI and the appearance of the lack of impartiality, and (3) being available to participate in-person in a two-day peer review meeting in the Washington DC area in the July or August 2014 timeframe. Following the screening process, Versar narrowed the list of potential reviewers to 15 candidates and provided to EPA the names of the candidates selected by Versar to be on the interim list. Additionally, information on the 15 candidates, including their professional affiliations, expertise, education, and professional experience were provided for the interim list and published in the Federal Register on April 30, 2014 (http://www.gpo.gov/fdsys/pkg/FR-2014-04-30/pdf/2014-09888.pdf). The Federal Register also requested the public to submit relevant information or documentation on the interim list of candidates that Versar should consider during the evaluation process of selecting the final six to seven reviewers.

Following the close of the public comment period on the interim list of potential reviewers, Versar re-evaluated each interim candidate's credentials to select the experts who, collectively, provided expertise spanning the multiple subject matter areas covered in the draft documents and provided a balance of perspectives. In addition, Versar evaluated the availability of each candidate to ensure all final peer reviewers were available on the same days for the meeting in the selected timeframe. Once the evaluation process was completed, Versar narrowed the interim list of 15 candidates and selected the seven final peer reviewers. In addition, Versar selected Dr. Deborah Cory-Slechta as Chair of the peer review meeting due to her expertise in toxicology as well as her strong record of chairing and participating in peer review panels, scientific meetings, and workshops. A list of the final seven peer reviewers who participated in this review is provided below. In addition, each reviewer's biographical sketch is included in Appendix A.

Following the selection process, Versar distributed EPA's draft PFOA and PFOS documents and 12 charge questions (see Section II) to the peer reviewers. The peer reviewers were asked to evaluate the scientific and technical merit of the draft documents and provide their responses to the 12 charge questions. This included evaluating the appropriateness of the quality, accuracy, and relevance of the data in the documents. Peer reviewers were not charged with making any regulatory recommendations or reaching consensus in either their written comments or public deliberations. In addition to being provided the draft documents and charge questions, comments submitted to EPA's public docket (Docket ID number EPA–HQ–OW–2014–0138) during each document's 60-day public comment period were provided to the peer reviewers ahead of the meeting for their consideration. Also, a brief summary of the public comments was developed by Versar and provided to the reviewers. However, peer reviewers were not asked to evaluate or respond to comments submitted to the docket.

Versar managed the pre-meeting peer review period, which provided the peer reviewers approximately two months to evaluate the draft health effects document and complete their written reviews. Following receipt of the peer reviewers' draft comments, Versar compiled the comments into a pre-meeting peer review report and distributed them to the peer reviewers and

EPA to prepare for the public peer review meeting. These preliminary responses to the charge questions formed the basis of reviewer discussions on Days 1 and 2 of the public meeting.

Peer Reviewers:

James V. Bruckner, Ph.D.
University of Georgia

Deborah A. Cory-Slechta, Ph.D.
University of Rochester School of Medicine and Dentistry

Jamie C. DeWitt, Ph.D.
East Carolina University

Jeffrey W. Fisher, Ph.D.
U.S. Food & Drug Administration

William L. Hayton, Ph.D.
The Ohio State University

Matthew P. Longnecker, Sc.D, M.D
National Institute of Environmental Health Sciences

Angela L. Slitt, Ph.D.
University of Rhode Island

I.3 Peer Review Meeting

On August 21 and 22, 2014, Versar convened a public peer review meeting in Arlington, Virginia. This meeting was held to discuss the scientific basis supporting EPA's draft health effects documents and to provide members of the public with an opportunity to observe the peer reviewer deliberations. The meeting followed both the documents' public comment period, during which members of the public were able to submit written comments, and the pre-meeting written peer review period, during which the seven selected peer reviewers evaluated EPA's draft health effects documents and provided preliminary comments in response to the charge questions.

Versar managed the pre-meeting registration period, which allowed members of the public to register to attend the meeting in person or remotely via teleconference and/or webinar. Members of the public were able to register online, via Versar's registration website (http://peerreview.versar.com/epa/pfoa/) as well as by telephone, email, or U.S. mail. In advance of the meeting, Versar provided all registered attendees with pre-meeting handouts, which included the agenda and logistics information.

On the first day of the meeting, Versar began by providing information on the overall peer review process and introducing the peer reviewers. In addition, EPA provided background

information on the draft documents and approach used in the development of the documents. Following opening remarks by Versar and EPA, the peer reviewers began their discussions on the PFOA draft document, which centered on individual responses to EPA's 12 charge questions. The remainder of the day was dedicated to discussions on the PFOA document, which were moderated by the Chair, Dr. Deborah Cory-Slechta. The second day of the meeting began with brief remarks from Versar followed by discussions focusing on the PFOS draft document. Approximately 23 public observers attended the peer review meeting in person and 21 observers attended the meeting via teleconference and/or webinar. Please see Appendix B for the meeting agenda and Appendix C for a list of public attendees.

Following the public peer review meeting, peer reviewers were given additional time to complete their individual written reviews. These final written comments are contained in Sections III, IV, and V of this report. Written peer review comments, as well as comments submitted to the EPA docket by members of the public, will be considered by EPA as it revises the draft health effects documents.

II. CHARGE TO REVIEWERS

PFOA and PFOS are environmentally persistent organic fluorocarbons (PFC) that have been identified in ambient waters, ground water, drinking water, and biosolids. They are metabolically inert but have the ability to bind to and interact with a variety of biomolecules leading to responses in living organisms. Both compounds have a substantial database of epidemiological, pharmacokinetic, toxicological and mechanistic studies. The two documents submitted for peer review include health assessment chapters that will be used 1) to provide information to drinking water treatment plant operators regarding the significance of monitoring results with respect to potential health outcomes and 2) to determine whether the perfluorinated compounds currently being monitored at Public Drinking Water Systems require regulation. The health information at that time will be accompanied with chapters on environmental fate, occurrence at public drinking water systems and occurrence in other media. The quantitative aspects of the Health Assessment documents will also be used to develop lifetime Health Advisories for both compounds.

Charge Questions

1. Please comment on the strengths, weaknesses, and characterization of the studies selected as key for quantification.

2. Please provide citations (and, where possible, pdfs or hard copies) for any references you suggest EPA consider adding to the document. Describe where you suggest these references be incorporated.

3. The OW concluded that the human epidemiology data for PFOS/PFOA do not provide adequate quantifiable dose-response information for use as the basis of a candidate RfD because of uncertainty regarding the routes, levels and timing of exposures plus the confounding influences of other PFCs present in serum. Please comment of the OW characterization of the data.

4. Please comment on the transparency and characterization of the epidemiological data.

5. The OW has concluded that the cancer classifications for PFOA and PFOS are most consistent with respective classifications of suggestive evidence for carcinogenicity as described the EPA Guidelines for Carcinogen Risk Assessment (pp. 2-56, 2-57). Please comment on the strengths and weaknesses of this classification.

6. Significant interspecies differences in pharmacokinetics exist for both PFOA and PFOS. Adjusting for interspecies differences was an important step in developing candidate RfDs given the totality of the human and animal data. Please comment on the strengths and weaknesses of the pharmacokinetic model adjustments to accommodate the impact of albumin binding and renal tubule transporters in determining average serum values.

7. Table 5-5 in the PFOA document and Table 5-7 in PFOS document list the parameters used for the ORD pharmacokinetic models that provide the final serum and AUC values for calculating the internal dose point of departure for the RfD calculation. Please comment on the strengths and weaknesses of the selected parameters.

8. The volume of distribution (Vd) and half-life values are critical in the derivation of the interspecies uncertainty factor applied in derivation of candidate RfDs from a NOAEL, LOAEL or a BMDL. The available data for both values are provided in Section 3.5.2 and 3.5.3 of both documents. Please comment the strengths and weaknesses of the values selected.

9. A variety of endpoints and studies were used to compare points of departure and the resultant RfDs for both PFOA and PFOS. In addition, comparisons were provided across RfD outcomes based on the model outputs compared to those for the NOAEL, LOAEL and BMDL points of departure. The range of candidate RfDs derived from the different points of departure is fairly narrow. Please comment on the strengths, weaknesses and transparency of this analysis.

10. The RfDs for PFOS and PFOA are derived from the modeled steady state serum concentrations and their association with effects that include short term and longer term exposures with associated diverse effects. The studies considered included effects due to exposure durations that ranged from 11 to 182 days, and occur at comparable human equivalent dose (HED) levels. The current, draft RfDs do not include an uncertainty factor for study duration because of the apparent concordance HEDs despite duration differences. Given this pattern of response, is it appropriate to conclude that the candidate RfDs are applicable to both short-term and lifetime exposures?

11. In addition to using the average serum values from animal studies to calculate internal doses for humans, the animal to human extrapolation can be accomplished by dividing animal average serum values by the human to animal clearance ratios to project a human average serum point of departure in units of mg/L serum. Please provide recommendations for applying uncertainty factors to the extrapolated average human serum values to determine serum-based thresholds that are protective for humans. A NOAEL expressed in average human serum units would be useful in interpreting NHANES population monitoring data.

12. Please describe any suggestions you have for improving the clarity, organization, and/or transparency of the draft documents.

III. GENERAL IMPRESSIONS

James V. Bruckner

PFOA-specific comments

This is one of the most comprehensive Health Effects Documents I have reviewed. The clarity and accuracy of accounts of pertinent research reports/publications are excellent. It is obvious considerable time and efforts were devoted to its composition. If anything, the amount of detail is so great that it is difficult to distill the mass of information on each topic and capture its "essence". This is likely the result of directions the authors were given for writing the document. Some topics in the Hazard Identification section do have summarizing sentences, in which the key/critical studies and their finding(s) are integrated and conclusions reached. It would be very helpful to devote much more attention to this for more topics, perhaps as an addition to Section 4.4 Hazard Characterization.

I do have a real problem with the scientific basis and soundness of certain conclusions in the document. The primary effect of PFOA in different species is increased absolute and/or relative liver weight. These are quite modest, reversible, non-specific effects that usually are not considered toxicologically significant. Livers of mice and rats dosed with PFOA typically exhibited hypertrophy characterized by increased peroxisomes, numerous mitochondria, reduced rough endoplasmic reticulum (RER), proliferation of smooth endoplasmic reticulum (SER), and increased autophagosomes or lipid-like droplets. Such morphological changes, particularly those in RER and SER, are manifestations of microsomal enzyme induction. This is considered adaptive, rather than adverse. Hall et al. (2012) points out that activation of a battery of genes involved in xenobiotic metabolism and transport serve to maintain homeostasis by enhancing the systemic elimination of the foreign chemical. Although PFOA is very poorly metabolized, it does persistently induce microsomal enzymes and the accompanying hepatocellular morphological changes. Upregulation of genes responsible for biliary excretion may be beneficial, since excretion of bilirubin, bile acids and conjugates of toxic chemicals/metabolites would be enhanced.

There are substantial qualitative and quantitative differences in responses of rodents and humans to PPARα activation. Therefore, many of the PFOA-induced alterations in lipid metabolism/homeostasis and associated biological processes in mice will be absent or an order of magnitude less pronounced at comparable doses in humans. Many of PFOA's effects on the liver of rodents are dependent on PPARα activation, though some effects appear to be PPARα-independent. Studies in PPARα-knockout mice show activation of other nuclear receptors by PFOA, including PXR, CAR, LXRA and FXR. Bjork et al. (2011) observed markedly lower transcriptional responses of PPARα, PXR, CAR and FXR to PFOA in cultured human than in cultured rat hepatocytes. These more subtle effects lead the investigators to conclude the changes in human cells reflected an adaptive metabolic remodeling rather than overt metabolic dysregulation, or disorder occurring in rat cells. The PFOA document's authors should go into detail discussing and summarizing the relative toxicological significance of non-PPARα effects in rodents versus humans.

It is important to recognize that clearly adverse effects of PFOA are seen. Lovelass et al. (2008), Cui et al. (2009) and others have seen focal necrosis and degenerative changes in the liver of mice and rats given relatively high doses of PFOA, as well as modest elevations in serum (hepatic) enzyme activities. Wolf et al. (2008a) observed a variety of degenerative structural changes in the liver of PPARα-null mice dosed with PFOA. Sakr et al. (2007a,b) and Olsen and Zobel (2007) reported associations between serum PFOA levels and slightly elevated serum enzyme activities in some occupationally-exposed populations. The increases in enzymes may have been attributable to factors other than PFOA. In light of the foregoing, it would be preferable to utilize hepatic morphological changes in rodents and/or elevated serum enzymes as the critical effect(s), rather than increased liver weight. These are clearly adverse effects seen in both rodents and humans.

An international panel of experts (Hall et al., 2012) opined that an increase in liver weight of ≤ 150%, at doses of chemical that do not produce structural or biochemical evidence of hepatocellular damage, would not be considered adverse. Absolute and relative liver weights were not increased as much as 50% by most PFOA doses in the majority rodent and monkey studies. Perkins et al. (2004), for example, reported dose-dependent increases in liver/body weight in rats fed 1, 10, 30 and 100 ppm PFOA for 13 weeks of 0, 10, 30, and 41%, respectively. Butenhoff et al. (2002) measured increases of 17, 21 and 37.5% and relative liver weight in monkeys given 3, 10 or 30/20 mg PFOA/kg/day for 26 weeks, respectively. Liver hypertrophy of this magnitude does not warrant such a low RfD. By adhering to EPA policies of calculating a $BMDL_{10}$ and using multiple UFs, regardless of the (lack of) severity of the critical effect and relatively low level of concern about other potential health effects, the end result is a vanishingly low RfD (i.e. 0.00002 mg/kg/day). A great deal of time and effort were spent on the PFOA hazard assessment, toxicokinetic modeling and extrapolations, dose metric and POD considerations, etc. Despite all of these scientifically-credible exercises and deliberations, the end result (RfD) seems to this reviewer to have been preordained-- to be extremely low.

Logic expressed on page 5-6, in support of use of liver weight gain as a critical effect and biomarker of loss of hepatocellular homeostasis seems flawed. As pointed out in the second paragraph, liver weight changes were not observed in PFOA-treated mice with a humanized PPARα receptor. It is noted that changes in gene products that modulate lipid metabolism do occur in these mice. EPA argues that this supports adoption of increased liver weight as a biomarker/critical effect. It has not been established that these changes in gene expression are adverse, or whether they are sufficient in magnitude to significantly alter lipid metabolism. It would be expected that repeated dosing with enough of a molecule (i.e., PFOA) that resembles a fatty acid would affect expression of such genes. Reversible changes in total cholesterol, bile acids, bilirubin, etc. have been observed. It has not been established, however, whether mild fluctuations in these indices are detrimental. No increases in mortality from cerebrevascular disease or ischemic heart disease have been found in PFOA-exposed humans. How then does the concurrence of alteration of expression of such genes and of liver weight gain support the latter as toxicologically-significant effect that should be prevented by setting the RfD low enough?

PFOS-specific comments

This Health Effects Document, like that for PFOA, is quite comprehensive. Its descriptions of the many studies of PFOS are clear, quite complete, and apparently quite accurate. As with the

PFOA document, so much detail is given about many studies in the Hazard Identification section, that is difficult to compare study designs/dosage regimens/species/indices/findings/etc. and to draw conclusions. The summary tables for single and multiple studies, however, are quite helpful in this regard. It would also be very useful to have more summary statements or paragraphs at the end of each topic. These should address the scientific importance of findings, their relevance to humans; and their impact on the weight of evidence on an issue.

The hazard characterization section (4.4) is, for the most part, inclusive and balanced in its presentation and integration of findings of the more important studies in each subject area. This is true for both non-cancer and cancer effects in humans and animals. It concerns me, however, that the document's authors do not focus in the remainder of the document on science (i.e., the candidate critical effects and their relevance to human health), but merely choose the most sensitive end-points and stress how similar the RfDs are after dosimetry modeling estimates and adjustments. I am not sure how this similarity of derived points of departure and other values, calculated from dissimilar endpoints, supports or validates the final RfD.

I recommend that an additional section be written, in which the primary adverse effects of PFOS are discussed-- in terms of their relative toxicological significance, their apparent mechanism(s), their relevance to humans, their likelihood in realistic exposure scenarios, and implications of altered experimental indices to actual organ dysfunction.

I am quite concerned about the increased rat pup mortality in several studies at relatively low maternal doses, but not about reversible liver weight changes or centrilobular hypertrophy. Is the decreased pup survival in several studies at relatively low maternal doses of PFOS relevant to humans?-- Is the dose-response curve steep, as suggested by Luebker et al. (2005a), such that there would be less concern about sub-threshold doses? -- What is the most likely mode of action (pulmonary surfactant or maturation, dietary, hormonal)? -- Is decreased survival PPARα-related? -- Is the mechanism in rats relevant to other species? -- Does pup mortality occur in other species at comparable doses? -- Might there be a dose-dependent alteration of maternal-fetal partitioning of PFOS?

Deborah A. Cory-Slechta

Both documents, although the PFOA document to some degree more than PFOS, overall are more of a tabulation of studies than a critical review of studies from which a rationale is presented for a choice of studies to model and from which to derive associated RfDs. The Executive summaries are too abbreviated and do not include sufficient rationale, description and detail to provide the reader with an understanding of how decisions described in Chapter 5 were made. Since in some cases, this will be the only sections read, they could provide a more informative summary.

It would be very helpful to provide a section up front that describes all of the parameters of the literature search, including the years that are included in the document review, as well as descriptions of criteria for studies that were included vs. those that were excluded. In addition, it should be indicated whether there was a criterion that studies be peer-reviewed. This is particularly important given the voluminous size of the data base that has accumulated for these

two chemicals. Given that revisions will be done and that such documents do not get updated with any frequency, it would be good to attempt to include as much of the new pertinent literature as possible.

The section on Toxicokinetics in the documents present studies in detail, but no real conclusions; this is true of most of the sections in these documents. Chapters 3 and 4 in particular read like tabulations of studies rather than critical reviews and because of that, the documents seem disjointed and Section 5, i.e., derivation of values, tend to be difficult to read through and require constant searching back to the original chapters in which they are described. It is critical to identify the strengths and weaknesses of the various studies, and which were given weight to use in the final determinations. It would be helpful if Sections 3, 4 and 5 included an introductory paragraph describing the goal of the chapter, and that each ends with an overall summary with conclusions. The tables in these chapters also would benefit from the inclusion of additional information that ultimately permits comparisons within the Table and does not require continually returning to the text to recall the species, sample sizes, etc.

In the sections on Hazard Identification, it is useful that studies are summarized by target organ, but there are almost no conclusions and no discussions of strengths or weaknesses of studies and therefore their use or not in future decisions. In fact, one is left with the impression that all studies are equal, especially in the section describing human studies. Within Chapter 4, the sub-sections entitled "evaluative and integrative" are actually neither. Data are presented simply as positive or negative with no real discussion of the strengths and limitations and what was concluded overall. For this reason, Chapter 5 is also lacking. It provides very little in the way of rationale and conclusions. Thus, the transparency of the process is really insufficient.

Jamie C. DeWitt

The information presented throughout the documents appears to be accurate (with one minor exception noted in Table 1 of these comments) and is presented clearly. For PFOA, a reference dose (RfD) of 0.00002 mg/kg/day was determined and evidence of carcinogenicity is considered suggestive with a human equivalent dose (HED) of 0.58 mg/kg/day. The RfD was based on changes in liver weight reported as a common denominator in four rodent (three rat and one mouse) studies and carcinogenicity was based on a limited number of epidemiology studies linking kidney and testicular tumors with exposure and evidence of tumor induction in the liver, testes, and pancreas (the "tumor triad") in rats. For PFOS, a RfD of 0.00003 mg/kg/day was determined and evidence of carcinogenicity is considered suggestive but with insufficient evidence to determine human carcinogenic potential. The RfD was based on developmental neurotoxicity and changes in liver weight.

While the carcinogenicity assessment seems appropriate for the two compounds given the limitations of the data sets, changes in liver weight as a basis of both of the RfDs is questionable in terms of its significance to exposed humans. Exposure to these agents increases liver weight and hepatocellular hypertrophy in rodents (and the definition of these endpoints as "adverse" or "toxic" also is contentious); this has been demonstrated across various rodent strains and under myriad exposure paradigms. However, there is no consensus in the scientific community regarding the mechanism by which exposure to these compounds increases liver weight and

induces hepatocellular hypertrophy in rodents and whether any of the putative mechanisms are sufficient to induce hepatotoxicity in exposed humans. Proposed mechanisms include peroxisome proliferator activated receptor alpha (PPARα) activation, activation of other nuclear receptors, peroxisome proliferation (which may or may not be dependent on PPARα activation), and oxidative stress. Humans can certainly respond to PPARα agonists (i.e., fibrate drugs are used as hypolipidemic agents) and a handful of epidemiological studies of highly exposed human populations have reported associations between PFOA/PFOS and alterations in liver enzymes, but the clinical relevance of the changes to the liver enzymes reported for these studies is uncertain. These liver-related changes in humans generally occur at higher doses than required to induce changes in the livers of rodents, which occurs at relatively lower doses than other observed effects. Therefore, a critical endpoint that occurs at very low doses in rodents, has no agreed upon mechanism that may or may not be relevant in humans at relatively high doses, may not be the best choice for the basis of a RfD. Liver weight change has been reported to occur in several species, including non-human primates, and at low doses, it may be an adaptive response and not a toxicological response. While this response may be protective of human health because it is common following low dose exposure to PFOA or PFOS, other endpoints may be more relevant to humans, especially endocrine system effects, including changes to thyroid hormones and mammary gland development, and immune system effects. Endocrine and immune system effects have been reported in exposed humans, suggesting that such endpoints may operate via a mechanism that is more relevant to humans than mechanisms related to changes in liver weights.

In addition, the one developmental neurotoxicity study used, in part, for the PFOS RfD is only weakly supported by additional studies in rodents or other species and is based on behavioral responses that could be influenced by factors other than direct effects on the nervous system. Additional confirmatory studies are necessary for this observation to be considered a critical effect of PFOS exposure.

Finally, while well-written overall, the documents lacked an overall critical analysis or depth required of a risk assessment. Why specific studies were included or not should be better explicated in the text.

Jeffrey W. Fisher

The document was well written in terms of balance and presenting information. Summary statements are needed for chapters; a synthesis/analyses of the data are needed in some cases. A more critical evaluation of the human and non-human responses to PFOA/PFOS is required to justify not using human or non-human primate data. A rationale for the modeling approaches is needed given the more recent PBPK models that are available.

William L. Hayton

The literature that pertains to the health effects of PFOA and PFOS is large and presents a major challenge to accurately summarize and analyze it and develop an RfD for PFOA and PFOS. Reported health effects in animals and humans, sometimes contradictory, include exposure-associated changes in serum cholesterol, lipids, uric acid, and thyroid hormones, obesity-related

metabolism, immune system function, and effects on reproduction, development of the mammary gland, the nervous system, and behavior. Target organ effects (e.g., liver, kidney) have been reported, as well as associations of PFOA and PFOS exposure with testicular, prostate and kidney cancer. Studies in several laboratory animal species have added the complications of interspecies comparisons and extrapolation of findings to humans. In humans, there have been a Phase I clinical trial of PFOA, and epidemiological studies of populations exposed to PFOA and PFOS occupationally and in communities with and without water supplies contaminated with PFOA and PFOS. The draft documents have accurately presented in summary form the results of many animal and human studies and used pharmacokinetic methods to link PFOA and PFOS exposure rates to internal dose metrics such as serum concentration. While the overall effort is commendable, there are two issues that the draft documents raise: 1) the literature cited does not include many apparently relevant published works. The cut-off date for cited literature was early 2013 (this should be indicated in the documents), but commenter's noted a number of pertinent publications in 2011 and 2012 that were not cited, and there have appeared several highly pertinent papers since the cut-off date, and 2) while the descriptions of individual studies are generally clear and accurate, there is a lack of independent, critical analysis of the studies and a lack of synthesis of results from multiple studies common to a particular health effect.

Matthew P. Longnecker

The PFOA and PFOS documents achieve the goal of identifying RfDs that are well founded. My main criticism is that the rationale for not using the human data to provide a POD needs to be strengthened.

For example, in the PFOA document, on page 5-19, first paragraph below the table, it says "human data … lack the exposure information for dose-response modeling." This statement is logically inconsistent with techniques that were used to estimate HED on the basis of serum concentration, as given on page 5-17, near the bottom. Or, in some cases, such as in the C8 study, the exposure estimates that were calculated based on water district were sufficiently good that a dose-response analysis would be possible. In other words, because many human studies have serum concentration of PFOA or reasonable estimated exposure values, the corresponding HED could be estimated, and hence the dose-response could be modeled. Granted, some assumptions would be needed, but the methods could be serviceable (see response to item 3 below). (Some of the above also applies to pages 5-1 and 5-2). More compelling arguments for not basing the POD on human data are, e.g., that: 1) the low probability that humans are 1,000 times more sensitive to PFOA than other species (the number is based on the last column in table 5-9 compared with PFOA values in the C8 study and background exposed populations), especially given the relatively tight agreement between LOAEL (average serum concentration basis) among other species, 2) the possibility that the observed associations in humans were due to unmeasured confounding factors or reverse causality, and 3) other weaknesses in the epidemiologic data such as inconsistent results across studies (selected outcomes), unreplicated findings, or associations with clinical chemistry results for which corresponding adverse clinical correlates (i.e., morbidity) are not clearly established.

Angela L Slitt

The documents provide a very thorough evaluation of PFOA and PFOS studies. It is logically organized, presenting findings in a way that the reader can understand the findings related to human, monkey, and rodents. The organization of the document allows the reader to easily find information about each species within the subchapters and summarizes key points in table form. PFOA is a well-studied compound, with a substantial amount of toxicokinetic and endpoint studies in rodents. Mechanistic data describing the role of membrane transporters to understand gender differences in PFOA elimination in rats is fairly well written. Little data exist regarding contribution of membrane transporters to PFOS disposition and elimination. The documents thoroughly describe species differences in PPAR-alpha signaling that might contribute to observed endpoints in rats, but not humans or monkeys. Overall, both documents are very thorough and provide a reliable basis for PFOS and PFOA evaluation.

For PFOA toxicokinetics, mechanisms of PFOA transport are important for understanding species differences in response to PFOA exposure, with focus placed on kidney. Figure 3-2 in the PFOA document does not adequately present the localization of renal transporters with relationship to their contribution to the urine compartment or renal reabsorption. A very nice diagram showing the subcellular localization of renal transporters presented by Klaassen and Aleksunes (Pharmacol Rev. 2010 Mar;62(1):1-96) clearly depicts the contribution of various transporters to filtrate or blood. This is an easier diagram to put PFOA elimination into context than the one presented. Contribution of membrane transporters to species differences in PFOA excretion Section 3 (specifically 3.4.1) would be put in better context if a table could be generated to compare Km and Vmax values for PFOA for various transporters, with specific focus on species information for OATPs and OATs. Data regarding information on contribution for OATps in liver accumulation of PFOS and PFOA is lacking, with specific regard to species differences. As PFOS is a likely candidate for hepatic uptake transport, understanding a mechanism to explain species differences in hepatic effects possibly due to difference in hepatic exposure is critical. Understanding impact species specific regulation of OATp expression in liver (e.g. whether species difference in PPAR-alpha signaling contributes) is also important in putting rodent distribution data into context.

Increased liver weight is considered to be a critical effect, but how increased liver weight relates to the observed human and monkey health effects needs to be further explained. First, use of liver weight alone might not be substantiate of an effect for point of departure compared to other liver effects observed at higher concentrations, such as increased serum ALT or AST.
The studies that have evaluated these endpoints are well conducted. In layman terms, if someone is walking around with an increased liver weight, is he or she at risk for disease? Will his/her life span be shortened? To increase transparency of the document, a more comprehensive explanation is needed to justify why increased liver weight should be considered as a critical endpoint for human health.

However, it should be noted for future consideration that there are a few publications in mice that do also ascribe liver weight changes in increased lipid accumulation along with increased expression of genes that contribute to fatty liver disease. This is considered to be a gap in knowledge for the field. Will relatively low dose PFOS exposures associated with hepatic

steatosis have other endocrine related effects known to be associated with NAFLD (insulin resistance, impaired glucose tolerance)? Emerging studies are evaluating whether PFOS induces hepatic steaosis and whether it is a PPAR-α mediated effect. For example a study performed by Wan et al. (2012) administered 0, 1, 5 or 10 mg/kg/day PFOS to adult male CD-1 mice for 3, 7, 14 or 21 days. Histological analysis of liver sections, and biochemical/molecular analysis of biomarkers for hepatic lipid metabolism were assessed. Overall, the study reported that PFOS-administration induced hepatic steatosis in a time- and dose-dependent manner. The study also shows a high correlation between liver weight and lipid content. Increased expression of a lipogenic target (CD36/FAT) was observed at 5 and 10 mg/kg PFOS. A second study by Bijland et al. (2011) illustrated that PFOS administration increased liver weight (+ 107%, $p < 0.0001$), which was accompanied by an increased hepatic TG content (+ 192%, $p < 0.0001$, respectively) in E3L.CETP mice on a C57Bl/6 background. At the age of 8–10 weeks, mice were fed a semisynthetic Western-type diet, containing 0.25% (wt/wt) cholesterol, 1% (wt/wt) corn oil, and 14% (wt/wt) bovine fat for 4 weeks in three independent experiments. Upon randomization according to body weight, plasma TC, and TG levels, mice were fed a Western-type diet without or with PFOS (0.003%, ~3 mg/kg/day) for 4–6 weeks. In summary, there is evidence that administration of relatively low PFOS doses to mice can result in hepatic lipid accumulation in the absence of overt "wasting."

Data is lacking as to whether higher species, such as monkeys or humans will also develop PFOS-induced steatosis, which is confounding. Studies have profiled gene expression in wild-type and PPARα-null mice administered PFOS, finding that there is pathology and gene expression consistent with lipid-promoting effects in liver that are independent of PPARα, as they are observed in PPARα-null mice (Rosen et al., 2010). Limitations to the studies are that they did not specifically quantify hepatic lipid content, but inferred that the PFOS-induced vacuolization in liver pathology observed was potentially related to triglycerides. Studies by Bjork et al. (2011) comparing rat and human primary hepatocytes treated with PFOS (25 μM) demonstrated that human hepatocytes were slightly less responsive to the induction of lipid oxidation and synthesis genes, as well as induction of carbohydrate metabolism. It should be noted that the hepatocytes from the study are from a single human donor, the hepatocyte lipid content was not determine, and hepatocyte culture conditions were standard and not optimized to induce steatosis. In summary, the current literature is lacking robust information regarding whether PFOS, which highly concentrates in liver, has a steatotic-inducing effect in human or monkey liver. The evidence in PPARα-null mice indicates that it might have some PPARα independent effects related to hepatic fat accumulation. Because evidence for hepatic lipid content in PPARα-null mice after PFOA or PFOS has been described only by pathology, more robust studies are needed to conclude whether the effects can occur independent of PPARα and the observed increased liver weight is due not only to hypertrophy due to nuclear receptor activation, but lipid accumulation. However, this reviewer is noting this as a concern for the future and area where a gap in knowledge exists.

Moreover, because the reviewer is noting a concern for hepatic fat accumulation that exists in the absence of PPARα, it should be appreciated that traditional markers, such as AST and ALT have poor prognosis for NAFLD or toxicant associated steatohepatitis. Most patients with NAFLD are asymptomatic and the disease is often diagnosed following findings of elevated aminotransferases, especially when combined with other features of metabolic syndrome. These

abnormal liver function tests usually require the physician to distinguish between NAFLD and alcoholic fatty liver disease (AFLD). Some have suggested that a serum AST/ALT ratio >1 are indicative of AFLD. Other possible signs of AFLD include elevated HDL-cholesterol with hypertriglyceridemia. While most diagnoses of NAFLD may result from altered liver function tests, normal serum aminotransferase tests can be seen in patients with both steatosis and NASH (Ipekci, et al., 2003; Mofrad, et al., 2003). Indeed, it is reported that two-thirds of NASH patients may have normal aminotransferase levels at any given time (Oh, et al., 2008; Delgado, 2008; Wieckowska and Feldstein, 2008). Kunde et al. investigated the accuracy of NASH diagnosis by serum ALT in women undergoing gastric bypass surgery (Kunde, et al., 2005). They compared two different reference laboratory cutoffs for "normal" ALT levels, the previous guideline of 30U/L, and new lower level of 19U/L that was suggested to aid in the diagnosis of NAFLD. Importantly, the authors reported that the diagnostic utility of serum ALT remained poor even at the new lower cutoff. Sensitivity and specificity of serum ALT levels were found to be 42% and 80% (ALT > 30U/L) versus 74% and 42% (ALT > 19U/L). These and other studies (Lizardi-Cervera, et al., 2006; Amarapurkar and Patel, 2004; Amarapurka, et al., 2006; Chen, et al., 2006; Fracanzani, et al., 2008; Sorrentino, et al., 2004; Mofrad, et al., 2003; Uslusoy, et al., 2009) illustrate the need for a more effective diagnostic measure for NAFLD, especially the NASH stage. In sum, use of ALT and AST elevation to base the point of departure must be taken into context because they are poor prognostic markers for increased liver accumulation, NAFLD, or even NASH. Use of ALT and AST might not be an appropriate biomarker for measurement of PFOA or PFOS-induced adverse effects on liver and should be considered as a gap in our knowledge for future work.

In the review panel discussion, there was discussion regarding a publication by Hall et al. (2012), which summarized the outcome of a workshop regarding liver hypertrophy and hepatocarcinogenesis. Overall, the workshop concluded that "hepatomegaly as a consequence of hepatocellular hypertrophy without histologic or clinical pathology alterations indicative of liver toxicity was considered an adaptive and a non-adverse reaction". This conclusion is taken in the context of hepatic hypertrophy caused by nuclear receptor activation. This differs from others that have concluded in mouse NTP studies where correlations between liver weight increases and histological parameters and carcinogenesis were assessed, the authors concluded that ''the best single predictor of liver cancer in mice was hepatocellular hypertrophy'' (Allen et al. 2004). Based on the conclusions presented in Hall et al. (2012), increased liver weight might not be considered an appropriate POD because of lack of overt toxicity and hepatomegaly being considered an adverse effect. It should be noted that this reviewer still has concerns regarding this conclusion for PFOS and PFOA because the pathology described in PPARα-null mice reflect increased hepatic lipids and not hepatomegaly due to nuclear receptor activation that is described in this opinion publication. If one considers the pathology examples of hypertrophy presented in Hall et al. (2012), it is quite different from the pathology described for PFOS and PFOA. For clarity, the document should try to delineate the cellular components that are contributing to increased liver weight caused by PFOA and PFOS administration, if such a publication exists (e.g. how much of the liver is associated with protein/peroxisome proliferation increase versus lipid increase). Because the literature is not clear regarding what exactly in the liver is causing increased liver weight, studies documenting hepatic lipid accumulation should not be currently considered for POD. Given the recent opinion cited by Hall et al. (2012), it is

EPA-HQ-2016-005679 02/14/2017

recommended that studies documenting liver damage, such as ALT and AST elevation be currently used as the POD.

The documents often have redundancy in information, especially in regard to hormone effects (there are very similar write ups in sections about effects on thyroid hormone) and metabolic/cardiovascular disease risk factors (e.g. lipid endpoints).

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IV. RESPONSE TO CHARGE QUESTIONS

Charge Questions

Question 1. Please comment on the strengths, weaknesses, and characterization of the studies selected as key for quantification.

James V. Bruckner

PFOA-specific comments
The document's authors have done a good job describing and integrating the findings of the numerous studies in which liver weight gain was observed. Although there is a consensus about the effect and the dosage required to elicit it in different species, this reviewer does not believe it should be utilized, as described above. There are several clearly adverse effects such as elevated serum (hepatic) enzyme activities, focal hepatocellular necrosis, bile duct degeneration and fibrosis, etc. These effects are generally seen in response to relatively high PFOA doses, so the PODs will be higher than with liver weight increase. Alternatively, a human endpoint such as elevated serum cholesterol could be considered. See responses to Charge Question 3.

PFOS-specific comments
There have been a substantial number of well-conducted toxicological studies of PFOS. My major concern, as expressed above, is its potential to cause adverse effects in children. Other than that, PFOS doesn't appear to produce effects other than those anticipated from a repetitive, cumulative dose of an 8-carbon fatty acid.

Deborah A. Cory-Slechta

In general, it appears that, at least with respect to the animal studies, the choices are appropriate both in the case of PFOA and PFOS. The derivation of the RfDs/RfCs are based on studies of sufficient strength, duration and represent the most sensitive endpoints.

Having said that, in both documents, the reader is forced to that conclusion with no real assistance from the text itself. There is virtually no discussion of the strengths and weaknesses of the studies overall. Human study outcomes for the most part are simply enumerated, although an occasional statement will be made about a limitation (usually) of one of those studies. There is no discussion in the human studies of the power to detect effects, the sample sizes, etc. Much weight seems to be given to occupational studies in some cases, being used to essentially dismiss effects in a community cohort as the same effect was not seen in occupationally exposed workers, when in fact finding effects in a population with seemingly longer, albeit lower exposure levels actually makes the outcome more robust. Also, population studies with smaller sample sizes that nevertheless find significant effects are in fact more compelling and suggest robust effects which can be detected even with a small sample size. This deficiency is manifest in statements such as those in the PFOS document (p. 5-1) that 'in most cases the findings are suggestive and not conclusive of an effect'.

There is a bit more discussion of the animal studies in both documents, at least with respect to methods, but as with the human studies, there is little text addressing which studies represent stronger studies or what the weaknesses are. From these increase liver weight has been chosen as the endpoint from which to derive RfDs. This reviewer does not have an issue with that choice, as while it has been described as adaptive by some, it represents a response to an involuntary exposure with a direction of effect that is potentially associated with adverse consequences. The fact that it is reversible when exposure ends seems irrelevant as reversal of exposure is not happening in the human environment.

Jamie C. DeWitt

Strengths: The studies selected as key for quantification were generally well-conducted studies, employing a range of doses and sample sizes large enough for detecting statistical differences. Additionally, the doses associated with LOAELs for the identified critical endpoints were not associated with signs of overt or systemic toxicity in the animal models and nearly all of the studies measured serum and/or tissue concentrations of the parent compounds.

Weaknesses: No obvious experimental design weaknesses were noted in any of the studies selected as key for quantification.

Characterization: The studies selected as key for quantification for PFOA are all rodent studies while at least one study selected for PFOS quantification includes a non-human primate study. It is therefore surprising that the PFOA database does not include, as a study key for quantification, the Butenhoff et al. 2002 study of non-human primates. Additionally variability in putative mechanisms among species was not adequately addressed in the characterization of the selective studies, although all of the selective studies were descriptive and not mechanistic.

Jeffrey W. Fisher

PFOA and PFOS: Data bases are massive and both need to be updated. Several human studies and a few non-human primate toxicity studies are available. The authors need to explain why these studies are not adequate for causality (dose-response).

William L. Hayton

An advantage to assessment of health effects for both PFOA and PFOS is the large amount of published work that informs the topic. While the draft health-effects documents have summarized the results of many pertinent studies, the literature reviewed was not comprehensive, which projects an appearance of weakness. The documents do not state whether the intention was to include all relevant health-effects literature, or to be selective and summarize those studies judged to be most relevant. Such a statement at the beginning of the documents would be helpful; a cut-off date for the literature review would also frame expectations of readers. If the intention was to be selective, a description of selection criteria would help allay concerns of readers about papers that were not included. If the intention was to comprehensively review all the PFOA and PFOS health-effects literature, then it appears that

more work should be done to include omitted works. Public comments list a number of works to consider for inclusion.

A general, albeit minor weakness of the literature is that PFOA and PFOS serum concentrations in control animals were not measured for many studies – they were likely non-zero and, since there is no information on how high they were, it is possible that baseline health-effects metrics were affected and that dose-response relationships were affected, especially in the low dose range. It is perhaps worthwhile to mention this shortcoming somewhere in the health effects documents.

Matthew P. Longnecker

EPA may want to consider the article by AP Hall et al. 2012, about liver hypertrophy. The defense of increased liver weight as the POD (or a POD) could be strengthened by evaluating the evidence in the context of Hall's Figure 9, where evidence regarding hepatoxicity and toxic mechanisms are also considered. In this case, the possibility of an unknown mechanism exists that could be relevant to humans, and long-term exposure could have effects that have not yet been detected. See Hall page 986, where it defines adverse as: "…affects [response] to an additional environmental challenge". Thus, an adverse effect, via an unknown mechanism, by this definition is possible and has not been studied in animals or humans.

While AP Hall's article is not all that supportive of using increased liver weight as a point of departure (unless certain criteria are met), they are focused on animal studies, especially those done in rodents. If increased relative liver weight were to occur in a human population, I suspect that it would be considered an adverse outcome, whether or not there was evidence of hepatotoxicity or a specific mechanism. Note also that for PFOA, in monkeys, there was an increase in relative liver weight with chronic exposure (PFOA document, page 4-66), so increase in liver weight in the animal experiments may be relevant to humans.

An additional comment of relevance here pertains to whether the human data support hepatoxicity. While there are studies that report elevated liver function tests in subjects with higher serum concentrations of perfluorakyl substances, these elevations do not clearly support the presence of toxicity. Again, AP Hall's discussion of what constitutes evidence of hepatoxicity is relevant here, and takes into account the number of LFTs elevated, the specific LFTs involved, and the magnitude of their elevation.

Finally, as discussed at the meeting, for the PFOA document on page 5-23 ("RfD Selection"), and the PFOS document on page 5-26 ("RfD selection"), I suggest minor editorial changes to deemphasize the "consistency of response" point and instead focus a little more on how the RfD is robust to choice of POD endpoints. If the selection of RfD does not hinge on increased liver weight as a POD, it will be more defendable.

Transparency might be increased by saying why (more clearly, or more clearly by implicit reasoning) the Macon et al. 2011 study, in which the LOAEL was 0.01 mg PFOA/kg from GD10 to GD17, based on delayed mammary gland development, was not considered as a POD, and why the Hines et al. 2009 study, in which the LOAEL was 0.01 mg PFOA/kg from GD1 to

GD17, based on various outcomes, was not considered as a POD. The PFOS studies with low LOAELs were considered in the dose-response assessment (no suggestions for improvement there).

Angela L Slitt

My response is basically the same as my General Impressions above.

The documents provide a very thorough evaluation of PFOA and PFOS studies. It is logically organized, presenting findings in a way that the reader can understand the findings related to human, monkey, and rodents. The organization of the document makes allows the reader to easily find information about each species within the subchapters and summarizes key points in table form. PFOA is a well-studied compound, with a substantial amount of toxicokinetic and endpoint studies in rodents. Mechanistic data describing the role of membrane transporters to understand gender differences in PFOA elimination in rats is fairly well written. Little data exist regarding contribution of membrane transporters to PFOS disposition and elimination. The documents thoroughly describe species differences in PPAR-alpha signaling that might contribute to observed endpoints in rats, but not humans or monkeys. Overall, both documents are very thorough are provide a reliable basis for PFOS and PFOA evaluation.

For PFOA toxicokinetics, mechanisms of PFOA transport are important for understanding species differences in response to PFOA exposure, with focus placed on kidney. Figure 3-2 in the PFOA document does not adequately present the localization of renal transporters with relationship to their contribution to the urine compartment or renal reabsorption. A very nice diagram showing the subcellular localization of renal transporters presented by Klaassen and Aleksunes (Pharmacol Rev. 2010 Mar;62(1):1-96) clearly depicts the contribution of various transporters to filtrate or blood. This is an easier diagram to put PFOA elimination into context than the one presented. Contribution of membrane transporters to species differences in PFOA excretion Section 3 (specifically 3.4.1) would be put in better context if a table could be generated to compare Km and Vmax values for PFOA for various transporters, with specific focus on species information for OATps and OATs. Data regarding information on contribution for OATps in liver accumulation of PFOS and PFOA is lacking, with specific regard to species differences. As PFOS is a likely candidate for hepatic uptake transport, understanding a mechanism to explain species differences in hepatic effects possibly due to difference in hepatic exposure is critical. Understanding impact species specific regulation of OATp expression in liver (e.g. whether species difference in PPAR-alpha signaling contributes) is also important in putting rodent distribution data into context.

Increased liver weight is considered to be a critical effect, but how increased liver weight relates to the observed human and monkey health effects needs to be further explained. In layman terms, if someone is walking around with an increased liver weight, is he or she at risk for disease? Will his/her life span be shortened? To increase transparency of the document, a more comprehensive explanation is needed to justify why increased liver weight should be considered as a critical endpoint for human health.

Use of humanized PPARα mice are a sexy tool to delineate species differences in effects associated with peroxisome proliferation. For transparency, the document should acknowledge the limitations of that model. Specifically, lack of response may not necessarily correlate to a lack of response for human PPARα because of species differences in binding to cogate DNA elements (e.g. a human receptor may have lower binding capacity to mouse DNA due to structural differences and species differences in co-activator/co-repressor interactions). Wording in the documents using these mice should acknowledge this limitation.

The documents often have redundancy in information, especially in regard to hormone effects (there are very similar write ups in sections about effects on thyroid hormone) and metabolic/cardiovascular disease risk factors (e.g. lipid endpoints).

Question 2. Please provide citations (and, where possible, pdfs or hard copies) for any references you suggest EPA consider adding to the document. Describe where you suggest these references be incorporated.

James V. Bruckner

PFOA-specific comments
Fabrega, F. et al. (2014). PBPK modeling for PFOS and PFOA: Validation with human experimental data. Toxicol. Lett. On line. (Hard copy available)

Stahl, T., Mattern D and Brunn, H. (2011). Toxicology of perfluorinated compounds. Environ. Sci. Europe 23: 38-60.

Hall, A. P., et al. 2012. Liver hypertrophy: A review of adaptive (adverse and non-adverse) changes- Conclusions from the 3rd International ESTP Expert Workshop. Toxicol. Pathol. 40: 971-994.

Bjork, J. A., Butenhoff, J. L., and Wallace, K. B. 2011. Multiplicity of nuclear receptor activation by PFOA and PFOS in primary human and rat hepatocytes. Toxicology 228: 8-17.

PFOS-specific comments
No additional references were located.

Deborah A. Cory-Slechta

For both PFOA and PFOS, the document should include a description of the process through which studies were identified and how they were processed for inclusion or not. It is not clear what the exact dates of the studies examined included, i.e., what the cut-off date was for these studies. This makes it difficult to evaluate whether there are missing studies. That said, this reviewer is not aware of any specific omissions in the peer-reviewed literature other than those that were discussed at the face-to-face meeting.

Jamie C. DeWitt

Granum, B., Haug, L.S., Namork, E., et al. 2013. Pre-natal exposure to perfluoroalkyl substances may be associated with altered vaccine antibody levels and immune-related health outcomes in early childhood. J Immunotoxicol. 10:373–379; Looker, C., Luster, M.I., Calafat, A.M., et al. 2014. Influenza vaccine response in adults exposed to perfluorooctanoate and perfluorooctanesulfonate. Toxicol. Sci. 138:76–88.

Any time the Grandjean et al. (2012) findings related to PFAS and vaccine responses are discussed, these references could/should be discussed as well as they report related findings in human populations. Although they also are confounded by multiple PFAS (as was the Grandjean et al. study), they lend additional support to immunotoxicity as an endpoint worthy of consideration. However, it is noted that these references were published after the cutoff date for consideration for inclusion in the document.

Lopez-Espinosa, M.J., et al. 2012. Thyroid function and perfluoroalkyl acids in children living near a chemical plant. Environ.Health Perspect. 120:1036–1041. This study is missing from the discussion of thyroid hormone disruption. It reports a positive correlation between hypothyroidism and PFOA in children from the C8 population aged 1-17.

Corsini E., et al. 2011. In vitro evaluation of the immunotoxic potential of perfluorinated compounds (PFCs). Toxicol. Appl. Pharmacol. 250:108-116. Corsini E. et al. 2012. In vitro characterization of the immunotoxic potential of several perfluorinated compounds (PFCs). Toxicol. Appl. Pharmacol. 258:248-255.These studies are in vitro/ex vivo studies of human-derived cells that provide evidence that in vitro measures of immunocompetence in mice may be relevant to the human experience.

Jeffrey W. Fisher

For completeness sake, at least, please update lab animal studies conducted since 2012.

William L. Hayton

A review of PFOA health-effects literature (GB Post et al. (2012) Environ. Res. 116: 93-117) provides an excellent, in-depth discussion of many issues covered in the PFOA health effects document. Consider citing this review in the document.

The literature on PFOA and PFOS toxicokinetics (Section 3) has been comprehensively covered in the health effects documents, with the notable omission of Wambaugh et al., Dosimetric Anchoring of *In Vivo* and *In Vitro* Studies for Perfluorooctanoate and Perfluorooctanesulfonate. Toxicol. Sci. 136:308-327, 2013. This paper informed a significant part of the health effects documents.

Commenter's have suggested a number of references to consider with regard to Section 4 Hazard Identification. Many recent publications report on toxicity associated with PFOA/PFOS exposure. For the Dose-Response Assessment (Section 5) it is desirable to focus on those toxicities that have occurred at the lowest PFOA/PFOS exposures. For PFOA, the literature that is used in Section 5 to determine an RfD was published prior to 2009 (Tables 5-8 – 5-11). The benchmark response chosen based on the Section 4 literature was a 10% increase in liver weight, which was the biological response that occurred at the lowest PFOA exposure; it was acknowledged that this response "… is a biomarker for systemic exposure in rodents, rather than a biomarker of adversity …" (p. 5-6). More recent studies of hazard have identified potential adverse effects that result from, or are associated with, PFOA exposures that are lower than the LOAEL for a 10% increase in liver weight. For example, adverse effects on fetal, neonatal and early childhood stages of development may occur at lower exposures than does liver weight gain, which suffers in addition from not being a biomarker of adversity, and which therefore raises a question about the validity of any RfD based upon it. Macon et al. 2011 reported an LOAEL for delayed mammary gland development of 0.01 mg/kg administered to pregnant CD-1 mice during GD10 – GD17. As this relatively brief exposure was well below that required for steady state, it is possible that had the dams been at steady state at the time of conception (about 9 weeks of exposure) a much lower LOAEL may have been observed; i.e., a much lower dose

rate at steady state would have produced the same exposure to the fetal pups as did the 0.01 mg/kg administered to the dams during GD10 – GD17. The steady state situation is more relevant to adverse effects in humans than is a brief exposure.

Matthew P. Longnecker

I suggest you include the following citation and include a discussion of the evidence presented: Paula I. Johnson, Patrice Sutton, Dylan S. Atchley, Erica Koustas, Juleen Lam, Saunak Sen, Karen A. Robinson, Daniel A. Axelrad, and Tracey J. Woodruff. The Navigation Guide—Evidence-Based Medicine Meets Environmental Health: Systematic Review of Human Evidence for PFOA Effects on Fetal Growth. Environ Health Perspect; DOI:10.1289/ehp.1307893 (in press and available through the journal's website).

Based on the meta-analysis in this paper, the evidence that PFOA is associated with lower birthweight is consistent. Thus, the rationale for not basing the POD on the human data needs to be strengthened, as noted above. The Johnson et al. report could be discussed in the section on anthropometric endpoints that begins on p 4-22.

The relationship between birthweight and PFOA or PFOS may be confounded because glomerular filtration (and hence excretion of the compounds) is proportional to birthweight, as discussed in:

Morken NH, Travlos GS, Wilson RE, Eggesbø M, Longnecker MP. Maternal glomerular filtration rate in pregnancy and fetal size. PLoS One. 2014 Jul 8;9(7):e101897

In the PFOA document, on page 4-18, you might want to also cite:

Taylor KW, Hoffman K, Thayer KA, Daniels JL. Polyfluoroalkyl chemicals and menopause among women 20-65 years of age (NHANES). Environ Health Perspect. 2014 Feb;122(2): 145-50.

The Taylor et al., like the Knox et al. report (already cited in the PFOA document) is from a large-cross sectional study. Both studies, in their discussion sections, note that the association of PFOA or PFOS concentration in serum with age at menopause could be expected because postmenopausal women have lost a route of excretion for the compound and will have higher serum concentrations on that basis. It would be worth noting this possible explanation in the PFOA document on page 4-18, and in the PFOS document on page 4-8.

Additional data are available on the potential carcinogenicity of PFOA:

- Steenland K, Woskie S. Cohort mortality study of workers exposed to perfluorooctanoic acid. Am J Epidemiol. 2012;176(10):909-17.
- Barry V, Winquist A, Steenland K. Perfluorooctanoic acid (PFOA) exposures and incident cancers among adults living near a chemical plant. Environ Health Perspect. 2013;121(11-12):1313-8
- Hall AP et al. Toxicol Pathol 2012:40:971-94. (About liver hypertrophy.)

The Steenland and Barry reports could be discussed in Section 4.1.2, on pages 4-28 and 4-29, respectively.

Angela L. Slitt

1) Evidence is presented for PFOA and PFOS as substrates for the related OATp1d1 in zebra fish. Establishing whether PFOS is an OATp transporter substrate is needed to better understand PFOS accumulation in liver. This study suggests that it might be. The following finding should be included in the PFOS document in Section 3.2.3 and the PFOA document in Section 3.0:
    a. Popovic M, Zaja R, Fent K, Smital T. Toxicol Appl Pharmacol. 2014 Interaction of environmental contaminants with zebrafish organic anion transporting polypeptide, OATp1d1 (Slco1d1).

2) This publication presents the finding that PFOS inhibits Pgp, Mrp1, and Mrp4 activity. The following finding should be included in the PFOS document in Section 3.2.3 and the PFOA document in Section 3.0:
    a. Dankers AC1, Roelofs MJ, Piersma AH, Sweep FC, Russel FG, van den Berg M, van Duursen MB, Masereeuw R. Toxicol Sci. 2013 Dec;136(2):382-91. Endocrine disruptors differentially target ATP-binding cassette transporters in the blood-testis barrier and affect Leydig cell testosterone secretion in vitro.

3) PFOS induced ABC transporters in grey mullets.
    a. de Cerio OD1, Bilbao E, Cajaraville MP, Cancio I. Gene. 2012 Apr 25;498(1):50-8. Regulation of xenobiotic transporter genes in liver and brain of juvenile thicklip grey mullets (Chelon labrosus) after exposure to Prestige-like fuel oil and to perfluorooctane sulfonate.

4) These are new publications regarding epidemiology findings for PFOS and PFOA exposure and serum lipids:
    a. Fitz-Simon N, Fletcher T, Luster MI, Steenland K, Calafat AM, Kato K, Armstrong B. Epidemiology. 2013 Jul;24(4):569-76. doi: 10.1097/EDE.0b013e31829443ee. Erratum in: Epidemiology. 2013 Nov;24(6):941.
    b. Starling AP, Engel SM, Whitworth KW, Richardson DB, Stuebe AM, Daniels JL, Haug LS, Eggesbø M, Becher G, Sabaredzovic A, Thomsen C, Wilson RE, Travlos GS, Hoppin JA, Baird DD, Longnecker MP. Perfluoroalkyl substances and lipid concentrations in plasma during pregnancy among women in the Norwegian Mother and Child Cohort Study. Environ Int. 2014 Jan;62:104-12.
    c. Fu Y, Wang T, Fu Q, Wang P, Lu Y. Associations between serum concentrations of perfluoroalkyl acids and serum lipid levels in a Chinese population. Ecotoxicol Environ Saf. 2014 Aug;106:246-52.
5) These are publications regarding PFOS exposure and hepatic steatosis:
    a. Lv Z, Li G, Li Y, Ying C, Chen J, Chen T, Wei J, Lin Y, Jiang Y, Wang Y, Shu B, Xu B, Xu S. Glucose and lipid homeostasis in adult rat is impaired by early-life exposure to perfluorooctane sulfonate. Environ Toxicol. 2013 Sep;28(9):532-42. doi: 10.1002/tox.20747. Epub 2011 Aug 24. PMID: 23983163 Select item 22484034

b. Wan HT, Zhao YG, Wei X, Hui KY, Giesy JP, Wong CK. PFOS-induced hepatic steatosis, the mechanistic actions on β-oxidation and lipid transport. Biochim Biophys Acta. 2012 Jul;1820(7):1092-101. doi: 10.1016/j.bbagen.2012.03.010. Epub 2012 Mar 28. PMID: 22484034 [PubMed - indexed for MEDLINE] Free Article
c. Bijland S, Rensen PC, Pieterman EJ, Maas AC, van der Hoorn JW, van Erk MJ, Havekes LM, Willems van Dijk K, Chang SC, Ehresman DJ, Butenhoff JL, Princen HM. Perfluoroalkyl sulfonates cause alkyl chain length-dependent hepatic steatosis and hypolipidemia mainly by impairing lipoprotein production in APOE*3-Leiden CETP mice. Toxicol Sci. 2011 Sep;123(1):290-303. doi: 10.1093/toxsci/kfr142. Epub 2011 Jun 24.

Question 3. The OW concluded that the human epidemiology data for PFOS/PFOA do not provide adequate quantifiable dose-response information for use as the basis of a candidate RfD because of uncertainty regarding the routes, levels and timing of exposures plus the confounding influences of other PFCs present in serum. Please comment of the OW characterization of the data.

James V. Bruckner

PFOA-specific comments
The document's authors have done a good job summarizing and accurately characterizing the epidemiology literature for various endpoints in Section 4.4 - Hazard Characterization. It is true there are a number of confounding factors that make estimation of PFOA exposures difficult. The EPA might consider, however, utilization of reverse dosimetry modeling. There is a reasonable body of data on serum PFOA levels, which could be used to estimate a range of PFOA exposures that would result in such internal doses.

PFOS-specific comments
I agree that human epidemiology data do not provide an adequate basis for calculation of a RfD or RfC. A reverse dosimetry modeling approach, however, could be used to estimate a range of PFOS exposures that could have resulted in measured body burdens. The human data might then be utilized in the risk assessment.

Deborah A. Cory-Slechta

It is not clear that such an assertion should be used in the construction of this document. It is not clear why the route of exposure should be raised to a concern in the calculations, in fact in the human environment, there are exposures from multiple routes, no doubt and thus this is consistent with human environmental exposures. Further, if there is data on serum levels, it should reflect that cumulative exposure across exposure routes. Indeed, at the end, the goal is to arrive at an RfD based on serum levels. There is, moreover, no guarantee that there is no contamination in studies in animals from food, glassware etc.

Furthermore, in many epidemiological studies in which mixed exposures are the norm, controlling for other exposures is utilized to address this concern and to therefore make conclusions about individual exposures. In point of fact, in every single human study, there will invariably be other exposures and not a single exposure, and thus this strategy essentially says that no human studies can ever be used for any risk assessments. The stated rationales for not using human data based on these statements is not adequate. This is why it is important as well to evaluate the strengths and weaknesses of each of the studies in terms of whether appropriate controlling for other known exposures was carried out and sample sizes sufficient etc. to arrive at some conclusions with respect to their ultimate usability in constructing RfDs.

Jamie C. DeWitt, Ph.D..

While the OW characterization of the epidemiological data for PFOA/PFOS is, technically, true, it also is somewhat misguided. Almost any epidemiological database will contain uncertainty regarding the routes, levels, and timing of exposures and will have confounding influences of other compounds. Very few epidemiological studies are free from these uncertainties, but when similar observations and conclusions are reached from multiple studies with these types of uncertainties, the database becomes useful for determining a candidate RfD or other value relevant to human health. What is particularly valuable about the PFOA/PFOS database is that it is relatively extensive in that it includes data not only from occupationally-exposed humans, but from people highly exposed to environmental concentrations of PFOA/PFOS and from people in the general population who have detectable concentrations of these compounds. Additionally, for establishing an RfD, do all of these uncertainties need to be absent? In other words, do animal studies used to derive RfDs lack these uncertainties?

What is missing from the OW characterization of the epidemiological data is a thorough evaluation concerning hepatotoxicity and developmental toxicity reported in human populations and how these endpoints are relevant to or related to animal studies.

Jeffrey W. Fisher

The use of non-human and human data is very important for interpreting exposure extrapolations from rats. I am not an epidemiologist so I cannot comment with authority on the epidemiology data for dose-response. Justify why human data are not suitable for use in the analysis of the health hazards of PFOA and PFOS.

William L. Hayton

There are a number of epidemiological studies that have been based on large numbers of subjects chronically exposed (over decades in some studies) to the subject compounds over a broad range of intakes. Steady state serum concentrations have also been available for quantification of the systemic exposure. While the route, levels and timing of the exposures may have been uncertain, the long half-lives of PFOA and PFOS in humans and the long periods of exposure to them indicate that 1) subject serum concentrations were generally at steady state, and 2) daily fluctuations in the amount and timing of the exposure would not produce much day-to-day fluctuation in the serum concentration of PFOA/PFOS. These consequences of the long exposure period and long half-life indicate that variability in the route and level of exposure would not have led to a measured serum concentration that was unrepresentative of the subjects' long-term average serum concentration. The serum concentration then should be relatively stable over time and it should reflect an integrated measure of the individual's exposure to PFOA and PFOS.

The serum concentration is a quantitative measure of systemic exposure to the subject chemicals, and is arguably a better metric of exposure than are intake rate. The over-all rate of intake (R) that produces a particular steady state serum concentration (Css) can readily be

calculated from the clearance (CL) of the chemicals, which is about 0.08 mL/d/kg body weight: R = Css x CL. The calculated rate of intake would represent all intake routes.

Confounding influences of other PFCs and indeed other chemicals and life-style factors such as smoking, diet, alcohol use, etc. would have to be considered, as is generally the case with epidemiological studies. Methodology exists for dealing with such influences.

Thus it appears that the epidemiological results should be used in the RfD determination. Their strength is that uncertainties associated with extrapolation from laboratory animal studies are avoided. Health effects that are positively associated with serum PFOA/PFOS concentration and that are observed in large populations of subjects should seriously be considered as potentially arising from PFOA/PFOS exposure. If mode of action studies in lab animals or in vitro studies support a cause-effect relationship, then the threshold serum concentration could inform the calculation of the RfD.

Matthew P. Longnecker

As noted in the General Impressions section above, the human studies with data on plasma or serum concentrations of PFOA and PFOS, especially for several categories of such levels, could be used to estimate dose-response information. However, there are other reasons why the human data may not be useful for setting the RfD (see above). Either PK or PBPK models might be useful for estimating the dose that human are exposed to; an advantage of a PBPK model is that it could incorporate information about routes and timing of exposure. Estimates of the contribution of various routes are available (e.g., Haug et al. 2011; Lorber & Egeghy 2011), and exposure trends could be assumed and evaluated in sensitivity analyses. Some occupational studies had data that allowed an estimate of serum levels, or measured them directly. Several reports show estimated exposure based on serum concentrations of PFOA or PFOS (Locissano et al. 2013; Lorber & Egeghy 2011; Thompson et al. 2010). With respect to confounding, the assessment of how likely this is could be informed by: 1) the correlation of serum concentration of PFOA, PFOS, and other compounds of this type in a particular study population (or in a series of studies), and 2) whether the other compound(s) has been associated with the particular outcome being considered. If the correlation is low or the other compound has not been associated with the outcome, concern about confounding may not be strongly justified. Without additional consideration of data that address these points, it may be premature to assume confounding would be a problem.

Angela L. Slitt

Strengths of the studies: Several studies, which all demonstrate a positive association between serum PFOA and/or PFOS and cholesterol or LDL levels are based on drinking water as a route of exposure. These studies are in agreement with Nelson et al., 2010, which was analyzing data from the 2003-4 NHANES study. Steenland et al., 2009 (*Environ Health Perspect.* Jul 2009; 117(7): 1083–1088) as part of the C8 Health Project collected data on 69,030 subjects with findings that serum PFOA was higher for males, those consuming local vegetables, and those using well water rather than public water, and *lower for those using bottled water.* The estimated response rate for participants >20 years of age was 81% and mean serum PFOA

concentration was 83 ng/l. Subjects were eligible to participate in the C8 Health Project if they had consumed drinking water for at least one year before 3 December 2004 supplied by Little Hocking Water Association (Ohio), City of Belpre (Ohio), Tuppers Plains Chester Water District (Ohio), Village of Pomeroy (Ohio), Lubeck Public Service District (West Virginia), Mason County Public Service District (West Virginia), or private water sources within these areas that were contaminated with PFOA. Subjects were also eligible if they could document that they had either worked in a contaminated water district or went to school there for at least one year. From this population, which the route of exposure is considered to be primarily via drinking water, serum lipids were analyzed with regard to PFOA levels and a positive correlation was observed for all serum lipids except HDL. Frisbee further characterized this cohort, analyzing 12, 476 children and adolescents included in the C8 Health Project, finding an increase in total cholesterol.

A recent epidemiology study (Fitz-Simon et al., 2013), not included in the current documents, described positive associations between PFOA and PFOS in serum LDL cholesterol. This study examined a study population that consisted of 560 adults living in parts of Ohio and West Virginia where public drinking water had been contaminated with PFOA. They had participated in a cross-sectional study in 2005-2006, and were followed up in 2010, by which time exposure to PFOA had been substantially reduced. Overall, the findings demonstrate a positive association between serum PFOA and PFOS levels and serum and LDL cholesterol.

Weaknesses: The studies did not appear to analyze PFOS or PFOA levels in drinking water from the participants analyzed and did not analyze data based on the length of exposure.

Question 4. Please comment on the transparency and characterization of the epidemiological data.

James V. Bruckner

PFOA-specific comments
See comments above.

PFOS-specific comments
The document's authors have done a good job describing and summarizing the designs and findings of the epidemiology studies.

Deborah A. Cory-Slechta

The PFOA document in particular and to some extent the PFOS document present all of the epidemiological studies but do not actually evaluate them; there is not a consistent indication of individual strengths and limitations of the studies, failures or not to adequately control potential confounding variables. Furthermore, there is no 'power analysis' type of evaluation, i.e., some of these studies included very small sample sizes and thus their power to actually detect effects may be limited, and yet they all appear to be weighted basically the same, i.e., studies with very small sample sizes with obviously extremely limited power to detect any effects appear to be considered the same as those with extremely large sample sizes. Studies with small sample sizes that nevertheless do find an effect of PFOA or PFOS actually suggest a robust type of effect.

The discarding of positive associations in human epidemiological studies because they do not produce frank clinical disease seems inappropriate and inconsistent with other EPA documents. For example, p. 4-3 in the PFOS document states that only a small number of ALT values were outside the normal range making the results difficult to interpret in terms of health. Physiological changes that are moving in the wrong direction, even if sub-clinical at the time, are still adverse effects. Are actual clinical diagnoses required for an adverse effect? This is especially the case given that the ranges of normal across populations are extremely broad.

The latter also raises the question of the cumulative toxicity of PFOA and PFOS and whether any consideration is being given to this.

Another such example is in the PFOS document, where it actually refers to a statistically significant, but not toxicologically significant effect (p.4-38); what does that mean? Also, p. 5-4 appears to dismiss any changes in thyroid function since no evidence of clinical hypothyroidism actually occurred. This whole approach with the human studies seems quite inconsistent with the reliance on increased liver weight in the absence of clinical pathology as the endpoint in the human studies.

Jamie C. DeWitt

It is not obviously or abundantly clear how the OW characterized the epidemiological data for either PFOA or PFOS. The studies were well-described, but the contribution of particular studies

to the overall assessment was not. The results of studies described in the hazard characterization section (4.4) need to be better characterized. For example, in the PFOA risk assessment:

- An increase in serum lipids associated with PFOA/PFOS exposure in humans is discussed as a risk factor for cardiovascular disease in humans; however, no evidence of increased cardiovascular disease has been observed in human populations. Additionally, serum lipids typically are decreased in animal models after PFOA/PFOS exposure, which is thought to be associated with/typical of exposure to agents that activate PPARα. If humans are known to respond to PPARα activators (i.e., fibrate drugs), why would the results between humans and animal models be discordant? This should be discussed.
- Several epidemiological studies reporting changes in liver enzymes clearly state that the clinical relevance of the changes in enzymes is unknown. Therefore, stating that the human studies "suggest effects on the liver as indicated by increases in liver enzymes" amounts to a mischaracterization of the data.
- No direct evidence of hepatotoxicity has been reported in epidemiological studies. This should be discussed.
- More in-depth characterizations are needed for the additional sections of the hazard characterization, with the exception of the thyroid section, which was well-described.

For example, in the PFOS risk assessment: Similarly to the PFOA risk assessment, the hazard characterization section needs to better discussion differences and similarities between effects reported in humans and effects reported in animal models.

Jeffrey W. Fisher

I am not an epidemiologist, but it appears to be adequate. Better characterization of the pros and cons of the human analyses and interpretation of the outcomes would be helpful.

William L. Hayton

The characterization of the individual epidemiological studies presented seems to be adequate. Public comments have identified the need to distinguish positive and negative associations with statistical significance, which seems to be a fair criticism. As noted in the response to Question 2, there are relevant studies that have not been described in the health-effects documents that ought to be considered and this includes some epidemiological studies. Most of the cited epidemiological studies have focused on healthy adults – workers exposed occupationally, residents of communities with or without contaminated water. These populations might be expected to be less sensitive to adverse effects than would early life stages and particular disease populations. Studies of potentially more sensitive populations would be desirable. The Frisbee et al. (2010) study of children 1-11.9 years and adolescents 12-17.9 years showed significant positive associations with serum lipid levels. Studies such as this one would be informative.

Matthew P. Longnecker

Please see the long paragraph above, under General Impressions, and some of the comments in response to item #2 above. Another point that the authors may want to consider is that studies that examine external exposure in relation to health outcomes may have special advantages in the case of PFOA and PFOS. While in general it is considered best to have a measure of exposure that is based on a biomarker of internal exposure, this may be problematic for several outcomes for PFOA and PFOS, because of the possibility of confounding or reverse causality that would not be an issue if an external estimate of exposure were used. For example, in Steenland K, Zhao L, Winquist A. A cohort study of workers exposed to PFOA. Occup Environ Med. 2014 Jun;71 Suppl 1:A55, when an external estimate of exposure was used for the Washington Works employees, no association with elevated cholesterol was found. The Viera et al. (2013) results are based on external estimates of exposure, whereas the similar study by Barry et al. (2013) are based on serum levels or estimates based on serum levels. The fact that association with kidney cancer is present in the Viera study decreases concern that the association was due to reverse causality. Steenland et al. 2012 used an external estimate of exposure to study cancer mortality and also found an association with kidney cancer. Lundin et al. (external estimate of exposure) had no cases of kidney cancer, though their study was also small.

Angela L. Slitt

The epidemiology data is well described and a thorough read. The data would be put in better context for the reader if there are average serum concentrations or ranges for the studies summarized in tables in addition to other key pieces of information.

A recent publication should be included in the document for consideration. Simon N, Fletcher T, Luster MI, Steenland K, Calafat AM, Kato K, Armstrong B. Epidemiology. 2013 Jul;24(4):569-76. doi: 10.1097/EDE.0b013e31829443ee.

Question 5. The OW has concluded that the cancer classifications for PFOA and PFOS are most consistent with respective classifications of suggestive evidence for carcinogenicity as described the EPA Guidelines for Carcinogen Risk Assessment (pp. 2-56, 2-57). Please comment on the strengths and weaknesses of this classification.

James V. Bruckner

PFOA-specific comments
I agree with EPA's choice of "Suggestive Evidence for Carcinogenicity." Epidemiological findings in occupationally-exposed and general populations to date are equivocal. Increases in Leydig cell tumors and liver adenomas have been reported in high-dose male rats. Increased incidences of pancreatic cell hyperplasia/adenomas and ovarian stromal hyperplasia/adenoma have been observed in female rats. More studies are necessary to confirm/expand these findings, and to assess carcinogenic potential in other species. Most mutagenicity and genotoxicity assays have been negative. Thus, there is some, but not undue cause for concern about the human carcinogenic potential of PFOA.

PFOS-specific comments
The document's authors have adequately and convincingly presented evidence for classifying PFOS as "suggestive of carcinogenicity."

Deborah A. Cory-Slechta

The classification of both PFOA and PFOS evidence for carcinogenicity as suggestive seems consistent with the clear limitations in the available data bases. In addition, the animal studies are limited to one species and mutagenicity does not occur in response to PFOA.

Jamie C. DeWitt

This classification is appropriate for both PFOA and PFOS given the epidemiological evidence, which is somewhat limited for PFOA and quite limited for PFOS. For PFOA, there is an association between kidney and testicular cancer, but there are limited data in animal models for these cancers and there is uncertainty that the mechanism of PFOA-induced carcinogenicity in animal models is applicable to humans. Studies of PFOS have the same limitations, but epidemiological studies have failed to find an association between PFOS exposure and cancer.

Jeffrey W. Fisher

I did not review the cancer studies for PFOA and PFOS.

William L. Hayton

The classification of "suggestive" is not unreasonable. The epidemiological studies, while showing apparent associations between PFOA exposure and cancer incidence in testicle and kidney as well as other tissues, do not provide a cause-effect relationship. However, they certainly do raise a concern about the carcinogenicity of the subject substances. Studies in animals have demonstrated conclusively that PFOA causes liver cancer in rats but the MOA that involves PPAR activation is absent in humans and it has been concluded that PFOA and PFOS cannot be carcinogenic in humans via this mechanism.

An EPA SAB panel (2006) consideration of this question resulted in a majority of the panel members favoring a classification of "likely to be carcinogenic" for PFOA. Board members acknowledged the PPAR MOA argument against causation of cancer in humans, but also found evidence that liver cancer in rats administered PFOA may also have had a MOA independent of PPAR activation. Recent epidemiological studies have added to the weight of evidence for an association between PFOA/PFOS exposure and cancer. Therefore a classification of "likely" is also not unreasonable to this reviewer. Lacking expertise in the nuances of applying the EPA's classification scheme, it is difficult for this reviewer to argue in favor of either "suggestive" or "likely".

Matthew P. Longnecker

The classification as "suggestive evidence for carcinogenicity" for both PFOA and PFOS is consistent with the guidelines put forth in the EPA Guidelines for Carcinogen Risk Assessment (2005). There are few pertinent data, including some suggestive but weak human evidence. There is clearly not enough evidence to classify these agents as likely human carcinogens.

Angela L. Slitt

Overall, the assessments for each PFOS and PFOA appear to be consistent with the EPA Guidelines for Carcinogen Risk Assessment (U.S. EPA, 2005a).

Strengths: Both classifications use evidence from human studies as guidance.

PFOS: The limited data that exist regarding PFOS and cancer were presented, the classification for PFOS under the EPA Guidelines for Carcinogen Risk Assessment (U.S. EPA, 2005a) is currently consistent with the suggestive evidence of carcinogenic potential descriptor. This assessment is reasonable, given that it is based on two studies that show a slight increase in adenomas that occurred in males and females.

PFOA: There is conflicting evidence regarding PFOA exposure and cancer risk. However, several human studies have found associations between PFOA exposure and elevation of cancer of the bladder and kidney. This is also supported by a chronic bioassay in rats, which demonstrated that PFOA was tumorigenic.

Question 6. Significant interspecies differences in pharmacokinetics exist for both PFOA and PFOS. Adjusting for interspecies differences was an important step in developing candidate RfDs given the totality of the human and animal data. Please comment on the strengths and weaknesses of the pharmacokinetic model adjustments to accommodate the impact of albumin binding and renal tubule transporters in determining average serum values.

James V. Bruckner

PFOA-specific comments
The adjustments made to accommodate the influence of albumin binding and saturable renal tubular resorption of PFOA seem reasonable. I would defer, however, to someone with more experience in providing for these processes in PBPK models.

PFOS-specific comments
The PBPK model adjustments to estimate human equivalent doses appear to be appropriate. I defer to someone more qualified on the subject.

Deborah A. Cory-Slechta

This falls outside my area of expertise and therefore no significant comments are provided. However, at the face-to-face meeting there was significant discussion regarding the modeling, including clarifications from Dr. Wambaugh that were found by those panel members with expertise to clarify these issues and these particular issues were considered adequately addressed.

Jamie C. DeWitt

Several PK models have been reported in the literature for these compounds and are relatively well described in the documents. The documents assert that the existing PK models do not consider the impact of renal tubule transporters and albumin binding; while, many of the existing models appropriately predict serum concentrations in humans and other species, but they are mostly based on empirical models. Please explain the weaknesses of such empirical models.

Additionally, numerous studies for both compounds report serum and tissue concentrations in humans and other species, which can be compared to existing models. Both documents present a revised model that amounts to a reanalysis of data from studies that report serum concentrations. A more thorough discussion of the improvements made by the reanalysis is needed to better understand if the improved model adequately estimates or predicts the clearance rate and other parameters for which confidence is low. Alternatively, the publication (Wambaugh et al., 2013) that thoroughly describes the reanalysis could be referenced.

Jeffrey W. Fisher

Serum protein binding: Both PFOA and PFOS are highly bound in serum proteins across species, thus model adjustments seem trivial for interspecies extrapolation. Steady-state conditions can be assumed to estimate the free fraction (e.g., 2% based on paper by Han et al., 2003 for humans). I did not find a discussion about the half-life of serum proteins, which may have some influence on the 'apparent' serum half-life of PFOA and PFOS. The estimated fraction of free PFOA or PFOS is important for describing urinary and fecal elimination in rats (and other species) and the plasma concentrations of total PFOA and PFOS. Thus, the model predicts total PFOA and PFOS in serum or plasma, but the free fraction estimates drive the gradual clearance of total BPA from plasma or serum by describing clearance of free.

Renal reabsorption: The renal reabsorption hypothesis involving species specific and sometimes gender specific transporters to describe the pharmacokinetic data represents sound judgment. This departure from normal allometric scaling is suggestive of active transport processes. Few PBPK models explicitly describe transporters with drugs or chemicals, although the field is moving in this direction. Thus, the approach used for PFOA and PFOS is adequate, that is, a hypothesis was evaluated by employing empirical PK-based kinetic analyses. Because the mechanistic details are missing for each species/gender, scaling of this biological phenomenon is not possible at this time. This is not a weakness, but represents the state of the science.

William L. Hayton

A very important strength of the documents is the attempt to deal with the interspecies differences in pharmacokinetics so that adverse effects across species are compared on the basis of internal, systemic exposure to PFOA and PFOS, instead of basing comparisons on the administered mg/kg dosages. PFOA and PFOS have complicated pharmacokinetics that have proven difficult to model. While a relatively simple one-compartment model appears adequate to analyze single, low doses, this model fails when it is extended to higher doses and repeated doses. Nonlinearities appear associated with saturable plasma protein binding and with saturation of transporters thought to be involved in the reabsorption of the compounds from renal filtrate.

A weakness of the pharmacokinetic model adjustments is the lack of robustness of the models. Despite the extensive efforts of talented pharmacokineticists, development of a model that scales across species and handles a range of dosages and a variety of administration routes has proven elusive. The two compartment model of Andersen et al. (2006) has formed the basis of the model used in the draft documents. The model incorporates saturable resorption of PFOA and PFOS from renal tubular filtrate. While protein binding is known to be saturable (fraction free increases with concentration), the model uses a species-specific but constant free fraction. Model parameter values for mouse, rat and monkey were used to predict reasonably well measured serum concentrations after a fixed daily dosing regimen, Tables 5-6 – 5-8 for PFOA and 5-8 and 5-10 for PFOS. This agreement between predicted and measured serum concentrations gives confidence that the model-calculated AUC values and final serum concentrations associated with adverse health effects (or in the case of liver weight, biological marker of exposure) are realistic and a basis for estimation of RfD. While the model used

appears adequate for the purpose, the model parameters that were used have some markedly non-physiological values. (Information subsequently provided at the reviewers meeting explained some of the departure from expected physiological values, as discussed in a following section.)

Matthew P. Longnecker

For PFOA and PFOA, the MCMC model results (predicted final serum value) were compared to the measured final serum values, and the agreement was fairly good. For PFOS, the MCMC model results were compared to those from Loccisano et al. (2012b) and were found to be similar, which is also reassuring. Because the PBPK models of PFOA and PFOS are empirical, and have been shown to give results that agree reasonably well with observed data, the adjustments to accommodate the impact of albumin binding and renal tubule transporters are not critical. More data on albumin binding and renal tubule transporters might allow improved understanding of the pharmacokinetics of these compounds, but may not necessarily cause substantial improvements in the empirical predictions from current models.

Angela L. Slitt

The current weakness of the models is that data on species differences in PFOA and PFOA for various key transporters is limited and the document is also using mRNA data for various transport proteins to explain gender differences in urinary elimination. First, with regard to PFOS accumulation in the liver compartment, it is necessary to compare affinity of human versus rat for OATp mediated transport. This alone is tricky because of species differences in OATps. If PFOS-induced liver effects are related to PFOS accumulation in liver, it is would be helpful to understand whether a lower affinity of human OATp1b1 and 1b3 compared to rat OATp1a1 predicts lower hepatic PFOS accumulation. More is known about PFOA, but a similar argument can be made for PFOA. In addition, more comprehensive, controlled assessment of renal transporter affinity for PFOA and PFOS is needed to better model the species difference in urinary elimination.

The document often speculates about PFOA or PFOS regulation of transporter expression, but some papers cited (Cheng and Klaasen) do not have enough data at the protein level to support whether these differences in transporter expression are the drivers of toxicokinetic differences between males and females.

Question 7. Table 5-5 in the PFOA document and Table 5-7 in PFOS document list the parameters used for the ORD pharmacokinetic models that provide the final serum and AUC values for calculating the internal dose point of departure for the RfD calculation. Please comment on the strengths and weaknesses of the selected parameters.

James V. Bruckner

PFOA-specific comments
Despite the complexities and unknowns involved in plasma protein binding and renal tubular functions (i.e., glomerular filtration, basolateral tubular excretion and resorption, and apical tubular excretion and resorption), it is necessary to: (a) simply model only for saturable tubular resorption; and (b) use a range, or distribution of parameter values consistent with existing kinetic data. Unfortunately, optimization sometimes results in selection of physiological parameters that are not biologically-realistic, or plausible.

PFOS-specific comments
The parameters used in the modeling are biologically plausible.

Deborah A. Cory-Slechta

This falls outside my area of expertise and therefore no significant comments are provided. However, at the face-to-face meeting there was significant discussion regarding the modeling, including clarifications from Dr. Wambaugh that were found by those panel members with expertise to clarify these issues and these particular issues were considered adequately addressed.

One unclear component of Table 5-7 in the PFOA document is the column labeled Species/Strain Used for prediction, which in every case is the same as the column labeled Species/Strain and is not otherwise adequately explained.

Jamie C. DeWitt

It is not clear that the parameters in Table 5-5 in the PFOA document and Table 5-7 in the PFOS document are from the Andersen et al. 2006 PK model or if they are parameters used in the reanalysis of the data. This needs to be better explained in both documents. Additionally, all of the units in the tables need to be explained and re-checked for accuracy.

Jeffrey W. Fisher

The authors should entertain the calculation of data derived AUC (e.g. Table 5-6) to compare to the model derived AUC, just as was done with measurement of total PFOA in serum. This works for the animal studies. The choice of using the empirical model over the more recent physiological models may be a weakness and our understanding of transporters advance.
The evolution of chemical-specific PBPK models for use in risk assessment and regulatory applications has repeated itself several times. This is, the first empirical non-physiological model(s) or PBPK models contain hypotheses generating ideas and later models test some of

these hypotheses, especially if additional experimental data become available. In the case of PFOA and PFOS, the EPA selected not to use the most recent PBPK models for PFOA and PFOS, but instead use a computational empirical based model (Andersen et al. 2006) that was the first attempt to quantitatively interpret the kinetics of PFOA and PFOS across species of laboratory animals. The authors did publish their model (Wambaugh et al., 2013). The authors chose not to use a human model because a lack of information for Bayesian analyses. The justification for their extrapolation methods should be stated and the published reference for the model should be cited.

Model parameter distributions (Bayesian analyses) appear to be biologically implausible in some cases, covering many orders of magnitude. The authors should discuss this issue and check the units of model parameters in Tables.

Both model parameters tables need to include a description of what the parameter represents and cite a figure. The figures showing the Andersen et al. 2006 model do not show all the model parameters and have different nomenclature.

The Andersen et al. 2006 paper is a critical paper offering a quantitative explanation for the PFOA and PFOS kinetic data sets.

William L. Hayton

In the "Pharmacokinetic Model Approach" sections of the documents, it is not made sufficiently clear that the parameter values in Table 5-5 (PFOA) and Table 5-7 (PFOS) were from re-fitting the published data, rather than using parameter values from the original literature reports.

PFOA Table 5-5, p. 5-12

Body Weight and Cardiac Output values are reasonable and typical.

k_a values for mouse and monkey seem extremely large; absorption half-lives would be on the order of 10 seconds, which is physiologically unrealistic. All of an oral dose would be absorbed within a minute, mimicking a rapid i.v. bolus dose. Serum concentration-time profiles may not be sensitive to these values, however so they are not disconcerting for the intended use of the models. The rat values appear reasonable.

V_{cc} values appear reasonable. The total steady-state volume of distribution value [$V_{ss} = V_{cc} \times (1 + R_{v2:v1})$] compares favorably with one-compartment V_d values for CD1 mouse, but V_{ss} values for the other columns (species) appear too large, due to the large $R_{v2:v1}$ values.

k_{12} values vary a lot across the columns, suggesting that k_{12} may be highly correlated with another parameter (e.g., $R_{v2:v1}$).

$R_{v2:v1}$ values also vary a lot across the columns.

T_{maxc} values are consistent across the columns; expressed in Gm/hr, they seem very large. For example, 2032 Gm/hr (4.91 moles x 414 Gm/mole) for the CD1 mouse. Even on a kg body weight basis could mouse renal tubules resorb 2 kg PFOA per hour? This maximum rate of resorption must far exceed the rate of filtration of PFOA at the glomerulus. (Clarification at the reviewers meeting explained this apparent departure from physiological reality. The units had been mis-specified in Tables 5-5 and 5-7. They were in fact micromole per hour

and micromolar for T_{maxc} and k_T instead of molar based. Thus T_{maxc} mouse value was 2 mg/hr, which is physiologically plausible.)

k_T values are the concentration in glomerular filtrate that half saturates the resorption transporters. Expressed in mg/mL, they seem large, much larger than the urine concentration that would be expected; e.g., for CD1 mouse, k_T is 15 mg/mL where free serum concentrations (Free x C_{serum}) would be about 0.3 µg/mL with 10 mg/kg in the mouse. So the transporter would not become saturated except at extreme doses. The value used by Andersen et al. (2006) for monkey was 0.00001 mg/mL. Unit specified in Tables 5-5 and 5-7 should be µM, not M.

Free fraction values measured in vitro are 0.01 or less at low PFOA serum concentrations (Table 3-1). The Free values for rat seem much higher than the measured values.

Q_{filc} is defined as a fraction of blood flow (renal or cardiac output?) to the filtrate (bottom of p. 5-11) but has units of flow in Table 5-5.

V_{filc} values are much smaller than the 0.01 L value used by Andersen et al. (2006), although Andersen et al. state that the model output is insensitive to this parameter and that their value was assumed.

<u>PFOS Table 5-7, p. 5-15</u>

Body Weight and Cardiac Output values are reasonable and typical.

k_a values for female mouse and monkey seem extremely large – see comment above for PFOA.

V_{cc} values appear reasonable. See comment above for PFOA.

k_{12} values vary a lot across the columns, suggesting that k_{12} may be highly correlated with another parameter.

$R_{v2:v1}$ values appear reasonable and consistent with other reports of V_{ss} values for PFOS.

T_{maxc} values are highly variable across the columns and seem much higher than physiological reality would allow. See comment above for PFOA.

k_T values are physiologically unrealistic and highly variable across columns. See comment above for PFOA.

Free fraction values have been measured in vitro and are 0.01 or less at low PFOS serum concentrations (Table 3-1, p. 3-3). The Free values in Table 5-7 are consistent with the measured values.

Q_{filc} is defined as a fraction of blood flow (renal or cardiac output?) to the filtrate (bottom of p. 5-14) but has units of flow in Table 5-7.

V_{filc} values are much smaller than the 0.01 L value used by Andersen et al. (2006), although Andersen et al. state that the model output is insensitive to this parameter and that their value was assumed.

While the parameter values for the pharmacokinetic models predict reasonable serum concentrations that generally agree with measured values (Tables 5-6 – 5-8 for PFOA and Tables 5-8 and 5-10 for PFOS), their high interspecies variability suggest that the models may be unreliable for prediction of internal exposures after other intake regimens and during a depuration phase.

Matthew P. Longnecker

Please see the answer to the previous question.

Angela L. Slitt

The parameters included appear to be appropriate, but this lies outside of my area of expertise.

Question 8. The volume of distribution (Vd) and half-life values are critical in the derivation of the interspecies uncertainty factor applied in derivation of candidate RfDs from a NOAEL, LOAEL or a BMDL. The available data for both values are provided in Section 3.5.2 and 3.5.3 of both documents. Please comment the strengths and weaknesses of the values selected.

James V. Bruckner

PFOA-specific comments
The adult male rat data of Kemper (2003), from which the rat half-life and clearance (CL) were obtained, appear to be solid. It is reasonable to select the human half-life of 2.3 years reported by Bartell et al. (2010), as their study population included equal numbers of males and females. Division of the rat CL by the human CL to yield a value of 219 is fine. I did not examine the publication of Bartell et al. (2010) to evaluate their data or methodology used to derive a human half-life of 2.3 years. Therefore, I am uncertain about its accuracy.

PFOS-specific comments
I would again defer to someone with more expertise.

Deborah A. Cory-Slechta

This falls outside my area of expertise and therefore no significant comments are provided. However, at the face-to-face meeting there was significant discussion regarding the modeling, including clarifications from Dr. Wambaugh that were found by those panel members with expertise to clarify these issues and these particular issues were considered adequately addressed.

Jamie C. DeWitt

While the overview of the individual studies that calculated Vd and half-life for each compound was detailed and complete, the rationale and analysis concerning *why* particular values were selected were insufficient. Additionally, as addressed in Charge Question 6, the rate of clearance/elimination likely contributes to the differences in half-life that are not associated with differences in the Vd. Therefore, a 3-fold uncertainty factor for species differences in pharmacodynamics (UF_A) was utilized for both compounds. What was the justification for using a UF_A of 3? The section on UF application needs a more thorough discussion regarding the choice of this value given differences in clearance. If the section on model adjustment (a suggestion in Charge Question 6) is better described, this comment may no longer be applicable.

Jeffrey W. Fisher

The use of this non-compartmental method should be justified. Why not use a PBPK model? Assuming steady state in the humans does allow for calculation a human equivalent serum concentration associated with a laboratory animal concentration. In what region of the exposure-dose range would nonlinearity occur in humans? Some type of discussion is needed

about the assumptions of this methodology and why it was used. I would like to see statements about if the NOAEL, LOAEL, and a BMDL doses are in the linear range for kinetics.

The authors should use the Bayesian analysis for animal studies to inform the UF. Use percentiles to explore Vd and half-life to support UF values. I did not see any attempt to use distribution information generated from the model beyond the central tendency or mean values. Please state why this is the case. It seems that the distribution information generated from the Bayesian analysis could be used to support UF development.

William L. Hayton

PFOA-specific comments

For male rat, the Kemper (2003) study appears to be the best source of pharmacokinetic parameter values, which were obtained by a model-independent analysis of serum concentration-time data from rats that were dosed by oral gavage at dosages of 0.1, 1.0, 5.0 and 25 mg/kg. In addition, there was a 1.0 mg/kg dosage administered intravenously, and a 0.1 mg/kg oral gavage dose with an extended sampling time. Each treatment used four animals. The CL and $t_{1/2}$ values appeared to be independent of dosage and route of administration. It would therefore be reasonable to average all 6 mean values for each parameter to give an over-all mean of 24 determinations. The average (n=24) values for male rat were CL = 0.0209 L/kg/d and $t_{1/2}$ = 7.83 d. These values can be used to calculate a V_d value ($t_{1/2}$ x CL / ln 2), which is 0.236 L/kg.

It is not apparent on p. 5-20 why a V_d value of 0.17 was used with half-life to calculate CL_{rat} when Kemper (2003) reported CL values and not $t_{1/2}$ values. (At the peer review meeting, it was clarified that the data of Kemper (2003) were re-analyzed and as a result the parameter values in the health effects documents differ somewhat from those published with the data in the original reports.)

The CL_{human} value was taken to be 0.00014 L/kg/d. There are no direct measurements of this parameter. Thompson et al. (2010) assumed that the intake rate of PFOA for subjects using PFOA-contaminated water was 91% of the PFOA in 1.4 L/d of water. This intake rate was used along with a PFOA half-life of 2.3 years to calculate a V_d value of 0.17 L/kg. This is the same value that was used in the health effects document for the rat (p. 5-20). The V_d values available in mouse, rat and monkey are about 0.2 L/kg, so the $V_{d,human}$ set at 0.17 L/kg is not unreasonable but it lacks the certainty of the rat V_d value.

The health effects document used a $t_{1/2}$ for PFOA in human of 839.5 d (2.3 years), which seems to be toward the low end of the range of values that have been reported. Along with Vd = 0.17 L/kg one arrives at CLhuman = ln 2 x 0.17 / 839.5 = 0.00014 L/kg/d.

The ratio CL_{rat} / CL_{human} calculated using the mean CL_{rat} from Kemper (2003) would be 0.0209 / 0.00014 = 149, which is about twice the value calculated on p. 5-21. This difference arises from the calculation of CL_{rat} using the $V_{d,human}$ and a half life of 11.5 d instead of using the CL_{rat} directly from Kemper (2003). The mean half life from Kemper (2003) was 7.8 d.

The CL_{mouse} / CL_{human} ratio is accurate, using Lou et al. (2009) data. A calculation for monkey is not shown.

PFOS-specific comments
Chang et al. (2012) appears to be the best source of pharmacokinetic parameter values for mouse, rat and monkey. Butenhoff and Chang (2007) is given as the reference for a 48-day half-life in rat; this is a final report, internal to 3M. The Chang paper gives half-life values for male and female Sprague-Dawley rat at 2 mg/kg and 15 mg/kg. The average V_d for the four groups of three/group was 0.71 L/kg. This is higher than the 0.23 L/kg value used in the draft document (p. 5-23). The 0.71 L/kg value is also higher than values for mouse, monkey and human, which are closer to the 0.23 L/kg value used in the draft document. The draft document ought to acknowledge this difference; it may be that the value in the 3M report is lower than the published value; Chang was a co-author for both sources. The Chang et al. (2012) paper gives CL_{rat} values that are 0.0051 L/h/kg for female (similar for 2 and 15 mg/kg doses) and for males, 0.022 and 0.011 L/h/kg for the 2 and 15 mg/kg doses. A single average value for CL_{rat} would be 0.011 L/h/kg, about 3 times the value used for the UF_A calculation in the draft document. The male value is about 2-3 times the female value and it may be appropriate to calculate a different UF_A value for each sex. Using the single CL_{rat} averaged across two doses and both sexes (0.011 L/H/kg) would give a CL_{rat} / CL_{human} ratio of 0.011 / 0.000081 = 135 and a UF_A = 407, substantially higher than the value of 123 in Table 5-15.

The UF_A values calculated for mouse and monkey appear to be in line with the literature values for PFOS CL values in these species.

Matthew P. Longnecker

The PBPK model of Loccisano et al. 2013 (for humans) can be used to calculate a volume of distribution for PFOA of 177 ml/kg, which is very close to the value of 170 ml/kg based on Thompson et al.'s 2010 one-compartment model . For PFOS, the corresponding value from the PBPK is 280 ml/kg, compared with the value of 230 ml/kg used in the Health Effects Document. This 22% difference could have an impact on some calculations. (Note: the PBPK model-based volumes of distribution were calculated by Marc-Andre Verner of the University of Montreal. He had calculated these values in the course of a separate project.)

For humans, the half-life data all depend on the assumption that ongoing exposure is negligible compared to baseline exposure, a reasonable assumption in most of the populations used to estimate half-life. While the Seals et al. (2011) gave estimates that were slightly different for PFOA in some cases, the methods employed in this study were not as strong as for Bartell et al. (2010) or the Burris et al. studies (2000; 2002). The agreement within species for the half-life estimates for PFOS are reassuring. The animal data on the half-life of PFOA are relatively sparse (2 rat studies that agreed reasonably well, 1 mouse study, 1 monkey study). For PFOA, the UF_As and RfD that were calculated based on the half-lives (expressed as clearance) would not have been substantially altered by alternate choices for specific values. The same is true for PFOS.

Angela L. Slitt

Strengths of the available data is that for the several species thorough evaluated, the half-life values are very consistent. For example, the several human studies cited report a range in calculated PFOS half-life in humans to be 4.1-8.67 years, two studies putting monkeys at 110-132 days, and rat generally has a narrow range with 3 out of 4 values provided ranging from 39.8-48.2 days for PFOS. An inconsistency is the Chang et al., 2012 describing a half-life of females of 66.7 days when in general female rodents may have faster elimination of PFOS.

Question 9. A variety of endpoints and studies were used to compare points of departure and the resultant RfDs for both PFOA and PFOS. In addition, comparisons were provided across RfD outcomes based on the model outputs compared to those for the NOAEL, LOAEL and BMDL points of departure. The range of candidate RfDs derived from the different points of departure is fairly narrow. Please comment on the strengths, weaknesses and transparency of this analysis.

James V. Bruckner

PFOA-specific comments
The procedure used to calculate PODs adheres to standard EPA guidelines and policy. The presentation of their derivation is clear, concise and transparent. It is certainly interesting that the range of PODs and resulting candidate RfDs is so narrow. Nevertheless, as discussed previously, I do not agree with their selection.

PFOS-specific comments
See my comments under General Impressions.

Deborah A. Cory-Slechta

While it is the case for both PFOA and PFOS that values from different points of departure are fairly narrow, the transparency of the analyses in neither case is clear. There is no rationale described even as to why these analyses were done on all of the studies, what was the primary study and how others related to that etc., i.e., this presentation does not follow the typical presentation format of IRIS documents in either its presentation of rationales and strategies, nor in the conclusions that it reaches. In both cases, it is only the single sentence indicating that modeling from one particular study will be protective of effects at other studies using higher exposures. This section in both documents needs introductory paragraphs that describe the specific strategy, choices of studies and the rationales for those choices.

As noted in response to Charge Question 3, the rationale for discarding the human epidemiological studies is not sufficient and requires rationale other than that stated and therefore, the question of using the human data remains open. As noted in response to Charge Question 1, in this reviewer's opinion, the increased liver weight can be justified as a departure point for assessment of RfDs, but as discussed at the face-to-face meeting, additional text supporting this choice is needed.

Jamie C. DeWitt

This particular section contained inadequate detail on why particular studies *were* or *were not* chosen. For example, immunotoxicity as an endpoint was not chosen for PFOS, based on "*in vitro* measures of immunocompetence on mice may not be relevant to the human experience and limited human data from epidemiology studies are inconclusive regarding the immunotoxicity of PFOS in humans"; however, the breadth of data from in vitro/ex vivo immunotoxicity studies for PFOS were not thoroughly discussed (please see Charge Question #2 for two additional in vitro studies).

For both compounds, an increase in absolute liver weight was selected as an endpoint as it was a common *effect* [sic] in both short and long term studies. However, the toxicological relevance of an increase in absolute liver weight was not discussed other than to indicate that it was a sign of altered homeostasis. Further, the co-occurrence of increases in absolute liver weight with other toxicologically-relevant endpoints (i.e., immunotoxicity and/or reproductive/developmental toxicity) is not a toxicologically valid justification for the use of liver weight as an endpoint for an RfD. Therefore, the analysis was not sufficiently transparent to deduce its relative strengths and weaknesses. Certainly, choosing an endpoint that occurs across species and occurs at relatively low doses will likely be protective of exposed humans; however, will it be a defensible endpoint? As currently written, the choice of this endpoint for an RfD is not adequately defended.

Jeffrey W. Fisher

I did not review the toxicity data.

William L. Hayton

For PFOA, a 10% increase in liver weight was selected as the metric for effect, which was "… not made based on toxicity but on the desire to find a common denominator against which to evaluate dose-response across studies and justified by the fact that other adverse effects accompanied the LOAEL for increased liver weight in some cases." The lowest serum concentration associated with an increase in liver weight was calculated for female mouse to be 20.33 mg/L (p. 5-16, PFOA document). These data are referenced to DeWitt (2008); this paper has only summary information on liver weights, all of which exceeded 20% weight gain, going as high as 70%; and it is not apparent in PFOA document how these liver weight gains were used to estimate an LOAEL for 10% liver weight gain.

Many of the animal studies of hazard assessment were conducted under conditions where the duration of the exposure was relatively short compared with the half-life, and steady state had not been achieved. It is not apparent how the NOAEL and LOAEL values from such studies were adjusted to account for the non-steady state situation. For example, the 20.33 mg/L PFOA concentration associated with a 10% increase in liver weight (Table 5-9) emanated from a 15 day drinking water exposure to 0.94 mg/kg/day that resulted in an average serum exposure of 20.33 mg/L (0 – 29.7 mg/L over 15 d, Tables 5-7, 5-9). For a fixed daily dose, the time to 90% steady state for mouse would be about 63 days (3.3 x half life, which was 19 days), and after 15 days the serum concentration would only be about 15% of its steady-state value. This seems to suggest that the RfD would have been over-estimated by a factor of 7, since the 0.94 mg/kg/day at steady state would have produced a serum concentration of about 150 mg/L, not 20.33 mg/L. This analysis is based upon the behavior expected from one-compartment model pharmacokinetics. As discussed on p.5-9 of the PFOA document, the steady-state serum concentration of PFOA is achieved in a much shorter time than one-compartment model kinetics would predict. Whether the target-site steady-state concentration of PFOA also occurs in a much shorter time than one-compartment model kinetics would predict (3.3 x half life) is apparently unknown.

Matthew P. Longnecker

This part of the document seemed especially strong and transparent. The agreement between methods was reassuring. The weaknesses and assumptions were well discussed. Please see the minor editorial comment on this issue given for Charge Question 1, above.

Angela L. Slitt

The RfD Point of Departure was based on animal studies that include monkey and rat.

Question 10. The RfDs for PFOS and PFOA are derived from the modeled steady state serum concentrations and their association with effects that include short term and longer term exposures with associated diverse effects. The studies considered included effects due to exposure durations that ranged from 11 to 182 days, and occur at comparable human equivalent dose (HED) levels. The current, draft RfDs do not include an uncertainty factor for study duration because of the apparent concordance HEDs despite duration differences. Given this pattern of response, is it appropriate to conclude that the candidate RfDs are applicable to both short-term and lifetime exposures?

James V. Bruckner

PFOA-specific comments
I do not believe it is appropriate to conclude that the candidate RfDs are applicable to both short-term and lifetime exposures. Steady-state is apparently achieved in monkeys within 4 - 6 weeks (Butenhoff et al., 2002). Steady-state likely takes considerably longer in humans. Thus, RfDs for shorter periods of exposure should be based upon results of studies of similar duration.

PFOS-specific comments
I do not believe the candidate RfDs, as calculated, are applicable to different durations of exposure.

Deborah A. Cory-Slechta

While initially believing that it was appropriate conclusion for PFOA and PFOS, based on the correspondences in RfDs across short and longer term exposure, discussion at the face-to-face meeting made clear that this approach is not reasonable and requires additional consideration.

Jamie C. DeWitt

This approach may be appropriate given the relative similarity of serum concentrations attained regardless of study duration, i.e., steady state in serum is attained after a relatively short period of exposure. This appears to be consistent across studies with various species of animal models. However, the document authors might need to reconsider given what we may or may not know about liver hypertrophy. In the Hall et al. (2012) paper on liver hypertrophy (discussed during the public meeting), increase in liver weight is an adaptive response that may not be adverse UNLESS weight increases >150% over a three month or longer period may. Following this large and prolonged increase in weight, the end result may be a hepatocarcinogenic response. However, none of the studies contained in the documents indicate that longer term exposures increase liver weight to this degree.

Jeffrey W. Fisher

The departure from K=CXT (Haber's law) should be based on the toxicity endpoints of concern and what is known about dose-exposure kinetics/responses for these chemicals and other chemicals that target the same endpoint, not that the HED values are comparable. The NAS

AEGL committee only considered primary irritation for inhaled chemicals as an endpoint that was independent of duration of exposure. There is an SOP if needed for reference.
The time to steady state should be included in a table for the lab animals. Toxicity studies conducted for less than 30 days (perhaps?) are not at steady state for the pharmacokinetics of PFOA. Thus the measured serum levels would be different than at steady state. The shorter the duration of the toxicity test, the more impact this could have on extrapolation to chronic exposures in humans. My personal preference would be to use PBPK models for all species and consider only long term exposures for extrapolation to humans.

William L. Hayton

This depends in part on how quickly the PFOA/PFOS concentrations at sites of toxicity come to steady state. Since the Vd for these chemicals is small (~ 0.3 L/kg) it seems likely that the concentrations in tissues rise in pseudo equilibrium with the rise in serum concentration. That said, the half lives are relatively long due to the very small clearance ($t_{1/2} = \ln 2 \times V_d / CL$). If one-compartment kinetics apply, then a guideline for time to 90% steady state is 3.3 $t_{1/2}$. For studies that expose animals for a period of time shorter than 3.3 $t_{1/2}$, the serum concentration would not be at steady state and the internal systemic exposure (serum concentration) would be less than what it would be if the exposure were longer than 3.3 $t_{1/2}$. This effect would seem to lead to overestimation of the intake rate that was associated with a particular internal exposure and associated biological endpoint. For example, the $t_{1/2}$ of PFOS in mouse is about 36 days and 3.3 $t_{1/2}$ is 120 days. Consider a 28-day exposure using a fixed daily dose that produced an LOAEL of "X" mg/kg/day. On Day 28, the body level would only be 42% of the steady state level, and the average body level over the 28-day period would be about 21% (approximating the increase as linear and not exponential). The true LOAEL would be 0.21 "X" mg/kg/day; i.e., intake of 0.21 "X" mg/kg/day would produce a body level at steady state that was the same as the average body level produced by X mg/kg/day administered over 28 days. The time to 90% steady state for a fixed intake rate is quite long; from the literature in the health effects documents, the times in the following table were calculated. From this line of reasoning, exposure times less than two half-lives begin to significantly overestimate intake rates associated with particular endpoints. This analysis is based upon the behavior expected from one-compartment model pharmacokinetics. As discussed on p.5-9 of the PFOA document, the steady-state serum concentration of PFOA is achieved in a much shorter time than one-compartment model kinetics would predict. Whether the target-site steady-state concentration of PFOA also occurs in a much shorter time than one-compartment model kinetics would predict (3.3 x half life) is apparently unknown.

	CL		Vd		$t_{1/2}$		Time to 90% steady state	
	[mL/d/kg]		[mL/kg]		[d]		[d]	
Species	PFOA	PFOS	PFOA	PFOS	PFOA	PFOS	PFOA	PFOS
Mouse	6.6	5	180	265	19	36	63	120
Rat - Male	23	16	273	947	8.4	40	28	92
Rat - Female	776	5.2	150	476	0.13	66	0.43	218
Monkey	6.3	1.4	190	238	27	121	89	400
Human	0.085	0.08	170	230	1378	2000	12.5 yr	18 yr

In addition, this line of reasoning may be incorrect if the assumption of one-compartment kinetics is incorrect. For multi-compartment models the serum concentration and target organ/tissue could come to their pseudo steady state levels relatively quickly while slowly equilibrating (deep) sites slowly approached steady state. Simulation with PBPK models for PFOS and PFOA may help answer this question.

Associated with the uncertainty introduced by exposures that were shorter than the time to achieve steady-state concentration at the target site is the exposure time required for the adverse effect to be expressed. While some adverse effects may occur immediately and directly in proportion to the concentration of PFOA or PFOS at the target site, other adverse effects may be slow to become manifest. These "indirect adverse response" behaviors are well known in the drug action arena; e.g., certain antidepressant drugs require several weeks exposure to the target site before the effect of the drug appears. This lag time is not associated with pharmacokinetics (time to steady state) but with indirect-response pharmacodynamics. It could be argued that uncertainty factors are needed for both pharmacokinetics (pre-steady state condition) and pharmacodynamics (or toxicodynamics) to account for possible indirect response behavior.

Matthew P. Longnecker

EPA might want to consider using an uncertainty factor for duration, for two reasons. First, the monkey data for PFOS used for the point of departure were from a study where the duration of exposure was relatively short-term relative to the half-life, and it appeared that duration of dose affected liver and other adverse outcomes detected at higher doses, and no monkey data were used in the POD for PFOA. Second, questions raised by Drs. Hayton and Fisher at the peer-review meeting made me less comfortable with the calculations that used average serum concentration derived from the AUC and duration of dosing to compare with humans, who are more likely to be near steady-state.

Angela L. Slitt

Yes, but this lies outside of my area of expertise.

Question 11. In addition to using the average serum values from animal studies to calculate internal doses for humans, the animal to human extrapolation can be accomplished by dividing animal average serum values by the human to animal clearance ratios to project a human average serum point of departure in units of mg/L serum. Please provide recommendations for applying uncertainty factors to the extrapolated average human serum values to determine serum-based thresholds that are protective for humans. A NOAEL expressed in average human serum units would be useful in interpreting NHANES population monitoring data.

James V. Bruckner

PFOA-specific comments
No comment.

PFOS-specific comments
No comment.

Deborah A. Cory-Slechta

In initial response to charge questions, I found it difficult to understand specifically what this charge question was asking for a response to: Does this refer to the data in Table 5-10 for PFOA? Wouldn't you include animal to human UF values at the least. Since the data for the studies listed in the Table is not clear as to their duration (columns are needed for this information, or add to the Study box), it is not clear whether a UF for study duration is warranted. It is not clear how sex differences are being accommodated in any of these.

At the face-to-face meeting, however, with some additional input from EPA, it was clear to all that there was no need to do such derivations from animal to human, which could instead be derived directly from the human data and thus presumably this is no longer an issue.

Jamie C. DeWitt

Would this approach take into account differences between animal studies that have a defined exposure duration and data from NHANES, where exposure duration is assumed to be continuous (although it may not be), if exposure duration does not appear significantly impact serum concentrations? Additionally, how would the half-life estimations from the Seals et al. (2011) study, which contained two half-life estimations based on concentration and time, impact this approach?

Jeffrey W. Fisher

Again, is the system linear in the exposure/dose ranges of interest? I would try to determine an UF by exploring a range of predicted human serum levels. Attempt to use 5,50, and 95% for animal serum concentrations with a 5,50, and 95% CL values in the animals and for the human perhaps use two CL values representing a high and low. The idea is to use as much information

as you can to determine the possible range of values. This will help guide the selection of uncertainty values.

William L. Hayton

This calculation is equivalent to dividing the animal dosage by the CLhuman, assuming that the animal serum concentration is at steady state ($C_{ss,animal}$) maintained by a constant dose rate (DR).

$$C_{ss,animal} / CL_{human} \div CL_{animal} = CL_{animal} * C_{ss,animal} / CL_{human} = DR / CL_{human}$$

This calculation would give the steady-state serum concentration in human that would be produced by the animal dose rate. (I will have to study this to understand the question; the calculation does not make sense to me.)

At the peer review meeting, the aim of this calculation was clarified. Authors desired a way to calculate a steady-state serum concentration ($C_{ss,human}$) that would result from the human equivalent dose rate (HED) administered until steady state. The appropriate calculation would be:

$$C_{ss,human} = HED / CL_{human}$$

Matthew P. Longnecker

The proposed division by animal clearance ratios does not make sense to me. The average serum values from animal studies is already taking pharmacokinetic variability in blood levels during the observation period into account, and human blood levels will be relatively constant. Thus, it would make sense to directly compare the POD estimated average serum concentrations from animal models to the blood levels in NHANES. With respect to uncertainty factors that would be need consideration for this approach, it seems that UF_H, UF_L (For LOAEL and HED_{LOAEL}), UF_D, and the component of UF_A that takes pharmacodynamics into account would all still be applicable.

Angela L. Slitt

This is outside of my area of expertise.

Question 12. Please describe any suggestions you have for improving the clarity, organization, and/or transparency of the draft documents.

James V. Bruckner

PFOA and PFOS-specific questions
See specific observations.

Deborah A. Cory-Slechta

While the EPA authors are aware of updates in the IRIS process, it might be very helpful to look at some of the new documents coming through that process for guidance as to the levels of critique and evaluation that are now included in these documents. They also include an introductory chapter focused specifically on the literature searches and literature that is included vs. excluded.

The Executive summary does not provide sufficient rationale and descriptions to lead a reader through the steps to what is concluded and reads more like an abstract than an Executive Summary. Since this may be the only section read by many reviewers, it is important that it provide a succinct journey through the process. Here again, the new IRIS documents (e.g., trimethylbenzene) could provide a useful template.

Chapters 3, 4 and 5 could each benefit from an opening paragraph describing what the section's goals are, and integration and conclusion sections at the end that establish the basis for the presentation in Chapter 5. Currently the Hazard Identification studies generally treat all studies as of equal strength/power, which is certainly not the case. These chapters should present that kind of critical and transparent assessment as it ultimately serves as the basis for decisions that are made.

The inclusion of sections on in Vitro data did not ultimately seem particularly relevant in the outcome for these compounds and could be significantly shortened to add more to Chapter 4 on study strengths and weaknesses. However, where pertinent, it would probably be more useful to break that section up and insert test where it follows an in vivo discussion.

Tables could be considerably improved and made far more useful to the reader for comparative assessments. As of now, they require going back and forth to the text to capture additional details of the studies, e.g., sample sizes, species etc. and could benefit the reader significantly with those additions. For the human assessments, it is equally important to include these details in the chapter as well as a column of study strengths and limitations.

While charge questions ask whether the appropriate studies were chosen as key studies, this reviewer does not remember that that term was even used in the documents, certainly no explicit mention was made as to which studies were considered key studies. This would seem to be a section that should be included in Chapter 4 more explicitly. Chapter 5 of both documents, more so PFOA, are confusing as almost all studies are subjected to modeling, for reasons that are

never presented in sufficient detail and simply followed by statements that a selected study (not really well presented in Chapter 4 as a selected study) will protect against other adverse effects.

Jamie C. DeWitt

The documents lack a critical analysis of differences between findings of epidemiological studies and findings of animal models. As stated in the comments to Charge Question #3, what is particularly valuable about the PFOA/PFOS database is that it is relatively extensive in that it includes data not only from occupationally-exposed humans, but from people highly exposed to environmental concentrations of PFOA/PFOS, and from people in the general population who have detectable concentrations of these compounds.

Critical to this analysis is a discussion of concordance and lack of concordance between human data and animal model data. For example, immunotoxicological findings appear to be consistent between humans and rodent models whereas serum lipids are not. How do these differences impact the overall confidence in the database and derivation of the RfD?

All of the sections related to the PK models developed by ORD need additional information for clarity and transparency. As written, it is not clear that the PK values presented throughout the document actually represent a reanalysis of existing data from studies that reported serum concentrations. The Wambaugh et al. (2013) study could be referenced to shorten this exercise as this publication provides details on the reanalysis of existing data.

Justifications for choosing or not choosing particular values or endpoints need to be more thoroughly detailed throughout both documents, especially for endpoints that appear to occur in both experimental animal models and exposed humans (i.e., thyroid hormone disruption and immunotoxicity).

Jeffrey W. Fisher

These documents represent an enormous undertaking to describe studies with PFOA and PFOS. Keep the same writing style for reporting studies. This was very good. A synthesis of the most important studies is needed and some statements about why other studies are not used by EPA. It is easy to get lost in the document because of its size, but if there was an analysis or synthesis section for the key toxicity studies and another for PK modeling rationale, it would help readers.

William L. Hayton

It would be helpful to use one set of units for test article amount and concentration. The draft documents use ng/mL, µg/mL, µg/L, ppb, ppm, and µM for PFOA/PFOS concentration in water, diet, and serum. It would be more straightforward to use one concentration term, preferably ng/mL, and perhaps µg/mL in addition as necessary. But making comparisons among ng/mL, ppm, and µM is a distraction.

In Section 3 of both documents, it would be helpful to include a summary table of primary pharmacokinetic parameter values for the species included in this section. Tables 3-17 – 3-20 in

the PFOS draft document are a good start. In the PFOA document, Table 3-23 lacks CL values, and Tables 3-24 and 3-25 lack V_d values. For the pharmacokinetic model analyses presented, primary parameters values could be limited to CL, Vd_{ss}, and half life (see table in response to question 10). The CL and V_{dss} values should be normalized to body weight. Where there are multiple models for a species, there should be separate entries for each study. Where there are multiple dosages for a species, there should be separate entries for each dosage. For the PBPK models, V_{dss} values are not available and therefore should not be included. Such a table would be helpful to show consistency or lack thereof among studies and would facilitate selection of the best available values for CL and V_{dss} for use in a human PK model that would predict steady-state serum concentration from intake (dosing) rate and, conversely, predict intake rate from steady-state serum concentration. These predictions are probably the primary reason to include a pharmacokinetics section in the documents.

The pharmacokinetic sections of both documents lack example graphs of serum concentration-time data on semilog coordinates for PFOA and PFOS. Inclusion of a few representative graphs would help the reader evaluate the consistency of the data used to generate the pharmacokinetic parameter values, and where model-based equations have been fitted to the data, the scatter of the measured concentrations around the model-predicted line would be informative as to the goodness of fit and the validity of the model and its parameters.

Matthew P. Longnecker

I can see advantages to treating this more like a systematic review of the literature, where the specific search algorithm for included articles is laid out, as are the range of dates of publication to be considered, and any other selection criteria applied for articles considered. In these documents, while the review of earlier literature appears to be comprehensive, after some point there must have been some decision making about which of the more recent articles to include.

The EPA has many guidelines about how data like these are to be evaluated, yet in the document few, if any, references to these guidelines were cited. Because so many guidelines exist, it could help readers if the authors cited specific places in critical documents that provide guidance for specific decisions.

Angela L. Slitt

The document reads very well. Although not included in the RfD determination, including a table of the observed human effects along with serum concentrations in Section 5.0 would put Tables 5-2 and 5-3 into context. Some sort of layman explanation to help understand why only non-human exposures are being included would be helpful to the general public.

V. SPECIFIC OBSERVATIONS

James V. Bruckner

Specific Observations for *Health Effects Document for Perfluorooctanoic Acid*		
Page	**Paragraph**	**Comment or Question**
3-11	5, lines 7-11	It is stated that the PFOA concentration in bile increased by a factor of 12.5 with the increase in PFOA dose from 12.5 to 25 umol/kg in wild-type mice and 19.5 in PPARα-null mice. These factors should be 2.8 for wild-type and 6.1 for PPARα-null mice. The document's authors may want to rethink their interpretation of the data. The results for the wild-type mice do suggest saturation of transport from liver to bile ducts, but the PPARα-null results do not, indicating a role for PPARα in this process. In contrast to the foregoing, the findings of Lou et al. (2009) (p. 3-11, pgr. 2) indicate their highest dose of PFOA is cleared from the blood of mice more rapidly than lower doses, suggesting saturation of hepatic and/or renal reuptake transporters. What is the relative importance of biliary and renal elimination of PFOA?
3-12	3, lines 2-4	It should also be stated that upregulation of MRP3&4 and the OATs may be <u>beneficial</u>, due to increased biliary excretion of bile acids, bilirubin, conjugated metabolites of toxic chemicals, etc.
3-14	1 & 2	It might be stated that the findings of Hinderliter (2004) support those of Han (2003), in regards to development of female rats.
3-14& 3-15		It is problematic to try to compare values in Table 3-14 with values referred to at the end of the second paragraph on p. 3-17. Whole pup and pup serum PFOA levels decrease between PND 1&18 for each dosage in the table. It would be preferable to include another table showing the PFOA levels with body weight taken into account. Table 3 – 14 and other tables should include the species in the title. It would also be helpful to include some details of the experimental protocol in the footnotes.
3-20		It would be useful at the end of this section (Distribution During Pregnancy and Lactation) to summarize the primary findings, or conclusions that can be drawn from the data that were presented.
3-23	4, line 2	It should be emphasized that urinary excretion of PFOA was substantially higher in female than male rats.
3-28	2, line 4	Replace "receptors" with "transporters".
3-28	6	Did 10 uM PFOA inhbit PAH and estrone uptake to a greater extent than 100 uM PFOA?
3-29	3 & 4	It is not clear what Yang et al. (2009) concluded about the role of OATp1a1 in the uptake of PFOA from glomerular filtrate.
3-32	3 & 4	These two summary paragraphs are very helpful.
3-37	1, line 1	Should "adsorption" be "absorption"?

Specific Observations for *Health Effects Document for Perfluorooctanoic Acid*		
Page	**Paragraph**	**Comment or Question**
4-7 & 4-9	Tables 4-1 & 4-2	Tables 4-1 and 4-2 are quite helpful in integrating the results of studies of occupationally-exposed populations.
4-13		A concluding sentence should be added to summarize the findings of a lack of association of PFOA with diabetes, metabolic syndrome, etc.
4-32		The NOAEL and/or LOAEL for this study should be stated at the end of the paragraph.
4-34	2	Is the LOAEL for liver effects 1 ppm in the study of Loveless et al. (2008)?
4-38	1	Include the meaning of the abbreviation "mPPARα".
4-39		Inclusion of the table for Minata et al. (2010) would be useful to help readers better comprehend the study findings.
4-40		A table of short-term LOAELs and NOAELs should be added here or in Section 5.
4-47	2	It is hard to believe, judging from the slight difference in mean values and their standard deviations, that absolute and relative liver weights are significantly higher than controls in the 1 mg/kg/day group.
4-67	2, line 5	Insert "absolute" before "liver weight".
4-69	1, lines 1 & 2	It might be worthwhile to point out that the actual study by Butenhoff et al. was conducted prior to 2004.
4-73		A summary sentence (or two) should be added at the end of the Mutagenicity and Genotoxicity section.
4-83		A summary paragraph should be included at the end of the Immunotoxicity section.
4-101	1, line 14	Insert the word "some" before "occupational studies". In order to present a more balanced perspective of findings in occupational studies, the following sentences could be added at the end of the paragraph: "Olson and Zobel (2007) examined groups of male workers at 3 fluorochemical production facilities. Serum PFOA concentrations were not associated with total cholesterol, LDL or HDL in workers at these facilities."
4-102	4	It should be stated that the increases in serum enzyme activity in workers were quite modest/small. The following sentence should be added at the end of the paragraph: "Emmett et al. (2006), however, found no association between serum PFOA and liver or renal enzymes".
4-103	2, line 2	Change "apoptotic or necrotic damage of" to "apoptosis or necrosis of". Apoptosis and necrosis are types of cell death, not damage/injury.
4-103	3, line 1	It is true that PFOA may interfere with the biliary excretion of other compounds that are transported by the same transporters. Upregulation of the genes for these transporters, however, may be beneficial in that the excretion of bile acids, bilirubin and conjugates

Specific Observations for *Health Effects Document for Perfluorooctanoic Acid*		
Page	**Paragraph**	**Comment or Question**
		of toxic chemicals/metabolites may be hastened.
4-103	4, line 2	I would avoid the word "critical" until the section on Dose-Response Assessment.
4-103	4	Increases in absolute and relative liver weights were dose-dependent (Cui et al., 2009; Elcombe et al., 2010; Wolf et al., 2008a)
4-103	5	It is important to distinguish between effects of PFOA on rough and smooth endoplasmic reticulum (RER and SER). RER content was diminished, but there was a proliferation of SER.
4-104	2, line 5	This last line should be amended to read "that PFOA has some effects of unknown toxicological significance that appear to be independent of PPARα activation.
4-104	4, line 3-5	The meaning of the sentence is not clear. Has something been omitted?
4-105	3, line 3	Add "of offspring" between "abilities" and "at 6 and 18". Include Fei and Olsen's (2011) finding of no association between prenatal PFOA exposure and behavioral or coordination problems in children at age 7.
4-109	3	The species (i.e., mice) studied by White et al. (2009) and by Wolf et al. (2007) should be stated.
4-111	4, line 2	Replace "examine" with "determine whether there was".
4-112	2, lines 1 & 2	The first sentence is misleading and should be rewritten. Butenhoff et al. (2012) did not see a significant increase in liver adenomas or carcinomas. Biegel et al. (2001) reported an increased incidence of hepatic adenoma but not carcinoma.
4-112	2, line 13	What is hepatic cystoid degeneration?
4-114	2, line 3	Insert "decreased" before "apoptosis".
4-115	5, line 2	What is meant by "PRAR exposures"?
4-116		There is no mention of PFOA-induced changes in expression of genes (e.g., cell cycle control, peroxisomes biogenesis, inflammation, etc.) that are PRARα-dependent. There is no mention of the role of PRARα or peroxisomes in oxidative injury and carcinogenesis.
4-120	1, lines 11 & 12	Insert "these" between "that" and "hormones".
4-121	3	It would be helpful to give the PFOA dosages of White et al. (2007) and one or two other studies, so the reader will have some idea of the magnitude of PFOA exposure required to alter mammary gland development.
5-1		RfD: Omit the word "wealth" from the bullet pertaining to epidemiology studies. There have been relatively few epidemiology studies of PFOA-exposed populations.

Specific Observations for *Health Effects Document for Perfluorooctanoic Acid*		
Page	**Paragraph**	**Comment or Question**
5-2	1, lines 2-6	Another obvious point should be made here, mainly that occupational exposures result in much higher plasma PFOA levels and body burdens than do environmental exposures. Thus, it would be anticipated that adverse effects would be more apparent in PFOA facility workers.
5-2	1, line 5	Include the words "in some instances" between the words "shown" and "between". Otherwise, it appears from this paragraph the serum PFOA concentrations are consistently/usually associated with the various maladies.
5-2	3, line 8	Insert "failure to attain" between the words "with" and "developmental".
5-7	2, line 4	Insert the word "rodent" between "between" and "species"
5-19	1, line 1	Insert "from some studies" between "data" and "have".

Specific Observations for *Health Effects Document for Perfluorooctane Sulfonate*		
Page	**Paragraph**	**Comment or Question**
3-2	5, lines 2 & 3	It is stated here that "the ratio of PFOS identified in serum and liver tissue are similar". Do the authors mean that PFOS concentrations in the serum and liver are similar?
3-2	6	How does PFOS distribute between plasma lipoproteins and proteins/albumin?
3-5	1, lines 9& 10	How much lower were milk PFOS levels than serum levels?
3-7	1, line 2	Oral and gavage are redundant.
3-16	Figure 3-1	This figure nicely illustrates relative PFOS levels in dams and fetuses/pups over time.
3-19	1,line 3	Insert "groups" between "day" and "on".
3-21	1, line 10	Substitute "longer" for "slower".
3-23	2	It is not clear who conducted the human PBPK modeling nor which model they used.
4-21	2, lines 1-3	What did the 2nd monkey die from?
4-26	3, line 3	The word "concentrations" should be replaced by "doses".
4-39	1	Does an increase in motor activity on PND 17, but no such effect on PND 13, 21 or 61, constitute a toxicologically-significant effect?
4-56	2, lines 1 & 2	It is stated that "taken together, these studies suggest a PPARα-independent mechanism…" Of the studies reviewed to this point in the document, only that of Abbott et al. (2009) supports this premise. Qazi et al. (2009), Rosen et al. (2010) and other groups of investigators have reported other PPARα-independent effects of PFOS.
4-60	2, lines 15-17	Is oxidative damage likely to be operative to a significant extent at lower PFOS doses?

Specific Observations for *Health Effects Document for Perfluorooctane Sulfonate*		
Page	**Paragraph**	**Comment or Question**
4-61	2, line 4	What is meant by "The concentration…"?
4-61	4, line 2	Change "dose of exposure is" to "levels of exposure are".
4-62	1, lines 2 & 3	What did Olsen et al. (2003) find correlation between?
4-62	3, lines 4 & 5	Identify the species (i.e., rat) studied by Chang et al. (2009) and Stein et al. (2012).
4-62	5	The liver of rats and monkeys was examined for histopathological changes, but the histological changes should not be considered lesions nor pathological.
4-68	4, lines 5 & 6	The elevated incidence of hepatocellular adenomas/ carcinomas was almost entirely due to adenomas. Only 1 of 60 high-dose female rats exhibited carcinoma.
4-69	5, lines 3 & 4	It is stated here that there was no increase in hepatocellular proliferation detected in the subchronic study of Seacat et al. (2003). It is stated previously on page 4-69 that "the data for PFOS are adequate to support some but not all key events…" I assume that cell proliferation is thought to be a missing event. Seacat et al. (2003) reported that the average hepatocyte proliferation index was not increased, but that some animals exhibited mild increases. It is clear in the current document that PFOS is not as potent a PPARα inducer as PFOA.
5-4	2 & 5, line 7	Again the terms "histopathological" and "lesions" are misnomers.
5-4	3, line 9	What is meant by a "biologically significant decrease in survival" at 0.8 mg/kg?

Deborah A. Cory-Slechta

Specific Observations for *Health Effects Document for Perfluorooctanoic Acid*		
Page	**Paragraph**	**Comment or Question**
Chapter 5		The text of Chapter 5 in the PFOA document (and other places) continues to state that a 10% increase in liver weight would not be an adverse effect, but merely a denominator for loss of homeostasis. On what basis was this conclusion derived? What is the support for this statement? It appears that benchmark dosing was applied to studies that had liver weight as the common denominator, but does this accommodate the lowest NOAELS and LOAELS observed for any endpoint in the long duration studies? Use of just studies with the common denominator because they provide replication ignores the fact that some other effect may occur at lower levels but simply hasn't been evaluated in as many studies as focused on PPARa-based targets. If this isn't the case, then the text should clearly address this.
5-7	2	States that the $BMDL_{10}$ values all fall below the experimental LOAELs. So, what does that mean, is there some conclusion that is supposed to be reached from this? IF so, please state it.
5-13	1	States "Generally these values were similar." What does similar mean? What is acceptable in this context?
5-16	3	States that the half-life value Bartell et al. (2010) was sued for half-life because it seemed more relevant to scenarios where exposure result from ingestion of contaminated drinking water by members of the general population. This rationale does not appear to consider the potential different strengths and weaknesses of the other potential studies. Is it necessarily the case that general population is more important than occupational studies? The rationale needs to be described in greater detail. Virtually no rationale is provided for the choice of the Thompson et al. (2010) study for a volume of distribution value.

Specific Observations for *Health Effects Document for Perfluorooctane Sulfonate*		
Page	**Paragraph**	**Comment or Question**
1-1	4	There are similar concerns for the PFOS document. Loose terminology should be eliminated, e.g., what is a 'finding of note' as used in the executive summary for PFOS.
Executive summary		The PFOS executive summary is of limited utility; for many readers this may be as much of the document as they read; as currently written it is not clear or transparent nor does it sufficiently explain how it arrived at an RfD.
3-3	Table 3-1	Couldn't a sentence essentially substitute for Table 3-1; it really isn't useful.
3-25	1	Loose terminology should be eliminated, e.g., what is "generally good"
3-26	Figure 3-7	Figure 3-7 has no explanation of what is the black vs. gray line.
All tables		There is a need to improve all of the tables; they should always include study name/year, sample size and exposure duration information on them; this would make all of the comparisons easier to evaluate and not require the reader to continue to go back and forth to the text.
4-4	Table 4-1	For example, table 4-1 has only study name and year, but what really matters is also exposure duration and sample sizes, because the comparisons of outcomes in the Table depend upon the power of the study to detect effects at the very least.
4-9	Table 4-2	The same comment applies to Table 4-2 and any others with this intended purpose.
4-11	Table 4-3	Table 4-3 needs sample sizes, exposure duration etc.
4-24	Table 4-7	Tables that summarize a significant amount of data from a single study (e.g., 4-7) should include the study authors and year in the Table title so it doesn't have to be searched for.
		In several instances in the PFOS document, adverse effects early that appear to be reversed at a later age are discounted with the suggestion that they therefore do not matter; given our increasing understanding of the importance of early changes in terms of epigenetic changes, this is no longer appropriate and in fact, misleading.
5-16	Table 5-8	What do the parentheses signify?
5-17	Table 5-9	What do the parentheses signify?

Jamie C. DeWitt

Specific Observations for *Health Effects Document for Perfluorooctanoic Acid*		
Page	**Paragraph**	**Comment or Question**
4-102	2 & 3	DeWitt et al. 2009 also included data on triglyceride levels in C57BL/6 mice exposed to PFOA for 15 days; triglyceride levels were dose-responsively decreased.

Jeffrey W. Fisher

No specific observations.

William L. Hayton

Specific Observations for *Health Effects Document for Perfluorooctanoic acid*		
Page	**Paragraph**	**Comment or Question**
1-2	Last, line 5	Delete "in"; should read "… in rats was analyzed …"
3-2	1, lines 6-8	Assumption that fecal excretion represented unabsorbed PFOA is problematic; suggest rephrasing this sentence.
3-3	Table 3-1	Protein binding is important for PK modeling, where the fraction unbound (fup) is the important parameter, not the fraction bound. Suggest listing fup values rather than percent bound.
3-6	Last, line 3	"concentration" should be "dose rate"
3-8	2, line 4	In addition to liver, kidney, and blood, other tissues are prominent. E.G., Table 42 of Kemper shows that in male at 1 mg/kg, t=Tmax, GI tract, GI contents, muscle, bone and skin contained a greater percentage of dose than did the kidney.
3-8	2, line 8	"Blood to kidney" should be "kidney to blood"
3-8	2, line 10-11	In Kemper, Tables 44-45, blood to kidney ratios are not 10 or higher in males.
3-8	2	This paragraph reports both percent of dose found in tissues, and concentrations found in tissues. But Tables 3-4 and 3-5 present only the former. When presenting tissue concentrations, please make it clear that those data are not shown.
3-18	Last, line 3	"were" is repeated.
3-19	1, line 1	Technically incorrect to say that the level peaked at PND7; that was the earliest sample time. The peak may have occurred before PND7.
3-19	Table 3-15	The last dose was on GD17; strange that at 1 and 3 mg/kg the serum concentration increases from PND7 to PND14.
3-22	4	Last sentence is garbled.
3-22	4, 5	Agree that biliary elimination is possible, but it could be that chloestyramine binds PFOA and PFOS in the GI tract lumen after they passively diffuse from the blood to the gut. There seems to be no direct evidence of biliary elimination, e.g., bile collected from treated animals.
3-23	Last, line 4	Should be Table 3-18.
3-34	Last, line 9	Should be "nonlinear least squares"
3-35	Table 3-23	Column 2, "Adsorption" should be "Absorption"
3-38 3-38	2 Figure 3-7	The arrow from Gut to Liver appears to point in the wrong direction; it should represent biliary excretion of PFOA from Liver to Gut.
3-43	Last line	"… indicating the absence of active excretion in human kidneys." This does not follow from the observation of renal clearance being about 0.001% of GFR. A plasma free fraction of 0.001 would

Specific Observations for *Health Effects Document for Perfluorooctanoic acid*		
Page	**Paragraph**	**Comment or Question**
		account for the CLr being 0.1% of GFR, and passive tubular reabsorption would make it 0.001% of GFR since urine flow is about 1% of GFR. Other scenarios are possible that do not invoke the absence or presence of active excretion.
3-44	Table 3-24	Should report all data values with three significant figures. For example, Lambda z values have only one sig. fig., while $T_{1/2}$ values have 5-6.
3-46	2	This reviewer does not follow the derivation and use of a value for volume of distribution with regard to intake rate and serum concentration of PFOA. If the subjects were at steady state, the body burden would have to be known. At steady state, the serum concentration would be independent of the volume of distribution, so any V value ought to match the intake rate to the steady state serum concentration.
4-9	1	Log transformed concentration was 1.51 and 1.48 ng/mL – are these the logarithms? IE, are the actual concentrations 10^1.51 = 32 and 10^1.48 = 30 ng/mL?
4-20	2, line 8	Anderson here is spelled Andersen in the reference list.
4-30	1, line 9	prostrate should be prostate.
4-31	4, line 10	decreased should be decrease.
4-112	1	It would be helpful to restate the serum concentrations for the Eriksen and Vieira studies, or refer reader to p. 4-29 where they are provided.
4-112	1, line 9	Delete "for".
4-112	2, line 12	Delete "were".
4-118	4	Delete "of actions" after MOAs
4-120	3	The broad range of half lives could also be due to person-to-person variability in the free fraction of PFOA in serum (fup). This is the case for highly bound drugs; e.g., warfarin.
5-1	3	Pharmacokinetic is misspelled.
5-1	5	Disagree – exposure assessment based on the human data is feasible. In fact, the serum concentrations are a better measure of exposure than are intake measures as they reflect all intake pathways and eliminate bioavailability and pharmacokinetic influences on internal exposure.
5-12	Last	Table numbers should be 5-6, 5-7, and 5-8.

Specific Observations for *Health Effects Document for Perfluorooctane Sulfonate*		
Page	**Paragraph**	**Comment or Question**
3-5	3	The low CSF : serum concentration ratio could also be due to an export transporter that pumps PFOS out of the CSF and/or to extensive serum protein binding, where only the free serum concentration of PFOS is in equilibrium with the free PFOS concentration in the CSF.
3-22	2	The free fraction used for the model is much larger than that determined experimentally, Table 3-1; this should be pointed out in the text.
3-22 3-23	2 Figure 3-5	The arrow from Gut to Liver appears to point in the wrong direction; it should represent biliary excretion of PFOS from Liver to Gut.
3-24	4	Anderson should be Andersen.
4-26	4	"concentrations" should be "dosages".
5.2	3	Should note for many of these studies, that steady state may not have been achieved due to the long half-life of PFOS. Half-life values from Section 3 are: mouse, 37 days; rat male, 40 days and female 64 days; monkey, 120 days. Using a one-compartment PK model, the time to 90% steady state is 3.3 half lives.
5-5	3	The NOAEL for liver effects in rats of 0.072 mg/kg/day is not consistent with p. 5.4, para. 2, which states that lesions of the liver <u>were</u> observed in male rats after 104 weeks at this dosage.
5-7	2	For female rat, the PFOS half life is about 60 d and the period of gestation is about 20 d or one-third of a half life. If PFOS is administered to the dam only during gestation at a fixed daily dose, the serum concentration of PFOS would rise from 0 to 21% of the steady-state serum concentration that the fixed dose rate would produce at steady state. The exposure of the fetus during gestation would average only about 10% of the exposure that would have occurred if the dam had received PFOS for 4 half-lives (240 days) prior to mating. BMDs based on such a fixed dose could be elevated by as much as a factor of 10 compared with the steady state situation. Steady state would be the relevant situation for humans. For the Luebker study (Table 5-3) the serum concentration during gestation would have increased from about 38% to 50% of the eventual steady state concentration.

Matthew P. Longnecker

Specific Observations for *Health Effects Document for Perfluorooctanoic*		
Page	**Paragraph**	**Comment or Question**
3-28	1st complete	Should the end of the sentence be "increase the transporters" rather than "increase the receptors"?
3-30	2nd complete	L 3, would insert "transfected" between "OAT3" and "cells"
3-39	1st complete	Next to last sentence: I doubt that Olsen assumed the major source of exposure was drinking water in the occupational study
3-41	4th complete	In the first formula listed, the plus sign should be an equal sign
4-9	1st complete	L 3 from bottom: the values of 1.51 and 1.48 given are probably better described as geometric means.
4-16	2nd complete	L 3 from bottom: would insert "draw" after "blood"
4-21	2nd complete	L 5: the value of 6.78 ug/L is a water level, not a serum level; this issue recurs on P 4-23, paragraph at bottom
4-30	1st complete	L 8: should read "exposure categories" rather than "cancer categories"?
4-37	Table	Would note dose of PFOA somewhere in table or footnote
4-55	Last para	L 3: should the ">" be a "<"?
4-79	Last para	Last sentence: should "50 and 25" be "50 and 250"?
4-80	1st complete	The last sentence does not accurately describe the table. E.G., the CD4+CD8+ cells decreased at the 47.21 mg/kg/d dose
4-82	Next to last para	Last sentence: the 37.5 mg/kg/dose is not mentioned earlier, so this is a little confusing.
4-85	Last para	L 2: should "0.5" be "0.05"?; Same issue for L 5.
4-89	4th para	How long were the animals dosed?
4-110	3rd complete para	L 5: should "serum" be "blood"?
4-113	3rd complete para	L 1: insert "in" before "liver cells"
5-4	Last para	Were the criteria for inclusion in Table 5.2 the same as for Table 5.1?
5-12	Para below table	8-6, 8-7, and 8-8 should be 5-6, 5-7, and 5-8
5-16	Last line	I do not see in the Thompson et al. (2010) study any mention of using exposure data from NHANES to calibrate the volume of distribution. Other sources of data were used, where the water had been contaminated.
5-17	1st formula	"/day" should be deleted from "0.17 $L/kg_{bw}/day$"
5-20	Table 5-12	The first three values in the UF_{total} column need to be corrected; they should be 21900, 219000, and 21900
5-21	Paragraph above table	Last sentence: UF_L should be UF_H
5-21	Last sentence	UD_S should be UF_S

Specific Observations for *Health Effects Document for Perfluorooctanoic*		
Page	**Paragraph**	**Comment or Question**
5-27	Calculations	The text says the body weight conversions should be based on the ¾ power. If so, the HED formulas are incorrect, and the HED should be 1.99 x 0.0254 = 0.0506, the dosimetric adjustment factor should be 0.0254, and the CSF should be 1.57. All the figures here should be checked as should the paragraph on P 5-28. The HED is 2,530-fold greater than the RfD, not 29,000.

Specific Observations for *Health Effects Document for Perfluorooctane Sulfonate*		
Page	**Paragraph**	**Comment or Question**
1-1	2nd	1st sentence: would revise for clarity. Do you mean uncertainties exist about whether PFOS-induced peroxisome proliferation is involved in causing PFOS-induced hepatic lesions?
1-1	3rd	1st sentence: would revise for clarity; the occupational studies were done at PFOS production plants, but to my knowledge there are no residential populations that have been studied for health effects who lived near PFOS production plants. (Mid-Ohio valley factory was a source of PFOA.) In the 2nd sentence, I do not believe that exposure was mainly through contaminated drinking water in any of these studies.
4-66	2nd	The earlier summary of the Bloom et al. study (P 4-10) said the results were not statistically significant, whereas here the interpretation appears to be that the study found an association. The interpretation does not seem consistent across the two sections.
5-17	Below table	L 3: the word "terminal" should be deleted from this sentence
5-20	1st formula	The "/day" should come out of "0.23 L/kg bw/day"
5-26	L 2 from bottom	This should be 35 ug/L not 35 mg/L

Angela L. Slitt

No specific observations.

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APPENDIX A: PEER REVIEWER BIOSKETCHES

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External Peer Review Meeting on EPA's Draft Health Effects Documents for Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS)

Crystal City Marriott at Reagan National Airport
1999 Jefferson Davis Highway
Arlington, VA, 22202-3526

August 21 and 22, 2014

LIST OF PEER REVIEWERS

James Bruckner, Ph.D.
University of Georgia

Deborah Cory-Slechta, Ph.D. (chair)
University of Rochester School of Medicine and Dentistry

Jamie DeWitt, Ph.D.
East Carolina University

Jeffrey Fisher, Ph.D.
U.S. Food & Drug Administration

William Hayton, Ph.D.
The Ohio State University (Emeritus)

Matthew Longnecker, Sc.D., M.D
National Institute of Environmental Health Sciences

Angela Slitt, Ph.D.
University of Rhode Island

BIOGRAPHICAL SKETCHES OF PEER REVIEWERS

James Bruckner, Ph.D.
University of Georgia

Dr. Bruckner is Professor of Pharmacology and Toxicology at the University of Georgia College of Pharmacy. He is also Professor in the Department of Physiology and Pharmacology at the University of Georgia College of Veterinary Medicine. He received his Ph.D. in Toxicology from the University of Michigan in 1974. He has previously held faculty positions at the University of Kansas and the University of Texas Medical School at Houston. He is actively engaged in graduate education and in federally-funded research projects. Dr. Bruckner's research focus is on the toxicology and toxicokinetics of solvents, drug-solvent interactions at occupational exposure levels, and toxicokinetic bases for susceptibility of children to insecticides and other chemicals. Dr. Bruckner has published more than 200 journal articles, book chapters, and abstracts. He has also served on a variety of expert panels and committees for the EPA, National Institutes of Health, National Aeronautics and Space Administration, Agency for Toxic Substances and Disease Registry, Food and Drug Administration, and the National Academy of Sciences.

Deborah Cory-Slechta, Ph.D. (chair)
University of Rochester School of Medicine and Dentistry

Dr. Cory-Slechta is a Professor in the Department of Environmental Medicine and the Department of Pediatrics at the University of Rochester School of Medicine and Dentistry, where she also serves as co-director of the Behavioral Sciences Facility Core and director of the Animal Behavior Core. Dr. Cory-Slechta received her Ph.D. from the University of Minnesota in 1977 and worked as a junior staff fellow of the National Center for Toxicological Research beginning in 1979. She was appointed to the faculty of the University of Rochester Medical School in 1982 and was appointed Chair of the Department of Environmental Medicine and Director of the NIEHS Environmental Health Sciences Center at the University of Rochester in 1998. From 2000 to 2002, she was the Dean for Research and Director of the AAB Institute for Biomedical Sciences. Following her appointment as Dean, she served from 2003 to 2007 as the Chair of the Department of Environmental and Occupational Medicine at Robert Wood Johnson Medical School and as Director of the Environmental and Occupational Health Sciences Institute, a joint Institute of the Robert Wood Johnson Medical School and Rutgers. Dr. Cory-Slechta's research has focused largely on environmental neurotoxicants as risk factors for behavioral disorders and neurodegenerative disease. These research efforts have resulted in over 170 papers and book chapters to date. Dr. Cory-Slechta has served on numerous national research review and advisory panels, including committees of the National Institutes of Health, the National Institute of Environmental Health Sciences, the Food and Drug Administration, the National Center for Toxicological Research, the Environmental Protection Agency, the National Academy of Sciences, the Institute of Medicine, and the Agency for Toxic Substances and Disease Registry, Centers for Disease Control. In addition, Dr. Cory-Slechta has served on the editorial boards of several journals including *Environmental Health Perspectives*, *Neurotoxicology*, *Toxicology*, *Toxicological Sciences*, *Fundamental and Applied Toxicology*, *Neurotoxicology* and *Teratology*, and *American Journal of Mental Retardation*. She has held the elected positions of President of the Neurotoxicology Specialty Section of the Society of Toxicology, President of the Behavioral Toxicology Society, and been named a Fellow of the American Psychological Association. She also previously served on the EPA Science Advisory Board Perfluorooctanoic Acid Risk Assessment Review Panel.

Jamie DeWitt, Ph.D.
East Carolina University

Dr. DeWitt is an Assistant Professor in the Department of Pharmacology and Toxicology at the Brody School of Medicine at East Carolina University (ECU). She is affiliated with The Harriet and John Wooten Laboratory for Alzheimer's and Neurodegenerative Diseases Research and holds an adjunct appointment in the ECU Department of Public Health. Dr. DeWitt received her Ph.D. in Environmental Science and Neural Science from the School of Public and Environmental Affairs and Program in Neural Science at Indiana University in 2004. She also completed postdoctoral training in Developmental Cardiotoxicity at Indiana University-Bloomington and in Immunotoxicology at EPA through a cooperative training agreement with the University of North Carolina at Chapel Hill. Dr. DeWitt's main research focus is on how toxicants found in the environment can lead to neurodevelopmental and neurodegenerative disorders via disruption of the developing immune system. Much of her past research has

involved the immunotoxicity of perfluorooctanoic acid (PFOA) and related polyfluroalkyl substances (PFASs). Dr. DeWitt has published seven peer reviewed research articles, three review papers and two book chapters that address the biological effects of PFOA, as well as one paper on the effects of PFOS on immune function. Her publications describe effects as well as underlying mechanisms following adult and developmental exposure. Her research experience and publication record (more than 25 peer reviewed manuscripts, 6 review articles, 9 book chapters) extend beyond the effects of PFAAs and working with rodent models. She is currently editing a book on the general toxicity of PFASs and is a current member of the mechanistic working group for Monograph 110 of the International Agency for Research on Cancer, which will include an assessment of PFOA. She is on the editorial boards of the *Journal of Immunotoxicology* and the *Journal of Environmental Toxicology and Health* and has reviewed grants for the Department of Defense and the National Institute of Occupational Safety and Health. She has also been manuscript reviewer for more than 20 journals. Dr. DeWitt is the current president of the North Carolina chapter of the Society of Toxicology and the Junior Councilor for the Immunotoxicology Specialty Section of the Society of Toxicology. She also was awarded the Outstanding Young Investigator Award from the Immunotoxicology Specialty Section in 2013.

Jeffrey Fisher, Ph.D.
U.S. Food & Drug Administration

Dr. Fisher is a Research Toxicologist at the U.S. Food and Drug Administration (FDA), National Center for Toxicological Research. He was formerly a Professor in the Department of Environmental Health Science, College of Public Health at the University of Georgia (UGA). He joined UGA in 2000 and served as Department Head of the Department of Environmental Health Sciences from 2000 to 2006 and Director of the Interdisciplinary Toxicology Program from 2006-2010. Prior to joining UGA, he spent most of his career at Wright Patterson Air Force Base (AFB), where he was Principal Investigator and Senior Scientist in the Toxics Hazards Division and Technical Advisor for the Operational Toxicology Branch. Dr. Fisher's research interests are in the development and application of biologically based mathematical models to ascertain health risks from environmental, food-borne and occupational chemical exposures. Dr. Fisher's modeling experience includes working with chlorinated and non-chlorinated solvents, fuels, pesticides, perchlorate and bisphenol A. He has developed physiologically-based pharmacokinetic (PBPK) models for use in cancer risk assessment, estimating lactational transfer of solvents, understanding in utero and neonatal dosimetry, quantifying metabolism of solvent mixtures and developing biologically motivated models for the hypothalamic-pituitary-thyroid axis in rodents and humans. Dr. Fisher has published over 140 papers on pharmacokinetics and PBPK modeling in laboratory animals and humans. He has served on several national panels and advisory boards for the DoD, ATSDR, USEPA and non-profit organizations. He was a U.S. delegate for the North Atlantic Treaty Organization. Dr. Fisher served on the International Life Sciences Institute Steering Committee, which evaluated chloroform and dichloroacetic acid using EPA-proposed Carcinogen Risk Guidelines. He is Past President of the Biological Modeling Specialty Section of the Society of Toxicology, reviewer for several toxicology journals, and was Co-Principal Investigator on a National Institutes of Health (NIH)-supported workshop on Mathematical Modeling at the University of Georgia in the fall of 2003. Dr. Fisher was also a member of the National Academy of Sciences subcommittee on Acute Exposure

Guideline Levels (AEGLs) from 2004-2010 and Science Advisory Board (SAB) for the US EPA (2007-2010). He is an ad hoc EPA SAB member for dioxin and perchlorate. Dr. Fisher is a Fellow of the Academy of Toxicological Sciences, an associate editor for Toxicological Sciences, and on the editorial board of *Journal of Environmental Science and Health Part C Environmental Carcinogenesis & Ecotoxicology Reviews*.

William Hayton, Ph.D.
The Ohio State University (Emeritus)

Dr. Hayton is a Professor Emeritus in the College of Pharmacy at The Ohio State University. Dr. Hayton received a Ph.D. in Pharmaceutics from the State University of New York at Buffalo in 1967. He was a member of the Washington State University College of Pharmacy faculty for 19 years, rising to Chair of the Pharmacology/Toxicology Graduate Program in 1982 and Acting Dean at the College of Pharmacy in 1987. In 1990, he transferred to the Ohio State University as Chair of the Division of Pharmaceutics, where he later served as Associate Dean for the Graduate Programs and Research until his retirement in 2010. Dr. Hayton's expertise is pharmacokinetics, particularly construction and validation of mathematical models that describe or explain the kinetics of complex biological systems. One recent research interest is characterization of the Fc receptor-mediated transport and catabolism of albumin and IgG in wild type and FcR knockout mice. A second recent project is the quantitative modeling of the female hypothalamus-pituitary-gonad (HPG) axis in the female rainbow trout (Oncorhynchus mykiss). The model is based on and integrates the biology of gonadotropin, estrogen, androgen and maturational hormone signaling systems, and it includes key intermediate steps in the signaling pathways; viz., gonadotropin and sex steroid synthesis, hormone receptors and their corresponding mRNA levels. Dr. Hayton's expertise extends to interspecies scaling of pharmacokinetic model parameter values and xenobiotic metabolism. Dr. Hayton is author or co-author of over 100 peer-reviewed scientific publications and has held peer-reviewed grant support from the National Institutes of Health, EPA, Air Force Office of Scientific Research, Food and Drug Administration, and U.S. Fish and Wildlife Service. He previously served on the EPA Science Advisory Board Perfluorooctanoic Acid Risk Assessment Review Panel.

Matthew Longnecker, Sc.D., M.D
National Institute of Environmental Health Sciences

Dr. Longnecker, M.D., Sc.D., is the head of the Biomarker-based Epidemiology Group at the National Institute of Environmental Health Sciences (NIEHS). Dr. Longnecker received an M.D. from Dartmouth Medical School and completed a residency in internal medicine at Temple University Hospital in Philadelphia. After receiving a Sc.D. in Epidemiology from Harvard School of Public Health in 1989, he served as an Assistant Professor in the Department of Epidemiology at the University of California, Los Angeles, School Of Public Health. Since 1996, Dr. Longnecker has served as Adjunct Professor/ Associate Professor in the Department of Epidemiology, School of Public Health, University of North Carolina at Chapel Hill. He came to the NIEHS Epidemiology Branch in 1995, as a tenure-track investigator. Dr. Longnecker's research program is focused on the health effects of persistent organic pollutants (e.g., the DDT metabolite p,p'-DDE, and polychlorinated biphenyls). He is particularly interested in the effects of intrauterine exposure to persistent organic pollutants in relation to intrauterine growth,

preterm birth, birth defects, neurologic findings at birth, growth, neurodevelopment, intelligence, and hearing. Recently, Dr. Longnecker has completed and has ongoing a series of studies on perfluorinated alkyl substances in relation to reproductive and pediatric outcomes. In addition, he has begun studying the effects of early, low-level exposure to the nonpersistent pollutants, bisphenol A and organophosphate pesticides. Dr. Longnecker's research efforts have resulted in over 180 papers and book chapters to date. He has served as a leader for numerous national and international committees, such as for the Society for Epidemiologic Research and the International Society for Environmental Epidemiology, and has been on numerous national and international scientific advisory boards, including the EPA Science Advisory Board for the Perfluorooctanoic Acid Risk Assessment Review.

Angela Slitt, Ph.D.
University of Rhode Island

Dr. Slitt is an Associate Professor in the Department of Biomedical and Pharmaceutical Sciences at the University of Rhode Island. Dr. Slitt received her Ph.D. in Pharmacology and Toxicology from the University of Connecticut in 2000, and then served until 2004 as a postdoctoral fellow at the University of Kansas Medical Center. Dr. Slitt has been a faculty member at the University of Rhode Island since 2006. Dr. Slitt's graduate and postdoctoral training was heavily focused on liver biology and health, with a focus in the area of toxicology, and included research in nuclear receptors, biotransformation, and transporter expression. Her current research interests focus on how 1) expression of drug transporters affects chemical disposition and toxicity, 2) nutrition and intake of dietary antioxidants affects the expression of drug transporters, 3) liver disease (i.e., diabetes, cholestasis, and ethanol cirrhosis) affects transporter expression and chemical disposition, and 4) transporter expression affects cholesterol transport and susceptibility to gallstone formation. She has also recently investigated the effect of PFOS on caloric restriction in mice. Dr. Slitt is presently on the Editorial Board of *BMC Pharmacology and Toxicology, Journal of Biochemical and Molecular Toxicology*, and *Toxicology Methods and Mechanism*, and is an ad-hoc reviewer for numerous other journals. She is author or co-author of over 50 peer-reviewed scientific publications, and was recently awarded the University of Rhode Island Early Career Faculty Research Excellence Award.

EPA-HQ-2016-005679 02/14/2017

APPENDIX B: MEETING AGENDA

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AGENDA

External Peer Review Meeting on EPA's Draft Health Effects Documents for Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS)

Crystal City Marriott at Reagan National Airport
1999 Jefferson Davis Highway
Arlington, VA, 22202-3526

August 21, 2014

Day 1 – PFOA Health Effects Document

8:30 AM	**Meeting Sign-In Begins**
9:00 AM	**Welcome, Goals of Meeting, and Introductions** David Bottimore, Versar, Inc.
9:15 AM	**Welcome by EPA and Overview of PFOA/PFOS Health Effects Documents** Elizabeth Doyle, Chief, EPA/OST/OW/HECD
9:30 AM	**Chair's Introduction and Review of Charge** Deborah Cory-Slechta, *Chair*
10:00 AM	**Discussion Session – "Round Table" General Overview Comments**
10:45 AM	*Break**
11:00 AM	**PFOA Discussion Session – Responses to Charge Questions:** Question 1: Strengths, Weaknesses, and Characterization of the Studies Selected Question 2: Additional References for Consideration Question 3: Conclusions on Human Epidemiology Data Question 4: Transparency and Characterization of Epidemiologic Data
12:15 PM	*Lunch**
1:15 PM	**PFOA Discussion Session – Response to Charge Questions:** Question 5: Cancer Classification Question 6: Pharmacokinetic Model Adjustments Question 7: Selected Parameters for Pharmacokinetic Model Question 8: Volume of Distribution (Vd) and Half-life Values
2:45 PM	*Break**
3:00 PM	**PFOA Discussion Session – Response to Charge Questions:** Question 9: Points of Departure and RfDs Question 10: RfDs and Applicability to Short-and Long-term Exposures Question 11: Uncertainty Factors Question 12: Other Suggestions for Improving the Document
5:00 PM	**Adjourn**

**Time for breaks and lunch are approximate and at the Chair's discretion.*

AGENDA

External Peer Review Meeting on EPA's Draft Health Effects Documents for Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS)

Crystal City Marriott at Reagan National Airport
1999 Jefferson Davis Highway
Arlington, VA, 22202-3526

August 22, 2014

Day 2 – PFOS Health Effects Document

9:00 AM	**Recap of Day 1 and Agenda for Day 2** David Bottimore, Versar, Inc.
9:15 AM	**Chair's Review of Charge for Day 2** Deborah Cory-Slechta, *Chair*
9:30 AM	**Discussion Session – "Round Table" General Overview Comments**
10:00 AM	*Break**
10:15 AM	**PFOS Discussion Session – Responses to Charge Questions:** Question 1: Strengths, Weaknesses, and Characterization of the Studies Selected Question 2: Additional References for Consideration Question 3: Conclusions on Human Epidemiology Data Question 4: Transparency and Characterization of Epidemiologic Data
11:30 PM	*Lunch Break**
12:30 PM	**PFOS Discussion Session – Response to Charge Questions:** Question 5: Cancer Classification Question 6: Pharmacokinetic Model Adjustments Question 7: Selected Parameters for Pharmacokinetic Model Question 8: Volume of Distribution (Vd) and Half-life Values
2:15 PM	*Break**
2:30 PM	**PFOS Discussion Session – Response to Charge Questions:** Question 9: Points of Departure and RfDs Question 10: RfDs and Applicability to Short-and Long-term Exposures Question 11: Uncertainty Factors Question 12: Other Suggestions for Improving the Document
5:00 PM	**Adjourn**

**Time for breaks and lunch are approximate and at the Chair's discretion.*

EPA-HQ-2016-005679 02/14/2017

APPENDIX C: MEETING ATTENDEE LIST

ED_000954(915)_Processed_PSTs-2_DD_00024885-00084

LIST OF OBSERVERS ATTENDING MEETING IN PERSON
August 21 and 22, 2014

External Peer Review Meeting on
EPA's Draft Health Effects Documents for
Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS)

Name	Affiliation
Janet Anderson, Ph.D.	U.S. Air Force
Robert Bilott	Taft Stettinius & Hollister LLP
Norman Birchfield	U.S. EPA
John Butenhoff, Ph.D.	3M Company
Sue Chang, Ph.D.	3M Company
C-H Selene Chou, Ph.D.	Agency for Toxic Substances and Registry
Steven Chranowski	Chemistry Council of New Jersey
Joyce Donohue	U.S. EPA
Elizabeth Doyle	U.S. EPA
Colleen Flaherty	U.S. EPA
Maria Hegstad	Inside EPA
Gerald Kennedy	DuPont
La Rae Landers	Dept of the Navy BRAC PMO
Willington Lin	Public
Angela Lynch, Ph.D.	American Chemistry Council
Geary Olsen, Ph.D.	3M Company
Gloria Post, Ph.D.	New Jersey Department of Environmental Protection
Resha Putzrath, Ph.D.	Navy and Marine Corps Public Health Center
Jennifer Seed	U.S. EPA
Shalene Thomas	AMEC Environment & Infrastructure
Steve Via	American Water Works Association
Anthony Walters	United Science, LLC
Carol Wood	Oak Ridge National Laboratory

LIST OF OBSERVERS ATTENDING VIA PHONE
August 21 and 22, 2014

External Peer Review Meeting on
EPA's Draft Health Effects Documents for
Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS)

Name	Affiliation
Matthew Bailor	Gilmore & Associates, Inc.
Victoria Binetti	U.S. EPA
Stuart Cagen, Ph.D.	Shell Health
Andrea Candara	New York State Department of Health
Tom Cleveland	Decatur Utilities
Jason Conder	ENVIRON International Corporation
Michelle Deveau	Health Canada
Stiven Foster	U.S. EPA
Helen Goeden	Minnesota Department of Health
Christopher Lau, Ph.D.	U.S. EPA
Anita Meyer, DABT	U.S. Army Corps of Engineers
Jonathan Naile, Ph.D.	Shell Health
Jessica Nelson	Minnesota Department of Health
Bridget O'Brien	U.S. EPA
Ramasamy Santhini	U.S. EPA
Robert Rickard	U.S. EPA
John Wambaugh	U.S. EPA
Carol Rowan West, Ph.D.	Massachusetts Department of Environmental Protection
Linda Wilson	New York State Office of the Attorney General
Virginia Yingling	Minnesota Department of Health
Tsedash Zewdie	Massachusetts Department of Environmental Protection

To: Burneson, Eric[Burneson.Eric@epa.gov]
From: BNA Highlights
Sent: Wed 7/22/2015 2:14:20 AM
Subject: Jul. 22 -- BNA, Inc. Daily Environment Report

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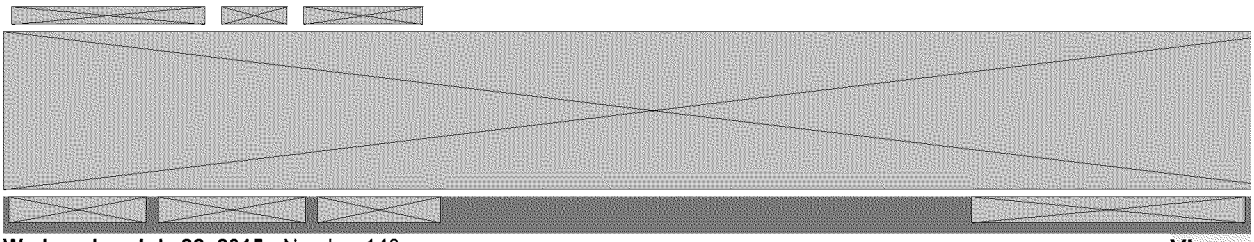

Wednesday, July 22, 2015 · Number 140

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NEWS

Air Pollution
Court Clarifies 100-Hour Engine Exemption Decision
A federal appeals court narrowed its opinion in litigation over an Environmental Protection Agency rule that allowed backup generators to operate up to 100 hours per year without emissions controls to clarify that maintenance and readiness...

Air Pollution
Court Upholds Revised Standards for Chromium
A federal appeals court July 21 upheld an Environmental Protection Agency rule that set more stringent emissions limits for chromium electroplating facilities and phased out use of fume suppressants that use the toxic compound perfluorooctyl...

Biotechnology
Monsanto Nears USDA Approval for High-Yield Modified Corn
Monsanto's new strain of high-yield, genetically modified corn is one step closer to winning approval from the Department of Agriculture, after the department's release of two favorable environmental studies....

Chemicals
Causation Proof Limited in PFOA Bellwether Trial
Medical causation experts slated to testify for Dupont Co. in upcoming bellwether trials over whether perfluorooctanoic acid in drinking water near a former Teflon plant harmed residents must limit their testimony to whether the substance...

Chemicals
Time Available to Complete TSCA Bill Before Recess
The Senate has enough time to complete work on a major overhaul of the Toxic Substances

Control Act before its August recess, Sen. Jim Inhofe (R-Okla.) told Bloomberg BNA July 21....

Climate Change
French Minister: Informal Climate Talks 'Constructive'
Two days of informal climate discussions in Paris were a "constructive" step toward crafting a global agreement to fight climate change at the end of the year, France's top diplomat said July 21....

Climate Regulation
Groups Sustain Lobbying Push on Clean Power Plan
More than 120 entities reported lobbying Congress on the Environmental Protection Agency's soon-to-be-finalized Clean Power Plan in the second quarter of 2015, maintaining the same level of interest as a year ago and in one of the final...

Climate Regulation
Methane Studies Should Spur EPA Action, EDF Says
Methane emissions from natural gas transmission and storage networks in the U.S. are more than double the Environmental Protection Agency's estimates, which should spur the agency to set aggressive emissions limits on the industry,...

Climate Regulation
Senators Probe EPA Role in Setting Social Cost of Carbon
Senate Republicans are seeking all documents related to the Environmental Protection Agency's participation in the development of the social cost of carbon figure since its inception in 2009....

Energy
Controversial Measures Left Out of House Energy Bill
A repeal of the 40-year crude oil export ban and other controversial measures were left out of an energy package that eaders of the House Energy and Commerce Committee released late July 20....

Energy
FERC's Chair Calls for More Gas, Electric Coordination
Federal Energy Regulatory Commission Chairman Norman Bay encouraged the natural gas and electric industries to continue working together to coordinate market scheduling....

Energy
Strategic Reserve Oil Sale Would Fund Highway Bill
Senate legislation to fund highways and other transportation programs would be partially funded by a $9 billion sale of crude oil from the Strategic Petroleum Reserve, the latest attempt by Congress to use the emergency stockpile as a funding...

Hydraulic Fracturing
EPA Flexibility Urged on Oil, Gas Effluent Standards
Proposed federal standards that would ban shale oil and shale gas wastewater from publicly owned treatment plants are an overreach, making no allowance for different waste streams or possible future needs, industry groups told the Environmental...

Hydraulic Fracturing
Oral Arguments Set in Longmont's Fracking Appeal
The Colorado Court of Appeals has scheduled oral arguments for Sept. 24 in the case of a voter-approved ban on hydraulic fracturing in Longmont, Colo. (Colorado Oil & Gas Ass'n v. Longmont, Colo. Ct. App., No. 14-CA-1759, , 7/17/15)....

Hydraulic Fracturing
Study: Shallow Fracking May Pose Higher Risk to Water
Additional safeguards may be needed to protect underground water supplies from shallow, high-volume hydraulic fracturing operations, according to a study released July 21....

International Climate
Former DOE Official Questions U.S. Emissions-Cutting Pledge
The U.S. would likely fall well short of its pledge to cut greenhouse gas emissions up to 28 percent by 2025 under a global climate accord even if all of the Obama administration's actions including power plant carbon pollution limits are...

International Climate
Local Officials Join Pope in Call for Climate Action
Around 70 environmentally minded mayors and regional political officials from around the world signed a pledge July 21 calling for a global agreement to confront climate change, including a nonequivocal statement that human-induced climate...

Oil & Gas
Shell Vessels Head for Chukchi Sea, Expect Drilling Permits
The two drilling rigs and many of the support vessels marshalled by Royal Dutch Shell Plc for its drilling plans in the Arctic offshore are on their way north from the Aleutians in anticipation of receiving two drilling permits imminently....

Oil & Gas
Utah Approves Revised Permit for Oil Sands Project
The Utah Division of Oil, Gas & Mining has approved a revised permit with new conditions for an oil sands extraction project in the state's Uintah Basin....

Radioactive Waste
Dow, Rockwell Lose Bid to Redo Radiation Appeal
Dow Chemical Co. and Rockwell International Corp. have failed to convince a federal appeals court to reconsider its decision allowing damages for nuisance to stand for contamination from Colorado's Rocky Flats nuclear weapons plant...

Renewable Energy
Distributed Solar Power Revenues May More Than Triple in Decade
Global revenues from distributed solar photovoltaic power are expected to more than triple in a decade as the technology becomes viable without subsidies, according to the industry analyst Navigant Research....

Right to Know
Preliminary Toxics Release Inventory Dataset Released by EPA
Preliminary 2014 Toxics Release Inventory data received by the Environmental Protection Agency was posted online by the agency July 21. ...

Risk Assessment
Canada Proposes New Regulations for Selenium
Canada has proposed new regulations for selenium and 28 of its compounds based on a draft environmental assessment that concluded the chemicals are toxic....

Solid Waste
Environmental Groups Seek Rehearing on Waste Rule
A coalition of nine environmental advocacy groups have asked a federal appeals court to rehear challenges to a regulation exempting certain nonhazardous secondary materials from stricter air

pollution requirements when burned in solid...

Solid Waste
White House Threatens to Veto House Coal Ash Bill
President Barack Obama would be urged to veto House legislation (H.R. 1734) on the management and disposal of coal ash if it reaches his desk, according to a July 21 statement of administration policy....

Taxes
Wind Tax Credit Survives Senate Finance Committee Markup
The Senate Finance Committee voted July 21 to reinstate and extend the wind production tax credit through 2016 as part of a broader $96 billion package of tax extenders....

Water Pollution
EPA Sued Over Hazardous Substances Spills
The Environmental Protection Agency was sued July 21 by a coalition of environmental justice groups that claim EPA failed to regulate spills of hazardous substances from onshore nontransportation facilities, such as above-ground storage...

Water Pollution
North Carolina Agency Justified in Finding Pollution Based on Circumstantial Evidence
The North Carolina Department of Environment and Natural Resources was justified in finding that a chicken processing facility polluted a nearby stream based on circumstantial evidence, the state court of appeals ruled (House of Raeford...

Water Pollution
States Can Revise Water Standards Using New Criteria
States will be able use the Environmental Protection Agency's newly revised national human health criteria for 94 chemical pollutants when updating their water quality standards, the association of state water regulators said. ...

BNA INSIGHTS

Waste Management Responsibilities for Producers and Importers in Russia
For 17 years, waste management in Russia has been regulated by the federal law dated June 24, 1998, N 89-FZ, On Production and Consumption Waste. It establishes the basic norms and principles of state policy for waste management, except for...

REGULATORY AGENDA

Comment Deadlines

JULY 21 FEDERAL REGISTER

JULY 22 FEDERAL REGISTER

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UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

MAR 22 2016

OFFICE OF
THE ADMINISTRATOR

Mr. Basil Seggos
Acting Commissioner
New York Department of Environmental Conservation
625 Broadway, 14th Floor
Albany, New York 12233-1010

Dear Mr. Seggos:

Thank you for your January 14, 2016, letter to U.S. Environmental Protection Agency Administrator Gina McCarthy regarding actions to address perfluorooctanoic acid in drinking water and groundwater. The EPA takes seriously concerns regarding PFOA contamination, and we are actively engaged in advancing the science related to PFOA to provide the best available information to the public, states, tribes and local governments and in working with the state of New York and others to address public-health risks associated with PFOA contamination at specific sites, including Hoosick Falls, New York. I am responding to your request by providing information on the EPA's current efforts related to each of the elements discussed in your letter.

Your letter requests that the EPA lower its provisional health advisory of 400 parts per trillion for PFOA in drinking water to take into account the most current scientific evidence; adopt a protective maximum contaminant level for PFOA under the Safe Drinking Water Act; list PFOA as a hazardous substance under the Comprehensive Environmental Response, Compensation, and Liability Act; and review the remaining uses of PFOA under the Toxic Substances Control Act.

As referenced above, the EPA since 2009 has had a provisional health advisory for short-term exposure to PFOA of 400 parts per trillion. In June 2015 four of the five samples collected from the Hoosick Falls Public Water Supply showed that the drinking water had more than 600 parts per trillion of PFOA in it. In light of these exceedances the EPA made a recommendation to Hoosick Falls residents that they not drink or cook with the water from the public water supply, and the EPA contacted the state health department to urge that the state make the same recommendation. Additionally, the EPA recommended that Hoosick residents with private wells that have not yet been tested for the presence of PFOA ask the state health department to test their wells and use bottled water in the meantime.

Moving forward, the EPA is currently developing an updated lifetime health advisory for PFOA based on the best available science. Health advisories provide federal, state, tribal and local governments with nonregulatory guidance to make decisions in cases where a contaminant is not federally regulated. Once finalized, the new lifetime health advisory will supersede the agency's provisional value for PFOA issued in 2009. The agency expects to finalize and publish the updated health advisory this spring. The EPA is also evaluating PFOA as a drinking-water contaminant in accordance with the process prescribed by the Safe Drinking Water Act. That law provides that, in order for the EPA to regulate a contaminant, the agency must find: (1) the contaminant may have an adverse effect on the health of persons; (2) it is known to occur or has a substantial likelihood to occur in public-water systems with a

frequency and at levels of public-health concern; and (3) in the sole judgment of the Administrator, regulation of the contaminant presents a meaningful opportunity for health-risk reductions for persons served by public-water systems.

In 2009 the EPA included PFOA on the third Contaminant Candidate List. The EPA uses the Contaminant Candidate List to identify priority contaminants for regulatory decision making and information collection. In 2012 the agency included PFOA among the contaminants for which water systems are required to monitor under the third Unregulated Contaminant Monitoring Rule. The agency evaluates data gathered from the Unregulated Contaminant Monitoring Rule to characterize the levels of unregulated contaminants that occur in drinking water and the frequency of that occurrence on a national basis. Once the EPA has received and analyzed the occurrence data from the third Unregulated Contaminant Monitoring Rule in the summer of 2016, the agency will consider that data along with the peer-reviewed health effects assessment supporting the forthcoming final PFOA health advisory to make a regulatory determination on whether to initiate the process to develop a national primary-drinking-water regulation. As it undertakes this process, the EPA will continue to use its health advisory to guide actions with regard to any specific sites involving PFOA contamination of drinking water.

CERCLA and the National Oil and Hazardous Substances Pollution Contingency Plan provide the agency broad authorities to address human-health risk from exposures to PFOA, even if it is not listed as a hazardous substance. In this case, when released into the environment, PFOA and all perfluorinated compounds generally fall within the definition of "pollutant or contaminant" in CERCLA Section 101(33). CERCLA Section 104(a)(1) authorizes the EPA and other federal agencies to respond to releases or threatened releases of pollutants or contaminants when the release or potential release may present an imminent and substantial danger to the public health or welfare. Section 104(e) authorizes the agency to obtain information and enter, inspect and sample premises regarding pollutants or contaminants in the same way as for hazardous substances. In addition, the agency has the authority to list a site on the National Priorities List based on risks due to PFOA exposures. Sections 105(c) and (d) of CERCLA require that the National Priorities listing consider risks due to hazardous substances, pollutants and contaminants.

The EPA has been considering actions to address PFOA under our Toxic Substances Control Act authorities. The agency has been working since the late 1990s to investigate and subsequently reduce exposure to long-chain perfluorinated compounds, including PFOA, to curtail such uses whenever less toxic alternatives are available. This has occurred primarily through Significant New Use Rules on long-chain perfluorinated compounds and the 2010/2015 PFOA Stewardship Program on PFOA and related perfluorinated compounds. You can see more at www.epa.gov/assessing-and-managing-chemicals-under-tsca/20102015-pfoa-stewardship-program. At the same time, under the TSCA New Chemicals program the agency is reviewing new substitutes for these substances.

Through the 2010/2015 PFOA Stewardship Program that commenced in 2006 the manufacturers of PFOA committed to voluntarily phase out PFOA and PFOA-related chemicals, including potential PFOA precursors, by the end of 2015. Companies in the program committed to reduce global facility emissions and product content of PFOA and related chemicals by 95 percent by 2010 and to work toward eliminating emissions and product content by 2015. Most recent progress reports indicate that companies have met the phase-out goal. The EPA is currently reviewing final progress reports that the companies submitted at the end of February. Leading up to the PFOA Stewardship Program and to complement it, the agency has developed Significant New Use Rules to allow the EPA to review new uses of long-chain perfluorinated compounds before they are commercialized.

These Significant New Use Rules require that anyone who intends to manufacture, including import, or process any chemicals for uses contained in the Significant New Use Rules, submit a notification to the agency at least 90 days before beginning the activity. This provides the agency with an opportunity to review and, if necessary, place limits on manufacturers or processors who intend to reintroduce or import products with these chemicals. Since 2000 the EPA has published Significant New Use Rules impacting several hundred perfluorooctyl sulfonyls and perfluoroalkyl sulfonates. The most recent finalized Significant New Use Rules were in 2013 and addressed long-chain perfluorinated compounds as part of carpets or carpet-treatment products. The agency anticipates finalizing a separate Significant New Use Rules later this year to ensure that perfluorinated compounds phased out as part of the 2010/2015 PFOA Stewardship Program do not re-enter the marketplace without review. The EPA is also assessing ongoing uses of perfluorinated compounds, particularly those that can degrade to PFOA, and will determine the appropriate action to address potential risks from these chemicals.

Finally, the EPA recently added perfluoroalkyl compounds to the multiyear agenda for the agency's Integrated Risk Information System program. The IRIS program is working with the Office of Water and other EPA programs and regions to determine the range of perfluoroalkyl compounds for which an IRIS assessment is needed. The IRIS multiyear agenda is available at www.epa.gov/iris/iris-agenda.

In summary, the EPA is currently engaged in numerous actions to protect the public from potential health risks posed by PFOA. I thank you once more for your letter. If you have further questions, please contact me or your staff may contact Cathy Davis in the EPA's Office of Congressional and Intergovernmental Relations at davis.catherinem@epa.gov or (202) 564-2703.

Sincerely,

A. Stanley Meiburg
Acting Deputy Administrator

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

MAR 22 2016

OFFICE OF
THE ADMINISTRATOR

Howard A. Zucker, M.D.
Commissioner
New York State Department of Health
Corning Tower
Empire State Plaza
Albany, New York 12237

Dear Dr. Zucker:

Thank you for your January 14, 2016, letter to U.S. Environmental Protection Agency Administrator Gina McCarthy regarding actions to address perfluorooctanoic acid in drinking water and groundwater. The EPA takes seriously concerns regarding PFOA contamination, and we are actively engaged in advancing the science related to PFOA to provide the best available information to the public, states, tribes and local governments and in working with the state of New York and others to address public-health risks associated with PFOA contamination at specific sites, including Hoosick Falls, New York. I am responding to your request by providing information on the EPA's current efforts related to each of the elements discussed in your letter.

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These Significant New Use Rules require that anyone who intends to manufacture, including import, or process any chemicals for uses contained in the Significant New Use Rules, submit a notification to the agency at least 90 days before beginning the activity. This provides the agency with an opportunity to review and, if necessary, place limits on manufacturers or processors who intend to reintroduce or import products with these chemicals. Since 2000 the EPA has published Significant New Use Rules impacting several hundred perfluorooctyl sulfonyls and perfluoroalkyl sulfonates. The most recent finalized Significant New Use Rules were in 2013 and addressed long-chain perfluorinated compounds as part of carpets or carpet-treatment products. The agency anticipates finalizing a separate Significant New Use Rules later this year to ensure that perfluorinated compounds phased out as part of the 2010/2015 PFOA Stewardship Program do not re-enter the marketplace without review. The EPA is also assessing ongoing uses of perfluorinated compounds, particularly those that can degrade to PFOA, and will determine the appropriate action to address potential risks from these chemicals.

Finally, the EPA recently added perfluoroalkyl compounds to the multiyear agenda for the agency's Integrated Risk Information System program. The IRIS program is working with the Office of Water and other EPA programs and regions to determine the range of perfluoroalkyl compounds for which an IRIS assessment is needed. The IRIS multiyear agenda is available at www.epa.gov/iris/iris-agenda.

In summary, the EPA is currently engaged in numerous actions to protect the public from potential health risks posed by PFOA. I thank you once more for your letter. If you have further questions, please contact me or your staff may contact Cathy Davis in the EPA's Office of Congressional and Intergovernmental Relations at davis.catherinem@epa.gov or (202) 564-2703.

Sincerely,

A. Stanley Meiburg
Acting Deputy Administrator

Margaret Wood Hassan
New Hampshire

Andrew M. Cuomo
New York

Peter Shumlin
Vermont

March 10, 2016

Gina McCarthy
Administrator
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, NW
Washington, DC 20460

Dear Administrator McCarthy,

We write as Governors whose states are all in the midst of addressing local drinking water contamination involving the federally unregulated chemical perfluorooctanoic acid (PFOA). We are deeply concerned for the health and well-being of our communities grappling with this contaminant. In New York and New Hampshire, tests have indicated the presence of this chemical in public drinking water systems, and in New York and Vermont the chemical has been detected in several private wells. It is clear that PFOA contamination is not a state problem or a regional problem – it's a national problem that requires federal guidelines and a consistent, science-based approach.

The EPA's PFOA health advisory was recently lowered in one village in New York by the EPA's Regional Office, though the higher advisory remains in the rest of the country. We urge the EPA, under your leadership, to expeditiously review the best available science on this contaminant, and provide uniform guidance to states that our health and environmental officials can use in assessing the safety of our drinking water. In addition, we seek your help and support for additional drinking water testing and analysis in communities exposed to PFOA.

We also are all strong supporters of full federal funding for water infrastructure. The Drinking Water State Revolving Fund is a critical tool for states to invest in modern and safe drinking water upgrades. Unfortunately, over the last six years funding for this program, and its companion Clean Water Revolving Fund, have been flat or declining. This comes even as the American Society of Civil Engineers points out massive gaps between our water infrastructure needs and our investment. We should invest more in both the Drinking Water State Revolving Fund and the Clean Water Revolving Fund, and we look forward to working with you to make that happen.

We respectfully request your personal attention to the challenges created by PFOA contamination in our states. Consistency, accuracy, and uniformity are paramount. We look

EPA-HQ-2016-005679 02/14/2017

forward to your response and assistance as we work to identify the polluters responsible for this contamination, and hold them accountable for their actions. It is unacceptable to us that any community should have to be concerned about the safety of their drinking water. Families in our states are worried about potentially tragic short and long-term health impacts, not to mention the potential loss in property values for homes in affected areas. It has been our priority to ensure that residents are being provided clean and safe water immediately, and that our infrastructure be modernized to eliminate these concerns in the future.

Sincerely,

Maggie Hassan

Margaret Wood Hassan
Governor of New Hampshire

Andrew M. Cuomo
Governor of New York

Peter Shumlin
Governor of Vermont

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To: Pillsbury, Sarah[Sarah.Pillsbury@des.nh.gov]
Cc: Freise, Clark[Clark.Freise@des.nh.gov]; Burack, Thomas[Thomas.Burack@des.nh.gov]; Grevatt, Peter[Grevatt.Peter@epa.gov]; Lovely, William[Lovely.William@epa.gov]
From: Burneson, Eric
Sent: Fri 3/18/2016 5:21:31 PM
Subject: RE: Invitation to a call at 11:45

Sarah:

Following up on your question about what constitutes a "short term" for purposes of the provisional health advisory. The Health advisory states:

"The relevant period of exposure for the Health Advisory is a short-term exposure. This time period is consistent with the toxicity data used for PFOA and PFOS, both of which rely upon subchronic data." See https://www.epa.gov/sites/production/files/2015-09/documents/pfoa-pfos-provisional.pdf.

However the fact sheet we prepared in 2014 clarifies that short term means "weeks to months" see https://www.epa.gov/sites/production/files/2015-09/documents/peer-review-of-health-effects-documents-for-pfoa-and-pfos-factsheet-february-2014.pdf.

I hope this information is helpful and look forward to speaking with you next week at the ASDWA Conference.

Eric Burneson, P.E.

Director of Standards and Risk Management

Office of Ground Water and Drinking Water

US Environmental Protection Agency

Phone: 202-564-5250

Fax: 202 564 3760

From: Pillsbury, Sarah [mailto:Sarah.Pillsbury@des.nh.gov]
Sent: Friday, March 18, 2016 11:23 AM
To: Burneson, Eric <Burneson.Eric@epa.gov>
Cc: Freise, Clark <Clark.Freise@des.nh.gov>; Burack, Thomas <Thomas.Burack@des.nh.gov>
Subject: RE: Invitation to a call at 11:45

Yes. Thank you. I am copying my Commissioner and Assistant Commissioner who may also join the call..

Sarah Pillsbury, Administrator

Drinking Water & Groundwater Bureau, NHDES

29 Hazen Drive, PO Box 95, Concord, NH 03302

Tel: (603) 271-1168 | Fax: (603) 271-5171

NHDES Be Well Informed Private Well App - An interactive web application for private well owners to interpret water quality test results and identify appropriate water treatment options

From: Burneson, Eric [mailto:Burneson.Eric@epa.gov]
Sent: Friday, March 18, 2016 11:18 AM
To: Pillsbury, Sarah
Subject: Invitation to a call at 11:45

Sarah:

I just sent an invitation to a call about the PFOA monitoring results for 11:45. Can you participate at this time? The call in number is 1 866 299 3188 code 202 564 5250

Eric Burneson, P.E.

Director of Standards and Risk Management

Office of Ground Water and Drinking Water

US Environmental Protection Agency

Phone: 202-564-5250

Fax: 202 564 3760

From: Burneson, Eric
Required Attendees: Lovely, William; Grevatt, Peter; sarah.pillsbury@des.nh.gov
Optional Attendees: Greene, Ashley; Spalding, Curt; Olson, Bryan; Barmakian, Nancy; Lindsay, Jane; Gray, Stuart
Location: 866-299-3188 code 202-564-5250
Importance: Normal
Subject: Monitoring Results PFOA
Start Date/Time: Fri 3/18/2016 3:45:00 PM
End Date/Time: Fri 3/18/2016 4:15:00 PM

Confirming the call at 11:45. Note that the call in code originally sent was incorrect. The code above is 202 564 5250 is the correct code.

To: sarah.pillsbury@des.nh.gov[sarah.pillsbury@des.nh.gov]
From: Burneson, Eric
Sent: Fri 3/18/2016 3:18:22 PM
Subject: Invitation to a call at 11:45

Sarah:

I just sent an invitation to a call about the PFOA monitoring results for 11:45. Can you participate at this time? The call in number is 1 866 299 3188 code 202 564 5250

Eric Burneson, P.E.

Director of Standards and Risk Management

Office of Ground Water and Drinking Water

US Environmental Protection Agency

Phone: 202-564-5250

Fax: 202 564 3760

From: Burneson, Eric
Required Attendees: Lovely, William; Grevatt, Peter; sarah.pillsbury@des.nh.gov
Optional Attendees: Greene, Ashley; Spalding, Curt; Olson, Bryan; Barmakian, Nancy
Location: 866-299-3188 code 202-564-5250
Importance: Normal
Subject: Monitoring Results PFOA
Start Date/Time: Fri 3/18/2016 3:45:00 PM
End Date/Time: Fri 3/18/2016 4:15:00 PM

From: Burneson, Eric
Required Attendees: Lovely, William; Grevatt, Peter; sarah.pillsbury@des.nh.gov
Optional Attendees: Greene, Ashley; Spalding, Curt; Olson, Bryan; Barmakian, Nancy
Location: 866-299-3188 code 202-564-5250
Importance: High
Subject: Canceled: Monitoring Results PFOA
Start Date/Time: Fri 3/18/2016 3:45:00 PM
End Date/Time: Fri 3/18/2016 4:15:00 PM

From: Burneson, Eric
Required Attendees: Lovely, William; Grevatt, Peter; sara.pillsbury@des.nh.gov
Optional Attendees: Greene, Ashley
Location: 866-299-3188 code 202-564-5250
Importance: Normal
Subject: Monitoring Results PFOA
Start Date/Time: Fri 3/18/2016 3:45:00 PM
End Date/Time: Fri 3/18/2016 4:15:00 PM

From: Burneson, Eric
Required Attendees: Lovely, William; Grevatt, Peter; sara.pillsbury@des.nh.gov
Optional Attendees: Greene, Ashley
Location: 866-299-3188; 202-565-5350
Importance: Normal
Subject: Monitoring Results PFOA
Start Date/Time: Fri 3/18/2016 3:45:00 PM
End Date/Time: Fri 3/18/2016 4:15:00 PM

Margaret Wood Hassan
New Hampshire

Andrew M. Cuomo
New York

Peter Shumlin
Vermont

March 10, 2016

Gina McCarthy
Administrator
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, NW
Washington, DC 20460

Dear Administrator McCarthy,

We write as Governors whose states are all in the midst of addressing local drinking water contamination involving the federally unregulated chemical perfluorooctanoic acid (PFOA). We are deeply concerned for the health and well-being of our communities grappling with this contaminant. In New York and New Hampshire, tests have indicated the presence of this chemical in public drinking water systems, and in New York and Vermont the chemical has been detected in several private wells. It is clear that PFOA contamination is not a state problem or a regional problem – it's a national problem that requires federal guidelines and a consistent, science-based approach.

The EPA's PFOA health advisory was recently lowered in one village in New York by the EPA's Regional Office, though the higher advisory remains in the rest of the country. We urge the EPA, under your leadership, to expeditiously review the best available science on this contaminant, and provide uniform guidance to states that our health and environmental officials can use in assessing the safety of our drinking water. In addition, we seek your help and support for additional drinking water testing and analysis in communities exposed to PFOA.

We also are all strong supporters of full federal funding for water infrastructure. The Drinking Water State Revolving Fund is a critical tool for states to invest in modern and safe drinking water upgrades. Unfortunately, over the last six years funding for this program, and its companion Clean Water Revolving Fund, have been flat or declining. This comes even as the American Society of Civil Engineers points out massive gaps between our water infrastructure needs and our investment. We should invest more in both the Drinking Water State Revolving Fund and the Clean Water Revolving Fund, and we look forward to working with you to make that happen.

We respectfully request your personal attention to the challenges created by PFOA contamination in our states. Consistency, accuracy, and uniformity are paramount. We look

EPA-HQ-2016-005679 02/14/2017

forward to your response and assistance as we work to identify the polluters responsible for this contamination, and hold them accountable for their actions. It is unacceptable to us that any community should have to be concerned about the safety of their drinking water. Families in our states are worried about potentially tragic short and long-term health impacts, not to mention the potential loss in property values for homes in affected areas. It has been our priority to ensure that residents are being provided clean and safe water immediately, and that our infrastructure be modernized to eliminate these concerns in the future.

Sincerely,

Maggie Hassan

Margaret Wood Hassan
Governor of New Hampshire

Andrew M. Cuomo
Governor of New York

Peter Shumlin
Governor of Vermont

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UNITED STATES
ENVIRONMENTAL PROTECTION AGENCY

REGION III
1650 Arch Street
Philadelphia, PA 19103-2029

REGION V
77 West Jackson Boulevard
Chicago, IL 60604

IN THE MATTER OF:	)	
	)	
The Chemours Company	)	
and	)	
E.I. du Pont de Nemours and Company	)	ORDER ON CONSENT
1007 Market Street	)	
Wilmington, DE 19898	)	
	)	Proceeding under Section 1431(a)(1)
Respondents.	)	of the Safe Drinking Water Act,
	)	42 U.S.C. § 300i(a)(1)
Washington Works Facility	)	
Route 892 South	)	
Washington, WV 26181	)	
	)	Docket Nos. SDWA-03-2016-___-DS
	)	SDWA-05-2016-_____

Pre-Decisional Draft

I. STATUTORY AUTHORITY

1. This Order on Consent ("Order") is issued pursuant to the authority vested in the Administrator of the United States Environmental Protection Agency ("EPA") by Section 1431(a)(1) of the Safe Drinking Water Act ("SDWA" or "the Act"), 42 U.S.C. § 300i(a)(1), and supersedes the Order on Consent (Docket Nos. SDWA-03-2009-0127DS and SDWA-05-2009-0001) issued on March 10, 2009.

2. The authority to issue this Order was delegated to the Regional Administrators by Delegation No. 9-17, dated May 11, 1994.

3. Under the SDWA, Congress has authorized EPA to exercise broad authority for the protection of public health from contaminants entering a public water system or an underground source of drinking water.

II. STIPULATIONS

4. The Chemours Company ("Chemours") and E.I. du Pont de Nemours and Company ("DuPont") (collectively, "Respondents") consent to EPA's jurisdiction to issue this Order. Chemours and DuPont do not admit to the EPA Findings in this Order.

5. Respondents waive any defenses they might have as to jurisdiction and venue and agree not to contest any of the findings of fact or conclusions of law herein in any action to enforce this Order. Except as to any proceeding brought by EPA to enforce this Order, in agreeing to this Order, Respondents make no admission of fact or law and reserve all rights and defenses available regarding liability or responsibility in any other legal proceeding related to the subject matter of this Order. Respondents further waive any rights to appeal this Order that would be otherwise applicable under the SDWA, including under Section 1448(a) of the Safe Drinking Water Act, 42 U.S.C. § 300j-7(a).

III. DEFINITIONS AND BACKGROUND

6. "Contaminant" means "any physical, chemical, biological, or radiological substance or matter in water." See 42 U.S.C. § 300f(6).

7. The term "underground source of drinking water" ("USDW") means an aquifer or a portion thereof which supplies a public water system ("PWS"), or which contains a sufficient quantity of ground water to supply a PWS and which currently supplies drinking water for human consumption, or contains fewer than 10,000 milligrams per liter total dissolved solids, and is not an exempted aquifer. See 40 C.F.R. § 144.3.

8. C-8, for purposes of this Order, is perfluorooctanoic acid, CAS # 335-67-1 (PFOA) and its salts, including ammonium perfluorooctanoate, CAS # 3825-26-1 (APFO). These are man-made perfluorinated compounds that do not occur naturally in the environment.

9. The term "day" means calendar day. When a stated time expires on a Saturday, Sunday or Federal Holiday, the stated time period shall be extended to include the next business day.

10. Micrograms per liter (µg/l) is the same as parts per billion (ppb), or may also be described in parts per trillion or (ppt). (.001 ppb is the same as 1 ppt)

11. The term "source water" shall mean water prior to any kind of treatment.

12. A "public water system," hereafter "PWS," provides piped drinking water for human consumption to persons within the meaning of Section 1401 (4) of the Act, 42 U.S.C. §300f(4) and 40 CFR §141.2.

13. A private water system is used by individual residents, or serves less than 25 persons per year from a well or other surface or ground water source and is otherwise not a "PWS."

14. The term "finished water" shall mean water that has passed through all the processes in a system's water treatment plant and is ready to be delivered to consumers.

15. "EDD" format is Electronic Delimited Data format for submission of all analytical data.

IV. EPA FINDINGS

16. Chemours and DuPont are both corporations and therefore are "persons" within the meaning of Section 1401(12) of the SDWA, 42 U.S.C. § 300f(12).

17. At all times relevant to this Order, Respondents own and operate, or previously owned and operated, a manufacturing facility known as the Washington Works facility ("Facility"), located in Washington, Wood County, West Virginia.

18. DuPont used C-8, in the form of APFO, in its manufacturing processes at the Facility since the early 1950s.

Pre-Decisional Draft

19. On November 15, 2001, DuPont, the West Virginia Department of Environmental Protection ("WVDEP") and the West Virginia Department of Health and Human Resources ("WVDHHR") entered into an agreement on consent ("WV Order"), which provided for, *inter alia*, a toxicological and human health risk assessment of C-8 to be conducted under the supervision of a C-8 Assessment of Toxicity ("CAT") Team. Ground water and surface water monitoring and plume identification in West Virginia and Ohio was conducted under the supervision of a Ground Water Investigation Steering ("GIS") Team.

20. In April 2002, the CAT Team conducted a toxicological and human health risk assessment of C-8 and developed a screening level of 150 ppb for C-8 in drinking water.

21. From 2000 to 2006 DuPont implemented recycling and abatement technologies that reduced both air emissions and water discharges of C-8 from the Facility. Annual emissions to air in 2005 were reported to be approximately 12,600 kilograms lower than annual air emissions in 2000. Annual discharges to water in 2005 were reported to be approximately 20,400 kilograms lower than annual water discharges in 2000. As of year-end 2006, DuPont had reduced annual air discharges by 99.1% and had reduced annual water discharges by 99.2% since 2000.[1]

[1] DuPont, "Data Assessment DuPont Washington Works (OPPT-2004-0113 PFOA Site-related Environmental Assessment Program)," (October 2, 2008).

22. From 2001 to 2006, sampling conducted through the GIS Team effort and by DuPont detected C-8 in private and public drinking water sources in Ohio and West Virginia at concentrations ranging from below the limits of quantitation up to 21.1 ppb.[2]

23. On November 20, 2006, DuPont and EPA entered into an Order on Consent ("2006 Order"), which required DuPont to offer, *inter alia*, alternative drinking water or treatment to public water systems or owners of residences using private water systems living in the vicinity of the Facility where levels of C-8 detected in the finished water of public and private drinking water systems were equal to or greater than 0.50 ppb.

24. The 0.50 ppb action level established in the 2006 Order was a precautionary level to reduce exposure from C-8 to the population living in the vicinity of the Facility.

25. On January 8, 2009, the EPA Office of Water issued a Provisional Health Advisory which established a national value of 0.4 ppb for PFOA.[3]

26. Provisional Health Advisory values reflect reasonable, health-based hazard concentrations above which action should be taken to reduce exposure to PFOA in drinking water.[4]

27. On March 11, 2009, DuPont and EPA entered into an Order on Consent ("2009 Order"), which required DuPont to offer, *inter alia*, alternative drinking water or treatment to public water systems or owners of residences using private water systems living in the vicinity of the Facility where levels of C-8 detected in the finished water of public and private drinking water systems were equal to or greater than 0.40 ppb.

28. The 2009 Order achieved comprehensive identification of private and public water systems in the vicinity of the Facility that were not being previously addressed and, based upon representations by Respondents, ensured that alternate water and/or treatment was offered, installed, and maintained at all public and private water systems that exceeded 0.40 ppb, or 400 ppt of C-8 in finished water.

29. Among other provisions, the 2009 Order provided that the Order was "binding upon DuPont and its agents, successors and assigns." 2009 Order at Para. 49.

---

[2] Hartten, Andrew S., Project Director, DuPont, "Amended 3Q05, and 4Q05 and 1Q06 Residential Sampling Results, West Virginia and Ohio DuPont Washington Works, Washington, WV (EPA Docket ID Number OPPT 2004-0113 PFOA Site-Related Environmental Assessment Program," submitted to Chad Board, West Virginia Department of Environmental Protection (April 5, 2006).

[3] United States Environmental Protection Agency's Office of Water, "Provisional Health Advisories for Perflurooctanioic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS)" (2009). (including Administrative Record thereto). Available: www.epa.gov/waterscience/criteria/drinking/pha-PFOA_PFOS.pdf

[4] Id.

30. Chemours is a successor to DuPont for liabilities incurred at the Facility as of July 1, 2015; therefore the 2009 Order is binding on Chemours.

31. Pursuant to the 2009 Order, Respondents have attempted to comply with the Work Plan that required DuPont to monitor for C-8 and provided treatment to owners of residences using private water systems for which data have demonstrated levels of C-8 at or above 0.40 ppb in finished water. For residents who so agreed, Respondents have either connected to a public water system or have installed and are operating granulated activated carbon treatment at approximately ___ private water systems with finished water that exceeded 0.40 ppb of C-8 and whose owners have accepted DuPont's offer.

32. Although Respondents attempted to comply with the 2009 Order requirements for the Work Plan, approximately ___ owners of private water systems in the vicinity of the Facility with finished water that exceeds 0.40 ppb of C-8 have declined or not responded to DuPont's offer for installation of treatment or connection to a public water system, or such residences were vacant.

33. On ___, 2016, EPA's Office of Water established a lifetime Health Advisory (HA) value for PFOA of ___ ppb.

34. EPA has identified private water systems and PWSs in the vicinity of the Facility where USDWs may contain levels of C-8 above the HA value.

Pre-Decisional Draft

35. C-8 is currently not a contaminant for which a national primary drinking water regulation, including a maximum contaminant level ("MCL"), has been established pursuant to the SDWA.

36. DuPont has released C-8 to the air, discharged C-8 to surface waters, and disposed of residues containing C-8 at the Facility. DuPont has also disposed of residues containing C-8 to its Dry Run, Local, and Letart Landfills in West Virginia and has otherwise shipped residues containing C-8 off-site for destruction and/or disposal.

37. The releases, discharges, and/or disposal referred to in Paragraph 36 have resulted in releases of C-8 to air, ground water, surface water, and soil.

38. The releases referred to in Paragraph 36 have entered USDWs and surface waters and resulted in levels of C-8 at concentrations at or above ___ ppb in some of the receiving waters.

39. Public and private water systems in the vicinity of the Facility are using water sources contaminated with C-8 at levels that may be at or above ___ ppb.

40. Section 1431 of the SDWA requires a finding that "a contaminant which is present in or is likely to enter a public water system or an underground source of drinking water…may present an imminent and substantial endangerment to the health of persons...." As required by Section 1431 of the SDWA and for purposes of this Order, EPA has determined that C-8 is a contaminant present in or likely to enter a PWS or a USDW which may present an imminent and substantial endangerment to human health at concentrations at or above [redacted] ppb in drinking water.[5]

41. State and local authorities rely on the expertise and resources of EPA to review and evaluate unregulated contaminants. The WVDEP, WVDHHR, OEPA, the Ohio Department of Health ("ODH"), and local authorities are relying on the EPA to establish a level for C-8 in drinking water that reduces exposure to C-8 for residents in the vicinity of the Facility. State agency actions taken to date, including actions taken by WVDEP, WVDHHR, OEPA, and ODH, have been based on the Action Level of 0.40 ppb established in the 2009 Order.

42. EPA has consulted with WVDEP, WVDHHR, OEPA, and ODH to confirm that the information upon which this Order is based is correct. The WVDEP, WVDHHR, OEPA, and ODH have requested that EPA take this action. Therefore, all requisite conditions have been satisfied for EPA action under Section 1431(a)(1) of the SDWA, 42 U.S.C. § 300i(a)(1).

V. ORDER ON CONSENT

Pre-Decisional Draft

43. Pursuant to the authority given to the EPA Administrator by Section 1431(a)(1) of the SDWA, 42 U.S.C. § 300i(a)(1), and delegated to the Regional Administrators, Respondents are ORDERED and hereby consent to the following:

a) Temporary Provision of Alternate Drinking Water. For those private water systems where existing validated data demonstrates levels of C-8 at or above [redacted] ppb in their finished water, Respondents shall provide an alternate drinking water supply as soon as practicable, but in any event no later than fourteen (14) days after the execution of this Order. Where Respondents conduct a water system survey pursuant to Paragraphs 43(e) or (f) and identifies private and public water systems where the level of C-8 in the finished water is at or above [redacted] ppb, Respondents shall provide an alternate drinking water supply as soon as practicable, but in any event no later than thirty (30) days, from the receipt of validated data. An "alternate drinking water supply" shall mean: water from some other source, acceptable to EPA, that meets the water quality requirements of 40 C.F.R. Part 141 and has a level of C-8 less than [redacted] ppb in finished water where applicable; is in sufficient quantity for drinking and cooking; and is

[5] United States Environmental Protection Agency's Office of Water, "Health Advisories for Perfluorooctanioic Acid (PFOA) and Perfluorooctane Sulfonate (PFOS)" (2016). (including Administrative Record thereto). Available: www.epa.gov/waterscience/criteria/drinking/pha-PFOA_PFOS.pdf

provided in a manner convenient to the users. Respondents shall continue to provide an alternate drinking water supply until it can fully implement the permanent remedies described *infra* pursuant to Paragraph 43 of this Order or the resident declines the offer or is non-responsive to the offer of treatment (as determined by EPA). Respondents shall be responsible for all costs of the provision of alternate drinking water.

b) Private Water Systems Receiving Treatment. For private water systems at which DuPont has already installed GAC Treatment, Respondents shall provide for operation and maintenance of each GAC Treatment system in good working order, including but not limited to timely replacement of carbon filters, until Respondents demonstrate to the satisfaction of EPA that the source prior to GAC Treatment contains less than [redacted] ppb of C-8 for four consecutive quarters, or the conditions of Paragraph 47 have been met. Respondents may also elect to satisfy any ongoing obligation under this Paragraph by connecting a particular location to a public water system that contains less than [redacted] ppb of C-8 in finished water.

c) Public Water Systems Receiving Treatment. For public water systems, at which DuPont has already installed GAC Treatment, Respondents shall provide for operation and maintenance of each GAC Treatment system in good working order, including but not limited to timely carbon bed changes, until Respondents demonstrate to the satisfaction of EPA that the source water in the system prior to GAC Treatment contains less than [redacted] ppb of C-8 for four consecutive quarters, or the conditions of Paragraph 47 have been met.

Pre-Decisional Draft

d) Action at Private Water Systems Based On Existing Data. For those private water systems where existing validated data demonstrates levels of C-8 at or above [redacted] ppb in their finished water, Respondents shall, within fourteen (14) days of execution of this Order, submit to EPA for approval, and to WVDHHR, WVDEP, OEPA, and ODH for review, a written Water Treatment Plan for each of these water systems in accordance with the provisions of Paragraph 43(g).

e) Survey and Identification of Additional Private and Public Water Systems. For geographical areas defined by EPA (upon consultation with West Virginia and Ohio), Respondents shall conduct a water system survey and where any private or public water system (not already sampled) is identified, monitor the finished and source waters for the presence of C-8. Respondents shall notify EPA of monitoring results immediately, but in any event no later than 7 days, after the data are finalized through Respondents' internal data quality control/quality assurance procedures. Respondents shall also notify owners or operators of private and public water systems of monitoring results within 7-10 days after

the data are finalized through Respondents' internal data quality control/quality assurance procedures.

f) Newly Activated or Permitted Water Systems. Upon notification by EPA of any newly activated public water system or any newly constructed/permitted/put into use private water system that conforms to state and local code and is located in the geographical areas defined by EPA (upon consultation with West Virginia and Ohio), Respondents shall monitor the finished and source waters for the presence of C-8 in accordance with the provisions of Paragraph 43(e). On the anniversary date of the effective date of this Order and annually thereafter, Respondents shall survey the geographical areas defined by EPA for any new private or public water systems until Respondents demonstrate to the satisfaction of EPA that the USDWs in these geographical areas (or a subset of those areas) contain less than [redacted] ppb of C-8 for four consecutive quarters, or the conditions of Paragraph 47 have been met. Respondents shall monitor the finished and source waters of any new systems for the presence of C-8 in accordance with the provisions of Paragraph 43(e).

g) Water Treatment Plan. If any additional private or public water systems covered by this Order contain C-8 at or above [redacted] ppb in their finished water, Respondents shall, within 30 days of receipt of validated data, submit to EPA for approval, and to WVDHHR, OEPA, and ODH for review, a written Water Treatment Plan for each of these water systems. Respondents shall perform all monitoring using Standard Method 537 as used in the Unregulated Contaminant Monitoring Rule list 3 (UCMR3) or an EPA-approved analytical method. The Water Treatment Plan shall include:

   i. a written offer to install and provide for operation and maintenance of GAC Treatment (including a draft operation and maintenance agreement);

   ii. identification of anticipated necessary permits;

   iii. a schedule for design and implementation of the GAC Treatment system; and

   iv. identification of technical and other information needed from the owner or operator of the water source in order for DuPont to design and install the system.

h) Implementation of Water Treatment Plan. Following approval from EPA, Respondents shall implement the Water Treatment Plan for any additional water system whose owner or operator accepts Respondents' offer. Respondents shall

act with all deliberate speed to design treatment, seek necessary regulatory permits, and install GAC Treatment or an alternative approved by EPA. If an owner or operator of a water system rejects Respondents' offer, either through express rejection or silence, Respondents shall inform EPA of this rejection and provide documentation.

i) Respondents' Operation and Maintenance Obligations. Respondents have or will execute operation and maintenance agreements ("O&M Agreements") with each water system owner or operator who has accepted the offer for treatment. Respondents will provide for operation and maintenance of the GAC Treatment or an alternative approved by EPA consistent with the specific terms of these O&M Agreements until it demonstrates to the satisfaction of EPA that the water system's source water prior to treatment is less than [redacted] ppb of C-8 for four consecutive quarters, or the conditions of Paragraph 47 have been met.

j) Follow-up Monitoring. After GAC Treatment is terminated, Respondents shall monitor annually the source water at EPA-specified public and private water systems for a period of five (5) years.

44. Progress Reports. Respondents shall submit Progress Reports as follows:

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a) Beginning [redacted], 2016, and quarterly thereafter, Respondents shall submit to EPA, WVDHHR, WVDEP, OEPA and ODH written reports summarizing all actions taken, with all data submitted in EDD format, in response to Paragraph 43 herein ("Progress Reports"). This reporting requirement shall remain in effect until Respondents submit a written request to EPA to submit Progress Reports on an annual basis and EPA approves such a request. Respondents shall continue to submit Progress Reports until such time as EPA provides written notice that the reports are no longer necessary, or this Order is terminated.

b) All Progress Reports required by this Paragraph shall contain the following certification, which shall be signed by a responsible corporate officer for both Chemours and DuPont:

> "I certify under penalty of law that this document and all attachments were prepared under my direction or supervision in accordance with a system designed to assure that qualified personnel properly gather and evaluate the information submitted. Based on my inquiry of the person or persons who manage the system, or those persons directly responsible for gathering the information, the information submitted is, to the best of my knowledge and belief, true, accurate, and complete. I am aware that there

are significant penalties for submitting false information, including the possibility of fine and imprisonment for knowing violations."

c) For purposes of this Order, a responsible corporate official shall be:

(A) a president, secretary, treasurer, or vice-president of each Respondent in charge of a principal business function, or any other person who performs similar policy or decision-making functions for each Respondent; or

(B) the manager of the Washington Works, West Virginia, Facility, so long as authority to sign documents has been delegated in writing to the manager in accordance with corporate procedures.

VI. GENERAL PROVISIONS

45. The Administrative Record to this Order is incorporated herein by reference.

46. Nothing in this Order is intended to supersede, impede, interfere with or otherwise affect the development of an MCL or other regulatory limit for C-8 that may be established by EPA through its regulatory processes in the future.

Pre-Decisional Draft

47. Notwithstanding any other provision of this Order, the EPA reserves the right to modify the Action Level identified in this Order if information previously unknown to EPA is received and EPA determines that this previously unknown information, together with any other relevant information, indicates that the Action Level may not be protective of human health, and Respondents reserve all rights and defenses should EPA take action under this Paragraph.

48. Respondents waive any and all remedies, claims for relief and otherwise available rights to judicial or administrative review that Respondents may have with respect to any issue of fact or law set forth in this Order on Consent, including any right of judicial review under Section 1448(a) of the Safe Drinking Water Act, 42 U.S.C. § 300j-7(a).

49. All submissions, including Progress Reports, required under this Order shall be submitted to the following addressees:

As to EPA:

Roger Reinhart
Groundwater and Enforcement Branch
U.S. EPA Region III
1650 Arch Street (3WP22)

Philadelphia, PA 19103-2029

Heather Shoven
Ground Water and Drinking Water Branch
U.S. EPA Region V
77 West Jackson Boulevard (WG-15J)
Chicago, IL 60604

As to WVDHHR:

Walter Ivey, Director
Division of Environmental Engineering
Office of Environmental Health Services
Dept. of Health and Human Resources
Capital and Washington Streets
One Davis Square, Suite 200
Charleston, WV 25301-1798

As to WVDEP:

Yogesh Patel
Groundwater Protection and Permitting Section
Division of Water and Waste Management
W.Va. Dept. of Environmental Protection
601 57th Street, SE
Charleston, WV 25304

Pre-Decisional Draft

As to OEPA:

Mike Baker, Chief
Division of Drinking and Ground Waters
Ohio EPA
122 South Front Street
Columbus, OH 43214

As to ODH:

W. Gene Phillips, RS, Bureau Chief
Bureau of Environmental Health
Ohio Department of Health
246 North High Street
P.O. Box 118

Columbus, OH 43216

50. This Order shall apply to and be binding upon Chemours and DuPont, and their agents, successors and assigns.

51. In order to ensure completion of the Work, Respondents shall secure financial assurance, initially in the amount of $[_____] for the benefit of EPA. The financial assurance must be one or more of the mechanisms listed below and satisfactory to EPA. Respondents may use multiple mechanisms if they are limited to surety bonds guaranteeing payment, letters of credit, trust funds, and/or insurance policies.

a. A surety bond guaranteeing payment and/or performance of the Work that is issued by a surety company among those listed as acceptable sureties on federal bonds as set forth in Circular 570 of the U.S. Department of the Treasury;

b. An irrevocable letter of credit, payable to or at the direction of EPA, that is issued by an entity that has the authority to issue letters of credit and whose letter-of-credit operations are regulated and examined by a federal or state agency;

c. A trust fund established for the benefit of EPA that is administered by a trustee that has the authority to act as a trustee and whose trust operations are regulated and examined by a federal or state agency;

d. A policy of insurance that provides EPA with acceptable rights as a beneficiary thereof and that is issued by an insurance carrier that has the authority to issue insurance policies in the applicable jurisdiction(s) and whose insurance operations are regulated and examined by a federal or state agency;

e. A demonstration by one or more Respondents that each such Respondent meets the relevant financial test criteria of 40 C.F.R. § 264.143(f) and reporting requirements of this Order for the sum of the estimated cost of the work and the amounts, if any, of other federal, state, or tribal environmental obligations financially assured through the use of a financial test or guarantee, accompanied by a standby funding commitment, which obligates Respondents to pay funds to or at the direction of EPA, up to the amount financially assured through the use of this demonstration in the event of a work takeover; or

f. A guarantee to fund or perform the work executed in favor of EPA by one of the following: (1) a direct or indirect parent company of a Respondent; or (2) a company that has a "substantial business relationship" (as defined in 40 C.F.R. § 264.141(h)) with a Respondent; provided, however, that any company providing such a guarantee must demonstrate to EPA's satisfaction that it meets the relevant financial test criteria of 40 C.F.R. § 264.143(f) and reporting requirements of this Order for the sum of the estimated cost of the work and the amounts, if any, of other federal,

state, or tribal environmental obligations financially assured through the use of a financial test or guarantee.

52. Respondents have selected, and EPA has found satisfactory, as an initial financial assurance a [insert type] prepared in accordance with Paragraph 51. Within 30 days after the Effective Date, or 30 days after EPA's approval of the form and substance of Respondents' financial assurance, whichever is later, Respondents shall secure all executed and/or otherwise finalized mechanisms or other documents consistent with the EPA-approved form of financial assurance and shall submit such mechanisms and documents to EPA.

53. If Respondents provide financial assurance by means of a demonstration or guarantee under Paragraph 51, the affected Respondents shall also comply and shall ensure that their guarantors comply with the other relevant criteria and requirements of 40 C.F.R. § 264.143(f) and this Section, including, but not limited to: (a) the initial submission to EPA of required documents from the affected entity's chief financial officer and independent certified public accountant no later than 30 days after the Effective Date; (b) the annual resubmission of such documents within 90 days after the close of each such entity's fiscal year; and (c) the notification of EPA no later than 30 days, in accordance with Paragraph 54, after any such entity determines that it no longer satisfies the relevant financial test criteria and requirements set forth at 40 C.F.R. § 264.143(f)(1). Respondents agree that EPA may also, based on a belief that an affected entity may no longer meet the financial test requirements of Paragraph 51, require reports of financial condition at any time from such entity in addition to those specified in this Paragraph.

Pre-Decisional Draft

54. Respondents shall diligently monitor the adequacy of the financial assurance. If any Respondent becomes aware of any information indicating that the financial assurance provided is inadequate or otherwise no longer satisfies the requirements of this Order, such Respondent shall notify EPA of such information within 7 days. If EPA determines that the financial assurance provided by Respondents is inadequate or otherwise no longer satisfies the requirements of this Order, EPA will notify the affected Respondent of such determination. Respondents shall, within 30 days after notifying EPA or receiving notice from EPA under this Paragraph, secure and submit to EPA for approval a proposal for a revised or alternative financial assurance mechanism that satisfies the requirements of this Order. EPA may extend this deadline for such time as is reasonably necessary for the affected Respondent, in the exercise of due diligence, to secure and submit to EPA a proposal for a revised or alternative financial assurance mechanism, not to exceed 60 days. Respondents shall follow the procedures of ¶ (Modification of Financial Assurance) in seeking approval of, and submitting documentation for, the revised or alternative financial assurance mechanism. Respondents' inability to secure and submit to EPA financial assurance in accordance with this Order shall in no way excuse performance of any other requirements of this Order.

55. Access to Financial Assurance.

a. If EPA issues a notice of implementation of a work takeover, then, in accordance with any applicable financial assurance mechanism and/or related standby funding commitment, EPA is entitled to require that any funds guaranteed be paid in accordance with ¶ .

b. If EPA is notified by the issuer of a financial assurance mechanism that it intends to cancel such mechanism, and the affected SD fails to provide an alternative financial assurance mechanism in accordance with this Section at least 30 days prior to the cancellation date, the funds guaranteed under such mechanism must be paid prior to cancellation in accordance with ¶ .

c. If, upon issuance of a notice of implementation of a work takeover, either: (1) EPA is unable for any reason to promptly secure the resources guaranteed under any applicable financial assurance mechanism and/or related standby funding commitment, whether in cash or in kind, to continue and complete the Work; or (2) the financial assurance is provided under ¶ or , then EPA may demand an amount, as determined by EPA, sufficient to cover the cost of the remaining work to be performed. Respondents shall, within __ days of such demand, pay the amount demanded as directed by EPA.

d. Any amounts required to be paid under this ¶ shall be, as directed by EPA: (i) paid to EPA in order to facilitate the completion of the work by EPA or by another person; or (ii) deposited into an interest-bearing account, established at a duly chartered bank or trust company that is insured by the FDIC, in order to facilitate the completion of the work by another person.

Pre-Decisional Draft

56. Modification of Amount, Form, or Terms of Financial Assurance. Respondents may submit, on any anniversary of the Effective Date or at any other time agreed to by the parties, a request to reduce the amount, or change the form or terms, of the financial assurance mechanism. Any such request must be submitted to EPA in accordance with ¶ , and must include an estimate of the cost of the remaining work, an explanation of the bases for the cost calculation, and a description of the proposed changes, if any, to the form or terms of the financial assurance. EPA will notify Respondents of its decision to approve or disapprove a requested reduction or change pursuant to this Paragraph. Respondents may reduce the amount of the financial assurance mechanism only in accordance with EPA's approval. Any decision made by EPA on a request submitted under this Paragraph to change the form or terms of a financial assurance mechanism shall be made in EPA's sole and unreviewable discretion, and such decision shall not be subject to challenge by Respondents. Within 30 days after receipt of EPA's approval of, or the agreement or decision resolving a dispute relating to, the requested modifications pursuant to this Paragraph, Respondents shall submit to EPA documentation of the reduced, revised, or alternative financial assurance mechanism in accordance with ¶ .

57. Release, Cancellation, or Discontinuation of Financial Assurance. Respondents may release, cancel, or discontinue any financial assurance provided under this Order only: (a) if EPA issues a termination of the Order pursuant to Paragraph 67.

58. Nothing in this Order shall be construed as prohibiting, altering or in any way eliminating the ability of EPA to seek any other remedies or sanctions available by virtue of any Respondent's violations of this Order or of the statutes and regulations upon which this Order is based or for any Respondent's violation of any applicable provision of law.

59. This Order shall not relieve Respondents of their obligation to comply with all applicable provisions of federal, state or local law, nor shall it be construed to be a ruling on, or determination of, any issue related to any federal, state or local permit.

60. Nothing in this Order is intended to nor shall be construed to operate in any way to resolve any criminal liability of Chemours or DuPont. Compliance with this Order shall not be a defense to any actions subsequently commenced for any violation of federal laws and regulations administered by EPA, and it is the responsibility of Chemours and DuPont to comply with such laws and regulations. EPA reserves the right to undertake action against any person, including Chemours and/or DuPont, in response to any condition which EPA determines may present an imminent and substantial endangerment to the public health, public welfare or the environment.

61. The undersigned representatives of Respondents certify that they are fully authorized to enter into the terms and conditions of this Order and to execute and legally bind Chemours and DuPont to it.

62. Pursuant to Section 1431(b) of the SDWA, 42 U.S.C. § 300i(b), and the Adjustment of Civil Monetary Penalties for Inflation, 40 C.F.R. Part 19, as revised (74 Fed. Reg. 626 (Jan.7, 2009)), the violation of any term of this Order, or failure or refusal to comply with this Order, may subject DuPont to a civil penalty not to exceed $16,500 for each day in which such violation occurs or failure to comply continues.

63. When Respondents know or should have known, by the exercise of due diligence, of an event that might delay completion of any requirement of this Order, Respondents shall provide notice to EPA, in writing, within two (2) business days after Chemours and/or DuPont knew, or in the exercise of due diligence, should have known, of such event. The notice shall describe in detail the basis for the delay, including whether it is a *force majeure* event, and describe the length of, precise cause(s) of, and measures taken or to be taken to prevent or minimize such delay. If EPA agrees that such event constitutes *force majeure*, EPA shall extend the time for performance of such requirement, in writing, to compensate for the delay caused by the *force majeure* event. Any Respondent's failure to notify in writing in accordance with this Paragraph shall render this Paragraph void and of no effect concerning such event. For purposes of this Order, *force majeure* is defined as an event arising from causes beyond the control of Chemours and/or DuPont, and any entity controlled by Chemours and/or DuPont, which delays or prevents the performance of any obligation under this Order. Unanticipated or increased costs or expenses associated with implementation of this Order and changed financial circumstances

shall not, in any event, be considered *force majeure* events. In addition, failure to apply for a required permit or approval or to provide in a timely manner all information required to obtain a permit or approval that is necessary to meet the requirements of this Order, or to obtain or approve contracts, shall not, in any event, constitute *force majeure* events.

64. This Consent Order may be executed in any number of counterpart originals, each of which shall be deemed to constitute an original agreement, and all of which shall constitute one agreement. The execution of one counterpart by any party shall have the same force and effect as if that party had signed all other counterparts.

65. All of the terms and conditions of this Order together comprise one agreement, and each of the terms and conditions is in consideration of all of the other terms and conditions. In the event that this Order is not executed by all of the signatories in identical form, or is not approved in such identical form by the Regional Administrators, then the entire Order shall be null and void.

66. The effective date of this Order is the date on which, after approval by the Regional Administrators, this Order is filed with the Regional Hearing Clerks of both Region III and Region V, if not, then on the same day.

67. This Order shall remain in effect until Respondents fulfill all obligations required by Paragraphs 43 and 44 herein, submit a written request to EPA to terminate this Order, and EPA approves such termination request.

Pre-Decisional Draft

68. This Order constitutes final agency action.

SO ORDERED:

_____ Date:_____

Shawn M. Garvin
Regional Administrator
U.S. Environmental Protection Agency,
Region III

_____ Date:_____

Robert Kaplan
Acting Regional Administrator
U.S. Environmental Protection Agency,
Region V

AGREED TO:

_____ Date:_____
The Chemours Company

_____ Date:_____
E.I. du Pont de Nemours and Company

Pre-Decisional Draft

Index to Administrative Record

1. Residential Well Location Map (Dec. 17, 2015).
2. List of residences above HA value (__ ppb) detection but below 0.40 ppb.

Pre-Decisional Draft

United States Environmental Protection Agency

The Third Unregulated Contaminant Monitoring Rule (UCMR 3): Data Summary

January 20

EPA uses the Unregulated Contaminant Monitoring Rule (UCMR) program to collect data for contaminants suspected to be present in drinking water, but that do not have health-based standards set under the Safe Drinking Water Act (SDWA). Every five years EPA develops a new list of UCMR contaminants, largely based on the Contaminant Candidate List (CCL). The SDWA Amendments of 1996 provide for:

- Monitoring no more than 30 contaminants per 5-year cycle
- Monitoring only a representative sample of public water systems serving less than or equal to 10,000 people
- Storing analytical results in a National Contaminant Occurrence Database (NCOD)

This dataset represents the ninth NCOD release of analytical results for UCMR 3. Updates will occur approximately quarterly and EPA anticipates that additional reference material will be made available to assist with the assessment of the UCMR 3 data. For more information about UCMR 3, please visit our website: http://water.epa.gov/lawsregs/rulesregs/sdwa/ucmr/ucmr3/index.cfm. Information regarding many of the UCMR 3 contaminants (including a description of their use) may also be found at the CCL website: http://water.epa.gov/scitech/drinkingwater/dws/ccl/ccl3.cfm#chemical.

UCMR 3 Data Considerations

- This dataset is not complete. UCMR 3 monitoring occurred through December 2015, and data are expected to be reported to EPA through the summer of 2016.
- Data are added and possibly removed or updated over the course of this reporting cycle. These results are subject to change following further review by the analytical laboratory, the public water system, the State and EPA.
- Data are presented as method-specific text files (UCMR3_200_8.txt, UCMR3_218_7.txt, UCMR3_300_1.txt, UCMR3_522.txt, UCMR3_524_3.txt, UCMR3_537.txt, UCMR3_539.txt, EPA_1615A, EPA_1615B, EPA_1615C, EPA_1615D, EPA_1615E, SM_9223B, ASTM_D6503_99, SM_9218, EPA_1602), one text file containing disinfectant residual type (UCMR3_DRT.txt), one text file containing the U.S. Postal Service Zip Code(s) for all areas served by a PWS (UCMR3_ZipCodes.txt) and one text file containing all UCMR 3 data to date (UCMR3_All.txt).
- These text files are tab delimited and have no text qualifier. Field names are included in the first row of each file.
- If you wish to perform additional data analyses, EPA suggests you import each field into your choice of software as text. Some of the IDs can be misinterpreted as long integer field types when they actually contain alpha characters.
- Samples collected at the maximum residence time in the distribution system (MR) are required to be analyzed for metals (including chromium-6) and chlorate.
- Water systems monitoring for Method 300.1 (chlorate) report disinfectant types.
- Population categories are based on retail population as indicated by the Safe Drinking Water Information System (Federal) (SDWIS/FED) as of December 31, 2010.
- In addition to reporting occurrence data for UCMR 3 target analytes, EPA tasked its small-system contract-support laboratories with reporting results for sec-butylbenzene, n-propylbenzene, tellurium, germanium and manganese. These additional unregulated analytes are within the scope of the methods already being performed for the UCMR analytes.

UCMR 3 Data Field Names and Definitions

Field Name	Definition
PWSID	Public Water System Identification Code, 9-character identification code (Begins with the standard 2-character postal State abbreviation or Region code, and the remaining seven numbers are unique to each PWS in the state)
PWSName	Name of the Public Water System (PWS)
Size	Size category of the PWS for UCMR, based on retail population as of December 31, 2010
	S: ≤ 10,000
	L: > 10,000
FacilityID	Public Water System Facility Identification Code, 5-digit identification code
FacilityName	Name of the facility at the PWS
FacilityWaterType	Source of water at the facility
	SW: Surface water
	GW: Ground water
	GU: Ground water under the direct influence of surface water
	MX: Any combination of: SW, GW and GU
SamplePointID	Identification code for each sample point location in the PWS
SamplePointName	Name of the sample point for every sample point ID at a PWS
SamplePointType	Sampling Point Type Code
	EP: Entry point to the distribution system
	MR: Distribution system at maximum residence time
AssociatedFacilityID	The facility ID of the associated DS/MRT
AssociatedSamplePointID	The sample point ID of the associated DS/MRT

Field Name	Definition
Disinfectant Type	CLGA: Gaseous Chlorine
	CLOF: Offsite Generated Hypochlorite (stored as liquid)
	CLON: Onsite Generated Hypochlorite (no storage)
	CAGC: Chloramine (formed from gaseous chlorine)
	CAOF: Chloramine (formed from offsite hypochlorite)
	CAON: Chloramine (formed from onsite hypochlorite)
	CLDO: Chlorine Dioxide
	OZON: Ozone
	ULVL: Ultraviolet Light
	OTHD: All other types of disinfectant
	NODU: No Disinfectant Used
CollectionDate	Date of sample collection (month, day, year)
SampleID	Identification code for each sample, as defined by the laboratory
Contaminant	Unregulated contaminant being analyzed in UCMR 3
MRL	Minimum Reporting Level defined by UCMR 3
MethodID	Identification code of the analytical method
AnalyticalResultsSign	Less than (<) the minimum reporting level (MRL) or equal to (=) a numeric value at or above the MRL
AnalyticalResultValue	Numeric value of the analytical result, null values represent less than MRL
SampleEventCode	Identification code for each sample event. Includes sample event one (SE1), sample event two (SE2), sample event three (SE3), and sample event four (SE4).

Field Name	Definition
MonitoringRequirement	AM: Assessment Monitoring (List 1)
	SS: Screening Survey (List 2)
	PST: Pre-Screen Testing (List 3)
Region	EPA Region: States
	1: CT, ME, MA, NH, RI, VT
	2: NJ, NY, PR (Puerto Rico), VI (Virgin Islands)
	3: DE, DC, MD, PA, VA, WV
	4: AL, FL, GA, KY, MS, NC, SC, TN
	5: IL, IN, MI, MN, OH, WI
	6: AR, LA, NM, OK, TX
	7: IA, KS, MO, NE
	8: CO, MT, ND, SD, UT, WY
	9: AZ, CA, HI, NV, AS (American Samoa), GU (Guam), MP (Northern Marianas Islands), NN (Navajo Nation)
	10: AK, ID, OR, WA
State	State abbreviation
ZipCode	U.S. Postal Service zip code(s) for all areas being served water by a PWS

UCMR 3 Chemical Contaminants and Methods

Contaminant	Contaminant Full Name	CAS[1] Number	Method ID	Method Name	Monitoring Requirement
1,2,3-trichloropropane	1,2,3-trichloropropane	96-18-4	524.3	Volatile Organic Compounds	AM
1,3-butadiene	1,3-butadiene	106-99-0	524.3	Volatile Organic Compounds	AM
Chloromethane	methyl chloride	74-87-3	524.3	Volatile Organic Compounds	AM
1,1-dichloroethane	1,1-dichloroethane	75-34-3	524.3	Volatile Organic Compounds	AM
Bromomethane	methyl bromide	74-83-9	524.3	Volatile Organic Compounds	AM
HCFC-22	chlorodifluoromethane	75-45-6	524.3	Volatile Organic Compounds	AM
Halon 1011	bromochloromethane	74-97-5	524.3	Volatile Organic Compounds	AM
1,4-dioxane	1,4-dioxane	123-91-1	522	Synthetic Organic Compound	AM
Vanadium	vanadium	7440-62-2	200.8	Metals	AM
Molybdenum	molybdenum	7439-98-7	200.8	Metals	AM
Cobalt	Cobalt	7440-48-4	200.8	Metals	AM
Strontium	Strontium	7440-24-6	200.8	Metals	AM
Chromium	total chromium	N/A	200.8	Metals	AM
Chromium-6	chromium-6	18540-29-9	218.7	Chromium-6	AM
Chlorate	Chlorate	14866-68-3	300.1	Oxyhalide Anion	AM
PFOS	perfluorooctanesulfonic acid	1763-23-1	537	Perfluorinated Compounds	AM
PFOA	perfluorooctanoic acid	335-67-1	537	Perfluorinated Compounds	AM
PFNA	perfluorononanoic acid	375-95-1	537	Perfluorinated Compounds	AM
PFHxS	perfluorohexanesulfonic acid	355-46-4	537	Perfluorinated Compounds	AM
PFHpA	perfluoroheptanoic acid	375-85-9	537	Perfluorinated Compounds	AM
PFBS	perfluorobutanesulfonic acid	375-73-5	537	Perfluorinated Compounds	AM
17β-estradiol	estradiol	50-28-2	539	Hormones	SS
17α-ethynylestradiol	ethinyl estradiol	57-63-6	539	Hormones	SS
Estriol	16-α-hydroxyestradiol	50-27-1	539	Hormones	SS
Equilin	Equilin	474-86-2	539	Hormones	SS
Estrone	Estrone	53-16-7	539	Hormones	SS
Testosterone	testosterone	58-22-0	539	Hormones	SS
4-androstene-3,17-dione	4-androstene-3,17-dione	63-05-8	539	Hormones	SS

[1]Chemical Abstract Service

UCMR 3 Microbiological Contaminants and Methods

Contaminant	Method ID	Method Name	Monitoring Requirement
Enteroviruses	EPA 1615A	Enterovirus cell culture	PST
Enteroviruses	EPA 1615B	Enterovirus RT-qPCR	PST
Noroviruses	EPA 1615C	Norovirus genogroup I with RT-qPCR primer set A	PST
Noroviruses	EPA 1615D	Norovirus genogroup I with RT-qPCR primer set B	PST
Noroviruses	EPA 1615E	Noroviruses genogroup II	PST
Total coliforms	SM 9223B	Colilert®	PST
E.coli	SM 9223B	Colilert®	PST
Enterococci	ASTM D6503-99	Enterolert®	PST
Aerobic spores	SM 9218	Aerobic endospores	PST
Somatic phage	EPA 1602	Bacteriophage	PST
Male specific phage	EPA 1602	Bacteriophage	PST

UCMR 3 Reference Concentrations for Chemical Contaminants

Under the current cycle of the Unregulated Contaminant Monitoring Rule (UCMR 3) chemicals are being studied at levels that are often significantly below those in prior UCMR cycles. Importantly, UCMR 3 minimum reporting levels (MRLs) were established based on the capability of the analytical method, not based on a level established as "significant" or "harmful." In fact, the UCMR 3 MRLs are often below current "health reference levels" (to the extent that HRLs have been established).

Results of UCMR 3 measurements should be interpreted accordingly. The detection of a UCMR 3 contaminant above the MRL does not represent cause for concern, in and of itself. Rather, the implications of the detection should be judged considering health effects information (which is often still under development or being refined for unregulated contaminants).

The intent of the following table is to identify draft UCMR reference concentrations, where possible, to provide context around the detection of a particular UCMR contaminant above the MRL. The draft reference concentration does not represent an "action level" (EPA requires no particular action[1,2] based simply on the fact that UCMR monitoring results exceed draft reference concentrations), nor should the draft reference concentration be interpreted as any indication of an Agency intent to establish a future drinking water regulation for the contaminant at this or any other level. Decisions as to whether or not to regulate the contaminant in drinking water will continue to be made following the Agency's Regulatory Determination process: [http://water.epa.gov/scitech/drinkingwater/dws/ccl/index.cfm].

The following key principles guided the development of the table:

(1) The reference concentrations are based on publically-available health information found in the following EPA resources: 2012 Drinking Water Standards and Health Advisories, the CCL 3 Contaminant Information Sheets, the Human Health Benchmark for Pesticides (HHBPs), the Integrated Information Risk System (IRIS), or the 2014 Preliminary Regulatory Determinations for Contaminants on CCL 3. The primary/secondary sources of health information vary with respect to scientific rigor from health assessment to single studies and are cited in the table.
(2) If health information was available from more than one of the EPA resources listed above, the most recent health information was used for the draft reference concentrations.
(3) Where both cancer and non-cancer draft reference concentrations existed, the lower (more conservative) of the two concentrations was used. For chemicals with reference concentrations based on a cancer endpoint, the table presents a range of values associated with 10^{-6} to 10^{-4} cancer risk. For chemicals with reference concentrations based on a non-cancer endpoint, the duration of exposure (short-term, intermediate/long-term, chronic) of the toxicity factor (e.g. Reference Dose) used as the basis for the reference concentration is shown.

Recognizing that additional health effects information will become available over time, EPA will periodically update the following table. Those attempting to assess UCMR occurrence data are encouraged to visit EPA's website for the most recent information.

[1] Consumer Confidence Report (CCR) and Public Notification (PN) reporting requirements (see 40 CFR 141.153(d) and 141.207, respectively) apply to public water systems; CCR requires particular reporting based on measurements relative to the UCMR method reporting limits (MRLs) defined in 40 CFR 141.40.
[2]States may establish requirements for drinking water contaminants not yet regulated by EPA, and those requirements may be based on State-established levels that differ from EPA's reference concentrations. Public Water Systems are responsible for being aware of and complying with their State's requirements, if any.

Contaminant	***MRL (µg/L)***	***Reference Concentration (µg/L)***	***Reference Concentration based on a Cancer Endpoint (Y/N)***	***EPA Reference(s)***
Cobalt[1]	1	70	N (intermediate exposure)	CCL 3 Contaminant Information Sheets
Molybdenum[2]	1	40	N (chronic exposure)	2012 Edition of the Health Advisories Table
Strontium[3]	0.3	1,500	N (chronic exposure)	Federal Register Notice for the Preliminary Regulatory Determinations for Contaminants on CCL 3
Vanadium[1,4]	0.2	21	N (intermediate exposure)	CCL 3 Contaminant Information Sheets
Chromium (Total)	0.2	100	N (chronic exposure)	The MCL for the National Primary Drinking Water Regulation
Chromium-6[1]	0.03	NA		
Chlorate	20	210	N (chronic exposure)	CCL 3 Contaminant Information Sheets
1,4-dioxane[5]	0.07	0.35 to 35	Y	2012 Edition of the Health Advisories Table
1,1-dichloroethane[5]	0.03	6.14 to 614	Y	CCL 3 Contaminant Information Sheets
1,2,3-trichloropropane[5,6,7]	0.03	0.0004 to 0.04	Y	2009 IRIS Assessment
1,3-butadiene[5,6]	0.1	0.0103 to 1.03	Y	CCL 3 Contaminant Information Sheets

[1] The contaminant is on the IRIS 20□2 Agenda for either a new assessment or an updated assessment (Federal Register Notice May 7, 20□2).
[2] The 20□2 Edition of the Health Advisories Table and the CCL 3 Contaminant Information Sheets (35 µg/L) have slightly different numbers due to rounding.
[3] The reference concentration has been updated based on the HRL cited in the preliminary regulatory determination for strontium [Docket No. EPAHQ-OW-20□2-0□55].
[4] The ATSDR, □992 used for the CCL 3 Contaminant Information Sheets is no longer publically available and has been replaced by a new assessment (ATSDR, 20□3).
The minimum risk level (RfD equivalent) was 0.003 mg/kg/day for minor renal effects in an animal study (ATSDR, □992) compared to 0.0□ mg/kg/day for lack of minor effects in blood pressure, body weight, and hematological parameters in a human study with a □2 weeks exposure (ATSDR, 20□3).
[5] Reference Concentration range based on cancer risk of □$0^{-□}$ to □0^{-4}.
[□] □$0^{-□}$ cancer risk < MRL < □0^{-4} cancer risk.
[7] To derive the reference concentration, age dependent adjustment factors were applied to the IRIS oral slope factor of 30 per mg/kg-day (calculated using adult exposure data) to address presumed early-life susceptibility for this chemical (per www.epa.gov/cancerguidelines/).

Contaminant	***MRL (µg/L)***	***Reference Concentration (µg/L)***	***Reference Concentration based on a Cancer Endpoint (Y/N)***	***EPA Reference(s)***
HCFC-22 (chlorodifluoromethane)[8]	0.08	NA		
Chloromethane (methyl chloride)[5]	0.2	2.69 to 269	Y	CCL 3 Contaminant Information Sheets
Halon 1011 (bromochloromethane)[9]	0.06	90	N (chronic exposure)	2012 Edition of the Health Advisories Table
Bromomethane (methyl bromide)	0.2	140	N (chronic exposure)	Human Health Benchmark for Pesticides (HHBPs)
PFBS	0.09	NA		
PFHpA	0.01	NA		
PFHxS	0.03	NA		
PFNA	0.02	NA		
PFOS	0.04	0.2	N (short-term exposure)	2012 Edition of the Health Advisories Table
PFOA	0.02	0.4	N (short-term exposure)	2012 Edition of the Health Advisories Table
17α-ethynylestradiol (ethinyl estradiol)[10]	0.0009	0.035	N (chronic exposure)	CCL 3 Contaminant Information Sheets
17β-estradiol (estradiol)[5]	0.0004	0.0009 to 0.09	Y	CCL 3 Contaminant Information Sheets
Equilin	0.004	0.35	N (chronic exposure)	CCL 3 Contaminant Information Sheets
Estriol (16-α-hydroxyestradiol)	0.0008	0.35	N (chronic exposure)	CCL 3 Contaminant Information Sheets
Estrone	0.002	0.35	N (chronic exposure)	CCL 3 Contaminant Information Sheets
4-androstene-3,17-dione	0.0003	NA		
Testosterone	0.0001	NA		

[8] The CCL 3 Contaminant Information Sheets provide a reference level of 3[illegible].5 µg/L; the number is based on a single LOAEL from a [illegible]983 study.

[9] The 20[illegible]2 Edition of the Health Advisories Table and the CCL 3 Contaminant Information Sheets (70 µg/L) have slightly different numbers due to rounding.

[10] This corrects the CCL 3 Contaminant Information Sheets reference level (originally listed as 0.28 µg/L).

Terms

a) UCMR Draft Reference Concentration = The reference concentrations are based on publically-available health information found in the following EPA resources: 2012 Drinking Water Standards and Health Advisories, the CCL 3 Contaminant Information Sheets, the Human Health Benchmark for Pesticides (HHBPs), or the 2014 Preliminary Regulatory Determinations for Contaminants on CCL 3. The primary/secondary sources of health information vary with respect to scientific rigor from health assessment to single studies. Many of the contaminants are currently under regulatory review or development and are subject to change as new health assessments are completed.

b) MRL = UCMR Minimum Reporting Level. *[Note that the Agency for Toxic Substances & Disease Registry (ATSDR) uses the term "MRL" for a different purpose (i.e., to describe "Minimal Risk Levels"). The UCMR term and the ATSDR term have no relationship to each other.]*

c) HRLs = Health Reference Levels. HRLs are not final determinations about the level of a contaminant in drinking water that is necessary to protect any particular population and are derived prior to development of a complete exposure assessment. HRLs are risk derived concentrations against which to evaluate the occurrence data to determine if contaminants occur at levels of potential public health concern.

d) MCL = Maximum Contaminant Level. The highest level of a contaminant allowed in drinking water. MCLs are enforceable standards.

e) Cancer Risk of 10^{-6} to 10^{-4} = the concentration of a contaminant in drinking water corresponding to an excess estimated lifetime cancer risk of one-in-a-million ($1x\ 10^{-6}$) to one-in-ten-thousand (1×10^{-4}). The 2012 Drinking Water Standards and Health Advisories provide the cancer risk at 1×10^{-4}. The CCL 3 Contaminant Information Sheets provide the cancer risk at $1x\ 10^{-6}$.

f) LOAEL = Lowest Observed Adverse Effect Level

g) NA = Not Available

h) Short-term = Typically refers to animal toxicological studies with an exposure duration of days to weeks.

i) Intermediate/Longer-term = Typically refers to animal toxicological studies with an exposure duration of weeks to months.

j) Chronic = Typically refers to animal toxicological studies with an exposure duration of months to years; representing a lifetime exposure in humans.

References

k) 2012 Drinking Water Standards and Health Advisories
l) CCL 3 Contaminant Information Sheets
m) Human Health Benchmark for Pesticides (HHBPs)
n) Announcement of Preliminary Regulatory Determinations for Contaminants on the Third Drinking Water Contaminant Candidate List
o) Integrated Risk Information System (IRIS)

January 2016 UCMR 3 Data Summary for Chemical Contaminants

Contaminant	MRL (µg/L)	Reference Concentration (µg/L)	Total number of results	Number of results ≥MRL	Number of results >Reference Concentration	% of total results >Reference Concentration	Total number of PWSs with results	Number of PWSs with results ≥MRL	Number of PWSs with results >Reference Concentration	% of PWSs with results >Reference Concentration
1,2,3-trichloropropane	0.03	0.0004 / 0.04[1]	34,749	243	243 / 186[1]	0.7% / 0.5%[1]	4,775	64	64 / 53[1]	1.3% / 1.1%[1]
1,3-butadiene	0.1	0.0103 / 1.03[1]	34,749	1	1 / 0[1]	0.003% / 0%[1]	4,775	1	1 / 0[1]	0.02% / 0%[1]
Chloromethane	0.2	2.69 / 269[1]	34,747	261	18 / 0[1]	0.05% / 0%[1]	4,775	130	7 / 0[1]	0.1% / 0%[1]
1,1-dichloroethane	0.03	6.14 / 614[1]	34,747	804	1 / 0[1]	0.003% / 0%[1]	4,775	237	1 / 0[1]	0.02% / 0%[1]
Bromomethane	0.2	140	34,748	110	0	0%	4,775	47	0	0%
HCFC-22	0.08	NA	34,749	799	- -	- -	4,775	278	- -	- -
Halon 1011	0.06	90	34,749	619	0	0%	4,775	297	0	0%
1,4-dioxane	0.07	0.35 / 35[1]	34,684	4,035	1,054 / 0[1]	3% / 0%[1]	4,773	1,041	333/ 0[1]	7% / 0%[1]
Vanadium	0.2	21	59,602	35,776	1,640	2.8%	4,789	3,526	159	3.3%
Molybdenum	1	40	59,607	24,308	135	0.2%	4,789	2,468	35	0.7%
Cobalt	1	70	59,590	809	3	0.005%	4,789	236	3	0.06%
Strontium	0.3	1,500	59,526	59,328	1,643	2.8%	4,789	4,789	275	5.7%
Chromium	0.2	100	59,504	30,171	1	0.002%	4,789	3,558	1	0.02%
Chromium-6	0.03	NA	59,442	44,884	- -	- -	4,787	4,276	- -	- -
Chlorate	20	210	59,357	32,746	9,248	15.6%	4,776	3,281	1,803	37.8%
PFOS	0.04	0.2	35,060	273	33	0.09%	4,788	91	17	0.4%
PFOA	0.02	0.4	35,060	345	0	0%	4,788	107	0	0%
PFNA	0.02	NA	35,060	19	- -	- -	4,788	14	- -	- -
PFHxS	0.03	NA	35,060	195	- -	- -	4,788	54	- -	- -
PFHpA	0.01	NA	35,060	225	- -	- -	4,788	82	- -	- -
PFBS	0.09	NA	35,060	14	- -	- -	4,788	6	- -	- -
17β-estradiol	0.0004	0.0009 / 0.09[1]	10,729	3	1 / 0[1]	0.009% / 0%[1]	1,146	1	1 / 0[1]	0.09% / 0%[1]
17α-ethynylestradiol	0.0009	0.035	10,730	4	0	0%	1,146	4	0	0%
Estriol	0.0008	0.35	10,729	1	0	0%	1,146	1	0	0%
Equilin	0.004	0.35	10,730	0	0	0%	1,146	0	0	0%
Estrone	0.002	0.35	10,730	0	0	0%	1,146	0	0	0%
Testosterone	0.0001	NA	10,729	58	- -	- -	1,146	52	- -	- -
4-androstene-3,17-dione	0.0003	NA	10,730	89	- -	- -	1,146	69	- -	- -

[1]Where two reference concentrations are listed, the first number is associated with a 10^{-6} cancer risk; the second number a 10^{-4} cancer risk.
Where two results are presented the first number is associated with the first reference concentration; the second number is associated with the second reference concentration.

January 2016 UCMR 3 Data Summary for Microbiological Contaminants

Contaminant	MRL	Unit	Total number of results	Number of results ≥MRL	Total number of PWSs with results	Number of PWSs with results ≥MRL
Aerobic spores	1	SFO[1]/100 mL[2]	794	219	772	216
E. coli	1	MPN[3]/100 mL	792	4	770	4
Enterococci	1	MPN/100 mL	791	40	769	40
Enteroviruses (cell culture)	0.002	MPN/L[4]	790	2	768	2
Enteroviruses (RT-qPCR[5])	0.398	GC[6]/L	790	6	768	6
Male specific phage	1	PFU[7]/100 mL	776	11	754	11
Noroviruses GIA[8]	0.398	GC/L	790	2	768	2
Noroviruses GIB[9]	0.398	GC/L	790	1	768	1
Noroviruses GII[10]	0.398	GC/L	790	4	768	4
Somatic phage	1	PFU/100 mL	776	5	754	5
Total coliforms	1	MPN/100 mL	792	41	770	40

[1]SFO = Spore Forming Units
[2]mL = milliliters
[3]MPN = Most Probable Number
[4]L = liters
[5]RT-qPCR = Reverse Transcription-Polymerase Chain Reaction
[6]GC = Genomic Copies
[7]PFU = Plaque Forming Units
[8]Noroviruses GIA = qPCR analysis of Norovirus genogroup I with RT-qPCR primer set A
[9]Noroviruses GIB = qPCR analysis of Norovirus genogroup I with RT-qPCR primer set B
[10]Noroviruses GII = qPCR analysis of Norovirus genogroup II

UCMR 3 Minimum Reporting Levels for Microbiological Contaminants

Under UCMR 3 microbe analytical results are reported as “below”, “at” or “above” MRL. UCMR 3 MRLs were established based on the capability of the analytical method.

It is important to note that microbial contamination can be transient in nature and microbial detections under UCMR 3 should be interpreted in the context of the time samples were collected. However, the presence of any UCMR 3 microbe indicates a potential vulnerability of the PWS to contamination.

United States Environmental Protection Agency

The Third Unregulated Contaminant Monitoring Rule (UCMR 3): Data Summary

October 2015

EPA uses the Unregulated Contaminant Monitoring Rule (UCMR) program to collect data for contaminants suspected to be present in drinking water, but that do not have health-based standards set under the Safe Drinking Water Act (SDWA). Every five years EPA develops a new list of UCMR contaminants, largely based on the Contaminant Candidate List (CCL). The SDWA Amendments of 1996 provide for:

- Monitoring no more than 30 contaminants per 5-year cycle
- Monitoring only a representative sample of public water systems serving less than or equal to 10,000 people
- Storing analytical results in a National Contaminant Occurrence Database (NCOD)

This dataset represents the eighth NCOD release of analytical results for UCMR 3. Updates will occur approximately quarterly and EPA anticipates that additional reference material will be made available to assist with the assessment of the UCMR 3 data. For more information about UCMR 3, please visit our website: http://water.epa.gov/lawsregs/rulesregs/sdwa/ucmr/ucmr3/index.cfm. Information regarding many of the UCMR 3 contaminants (including a description of their use) may also be found at the CCL website: http://water.epa.gov/scitech/drinkingwater/dws/ccl/ccl3.cfm#chemical.

UCMR 3 Data Considerations

- This dataset is not complete. UCMR 3 monitoring occurs through December 2015, and data are expected to be reported to EPA through the summer of 2016.
- Data are added and possibly removed or updated over the course of this reporting cycle. These results are subject to change following further review by the analytical laboratory, the public water system, the State and EPA.
- Data are presented as method-specific text files (UCMR3_200_8.txt, UCMR3_218_7.txt, UCMR3_300_1.txt, UCMR3_522.txt, UCMR3_524_3.txt, UCMR3_537.txt, UCMR3_539.txt, EPA_1615A, EPA_1615B, EPA_1615C, EPA_1615D, EPA_1615E, SM_9223B, ASTM_D6503_99, SM_9218, EPA_1602), one text file containing disinfectant residual type (UCMR3_DRT.txt), one text file containing the U.S. Postal Service Zip Code(s) for all areas served by a PWS (UCMR3_ZipCodes.txt) and one text file containing all UCMR 3 data to date (UCMR3_All.txt).
- These text files are tab delimited and have no text qualifier. Field names are included in the first row of each file.
- If you wish to perform additional data analyses, EPA suggests you import each field into your choice of software as text. Some of the IDs can be misinterpreted as long integer field types when they actually contain alpha characters.
- Samples collected at the maximum residence time in the distribution system (MR) are required to be analyzed for metals (including chromium-6) and chlorate.
- Water systems monitoring for Method 300.1 (chlorate) report disinfectant types.
- Population categories are based on retail population as indicated by the Safe Drinking Water Information System (Federal) (SDWIS/FED) as of December 31, 2010.
- In addition to reporting occurrence data for UCMR 3 target analytes, EPA tasked its small-system contract-support laboratories with reporting results for sec-butylbenzene, n-propylbenzene, tellurium, germanium and manganese. These additional unregulated analytes are within the scope of the methods already being performed for the UCMR analytes.

UCMR 3 Data Field Names and Definitions

Field Name	Definition
PWSID	Public Water System Identification Code, 9-character identification code (Begins with the standard 2-character postal State abbreviation or Region code, and the remaining seven numbers are unique to each PWS in the state)
PWSName	Name of the Public Water System (PWS)
Size	Size category of the PWS for UCMR, based on retail population as of December 31, 2010
	S: ≤ 10,000
	L: > 10,000
FacilityID	Public Water System Facility Identification Code, 5-digit identification code
FacilityName	Name of the facility at the PWS
FacilityWaterType	Source of water at the facility
	SW: Surface water
	GW: Ground water
	GU: Ground water under the direct influence of surface water
	MX: Any combination of: SW, GW and GU
SamplePointID	Identification code for each sample point location in the PWS
SamplePointName	Name of the sample point for every sample point ID at a PWS
SamplePointType	Sampling Point Type Code
	EP: Entry point to the distribution system
	MR: Distribution system at maximum residence time
AssociatedFacilityID	The facility ID of the associated DS/MRT
AssociatedSamplePointID	The sample point ID of the associated DS/MRT

Field Name	Definition
Disinfectant Type	CLGA: Gaseous Chlorine
	CLOF: Offsite Generated Hypochlorite (stored as liquid)
	CLON: Onsite Generated Hypochlorite (no storage)
	CAGC: Chloramine (formed from gaseous chlorine)
	CAOF: Chloramine (formed from offsite hypochlorite)
	CAON: Chloramine (formed from onsite hypochlorite)
	CLDO: Chlorine Dioxide
	OZON: Ozone
	ULVL: Ultraviolet Light
	OTHD: All other types of disinfectant
	NODU: No Disinfectant Used
CollectionDate	Date of sample collection (month, day, year)
SampleID	Identification code for each sample, as defined by the laboratory
Contaminant	Unregulated contaminant being analyzed in UCMR 3
MRL	Minimum Reporting Level defined by UCMR 3
MethodID	Identification code of the analytical method
AnalyticalResultsSign	Less than (<) the minimum reporting level (MRL) or equal to (=) a numeric value at or above the MRL
AnalyticalResultValue	Numeric value of the analytical result, null values represent less than MRL
SampleEventCode	Identification code for each sample event. Includes sample event one (SE1), sample event two (SE2), sample event three (SE3), and sample event four (SE4).

Field Name	Definition
MonitoringRequirement	AM: Assessment Monitoring (List 1)
	SS: Screening Survey (List 2)
	PST: Pre-Screen Testing (List 3)
Region	EPA Region: States
	1: CT, ME, MA, NH, RI, VT
	2: NJ, NY, PR (Puerto Rico), VI (Virgin Islands)
	3: DE, DC, MD, PA, VA, WV
	4: AL, FL, GA, KY, MS, NC, SC, TN
	5: IL, IN, MI, MN, OH, WI
	6: AR, LA, NM, OK, TX
	7: IA, KS, MO, NE
	8: CO, MT, ND, SD, UT, WY
	9: AZ, CA, HI, NV, AS (American Samoa), GU (Guam), MP (Northern Marianas Islands), NN (Navajo Nation)
	10: AK, ID, OR, WA
State	State abbreviation
ZipCode	U.S. Postal Service zip code(s) for all areas being served water by a PWS

UCMR 3 Chemical Contaminants and Methods

Contaminant	Contaminant Full Name	CAS[1] Number	Method ID	Method Name	Monitoring Requirement
1,2,3-trichloropropane	1,2,3-trichloropropane	96-18-4	524.3	Volatile Organic Compounds	AM
1,3-butadiene	1,3-butadiene	106-99-0	524.3	Volatile Organic Compounds	AM
Chloromethane	methyl chloride	74-87-3	524.3	Volatile Organic Compounds	AM
1,1-dichloroethane	1,1-dichloroethane	75-34-3	524.3	Volatile Organic Compounds	AM
Bromomethane	methyl bromide	74-83-9	524.3	Volatile Organic Compounds	AM
HCFC-22	chlorodifluoromethane	75-45-6	524.3	Volatile Organic Compounds	AM
Halon 1011	bromochloromethane	74-97-5	524.3	Volatile Organic Compounds	AM
1,4-dioxane	1,4-dioxane	123-91-1	522	Synthetic Organic Compound	AM
Vanadium	vanadium	7440-62-2	200.8	Metals	AM
Molybdenum	molybdenum	7439-98-7	200.8	Metals	AM
Cobalt	Cobalt	7440-48-4	200.8	Metals	AM
Strontium	Strontium	7440-24-6	200.8	Metals	AM
Chromium	total chromium	N/A	200.8	Metals	AM
Chromium-6	chromium-6	18540-29-9	218.7	Chromium-6	AM
Chlorate	Chlorate	14866-68-3	300.1	Oxyhalide Anion	AM
PFOS	perfluorooctanesulfonic acid	1763-23-1	537	Perfluorinated Compounds	AM
PFOA	perfluorooctanoic acid	335-67-1	537	Perfluorinated Compounds	AM
PFNA	perfluorononanoic acid	375-95-1	537	Perfluorinated Compounds	AM
PFHxS	perfluorohexanesulfonic acid	355-46-4	537	Perfluorinated Compounds	AM
PFHpA	perfluoroheptanoic acid	375-85-9	537	Perfluorinated Compounds	AM
PFBS	perfluorobutanesulfonic acid	375-73-5	537	Perfluorinated Compounds	AM
17β-estradiol	estradiol	50-28-2	539	Hormones	SS
17α-ethynylestradiol	ethinyl estradiol	57-63-6	539	Hormones	SS
Estriol	16-α-hydroxyestradiol	50-27-1	539	Hormones	SS
Equilin	Equilin	474-86-2	539	Hormones	SS
Estrone	Estrone	53-16-7	539	Hormones	SS
Testosterone	testosterone	58-22-0	539	Hormones	SS
4-androstene-3,17-dione	4-androstene-3,17-dione	63-05-8	539	Hormones	SS

[1]Chemical Abstract Service

UCMR 3 Microbiological Contaminants and Methods

Contaminant	Method ID	Method Name	Monitoring Requirement
Enteroviruses	EPA 1615A	Enterovirus cell culture	PST
Enteroviruses	EPA 1615B	Enterovirus RT-qPCR	PST
Noroviruses	EPA 1615C	Norovirus genogroup I with RT-qPCR primer set A	PST
Noroviruses	EPA 1615D	Norovirus genogroup I with RT-qPCR primer set B	PST
Noroviruses	EPA 1615E	Noroviruses genogroup II	PST
Total coliforms	SM 9223B	Colilert®	PST
E.coli	SM 9223B	Colilert®	PST
Enterococci	ASTM D6503-99	Enterolert®	PST
Aerobic spores	SM 9218	Aerobic endospores	PST
Somatic phage	EPA 1602	Bacteriophage	PST
Male specific phage	EPA 1602	Bacteriophage	PST

UCMR 3 Reference Concentrations for Chemical Contaminants

Under the current cycle of the Unregulated Contaminant Monitoring Rule (UCMR 3) chemicals are being studied at levels that are often significantly below those in prior UCMR cycles. Importantly, UCMR 3 minimum reporting levels (MRLs) were established based on the capability of the analytical method, not based on a level established as "significant" or "harmful." In fact, the UCMR 3 MRLs are often below current "health reference levels" (to the extent that HRLs have been established).

Results of UCMR 3 measurements should be interpreted accordingly. The detection of a UCMR 3 contaminant above the MRL does not represent cause for concern, in and of itself. Rather, the implications of the detection should be judged considering health effects information (which is often still under development or being refined for unregulated contaminants).

The intent of the following table is to identify draft UCMR reference concentrations, where possible, to provide context around the detection of a particular UCMR contaminant above the MRL. The draft reference concentration does not represent an "action level" (EPA requires no particular action[1,2] based simply on the fact that UCMR monitoring results exceed draft reference concentrations), nor should the draft reference concentration be interpreted as any indication of an Agency intent to establish a future drinking water regulation for the contaminant at this or any other level. Decisions as to whether or not to regulate the contaminant in drinking water will continue to be made following the Agency's Regulatory Determination process: [http://water.epa.gov/scitech/drinkingwater/dws/ccl/index.cfm].

The following key principles guided the development of the table:

(1) The reference concentrations are based on publically-available health information found in the following EPA resources: 2012 Drinking Water Standards and Health Advisories, the CCL 3 Contaminant Information Sheets, the Human Health Benchmark for Pesticides (HHBPs), the Integrated Information Risk System (IRIS), or the 2014 Preliminary Regulatory Determinations for Contaminants on CCL 3. The primary/secondary sources of health information vary with respect to scientific rigor from health assessment to single studies and are cited in the table.
(2) If health information was available from more than one of the EPA resources listed above, the most recent health information was used for the draft reference concentrations.
(3) Where both cancer and non-cancer draft reference concentrations existed, the lower (more conservative) of the two concentrations was used. For chemicals with reference concentrations based on a cancer endpoint, the table presents a range of values associated with 10^{-6} to 10^{-4} cancer risk. For chemicals with reference concentrations based on a non-cancer endpoint, the duration of exposure (short-term, intermediate/long-term, chronic) of the toxicity factor (e.g. Reference Dose) used as the basis for the reference concentration is shown.

Recognizing that additional health effects information will become available over time, EPA will periodically update the following table. Those attempting to assess UCMR occurrence data are encouraged to visit EPA's website for the most recent information.

[1] Consumer Confidence Report (CCR) and Public Notification (PN) reporting requirements (see 40 CFR 141.153(d) and 141.207, respectively) apply to public water systems; CCR requires particular reporting based on measurements relative to the UCMR method reporting limits (MRLs) defined in 40 CFR 141.40.
[2] States may establish requirements for drinking water contaminants not yet regulated by EPA, and those requirements may be based on State-established levels that differ from EPA's reference concentrations. Public Water Systems are responsible for being aware of and complying with their State's requirements, if any.

Contaminant	***MRL (µg/L)***	***Reference Concentration (µg/L)***	***Reference Concentration based on a Cancer Endpoint (Y/N)***	***EPA Reference(s)***
Cobalt[1]	1	70	N (intermediate exposure)	CCL 3 Contaminant Information Sheets
Molybdenum[2]	1	40	N (chronic exposure)	2012 Edition of the Health Advisories Table
Strontium[3]	0.3	1,500	N (chronic exposure)	Federal Register Notice for the Preliminary Regulatory Determinations for Contaminants on CCL 3
Vanadium[1,4]	0.2	21	N (intermediate exposure)	CCL 3 Contaminant Information Sheets
Chromium (Total)	0.2	100	N (chronic exposure)	The MCL for the National Primary Drinking Water Regulation
Chromium-6[1]	0.03	NA		
Chlorate	20	210	N (chronic exposure)	CCL 3 Contaminant Information Sheets
1,4-dioxane[5]	0.07	0.35 to 35	Y	2012 Edition of the Health Advisories Table
1,1-dichloroethane[5]	0.03	6.14 to 614	Y	CCL 3 Contaminant Information Sheets
1,2,3-trichloropropane[5,6,7]	0.03	0.0004 to 0.04	Y	2009 IRIS Assessment
1,3-butadiene[5,6]	0.1	0.0103 to 1.03	Y	CCL 3 Contaminant Information Sheets

[1] The contaminant is on the IRIS 2012 Agenda for either a new assessment or an updated assessment (Federal Register Notice May 7, 2012).
[2] The 2012 Edition of the Health Advisories Table and the CCL 3 Contaminant Information Sheets (35 µg/L) have slightly different numbers due to rounding.
[3] The reference concentration has been updated based on the HRL cited in the preliminary regulatory determination for strontium [Docket No. EPA-HQ-OW-2012-0155].
[4] The ATSDR, 1992 used for the CCL 3 Contaminant Information Sheets is no longer publically available and has been replaced by a new assessment (ATSDR, 2013).
The minimum risk level (RfD equivalent) was 0.003 mg/kg/day for minor renal effects in an animal study (ATSDR, 1992) compared to 0.01 mg/kg/day for lack of minor effects in blood pressure, body weight, and hematological parameters in a human study with a 12 weeks exposure (ATSDR, 2013).
[5] Reference Concentration range based on cancer risk of 10^{-6} to 10^{-4}.
[6] 10^{-6} cancer risk < MRL < 10^{-4} cancer risk.
[7] To derive the reference concentration, age dependent adjustment factors were applied to the IRIS oral slope factor of 30 per mg/kg-day (calculated using adult exposure data) to address presumed early-life susceptibility for this chemical (per www.epa.gov/cancerguidelines/).

Contaminant	***MRL (µg/L)***	***Reference Concentration (µg/L)***	***Reference Concentration based on a Cancer Endpoint (Y/N)***	***EPA Reference(s)***
HCFC-22 (chlorodifluoromethane) [8]	0.08	NA		
Chloromethane (methyl chloride)[5]	0.2	2.69 to 269	Y	CCL 3 Contaminant Information Sheets
Halon 1011 (bromochloromethane)[9]	0.06	90	N (chronic exposure)	2012 Edition of the Health Advisories Table
Bromomethane (methyl bromide)	0.2	140	N (chronic exposure)	Human Health Benchmark for Pesticides (HHBPs)
PFBS	0.09	NA		
PFHpA	0.01	NA		
PFHxS	0.03	NA		
PFNA	0.02	NA		
PFOS	0.04	0.2	N (short-term exposure)	2012 Edition of the Health Advisories Table
PFOA	0.02	0.4	N (short-term exposure)	2012 Edition of the Health Advisories Table
17α-ethynylestradiol (ethinyl estradiol)[10]	0.0009	0.035	N (chronic exposure)	CCL 3 Contaminant Information Sheets
17β-estradiol (estradiol)[5]	0.0004	0.0009 to 0.09	Y	CCL 3 Contaminant Information Sheets
Equilin	0.004	0.35	N (chronic exposure)	CCL 3 Contaminant Information Sheets
Estriol (16-α-hydroxyestradiol)	0.0008	0.35	N (chronic exposure)	CCL 3 Contaminant Information Sheets
Estrone	0.002	0.35	N (chronic exposure)	CCL 3 Contaminant Information Sheets
4-androstene-3,17-dione	0.0003	NA		
Testosterone	0.0001	NA		

[8] The CCL 3 Contaminant Information Sheets provide a reference level of 31.5 µg/L; the number is based on a single LOAEL from a 1983 study.
[9] The 2012 Edition of the Health Advisories Table and the CCL 3 Contaminant Information Sheets (70 µg/L) have slightly different numbers due to rounding.
[10] This corrects the CCL 3 Contaminant Information Sheets reference level (originally listed as 0.28 µg/L).

Terms

a) UCMR Draft Reference Concentration = The reference concentrations are based on publically-available health information found in the following EPA resources: 2012 Drinking Water Standards and Health Advisories, the CCL 3 Contaminant Information Sheets, the Human Health Benchmark for Pesticides (HHBPs), or the 2014 Preliminary Regulatory Determinations for Contaminants on CCL 3. The primary/secondary sources of health information vary with respect to scientific rigor from health assessment to single studies. Many of the contaminants are currently under regulatory review or development and are subject to change as new health assessments are completed.
b) MRL = UCMR Minimum Reporting Level. *[Note that the Agency for Toxic Substances & Disease Registry (ATSDR) uses the term "MRL" for a different purpose (i.e., to describe "Minimal Risk Levels"). The UCMR term and the ATSDR term have no relationship to each other.]*
c) HRLs = Health Reference Levels. HRLs are not final determinations about the level of a contaminant in drinking water that is necessary to protect any particular population and are derived prior to development of a complete exposure assessment. HRLs are risk derived concentrations against which to evaluate the occurrence data to determine if contaminants occur at levels of potential public health concern.
d) MCL = Maximum Contaminant Level. The highest level of a contaminant allowed in drinking water. MCLs are enforceable standards.
e) Cancer Risk of 10^{-6} to 10^{-4} = the concentration of a contaminant in drinking water corresponding to an excess estimated lifetime cancer risk of one-in-a-million ($1x\ 10^{-6}$) to one-in-ten-thousand (1×10^{-4}). The 2012 Drinking Water Standards and Health Advisories provide the cancer risk at 1×10^{-4}. The CCL 3 Contaminant Information Sheets provide the cancer risk at $1x\ 10^{-6}$.
f) LOAEL = Lowest Observed Adverse Effect Level
g) NA = Not Available
h) Short-term = Typically refers to animal toxicological studies with an exposure duration of days to weeks.
i) Intermediate/Longer-term = Typically refers to animal toxicological studies with an exposure duration of weeks to months.
j) Chronic = Typically refers to animal toxicological studies with an exposure duration of months to years; representing a lifetime exposure in humans.

References

k) 2012 Drinking Water Standards and Health Advisories
l) CCL 3 Contaminant Information Sheets
m) Human Health Benchmark for Pesticides (HHBPs)
n) Announcement of Preliminary Regulatory Determinations for Contaminants on the Third Drinking Water Contaminant Candidate List
o) Integrated Risk Information System (IRIS)

October 2015 UCMR 3 Data Summary for Chemical Contaminants

Contaminant	MRL (µg/L)	Reference Concentration (µg/L)	Total number of results	Number of results ≥MRL	Number of results >Reference Concentration	% of total results >Reference Concentration	Total number of PWSs with results	Number of PWSs with results ≥MRL	Number of PWSs with results >Reference Concentration	% of PWSs with results >Reference Concentration
1,2,3-trichloropropane	0.03	0.0004 / 0.04[1]	32,893	228	228 / 174[1]	0.7% / 0.5%[1]	4,750	62	62 / 52[1]	1.3% / 1.1%[1]
1,3-butadiene	0.1	0.0103 / 1.03[1]	32,893	1	1 / 0[1]	0.003% / 0%[1]	4,750	1	1 / 0[1]	0.02% / 0%[1]
Chloromethane	0.2	2.69 / 269[1]	32,891	251	18 / 0[1]	0.05% / 0%[1]	4,750	127	7 / 0[1]	0.1% / 0%[1]
1,1-dichloroethane	0.03	6.14 / 614[1]	32,891	764	1 / 0[1]	0.003% / 0%[1]	4,749	230	1 / 0[1]	0.02% / 0%[1]
Bromomethane	0.2	140	32,892	109	0	0%	4,750	46	0	0%
HCFC-22	0.08	NA	32,893	742	- -	- -	4,750	266	- -	- -
Halon 1011	0.06	90	32,893	596	0	0%	4,750	286	0	0%
1,4-dioxane	0.07	0.35 / 35[1]	32,740	3,796	989 / 0[1]	3% / 0%[1]	4,746	1,025	325/ 0[1]	6.8% / 0%[1]
Vanadium	0.2	21	56,298	33,595	1,563	2.8%	4,756	3,454	159	3.3%
Molybdenum	1	40	56,307	23,089	133	0.2%	4,756	2,438	34	0.7%
Cobalt	1	70	56,290	772	3	0.005%	4,756	226	3	0.06%
Strontium	0.3	1,500	56,227	56,038	1,546	2.7%	4,756	4,755	272	5.7%
Chromium	0.2	100	56,161	28,378	1	0.002%	4,756	3,517	1	0.02%
Chromium-6	0.03	NA	56,039	42,151	- -	- -	4,756	4,229	- -	- -
Chlorate	20	210	55,990	30,991	8,608	15.4%	4,749	3,236	1,744	36.7%
PFOS	0.04	0.2	33,178	260	33	0.1%	4,764	90	17	0.4%
PFOA	0.02	0.4	33,178	324	0	0%	4,764	103	0	0%
PFNA	0.02	NA	33,178	19	- -	- -	4,764	14	- -	- -
PFHxS	0.03	NA	33,178	192	- -	- -	4,764	54	- -	- -
PFHpA	0.01	NA	33,178	216	- -	- -	4,764	80	- -	- -
PFBS	0.09	NA	33,178	13	- -	- -	4,764	6	- -	- -
17β-estradiol	0.0004	0.0009 / 0.09[1]	10,234	3	1 / 0[1]	0.01% / 0%[1]	1,145	1	1 / 0[1]	0.09% / 0%[1]
17α-ethynylestradiol	0.0009	0.035	10,235	4	0	0%	1,145	4	0	0%
Estriol	0.0008	0.35	10,234	1	0	0%	1,145	1	0	0%
Equilin	0.004	0.35	10,235	0	0	0%	1,145	0	0	0%
Estrone	0.002	0.35	10,235	0	0	0%	1,145	0	0	0%
Testosterone	0.0001	NA	10,234	57	- -	- -	1,145	51	- -	- -
4-androstene-3,17-dione	0.0003	NA	10,235	84	- -	- -	1,145	64	- -	- -

[1]Where two reference concentrations are listed, the first number is associated with a 10^{-6} cancer risk; the second number a 10^{-4} cancer risk.
Where two results are presented the first number is associated with the first reference concentration; the second number is associated with the second reference concentration.

October 2015 UCMR 3 Data Summary for Microbiological Contaminants

Contaminant	MRL	Unit	Total number of results	Number of results ≥MRL	Total number of PWSs with results	Number of PWSs with results ≥MRL
Aerobic spores	1	SFO[1]/100 mL[2]	732	201	710	198
E. coli	1	MPN[3]/100 mL	730	3	708	3
Enterococci	1	MPN/100 mL	729	37	707	37
Enteroviruses (cell culture)	0.002	MPN/L[4]	728	2	706	2
Enteroviruses (RT-qPCR[5])	0.398	GC[6]/L	728	6	706	6
Male specific phage	1	PFU[7]/100 mL	714	10	692	10
Noroviruses GIA[8]	0.398	GC/L	728	0	706	0
Noroviruses GIB[9]	0.398	GC/L	728	1	706	1
Noroviruses GII[10]	0.398	GC/L	728	4	706	4
Somatic phage	1	PFU/100 mL	714	3	692	3
Total coliforms	1	MPN/100 mL	730	33	708	32

[1]SFO = Spore Forming Units
[2]mL = milliliters
[3]MPN = Most Probable Number
[4]L = liters
[5]RT-qPCR = Reverse Transcription-Polymerase Chain Reaction
[6]GC = Genomic Copies
[7]PFU = Plaque Forming Units
[8]Noroviruses GIA = qPCR analysis of Norovirus genogroup I with RT-qPCR primer set A
[9]Noroviruses GIB = qPCR analysis of Norovirus genogroup I with RT-qPCR primer set B
[10]Noroviruses GII = qPCR analysis of Norovirus genogroup II

UCMR 3 Minimum Reporting Levels for Microbiological Contaminants

Under UCMR 3 microbe analytical results are reported as “below”, “at” or “above” MRL. UCMR 3 MRLs were established based on the capability of the analytical method.

It is important to note that microbial contamination can be transient in nature and microbial detections under UCMR 3 should be interpreted in the context of the time samples were collected. However, the presence of any UCMR 3 microbe indicates a potential vulnerability of the PWS to contamination.

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February 16, 2016

BY EMAIL AND REGULAR U.S. MAIL

Gina McCarthy
Administrator
United States Environmental Protection
Agency
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1200 Pennsylvania Ave., N.W.
Mail Code 1101A
Washington, D.C. 20460

Re: PFOA Drinking Water Contamination

Dear Administrator McCarthy:

As indicated in the attached examples of our prior correspondence to EPA, including our letters of November 9, 2015, and January 20, 2015, to EPA Regions III and V (extra copies attached), we have been writing to EPA since March of 2001 – for almost *fifteen years* – to try to focus the Agency's attention on the imminent and substantial threat to human health and the environment posed by the contamination of human drinking water supplies with perfluorooctanoic acid ("PFOA" a/k/a "C-8").[1] In our letter of November 9, 2015, we specifically highlighted the fact that recent testing of public water supplies across the country indicated the presence of PFOA in numerous locations at levels exceeding the level of PFOA (0.05 ppb) where six serious diseases, including cancer, were found by independent scientists to be linked to PFOA exposures in community residents. (*See* attached. *See also* www.c8sciencepanel.org .) In response, EPA indicated in a letter dated December 8, 2015, that it was working on a guideline for "lifetime" exposures to PFOA in drinking water, which it expected to be able to release sometime "by early 2016." (*See* attached.)

On January 28, 2016, EPA noted that it was still working on "developing a lifetime health advisory level for PFOA," but that,"[w]hile this work continues," EPA

[1] We also recently have communicated with EPA Region IV to try to obtain EPA's assistance in investigating and addressing significantly elevated PFOA blood levels among Northern Kentucky residents, (*see* attached), but, so far, EPA has still not responded.

"recommends" that water containing more than 0.1 ppb PFOA not be used for drinking or cooking and that those with such PFOA-contaminated water sources use bottled water. (*See* attached.) Thus, at a minimum, it appears that EPA has revised its guideline for short-term, temporary exposures to PFOA in drinking water from 0.4 ppb to 0.1 ppb. What is not clear, however, is the extent to which members of the public exposed to levels of PFOA exceeding 0.1 ppb in different areas across the country (particularly those with long-term, "lifetime" exposures) have been informed of those exposures or have seen the EPA's recommendation to use bottled water or some other alternative water source in those situations.

In contrast to the public, EPA is aware of several such exposure scenarios by virtue of the data supplied to EPA by various public water supplies under EPA's unregulated contaminant monitoring program ("UCMR program"). Under the UCMR program, certain public water supplies have been sampling for and reporting to EPA the presence of PFOA in their drinking water since at least 2013, but not all of those water supplies have necessarily informed their customers of the detections of PFOA, believing that, because the chemical is not one of the "regulated" water contaminants for testing, the chemical is not "required" to be included on the annual customer water reports. We have attached a list of what we believe to be the currently-available public water supply sampling results for PFOA available in the large file of raw data posted to EPA's UCMR program webpage.[2]

As noted in our prior correspondence, we request that EPA take those steps necessary to immediately and properly disclose, investigate, and address elevated levels of PFOA in impacted communities, whether reflected in elevated drinking water exposures or elevated blood levels. At a minimum, such steps should include an immediate revision to EPA's March 2009 Consent Order with DuPont to incorporate EPA's latest 0.1 ppb guideline for PFOA in drinking water, given recent detections of PFOA above that level in at least one impacted local community - Vienna, West Virginia – as we requested in our January 29, 2016, email to EPA . (*See* attached.)

Thank you.

Very truly yours,

Robert A. Bilott

RAB:mdm
Attchs.

[2] We understand that EPA is only currently requiring the reporting of PFOA at concentrations at or above 0.02 ppb (even though current analytical methods allow quantification and detection at much lower levels), so this list does not include any detection below that arbitrary 0.02 ppb reporting level.

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January 20, 2015

BY EMAIL AND REGULAR U.S. MAIL

Susan Hedman
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United States Environmental Protection Agency
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Chicago, IL 60604-3507

Randy C. Huffman
Cabinet Secretary
West Virginia Department of Environmental Protection
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Charleston, WV 25304

Shawn M. Garvin
Regional Administrator
United States Environmental Protection Agency
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1650 Arch Street
Mail Code: 3RA00
Philadelphia, PA 19103-2029

Re: *In the Matter of: E.I. du Pont de Nemours and Company*
(Docket Nos. SDWA-03-2009-0127 DS – SDWA-05-2009-0001)

Dear Ms. Hedman, Mr. Garvin and Mr. Huffman:

We first wrote to US EPA and WVDEP in March of 2001 – over 13 years ago – to alert your Agencies to the imminent and substantial threat to human health and the environment posed by the contamination of human drinking water supplies with perfluorooctanoic acid ("PFOA" a/k/a "C-8") released from E. I. du Pont de Nemours and Company's ("DuPont's) Washington Works Plant in Wood County, West Virginia (the "DuPont Plant"). (*See* Ex. A.) In that original letter, we alerted your Agencies to the fact that PFOA was poisoning drinking water supplies in the vicinity of the DuPont Plant at levels exceeding a 1 part per billion (1 ppb) exposure guideline that DuPont had adopted for PFOA in community water more than a decade earlier, and asked your

Agencies to take immediate action to address and abate that health threat under applicable state and federal laws, including the Clean Water Act ("CWA"), the Safe Drinking Water Act ("SDWA"), the Toxic Substances Control Act ("TSCA"), and the Resource Conservation and Recovery Act ("RCRA"). (*See id.*) Soon thereafter, US EPA launched a "priority review" of PFOA under TSCA and began the process to establish federal safety limits for PFOA in drinking water, beginning with the release of a draft PFOA risk assessment in 2003. WVDEP, on the other hand, has still not even begun the process of trying to establish or set any regulatory safety levels for PFOA, choosing, instead, to defer to whatever US EPA ultimately decides. In the meantime, given the lack of any enforceable federal or state regulatory safety limits for PFOA in drinking water, US EPA was left with having to address this serious health threat by negotiating "Consent Orders" with DuPont through which US EPA could incorporate only such terms as to which DuPont ultimately would "consent."

The first such US EPA Consent Order was entered in 2002, soon after US EPA received our original letter. Rather than require clean water whenever DuPont's own 1 ppb drinking water exposure level was exceeded (which 1 ppb level had been created by DuPont's own scientists, had been followed internally by DuPont for more than a decade, and was still being followed internally by DuPont at that time), DuPont would only "consent" to providing clean water through this new Consent Order, if the level of PFOA exceeded a significantly higher 14 ppb level that DuPont's outside consultants had generated.

Just two months later, in May 2002, DuPont succeeded in forcing US EPA to raise that 14 ppb level to 150 ppb, based on the terms of a separate, privately-negotiated deal between DuPont and WVDEP under which WVDEP allowed DuPont to collaborate with WVDEP and its consultant to create a new, higher trigger level for clean water. DuPont then held that 150 ppb number out to the public for the next several years as the appropriate, government-endorsed safety number for PFOA in drinking water, even though, internally, DuPont's own scientists still supported a 1 ppb exposure guideline for PFOA in community drinking water supplies.

DuPont only "consented" to a new Consent Order with US EPA on these issues in 2006, after significant additional health risk information had been released on PFOA, including a final report from US EPA's own Science Advisory Board, where the majority of the Board recommended that PFOA be classified as a "likely" human carcinogen. Upon review of this new data, US EPA's scientists had determined that the 150 ppb trigger picked by DuPont and WVDEP was "not protective of human health and must be replaced by a lower threshold value of 0.20 ppb." (Ex. B. at 1.) DuPont informed US EPA at the time that it agreed, based on this new data, that "it is prudent to minimize, where possible, exposure to biopersistent materials such as PFOA," and that a new, lower clean water trigger number should be adopted "to help promote reductions of PFOA in blood levels through alternate drinking supplies." (Ex. C at 3-4.) According to DuPont, a "median serum/drinking water ratio for PFOA was calculated to be 105, i.e.,

for every 1 ppb of PFOA in drinking water ingested by community residents; 105 ppb of PFOA will be present in serum." (*Id.* at 9.) At the 150 ppb trigger level then in effect, DuPont noted that "a serum level of approximately 15 ppm [15,000 ppb] can be predicted," which "exceeds the current occupational exposures" where adverse health effects were being reported in the new data. (*Id.* at 11.) According to DuPont, reducing the clean water trigger from 150 ppb to 0.5 ppb - not 0.20 ppb – would be sufficient, as it "would result in approximately 50 ppb of PFOA in serum," which DuPont argued was "within the range found in the general population" where no such adverse health effects were purportedly being found at the time. (*Id.*) Thus, in light of DuPont's refusal to agree to a safe drinking water trigger level any lower than 0.5 ppb at that time, the new US EPA/DuPont Consent Order in 2006 lowered the PFOA clean drinking water threshold from 150 ppb to 0.5 ppb PFOA. US EPA was not able to obtain DuPont's "consent" to lower the threshold for safe water any further until 2009, after US EPA released its first "provisional health advisory" ("PHA") for short-term, temporary exposure to C-8 in drinking water of 0.4 ppb. At that point, DuPont finally agreed to lower the clean water trigger in its Consent Order with US EPA - but only from 0.5 ppb to 0.4 ppb.

US EPA made clear in its 2009 Consent Order with DuPont that the 0.4 ppb C-8 trigger level for clean water was a "temporary value that will be re-evaluated when EPA determines a reference dose under TSCA or establishes a drinking water standard for C-8, whichever comes first." (2009 Consent Order, at ¶ 46.) US EPA also made clear that it reserved "the right to modify the [0.4 ppb C-8 clean water trigger] identified in this Order if information previously unknown to EPA is received and EPA determines that this previously unknown information, together with any other relevant information, indicates that [such trigger level] may not be protective of human health." (*Id.* at ¶ 47.)

Since entry of the current Consent Order in March of 2009, extensive additional information has been released in the scientific and peer-reviewed literature confirming that the 0.4 ppb trigger level for clean water is not protective of human health for long-term exposures and should be revised. For example, in December 2009, US EPA released its Long-Chain Perfluorinated Chemicals (PFCs) Action Plan, identifying C-8 as "raising serious health and environmental concerns," which could justify significant "risk reduction measures to protect human health and the environment." Then, in 2011-2012, an independent C-8 Science Panel – jointly selected and fully-funded by DuPont – confirmed probable links between exposure to PFOA in drinking water as low as 0.05 ppb and six serious human diseases: 1) kidney cancer; 2) testicular cancer; 3) ulcerative colitis; 4) thyroid disease; 5) pregnancy-induced hypertension/preeclampsia; and 6) hypercholesterolemia. Each of those links was based on the independent Science Panel's review of data (including PFOA blood tests, blood chemistries, and medical records reviews/verifications) from approximately 70,000 people actually exposed to PFOA in drinking water in the vicinity of the DuPont Plant, along with all other available data, including peer-reviewed studies from all over the world and DuPont's own worker data. Each of the Science Panel's findings ultimately was

confirmed in published, peer-reviewed papers. US EPA was encouraged through public comments and formal peer reviewers to consider and incorporate all such important new data (along with additional, significant new toxicological data, including new data on mammary gland impacts and from studies in mice), in the context of finalizing US EPA's "Health Effects Document for Perfluorooctanoic Acid," which was released in draft form to the public in 2014 but, as of today's date, still has not been finalized.

Although US EPA still has not released a guideline for long-term, chronic exposure to PFOA in drinking water or finalized its PFOA health effects document, European regulators have moved forward. Just this month, the European Chemicals Agency (ECHA) publicly released a report from Germany and Norway recommending significant new restrictions on PFOA in light of the more current health effects data, specifically including the findings of the C8 Science Panel linking very low level PFOA exposure in drinking water (as low as 0.05 ppb) with 6 diseases, including two forms of cancer. (*See* http://echa.europa.eu/documents/10162/e9cddec6-3164-473d-b590-8f9caa50e7 .) Particularly significant in this new European report are new risk calculations revealing that levels of PFOA in the blood of people exposed to PFOA at the levels allowed under the existing 2009 Consent Order (PFOA drinking water levels as high as 0.5 ppb) would far exceed the blood risk levels derived using the latest health effects data. This is because significant adverse health effects (including cancer) were found to be linked to PFOA exposures in humans as low as 0.05 ppb in drinking water – some *ten times lower* than the current level allowed under the 2009 Consent Order. (*See also* Post, G.B., *et al.*, "Perfluorooctanoic acid (PFOA), an emerging drinking water contaminant: A critical review of recent literature," 116 *Environ. Res.* 93-117 (July 2012).)

Although neither the European report nor US EPA's work to set a safety level for long-term chronic exposure to PFOA in drinking water has been completed, US EPA retains both the right and responsibility to modify the 2009 Consent Order in light of new health data on PFOA to make sure that human health is protected. US EPA should consider the new PFOA health effects data and European safety calculations noted above to evaluate whether there is a current or imminent and substantial threat or endangerment to human health that mandates steps be taken to modify the 2009 Consent Order to require DuPont to provide for alternate/clean drinking water for any human drinking water supply in the vicinity of the DuPont Washington Works Plant where PFOA has been detected at levels below the current 0.4 ppb trigger level established in that Consent Order. In New Jersey, for example, state regulators already are evaluating the safety of drinking water supplies by comparing PFOA water levels to a 0.04 ppb "health-based drinking water guidance level" developed specifically for the purpose of assessing long-term, chronic exposures to PFOA in human drinking water supplies. (*See, e.g.,* Ex. D.)

As both US EPA and WVDEP are aware, there are at least two public drinking water supplies in the vicinity of the DuPont Plant in West Virginia where sampling for

PFOA revealed levels of PFOA in the treated water above the 0.05 ppb level of exposure considered in the C8 Science Panel studies: 1) the City of Parkersburg, West Virginia (most recent rounds of CCL3 sampling data submitted to US EPA and now posted on US EPA's website revealed PFOA as high as 0.0631 ppb after treatment on 3/25/14); and 2) the City of Vienna, West Virginia (reports submitted by DuPont to US EPA and posted in US EPA' public dockets confirm 0.056 ppb PFOA after treatment on last-known PFOA sampling date of 5/10/07). (*See* Ex. E). DuPont successfully used US EPA's and WVDEP's continuing failure to adopt any final long-term, chronic exposure limits for PFOA in drinking water to thwart all efforts by impacted Parkersburg residents to require DuPont to provide clean water through the court system. (*See, e.g.,* 9/30/08 Memorandum Opinion and Order in *Rhodes, et al., v. E.I. du Pont de Nemours and Co.,* Civil Action No. 6:06-cv-00530 (S.D. W. Va.) at 1 (West Virginia federal court denied Parkersburg residents' attempts to bring community/class-wide claims against DuPont for clean water through the judicial system, noting that, although the "plaintiffs have presented compelling evidence that exposure to C-8 may be harmful to human health, and the evidence certainly justifies the concerns expressed by the plaintiffs in this case," the Court could not certify those claims to proceed through the Court system at that time: "The fact that a public health risk may exist is more than enough to raise concern in the community and call government agencies to action, but it does not show the common individual injuries needed to certify a class action" for relief through the judicial system.).)

Thus, despite DuPont's acknowledgment to US EPA by at least 2006 that "it is prudent to minimize, where possible, exposure to biopersistent materials such as PFOA" and purported desire "to help promote reductions in PFOA in blood levels through alternate drinking supplies," (Ex. C at 3-4), DuPont aggressively fought and ultimately succeeded in preventing Parkersburg residents from obtaining clean water through the court system, even though DuPont knew that failure to remove PFOA from that water would allow PFOA to steadily build up and accumulate in the blood of the residents drinking that water at a ratio of approximately 105 ppb PFOA in blood for every 1 ppb PFOA in their drinking water. US EPA and WVDEP, likewise, have not required any action to date to abate these on-going exposures in either Parkersburg or Vienna, despite knowledge of the on-going contamination (and associated accumulation and build- up of PFOA in residents' blood) for almost a decade.

US EPA should re-assess its position with respect to these on-going PFOA exposures in light of existing health data. US EPA also should consider whether any steps need to be taken to insure that the appropriate parties remain bound under its existing Consent Orders and Memoranda of Understanding with DuPont on PFOA issues, in light of DuPont's recently announced intentions to soon "spin-off" and/or jettison certain operations and liabilities of DuPont relating to PFOA to a new entity to be known as "Chemours," (*see* Ex. F).

Very truly yours,

Robert A. Bilott

RAB:mdm
Encls: Exs. A-F
cc: Elizabeth Doyle, USEPA (w/encls.)(by regular U.S. mail)

United States Environmental Protection Agency
Regional Administrator
Region 5
77 West Jackson Boulevard
Chicago, IL 60604-3590

FEB 2 3 2015

Robert A. Bilott
Taft Stettinius & Hollister LLP
425 Walnut Street, Suite 1800
Cincinnati, Ohio 45202-3957

Dear Mr. Bilott:

Thank you for your January 20, 2015 letter requesting that the U.S. Environmental Protection Agency consider new health effects data to evaluate the 0.40 parts per billion (ppb) site-specific action level for perfluorooctanoic acid (PFOA) in EPA's 2009 consent order with DuPont's Washington Works Facility in West Virginia.

On February 28, 2014, EPA released a draft health effects document for PFOA for public comment and peer review: https://peerreview.versar.com/epa/pfoa/. When the document is finalized later this year a lifetime health advisory will be developed, which may replace our 2009 PFOA provisional (short-term) health advisory of 0.4 ppb. When that process is complete the action level established in the March 10, 2009 consent order between US EPA and DuPont may be re-evaluated.

Again, thank you for your letter. If you have further questions, please contact Debra Klassman, Associate Regional Counsel, of my staff, at 312-886-6742, or Lori Kier, Senior Assistant Regional Counsel, of Mr. Garvin's staff in Region 3, at 215-814 2656.

Sincerely,

Susan Hedman
Regional Administrator

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513.357.9638
bilott@taftlaw.com

November 9, 2015

BY EMAIL AND REGULAR U.S. MAIL

Susan Hedman
Regional Administrator
United States Environmental Protection Agency
Region V
77 West Jackson Blvd.
Mail Code: R-19J
Chicago, IL 60604-3507

Randy C. Huffman
Cabinet Secretary
West Virginia Department of Environmental Protection
601 57th Street, SE
Charleston, WV 25304

Shawn M. Garvin
Regional Administrator
United States Environmental Protection Agency
Region III
1650 Arch Street
Mail Code: 3RA00
Philadelphia, PA 19103-2029

Re: *In the Matter of: E.I. du Pont de Nemours and Company*
(Docket Nos. SDWA-03-2009-0127 DS – SDWA-05-2009-0001)

Dear Ms. Hedman, Mr. Garvin and Mr. Huffman:

This letter serves as a follow up to my letter to you dated January 20,2015, (extra copy enclosed), to which US EPA Region 5 responded on February 23, 2015, (extra copy enclosed), but to which neither US EPA Region 3 nor the West Virginia Department of Environmental Protection ("WVDEP") ever responded.

As noted in my prior letter, we first asked your agencies to take immediate action to address on-going contamination of human drinking water supplies with PFOA more than 14 years ago. Since that time, WVDEP has done nothing to establish any regulatory standards or limits for PFOA in drinking water, choosing, instead, to simply

defer to whatever US EPA eventually decides to do. US EPA, however, *still* has not established – nor even proposed – any standards or limits for long term (more than a few weeks or even a few months) exposure to PFOA in drinking water.

In the meantime, data collected from public drinking water supplies across the country and provided directly to US EPA has confirmed the presence of PFOA in public drinking water supplies in over 20 states in all regions of the Country. (*See* http://water.epa.gov/lawsregs/rulesregs/sdwa/ucmr/data.cfm.) As US EPA is aware, the levels of PFOA found in many of these water supplies – more than a dozen – exceed the 0.05 ppb PFOA level where probable links were found between such PFOA drinking water exposure levels and six serious diseases, including cancer. (*See id.*) Even more exceed the levels that have been set or recommended by other regulatory bodies (such as the State of New Jersey) for long-term PFOA exposures, or the levels that the most recent scientific research now indicates may be excessive. (*See* enclosed 1/20/15 letter (and references).)

Nevertheless, when US EPA Region 5 responded to my prior letter, it indicated that no further action would be taken by US EPA to address any of this on-going contamination of public water supplies until its previously-released "draft health effects document for PFOA" was finalized, which Region 5 stated would occur "later this year." As we are now nearing the end of the year and the document still has not been finalized (nor has any information been released suggesting when any such action might occur), we request that US EPA confirm the schedule for finalizing the document and moving forward with appropriate actions to protect the public health.

Very truly yours,

Robert A. Bilott

RAB:mdm
Ecls:
cc: Elizabeth Doyle, USEPA (w/encls.)(by regular U.S. mail)

13988385.1

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION III
1650 Arch Street
Philadelphia, Pennsylvania 19103-2029

DEC 3 8 2015

Robert A. Bilott, Partner
Taft Stettinius & Hollister LLP
425 Walnut Street, Suite 1800
Cincinnati, Ohio 45202-3957

Dear Mr. Bilott:

Thank you for your November 9, 2015 letter requesting that the U.S. Environmental Protection Agency provide you with an update on EPA's draft perfluorooctanoic acid (PFOA) health effects document, which addresses long-term PFOA exposure. EPA released the draft PFOA health effects document in February 2014 for purposes of public comment (scientific views) and peer review.

Please be advised that the timeline for developing a lifetime health advisory level for PFOA has changed slightly since EPA Regions 3 and 5's combined response to you dated February 23, 2015. The EPA Office of Water now expects to complete its efforts to develop a revised Health Advisory for both PFOA by early 2016.

Once a final risk assessment is completed, or if further information about the health effects of PFOA indicates that it is necessary, EPA may re-evaluate the PFOA action level established in the March 9, 2010, Safe Drinking Water Act Order on Consent between EPA and DuPont.

Thank you for your continued interest in these matters. If you have further questions, please contact Lori Kier, Esq., Senior Assistant Regional Counsel, of Region 3 at 215-814 2656, or Jacqueline Clark, Esq., Associate Regional Counsel, in Region 5, at 312-353-4191.

Sincerely,

Jon M. Capacasa
Director, Water Protection Division

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December 16, 2015

BY EMAIL AND REGULAR U.S. MAIL

Heather McTeer Toney
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Atlanta, GA 30303-3104

Ileana Arias, PhD
Director
Division of Community Health Investigations
ATSDR
4770 Buford Hwy, NE (MSF59)
Atlanta, GA 30341-3717

Re: Request For Investigation/Disclosure of PFOA Human Drinking Water Impacts in Northern Kentucky

Dear Ms. McTeer Toney and Dr. Arias:

On November 11, 2015, researchers at Brown University publicly announced the release of a study finding that children born to mothers in a Cincinnati, Ohio, area cohort with elevated levels of PFOA in their blood had increased adiposity at age 8 and had greater BMI gains from age 2 to 8 than children with lower PFOA exposures. (*See* Ex. A.) As for the extent of the elevated PFOA exposures in the cohort being studied, the researchers reported that the "average exposure measured among the mothers in the cohort was more than twice that of a representative sample of pregnant women from the United States." (*Id.* at 2.)

With respect to the potential source of the elevated levels of PFOA found in the blood of this Cincinnati-area cohort, the researchers referred to an earlier study that purportedly noted increased PFOA blood levels among school-age Cincinnati-area girls whose drinking water may have been impacted by PFOA contamination coming from the Ohio River. (*See id.* (published paper at 5).) That earlier study, published in 2014, reported that the Cincinnati-area girls receiving their water from the public water supply

in Northern Kentucky (directly across the Ohio River from downtown Cincinnati) had significantly increased levels of PFOA in their blood. (*See* Ex. B at 329.) More specifically, the researchers reported finding a median serum PFOA concentration of 22.0 ppb in the young girls consuming water from the Northern Kentucky water supply, with 48 out of 51 of those girls having PFOA serum concentrations above the 95th percentile of PFOA serum concentrations reported from a 2005-2006 nation-wide survey of PFOA serum concentrations. (*Id.*) According to these researchers, this elevated "PFOA serum concentration was highly associated ... with cumulative years of drinking water from the NKY water distribution zone." (*Id.* at 330-331.)

In support of their hypothesis that the elevated level of PFOA found among the girls in Northern Kentucky was attributable to elevated levels of PFOA in their drinking water, the researchers compared the PFOA serum concentrations to those found among girls living directly across the Ohio River who received their drinking water from the City of Cincinnati's public water supply. (*See id.*) This was done because both the Cincinnati and Northern Kentucky public water supplies draw water from the approximately same general area of the Ohio River, but only the Cincinnati public water supply was being treated at the time by using the type of granular activated carbon ("GAC") filtration system that has been shown to reduce PFOA levels prior to entry into the public water distribution system. (*See id.* at 332.) After confirming that the PFOA serum level among the girls in Northern Kentucky was significantly higher than the levels found in the serum of the girls drinking Cincinnati water, the researchers concluded that the lack of GAC filtration of the drinking water in Northern Kentucky at the time "could be a plausible explanation for the differences ... found in serum concentrations." (*Id.*) According to these same researchers, the Northern Kentucky public water district first initiated this type of GAC treatment in 2012. (*Id.*)[1]

As for the amount of PFOA purportedly present in the Ohio River raw source water for the Northern Kentucky drinking water supply, the researchers noted that PFOA had been found in detectable amounts in the untreated Ohio River water near the Greater Cincinnati/Northern Kentucky water supply area. (*See id.* at 332.) Based on responses to our recent public file requests, we have now learned that this PFOA was detected at levels between 0.016 ppb and 0.1 ppb in samples of the untreated Ohio River water taken near the Northern Kentucky public water supply between 2005 and 2009, (*see* Ex. D), with PFOS also being found in such water as high as 0.0012 ppb in 2010, (*see* Ex. E).

As for the origin or source of the PFOA in the Ohio River, the researchers stated that they "know of no PFC compound manufacturing sites in the GC [Greater Cincinnati]

[1] According to documents produced by the Northern Kentucky Water Service District in late 2015 in response to our public file request, the water district never sampled any of its finished drinking water (as supplied to customers) for PFOA or PFOS prior to the initiation of GAC treatment in 2012, thus we have been unable to locate any data confirming what amount (if any) PFOS or PFOS was present in any of that water prior to such treatment. (*See* Ex. C.)

area," and thus hypothesized that "a source upriver from the GC [Greater Cincinnati] area may have contributed to exposures in girls from NKY through their drinking water." (Ex. B at 331-32.) With respect to the identify of any such "source upriver," the researchers noted only that "an industrial facility" located "about 285 miles upriver ...may represent a potential source of exposure via the Ohio River for the girls from NKY," and that sampling for PFOA in the Ohio River in 2009 revealed that "levels of PFOA progressively decreased with increasing distance downriver from Parkersburg WV to the Greater Cincinnati area." (*Id.* at 332.) Although the researchers did not mention the name of that facility near Parkersburg, they did reference a "legal settlement" involving the facility that spurred creation of the "C8 Health Project," which is well-known to involve extensive investigation of the community residents impacted by PFOA released by the Washington Works manufacturing facility originally owned and operated by E. I. du Pont de Nemours and Company, and now owned and operated by the Chemours Company. (*See id.*) As of today's date, however, we are unaware of any steps taken or ordered by either or your agencies (or any other regulatory entity) to investigate and address or require any other party to investigate address the nature, extent, and source of the elevated PFOA levels found in the Ohio River in the Greater Cincinnati Area.

Moreover, we are unaware of any efforts being taken or ordered by your Agencies (or any other entity) to evaluate the nature and extent of any elevated levels of PFOA in the serum of the tens of thousands of people in Northern Kentucky who may have been exposed to PFOA in their drinking water. Even though GAC filtration may be in place now, that treatment does not address past exposures to PFOA in drinking water, which, as your Agencies are aware, would result in the steady accumulation of elevated levels of PFOA in serum from even the smallest PFOA exposures, given the extremely persistent nature of the chemical. Moreover, because of the long half-life of the chemical, any such elevated levels of PFOA in serum would be expected to remain in an exposed person's blood/body for many years and only slowly decrease over time once all exposures stopped. Although blood levels were evaluated from a small group of young girls in the study referenced above, we are unaware of any investigation having been undertaken or ordered of the larger community in Northern Kentucky – adults, children, elderly, infirm, etc. – to assess the extent and nature of any elevation of PFOA serum levels.

Most troubling, however, is that we are unaware of any meaningful public disclosure of potentially elevated PFOA serum levels and associated adverse health effects to all of the residents potentially impacted in Northern Kentucky. Although we understand that the families of the actual participants in the study involving the several dozen young girls in Northern Kentucky were informed of their serum results by the study sponsors and were provided some related information in private meetings with those families back in 2007, we are unaware of any disclosures to the larger Northern Kentucky community having occurred at that time (or since). (*See* Ex. F.) The researchers reported that the "superintendent of the school district attended" one of the

meetings in 2007 and even "commented that the findings might have implications beyond the study families and perhaps the county" and apparently inferred "that there would be follow-up if health concerns came to light." (*Id.* at 5.) The researchers also noted that they "met with relevant water district personnel in Greater Cincinnati to notify them of the biomarker [PFOA serum level] findings," and even drafted a press release "should the need arise for a more public statement concerning the findings." (*Id.*) As of today's date, however, we are unaware of any such press release having been released or any "more public statement" being made to the broader Northern Kentucky community, prior to the November 11, 2015, press release announcing results of the most recent adiposity study results, which was not to our knowledge reported by any of the local newspapers serving Northern Kentucky. As a result, the Northern Kentucky community (with the possible exception of some of the original study participants and their families) remains completely unaware that they may have elevated PFOA serum levels or how any such elevated PFOA serum levels may relate to their health.

The need for immediate, meaningful, community-wide public disclosure in this regard is imperative to protect public health. Since the original meetings were held in 2007 with the families of the Northern Kentucky study participants, school superintendent, and water district personnel, significant additional health information has been released confirming links between elevated exposures to PFOA through drinking water and serious human disease. More specifically, independent, published, peer-reviewed scientific research has confirmed that community-level exposure to PFOA in drinking water at concentrations as low as 0.05 ppb for as little as one year is capable of causing: 1) testicular cancer; 2) kidney cancer; 3) ulcerative colitis; 4) thyroid disease; 5) preeclampsia/pregnancy-induced hypertension; and medically-diagnosed high cholesterol (hypercholesterolemia). (*See* www.C8sciencepanel.org for copies of or links to all such data.) These links between PFOA exposure in drinking water and serious disease were found after independent evaluation of tens of thousands of residents – adults, children, elderly, and the infirm – exposed to PFOA through contamination caused by DuPont's manufacturing operations at its Washington Work's plant upriver near Parkersburg, West Virginia. (*See id.*) The average level of PFOA found in the serum of all of the female community residents who participated in those studies where the disease links were found was reported to be approximately 30 ppb. (*See* Ex. B at 332.) In October of 2015, a woman who had been exposed to PFOA in her drinking water in that area and had a PFOA serum test result of 19 ppb won a $1.6 Million verdict against DuPont after an Ohio jury found that DuPont's contamination of her drinking water with PFOA caused her kidney cancer. (*See* Ex. G.)

We, therefore, request that your Agencies immediately take those steps necessary to inform the Northern Kentucky community of the potential health risks posed by potentially-elevated PFOA serum levels, and to investigate, assess, and determine the full extent, nature, and source of any such elevated PFOA serum levels so that steps can be taken to insure that any potential for continuing, on-going exposure is stopped. Given our extensive involvement in PFOA scientific, legal, and regulatory

issues over the last 15 years, we would be happy to assist in any way we can so that complete and accurate information is provided in the most appropriate and timely manner possible.

Thank you.

Very truly yours,

Robert A. Bilott

RAB:mdm
Ecls:

13988385.1

EPA Statement on Private Wells in

The Town of Hoosick and Village of Hoosick Falls, NY

January 28, 2016

The EPA is developing a lifetime health advisory level for PFOA. While this work continues, the EPA recommends that people in the Town of Hoosick and the Village of Hoosick Falls who have private wells at which PFOA has been found to be present at a level greater than 100 parts per trillion not use that water for drinking or cooking, and instead take advantage of the free bottled water that is being made available at the Tops Market in Hoosick Falls. In addition, the EPA recommends that people in the Town of Hoosick and the Village of Hoosick Falls who have private wells that have not yet been tested for the presence of PFOA ask the New York State Department of Health to test their well and, in the meantime, take advantage of the bottled water available at the Tops Market in Hoosick Falls.

Bilott, Robert A.

From: Bilott, Robert A.
Sent: Friday, January 29, 2016 4:50 PM
To: 'Kaplan.robert@Epa.gov'; 'Garvin.shawn@Epa.gov'; 'Randy.C.Huffman@Wv.Gov'; 'Klassman.debra@Epa.gov'; 'Kier.lori@Epa.gov'
Subject: FW: In the Matter of: E.I. du Pont de Nemours and Comapny (Docket Nos: SDWA-03-2009-0127 DS/ SDWA-05-2009-0001)
Attachments: EPA Response Letter 2 23 15.pdf; epa_statement_on_private_wells_in_the_town_of_hoosick.pdf; [Untitled].pdf

Ladies and Gentlemen:

Given the updated guidance released by US EPA last night (attached) referencing a 0.1 ppb action level for PFOA in human drinking water, we repeat the request we made over a year ago (as set forth in our attached letter) that steps be taken immediately to insure that the 2009 Consent Order referenced in our letter is modified accordingly to reference and incorporate the new 0.1 ppb guideline and that all appropriate parties are and/or remain bound by its terms. (According to EPA's earlier response (also attached), such actions would be considered when such a new guidance number was released.) We also request that any water supply previously tested or to be tested under that Consent Order with results at or above 0.1 ppb that is not already being treated to remove any such PFOA be addressed accordingly. Thank you.

Rob Bilott

lott, Robert A.
Sent: Tuesday, January 20, 2015 4:32 PM
To: hedman.susan@epa.gov; Garvin.shawn@Epa.gov; Randy.C.Huffman@Wv.Gov
Cc: Elizabeth A. Doyle (doyle.elizabeth@epa.gov)
Subject: In the Matter of: E.I. du Pont de Nemours and Comapny (Docket Nos: SDWA-03-2009-0127 DS/ SDWA-05-2009-0001)

Document attached.

Taft /

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Summary of Public Water Supply PFOA Data as Reported to EPA in UCMRR Program

No.	PWS	State	Sample Location	Date	Results
1	Warminster Municipal Authority	PA	Well 26 EPTDS	2014/06/09	0.349
1	Warminster Municipal Authority	PA	Well 26 EPTDS	2013/11/19	0.291
1	Warminster Municipal Authority	PA	Well 2 EPTDS	2013/11/19	0.0343
1	Warminster Municipal Authority	PA	Well 2 EPTDS	2014/06/09	0.0216
1	Warminster Municipal Authority	PA	Well 13 EPTDS	2013/11/19	0.122
1	Warminster Municipal Authority	PA	Well 13 EPTDS	2014/06/09	0.0796
1	Warminster Municipal Authority	PA	Well 10 EPTDS	2013/11/19	0.0885
1	Warminster Municipal Authority	PA	Well 10 EPTDS	2014/06/09	0.0822
1	Warminster Municipal Authority	PA	Well 15 EPTDS	2013/11/19	0.0246
1	Warminster Municipal Authority	PA	Well 5 EPTDS	2013/11/19	0.0231
1	Warminster Municipal Authority	PA	Well 5 EPTDS	2014/06/09	0.0201
1	Warminster Municipal Authority	PA	Well 14 EPTDS	2013/11/19	0.0228
1	Warminster Municipal Authority	PA	Well 9 EPTDS	2013/11/19	0.0203

No.	PWS	State	Sample Location	Date	Results
2	Oakdale	MN	Well 7 Entry Point	2015/01/05	0.338
2	Oakdale	MN	Well 7 Entry Point	2015/07/14	0.322
2	Oakdale	MN	Well 1 Entry Point	2015/07/14	0.0986
2	Oakdale	MN	Well 1 Entry Point	2015/01/05	0.0929
2	Oakdale	MN	Well 2 Entry Point	2015/01/05	0.0614
2	Oakdale	MN	Well 2 Entry Point	2015/0714	0.0583
3	Horsham Water & Sewer Authority	PA	Well 26 EPTDS	2014/06/24	0.29
3	Horsham Water & Sewer Authority	PA	Well 40 EPTDS	2014/06/24	0.063
3	Horsham Water & Sewer Authority	PA	Well 17 EPTDS	2013/06/24	0.023
3	Horsham Water & Sewer Authority	PA	Well 17 EPTDS	2014/12/09	0.026
3	Horsham Water & Sewer Authority	PA	Well 10 EPTDS	2014/06/24	0.02
3	Horsham Water & Sewer Authority	PA	Well 10 EPTDS	2014/12/09	0.026
4	Doylestown Twp. Muni. Authority	PA	Cross Keys Well EP #113	2014/02/18	0.20605
5	Artesian Water Supply	DE	Wilmington Manor 3 Plant EPTDS	2013/07/17	0.14
5	Artesian Water Supply	DE	Wilmington Manor 3 Plant EPTDS	2014/01/28	0.14

No.	PWS	State	Sample Location	Date	Results
5	Artesian Water Supply	DE	Jefferson Farm Plant EPTDS	2013/07/07	0.06
5	Artesian Water Supply	DE	Jefferson Farm Plant EPTDS	2014/01/28	0.04
5	Artesian Water Supply	DE	Castle Hills EPTDS	2014/07/16	0.05
5	Artesian Water Supply	DE	Castle Hills EPTDS	2013/07/17	0.04
5	Artestian Water Supply	DE	Midvale Plant EPTDS	2013/08/12	0.04
5	Artestian Water Supply	DE	Midvale Plant EPTDS	2014/01/28	0.04
5	Artesian Water Supply	DE	Wilmington Manor 1 Plant EPTDS	2013/07/17	0.03
5	Artesian Water Supply	DE	Wilmington Manor 1 Plant EPTDS	2014/01/28	0.03
6	City of Vienna	WV	Treatment plant for Well 11/14 (EP#3 after treatment)	2015/05/27	0.129
6	City of Vienna	WV	Treatment plant for Well 7/8 (EP #1 after treatment)	2015/05/27	0.0991
6	City of Vienna	WV	Treatment plant for Well 9/10 (EP #2 after treatment)	2015/05/27	0.0758
7	Warrington Township Water & Sewer Department	PA	Wells 1, 2 & 6 EP	2014/11/11	0.11839
7	Warrington Township Water & Sewer Department	PA	Well 3 EP	2014/11/11	0.02015
7	Warrington Township Water & Sewer Department	PA	Well 9 EP	2015/05/11	0.02883

No.	PWS	State	Sample Location	Date	Results
8	West Morgan-East Lawrence	AL	Tenn. River + Treatment	2014/02/05	0.1
9	Security WSD	CO	EPTDS from Air Stripping Plant	2014/07/31	0.09
9	Security WSD	CO	EPTDS from Air Stripping Plant	2014/01/29	0.08
9	Security WSD	CO	EPTDS from Chlorinator for W9 Well	2014/07/30	0.08
9	Security WSD	CO	EPTDS from Chlorinator for W9 Well	2014/01/15	0.07
9	Security WSD	CO	EPTDS from Chlorinator for W8 Well	2014/07/31	0.08
9	Security WSD	CO	EPTDS from Chlorinator for W8 Well	2014/01/15	0.08
9	Security WSD	CO	EPTDS from CT 150,000 Gallon Steel Tank	2014/07/15	0.07
9	Security WSD	CO	EPTDS from CT 150,000 Gallon Steel Tank	2014/01/22	0.07
9	Security WSD	CO	EPTDS from Chlorinator for S16 Well	2014/07/30	0.07
9	Security WSD	CO	EPTDS from Chlorinator for S16 Well	2014/01/22	0.06
9	Security WSD	CO	EPTDS from Chlorinator for S15 Well	2014/07/30	0.07
9	Security WSD	CO	EPTDS from Chlorinator for S15 Well	2014/01/21	0.06

No.	PWS	State	Sample Location	Date	Results
9	Security WSD	CO	EPTDS from Chlorinator for R2 Well	2014/07/29	0.06
9	Security WSD	CO	EPTDS from Chlorinator for R2 Well	2014/01/14	0.06
9	Security WSD	CO	EPTDS from Chlorinator for W12 Well	2014/07/30	0.05
9	Security WSD	CO	EPTDS from Chlorinator for W12 Well	2014/01/15	0.04
9	Security WSD	CO	EPTDS from Chlorinator for FV4 Well	2014/07/29	0.05
9	Security WSD	CO	EPTDS from Chlorinator for FV4 Well	2014/01/14	0.05
9	Security WSD	CO	EPTDS from Chlorinator for S9 Well	2014/01/14	0.05
9	Security WSD	CO	EPTDS from Chlorinator for S9 Well	2014/07/29	0.04
9	Security WSD	CO	EPTDS from Chlorinator for S2 Well	2014/07/29	0.05
9	Security WSD	CO	EPTDS from Chlorinator for S2 Well	2014/01/21	0.05
9	Security WSD	CO	EPTDS from Chlorinator for S8 Well	2014/07/30	0.04
9	Security WSD	CO	EPTDS from Chlorinator for S10 Well	2014/07/30	0.04

No.	PWS	State	Sample Location	Date	Results
9	Security WSD	CO	EPTDS from Chlorinator for S10 Well	2014/01/14	0.04
9	Security WSD	CO	EPTDS from Chlorinator for R1 Well	2014/01/14	0.04
9	Security WSD	CO	EPTDS from Chorinator for R1 Well	2014/08/11	0.04
9	Security WSD	CO	EPTDS from FVA Intertie	2014/07/30	0.03
9	Security WSD	CO	EPTDS from Chlorinator for S11 Well	2014/07/30	0.03
9	Security WSD	CO	EPTDS from Chlorinator for S11 Well	2014/04/22	0.03
9	Security WSD	CO	EPTDS from Chlorinator for S7 Well	2014/07/30	0.03
9	Security WSD	CO	EPTDS from Chlorinator for S7 Well	2014/01/22	0.03
9	Security WSD	CO	EPTDS from Chlorinator for S4 Well	2014/07/30	0.03
9	Security WSD	CO	EPTDS from Chlorinator for S4 Well	2014/02/10	0.03
10	VAW Water System, Inc.	AL	West Morgan-East Lawrence EPTDS	2014/02/18	0.09
10	VAW Water System, Inc.	AL	West Morgan-East Lawrence EPTDS	2014/05/28	0.02
10	VAW Water System, Inc.	AL	West Morgan-East Lawrence Intertie EPTDS	2014/08/05	0.02

No.	PWS	State	Sample Location	Date	Results
11	Town of Cumberland	RI	Abbott Run #1 TP EPTDS	2015/02/18	0.081
12	Dover Water Department	NH	Griffin Well Treatment Plant (finished water sample)	2014/03/24	0.067
13	Emerald Coast Utilities Authority	FL	Hagler (EP tap)	2014/11/11	0.065
13	Emerald Coast Utilities Authority	FL	Hagler (EP tap)	2014/06/17	0.043
13	Emerald Coast Utilities Authority	FL	Bronson East (EP tap)	2014/05/14	0.024
14	Parkersburg Utility Board	WV	Treatment Plant (Entry Point after Treatment)	2014/03/25	0.0631
14	Parkersburg Utility Board	WV	Treatment Plant (Entry Point after Treatment)	2014/09/09	0.0412
15	Hyannis Water System	MA	Mary Dunn Well 2 EPTDS	2014/05/22	0.062
15	Hyannis Water System	MA	Mary Dunn Well 2 EPTDS	2013/11/20	0.02
15	Hyannis Water System	MA	Maher Treatment Plant EPTDS	2014/05/22	0.02
15	Hyannis Water System	MA	Mary Dunn Well 3 EPTDS	2014/05/22	0.02
16	Colbert County Rural Water System	AL	West Lawrence (WL meter)	2014/12/10	0.06
16	Colbert County Rural Water System	AL	West Lawrence (WL meter)	2014/06/16	0.02
16	Colbert County Rural Water System	AL	Cherokee (North Pike meter)	2014/06/16	0.02
16	Colbert County Rural Water System	AL	Muscle Shoals (Muscle Shoals meter)	2014/06/16	0.02

No.	PWS	State	Sample Location	Date	Results
17	Rome	GA	Bruce Hamler Water Plant (finished water tap)	2015/02/12	0.06
17	Rome	GA	Bruce Hamler Water Plant (finished water tap)	2015/05/12	0.05
18	Freeport	IL	Filter Plant EPTDS	2014/12/10	0.059
19	South Orange Water Dept.	NJ	Meadowbrook Well TP EPTDS	2015/03/25	0.058
20	New Jersey American (Raritan)	NJ	Hummocks Station Plant EPTDS	2015/03/09	0.054
20	New Jersey American (Raritan)	NJ	Quinton Avenue Well (Kenilworth)	2015/03/13	0.035
20	New Jersey American (Raritan)	NJ	Springfield Plant EPTDS	2015/03/03	0.034
20	New Jersey American (Raritan)	NJ	Clinton Avenue Well EPTDS	2015/03/13	0.032
20	New Jersey American (Raritan)	NJ	Charles St. Comp. Del. EPTDS	2015/03/09	0.028
20	New Jersey American (Raritan)	NJ	Rock Avenue Well (Piscataway)	2015/03/11	0.026
20	New Jersey American (Raritan)	NJ	Netherwood Plant EPTDS	2015/03/03	0.023
20	New Jersey American (Raritan)	NJ	Greenbrook Plant (Jefferson Ave.)	2015/03/03	0.02

No.	PWS	State	Sample Location	Date	Results
21	Eastern Municpal Water District	CA	Well 59 (Indian Ave.) EP #82: Treated	2013/06/18	0.053
21	Eastern Municpal Water District	CA	Well 59 (Indian Ave.) EP #82: Treated	2013/10/08	0.044
22	Fort Lewis Water - Cantonment	WA	Well 17 EPTDS	2014/12/29	0.0511
23	City of Tempe	AZ	EPDS001	2014/01/14	0.05
23	City of Tempe	AZ	EPDS001	2013/07/16	0.044
23	City of Tempe	AZ	EPDS004	2013/07/16	0.032
23	City of Tempe	AZ	EPDS004	2014/01/14	0.023
24	Liberty Water LPSCO (AZ)	AZ	Well 02 Airline POE	2014/11/18	0.05
24	Liberty Water LPSCO (AZ)	AZ	Well 02 Airline POE	2014/05/29	0.05
25	Hudson Water Supply	MA	Chesnut St. WTP (EPTDS)	2015/04/30	0.05
26	West Lawrence Water Co-op	AL	West Morgan-East Lawrence Water Intertie (EPTDS)	2015/05/26	0.05
26	West Lawrence Water Co-op	AL	West Morgan-East Lawrence Water Intertie (EPTDS)	2015/02/16	0.03
27	United Water Delaware	DE	Stanton Plant DEP	2014/11/06	0.0499
27	United Water Delaware	DE	Stanton Plant DEP	2014/02/14	0.0263
28	Montclair Water Bureau	NJ	Glenfield Well (Maple Ave.)	2014/11/21	0.0485
28	Montclair Water Bureau	NJ	Glenfield Well (Maple Ave.)	2015/05/21	0.04212
28	Montclair Water Bureau	NJ	Rand Well (Fullerton Ave.)	2014/11/21	0.04653

No.	PWS	State	Sample Location	Date	Results
28	Montclair Water Bureau	NJ	Rand Well (Fullerton Ave.)	2015/05/21	0.04274
28	Montclair Water Bureau	NJ	Lorraine Well (Lorraine Ave. & N. Mt. Ave.)	2014/11/21	0.04405
28	Montclair Water Bureau	NJ	Lorraine Well (Lorraine Ave. & N. Mt. Ave.)	2015/05/21	0.03532
29	Town of Hempstead Water District	NY	Bowling Green TP EPTDS	2014/06/09	0.048
29	Town of Hempstead Water District	NY	Bowling Green TP EPTDS	2015/01/30	0.045
30	Widefield WSD	CO	Chlorinator for W3 Well (EPTDS)	2014/05/14	0.048
30	Widefield WSD	CO	Chlorinator for W3 Well (EPTDS)	2013/11/12	0.031
30	Widefield WSD	CO	Chlorinator for W4 Well (EPTDS)	2014/05/19	0.045
30	Widefield WSD	CO	Chlorinator for W4 Well (EPTDS)	2013/11/12	0.037
30	Widefield WSD	CO	Chlorinator for E2 Well (EPTDS)	2014/05/14	0.036
30	Widefield WSD	CO	Chlorinator for W7 Well (EPTDS)	2014/05/14	0.036
30	Widefield WSD	CO	Chlorinator for C36 Well (EPTDS)	2013/11/06	0.03
30	Widefield WSD	CO	Chlorinator & Aeration for Well W1 (EPTDS)	2013/11/20	0.024
30	Widefield WSD	CO	Chlorinator & Aeration for Well W1 (EPTDS)	2014/06/19	0.021
31	Town of Payson	AZ	Country Club #2 Well (EPTDS)	2014/06/09	0.048

No.	PWS	State	Sample Location	Date	Results
31	Town of Payson	AZ	Country Club #2 Well (EPTDS)	2014/12/10	0.042
31	Town of Payson	AZ	Lake Drive Well (EPTDS)	2014/06/04	0.042
31	Town of Payson	AZ	Lake Drive Well (EPTDS)	2014/12/15	0.04
31	Town of Payson	AZ	Mountain View Well (EPTDS)	2014/06/26	0.038
31	Town of Payson	AZ	Mountain View Well (EPTDS)	2014/12/10	0.036
31	Town of Payson	AZ	Country Club #1 Well (EPTDS)	2014/12/10	0.02
32	Orange Water Dept.	NJ	Gist Place (EPTDS)	2015/02/03	0.044
33	West Bend Waterworks	WI	(Tower 2 Air Stripper)	2014/11/03	0.04317
33	West Bend Waterworks	WI	Treatment Plant (Tower 1 Air Stripper)	2014/05/12	0.02393
33	West Bend Waterworks	WI	Treatment Plant (Tower 2 Air Stripper)	2014/05/12	0.02155
34	Westfield Water Dept.	MA	Well 7 EPTDS	2013/02/27	0.043
35	Atlantic City MUA	NJ	Treatment Plant	2015/02/25	0.043
36	Merrimack Village District	NH	Pump Station 5 (Blend 4 EPTDS)	2014/04/14	0.042
37	City of Corona	CA	R-3 Blend (EP #35: SB-9)	2014/07/10	0.042
37	City of Corona	CA	R-3 Blend (EP #35: SB-9)	2014/01/16	0.038
37	City of Corona	CA	19579 Temescal Canyon Rd. (D3-1)	2014/01/16	0.036

No.	PWS	State	Sample Location	Date	Results
37	City of Corona	CA	19579 Temescal Canyon Rd. (D3-1)	2013/10/09	0.023
37	City of Corona	CA	Ontario/Garretson Zone 3 (SB-3)	2013/10/23	0.029
37	City of Corona	CA	Lester SWTP (EP#19: SS-1)	2014/01/15	0.026
38	Elsinore Valley Municpal Water District	CA	Canyon Lake WTP (EP#2: Combined Effluent)	2014/12/23	0.041
38	Elsinore Valley Municpal Water District	CA	Canyon Lake WTP (EP#2: Combined Effluent)	2013/09/19	0.038
38	Elsinore Valley Municpal Water District	CA	Canyon Lake WTP (EP#2: Combined Effluent)	2014/06/19	0.038
38	Elsinore Valley Municpal Water District	CA	Canyon Lake WTP (EP#2: Combined Effluent)	2014/10/24	0.035
39	Hastings	MN	Well 8 Entry Point	2014/12/10	0.0405
39	Hastings	MN	Well 8 Entry Point	2015/06/09	0.0324
40	Bloomington	IL	Plant on South Side of Dam (EPTDS from plant)	2013/03/06	0.04
41	City of Fountain	CO	Fountain Valley Authority Intertie (EPTDS)	2013/04/16	0.04
41	City of Fountain	CO	Chlorination for Well 4 (EPTDS)	2013/07/17	0.04
41	City of Fountain	CO	Chlorination for Well 4 (EPTDS)	2013/01/15	0.03
41	City of Fountain	CO	Chlorination for Well 2 (EPTDS)	2013/07/17	0.04
41	City of Fountain	CO	Chlorination for Well 2 (EPTDS)	2013/01/15	0.04

No.	PWS	State	Sample Location	Date	Results
41	City of Fountain	CO	Chlorination for Well 1 (EPTDS)	2013/07/17	0.04
41	City of Fountain	CO	Chlorination for Well 1 (EPTDS)	2013/01/15	0.04
41	City of Fountain	CO	Chlorination for Well 3 (EPTDS)	2013/07/17	0.03
41	City of Fountain	CO	Chlorination for Well 3 (EPTDS)	2013/01/15	0.02
42	Southside Waterworks/ Rainbow City Utilities Board (Gadsden, AL)	AL	Gadsden Water Intertie (EPTDS)	2013/04/15	0.04
42	Southside Waterworks/ Rainbow City Utilities Board (Gadsden, AL)	AL	Gadsden Water Intertie (EPTDS)	2013/07/22	0.04
42	Southside Waterworks/ Rainbow City Utilities Board (Gadsden, AL)	AL	Gadsden Water Intertie (EPTDS)	2013/10/21	0.03
42	Southside Waterworks/ Rainbow City Utilities Board (Gadsden, AL)	AL	Gadsden Water Intertie (EPTDS)	2013/01/21	0.03
42	Southside Waterworks/ Rainbow City Utilities Board (Gadsden, AL)	AL	Gadsden Water Intertie (EPTDS)	2013/04/15	0.03
42	Southside Waterworks/ Rainbow City Utilities Board (Gadsden, AL)	AL	Gadsden Water Intertie (EPTDS)	2013/10/21	0.03
42	Southside Waterworks/ Rainbow City Utilities Board (Gadsden, AL)	AL	Gadsden Water Intertie (EPTDS)	2013/07/08	0.02
42	Southside Waterworks/ Rainbow City Utilities Board (Gadsden, AL)	AL	Gadsden Water Intertie (EPTDS)	2014/01/15	0.02
43	Gadsden Waterworks & Sewer Board	AL	Water Treatment Plant	2014/10/02	0.04
43	Gadsden Waterworks & Sewer Board	AL	Water Treatment Plant	2015/04/21	0.03
43	Gadsden Waterworks & Sewer Board	AL	Water Treatment Plant	2014/01/27	0.02
44	Fort Drum	NY	Water Treatment Plant (WTP01EP)	2015/03/04	0.04
44	Fort Drum	NY	Water Treatment Plant (WTP01EP)	2014/09/08	0.03

No.	PWS	State	Sample Location	Date	Results
45	Northeast Alabama Water System	AL	Centre Water (MM)	2015/03/12	0.04
45	Northeast Alabama Water System	AL	Centre Water (MM)	2015/02/27	0.04
45	Northeast Alabama Water System	AL	Centre Water (MM)	2014/06/12	0.03
45	Northeast Alabama Water System	AL	Centre Water (MM)	2014/12/16	0.03
45	Northeast Alabama Water System	AL	Albertville (MM)	2015/03/12	0.02
46	Bethany	OK	Water Treatment Plant EP	2015/05/11	0.04
46	Bethany	OK	Water Treatment Plant EP	2015/02/10	0.034
47	United Water PA	PA	Airport EPTDS	2014/02/04	0.038
48	Woodbury	MN	Well 9 Entry Point	2015/0415	0.0373
48	Woodbury	MN	Well 9 Entry Point	2014/10/14	0.0279
49	Fair Lawn Water Dept.	NJ	Westmoreland TP (EPTDS)	2013/01/29	0.03678
49	Fair Lawn Water Dept.	NJ	Westmoreland TP (EPTDS)	2013/07/30	0.0304
49	Fair Lawn Water Dept.	NJ	Water Comm. (EPTDS)	2013/11/26	0.03138
49	Fair Lawn Water Dept.	NJ	Dorothy St. TP (EPTDS)	2013/11/26	0.02699
49	Fair Lawn Water Dept.	NJ	Dorothy St. TP (EPTDS)	2013/05/28	0.0253
49	Fair Lawn Water Dept.	NJ	Well 28 TP (treatment house)	2013/01/29	0.02684
49	Fair Lawn Water Dept.	NJ	Well 28 TP (treatment house)	2013/07/30	0.02062
49	Fair Lawn Water Dept.	NJ	Cadmus TP (EPTDS)	2013/07/30	0.0217

No.	PWS	State	Sample Location	Date	Results
50	United Water - Rahway	NJ	Treatment Plant (EPTDS)	2014/10/07	0.033
50	United Water - Rahway	NJ	Treatment Plant (EPTDS)	2014/07/21	0.025
50	United Water - Rahway	NJ	Treatment Plant (EPTDS)	2014/04/08	0.023
50	United Water - Rahway	NJ	Treatment Plant (EPTDS)	2014/01/15	0.022
51	Oatman Water Company	AZ	Well #1 (Finished Tap Water)	2014/10/29	0.032
51	Oatman Water Company	AZ	Well #1 (Finished Tap Water)	2014/04/12	0.03
52	Garfield Water Dept.	NJ	Well 8C Treatment Plant	2014/04/28	0.031
52	Garfield Water Dept.	NJ	Well 8C Treatment Plant	2013/10/21	0.026
52	Garfield Water Dept.	NJ	Elmwood Park Station Treatment Plant	2014/04/28	0.03
52	Garfield Water Dept.	NJ	Elmwood Park Station Treatment Plant	2013/10/21	0.026
53	CA American Water Co. - Suburban	CA	Nut Plains (EP #35: Treated)	2014/10/08	0.031
53	CA American Water Co. - Suburban	CA	Nut Plains (EP #35: Treated)	2015/04/07	0.026
54	Orange Water & Sewer Authority	NC	Plant Clearwell (Effluent)	2014/02/11	0.03
55	Calhoun	GA	Mauldin Rd. Water Plant (finished water tap)	2015/02/04	0.03

No.	PWS	State	Sample Location	Date	Results
55	Calhoun	GA	Brittany Drive Water Plant (finished water tap)	2015/08/05	0.02
55	Calhoun	GA	Brittany Drive Water Plant (finished water tap)	2015/05/11	0.02
55	Calhoun	GA	Brittany Drive Water Plant (finished water tap)	2015/02/03	0.02
56	City of DuPont Water System	WA	Hoffman Hill Well #1 (EPTDS)	2014/10/13	0.03
56	City of DuPont Water System	WA	Hoffman Hill Well #2 (EPTDS)	2014/10/13	0.027
56	City of DuPont Water System	WA	Hoffman Hill Well #2 (EPTDS)	2014/04/08	0.024
57	Town of Fuquay-Varina	NC	Water Intertie (EPTDS)	2014/09/23	0.03
57	Town of Fuquay-Varina	NC	Water Intertie (EPTDS)	2014/12/10	0.02
58	Summerville	GA	Summerville Water Plant, (finished water tap)	2015/02/24	0.03
59	Middlesex Water Company	NJ	Park Ave. Treatment (EPTDS)	2013/10/15	0.02926
59	Middlesex Water Company	NJ	Park Ave. Treatment (EPTDS)	2014/04/14	0.02234
59	Middlesex Water Company	NJ	South Tingley Lane (EPTDS)	2013/10/15	0.02859
60	City of Orange	CA	Well 3 (EP#36: EP-O3-01)	2014/05/14	0.0291

No.	PWS	State	Sample Location	Date	Results
60	City of Orange	CA	Well 3 (EP#36: EP-O3-01)	2014/10/13	0.0268
60	City of Orange	CA	Well 15 (EP #40: EP-O15-01)	2014/05/14	0.0261
60	City of Orange	CA	Well 15 (EP #40: EP-O15-01)	2014/10/13	0.0252
60	City of Orange	CA	Well 23 (EP#46: EP-O23-01)	2014/10/14	0.0224
60	City of Orange	CA	Wells 4 & 5 Blend (EP#24: RES-OWATER-01)	2014/10/14	0.021
61	City of Pico Rivera Water Dept.	CA	Well 4 (EP #23: Well 4 Treated)	2014/07/30	0.029
61	City of Pico Rivera Water Dept.	CA	Well 6 (EP #25: Well 6 Treated)	2014/06/04	0.027
61	City of Pico Rivera Water Dept.	CA	Well 12 (EP #20: Well 12 Treated)	2013/01/24	0.021
61	City of Pico Rivera Water Dept.	CA	Well 11 (EP #19: Well 11 Treated)	2013/01/24	0.02
61	City of Pico Rivera Water Dept.	CA	Well 7 (EP #26: Well 7 Treated)	2014/07/30	0.02
62	Atascadero Mutual Water Company	CA	Treatment Bldg. C (EP #53)	2013/10/30	0.028
63	Camp Pendleton (South)	CA	Chlorin. Station 410618 (Eff.)	2014/05/21	0.028
63	Camp Pendleton (South)	CA	Chlorin. Station 410618 (Eff.)	2013/11/13	0.021
64	City of Anaheim	CA	Well 46 (EP #72)	2015/01/06	0.028

No.	PWS	State	Sample Location	Date	Results
64	City of Anaheim	CA	Well 41 (EP #74)	2015/01/06	0.023
64	City of Anaheim	CA	Well 55 (EP#57)	2015/01/07	0.02
65	Cottage Grove	MN	Combined Discharge (EPTDS)	2013/08/28	0.0278
66	Santa Clarita Water Division	CA	Santa Clara Well (EP #52: Treated)	2014/04/09	0.027
67	Ridgewood Water	NJ	Russell (EPTDS)	2015/06/04	0.027
67	Ridgewood Water	NJ	Russell (EPTDS)	2014/06/25	0.025
67	Ridgewood Water	NJ	Lakeview (EPTDS)	2015/06/04	0.024
67	Ridgewood Water	NJ	Stevens (EPTDS)	2014/06/30	0.024
67	Ridgewood Water	NJ	Stevens (EPTDS)	2014/12/29	0.023
67	Ridgewood Water	NJ	Fairview (EPTDS)	2015/06/04	0.022
67	Ridgewood Water	NJ	Carr Treatment Plant (EPTDS)	2014/06/25	0.022
67	Ridgewood Water	NJ	Weisch (EPTDS)	2014/06/30	0.022
67	Ridgewood Water	NJ	Weisch (EPTDS)	2014/12/15	0.02
67	Ridgewood Water	NJ	Meer Treatment House (EPTDS)	2014/12/15	0.026
67	Ridgewood Water	NJ	Meer Treatment House (EPTDS)	2014/06/25	0.024
67	Ridgewood Water	NJ	Prospect (EPTDS)	2014/06/30	0.025
67	Ridgewood Water	NJ	Prospect (EPTDS)	2014/12/17	0.025
67	Ridgewood Water	NJ	Irving (EPTDS)	2014/12/17	0.025
67	Ridgewood Water	NJ	Irving (EPTDS)	2014/06/30	0.024

No.	PWS	State	Sample Location	Date	Results
67	Ridgewood Water	NJ	Glen Rock (EPTDS)	2014/06/30	0.023
67	Ridgewood Water	NJ	Glen Rock (EPTDS)	2014/12/17	0.02
67	Ridgewood Water	NJ	East Ridgewood Treatment Facility (EPTDS)	2014/12/29	0.021
67	Ridgewood Water	NJ	East Ridgewood Treatment Facility (EPTDS)	2014/06/30	0.02
67	Ridgewood Water	NJ	Lafayette (EPTDS)	2014/06/30	0.02
67	Ridgewood Water	NJ	Cedar Hills Wells (EPTDS)	2014/12/29	0.02
67	Ridgewood Water	NJ	Mountain Treatment House (EPTDS)	2014/06/25	0.02
68	Aqua OH - Struthers	OH	Poland Filtration Plant (EPTDS)	2014/10/13	0.027
69	City of Port Lavaca	TX	GBRA Water Intertie (EPTDS)	2014/02/24	0.0264
70	Aqua PA - Bristol	PA	Filter Plant 2 (Edgely Wellfield)	2013/12/20	0.026
70	Aqua PA - Bristol	PA	Filter Plant 2 (Edgely Wellfield)	2014/06/20	0.02
71	Aqua NJ - Blackwood	NJ	Treatment Plant (Well 20)	2014/04/17	0.026
72	City of Norco	CA	MWD Intertie (EPTDS)	2015/02/03	0.026
73	Valencia Water Company	CA	Well N-7 (EP #54: Treated)	2014/10/16	0.026

No.	PWS	State	Sample Location	Date	Results
73	Valencia Water Company	CA	Well S-8 (EP #44: Treated)	2014/10/16	0.024
73	Valencia Water Company	CA	Well N (EP #29: Treated)	2014/05/28	0.024
73	Valencia Water Company	CA	Well S-6 (EP #43: Treated)	2014/03/12	0.024
73	Valencia Water Company	CA	Well S-7 (EP #45: Treated)	2014/03/12	0.023
74	Yorba Linda Water District	CA	Highland Reservoir (EP 12: RES-YLWDHIGHLAND-01)	2013/07/10	0.0259
74	Yorba Linda Water District	CA	Highland Reservoir (EP 12: RES-YLWDHIGHLAND-01)	2013/01/09	0.0241
75	Danvers Water Dept.	MA	Well 1 Treatment Facility (EPTDS)	2013/11/19	0.025
75	Danvers Water Dept.	MA	Well 1 Treatment Facility (EPTDS)	2014/05/21	0.022
76	City of Lathrop	CA	Well 21 (EP #14: Treated)	2013/04/17	0.025
76	City of Lathrop	CA	Well 21 (EP #14: Treated)	2013/10/15	0.02
77	Wright-Patterson AFB Area A/C	OH	Building 10855-STU 1 (EPTDS)	2014/10/07	0.025
78	Point Pleasant Water Dept.	NJ	Bricktown Intertie (MUA)	2013/11/13	0.02455
78	Point Pleasant Water Dept.	NJ	Bricktown Intertie (MUA)	2013/09/16	0.0244
78	Point Pleasant Water Dept.	NJ	Connection (NJEMS 15-201)	2015/06/10	0.02

No.	PWS	State	Sample Location	Date	Results
79	Woodruff Roebuck Water District	SC	Woodruff-Roebuck WTP (1101 Kitchens Rd.)	2014/09/02	0.024
80	Galesburg	IL	Water Treatment Plant (EPTDS)	2013/10/14	0.02338
81	Moore County Public Utilities	NC	EMWD Intertie (EPTDS)	2013/05/16	0.023
82	Hawthorne Water Dept.	NJ	S. Station Tower 1 (Wagaraw Wellfield)	2013/05/22	0.023
82	Hawthorne Water Dept.	NJ	Utter Ave. Treatment (EPTDS)	2013/05/22	0.023
82	Hawthorne Water Dept.	NJ	N. Station Goffle Field (EPTDS)	2013/05/22	0.022
83	Lakewood Township MUA	NJ	BTMUA Intertie (EPTDS)	2014/08/27	0.0225
83	Lakewood Township MUA	NJ	BTMUA Intertie (EPTDS)	2015/02/19	0.0204
84	Washington County Service Authority	VA	BVUB - Exit 14 (EPDS)	2014/05/06	0.0222
85	Wallington Water Dept.	NJ	Passaic Valley Water Comm. (NJEMS 02-050)	2013/11/13	0.022
86	Issaquah Water System	WA	Gillman Well 4 (EPTDS)	2013/07/22	0.0215
86	Issaquah Water System	WA	Gillman Well 4 (EPTDS)	2014/01/08	0.02
87	New Windsor Consolidated Water District	NY	Newburgh City Water Intertie (EPTDS)	2014/12/05	0.0215

No.	PWS	State	Sample Location	Date	Results
88	Chatsworth	GA	Eaton Spring Plant (Finished Water Tap)	2015/06/15	0.02148
89	Hartford County DPW	MD	Perryman WTP (POE)	2013/09/03	0.02123
90	Brookwood Comm. Water System	NC	Well 39 (EPTDS)	2014/02/25	0.021
90	Brookwood Comm. Water System	NC	Well 34 (EPTDS)	2014/02/24	0.02
91	City of Miramar	FL	East Plant (POE)	2014/07/10	0.021
92	Orchard Dale Water District	CA	Mills (EPTDS)	2014/07/21	0.021
93	Gallia County Rural Water Association	OH	Gallia Co. WTP (EPTDS)	2014/09/04	0.02044
94	Consolidated Utility District of Rutherford	TN	E. Fork Stones River Plant (EPTDS)	2013/11/12	0.02
95	Louisville Water Company	KY	BE Payne Water Treatment (plant tap EPTDS)	2013/02/11	0.02
95	Louisville Water Company	KY	Crescent Hill Filter Plant (plant tap EPTDS)	2013/08/12	0.02
96	Florence Water-Wastewater Dept.	AL	Wilson Lake + Treatment Plant	2014/04/16	0.02
97	Albertville Utilities Board	AL	Old Plant (12 MGD)	2015/01/29	0.02
97	Albertville Utilities Board	AL	New Plant (9 MGD)	2015/01/29	0.02

No.	PWS	State	Sample Location	Date	Results
98	Boaz Water & Sewer Board	AL	Albertville Utilities Intertie (EPTDS)	2015/01/27	0.02
99	Clanton Water Department	AL	Clanton Water Treatment Plant	2014/11/24	0.02
100	Harnett County Department of Public Utilities	NC	Clearwell (Eflluent)	2014/09/17	0.02
100	Harnett County Department of Public Utilities	NC	Clearwell (Eflluent)	2014/12/10	0.02
101	City of Dunn	NC	A.B. Uzzell WTP (EPTDS)	2015/06/17	0.02

United States Environmental Protection Agency

The Third Unregulated Contaminant Monitoring Rule (UCMR 3): Data Summary

January 20

EPA uses the Unregulated Contaminant Monitoring Rule (UCMR) program to collect data for contaminants suspected to be present in drinking water, but that do not have health-based standards set under the Safe Drinking Water Act (SDWA). Every five years EPA develops a new list of UCMR contaminants, largely based on the Contaminant Candidate List (CCL). The SDWA Amendments of 1996 provide for:

- Monitoring no more than 30 contaminants per 5-year cycle
- Monitoring only a representative sample of public water systems serving less than or equal to 10,000 people
- Storing analytical results in a National Contaminant Occurrence Database (NCOD)

This dataset represents the ninth NCOD release of analytical results for UCMR 3. Updates will occur approximately quarterly and EPA anticipates that additional reference material will be made available to assist with the assessment of the UCMR 3 data. For more information about UCMR 3, please visit our website: http://water.epa.gov/lawsregs/rulesregs/sdwa/ucmr/ucmr3/index.cfm. Information regarding many of the UCMR 3 contaminants (including a description of their use) may also be found at the CCL website: http://water.epa.gov/scitech/drinkingwater/dws/ccl/ccl3.cfm#chemical.

UCMR 3 Data Considerations

- This dataset is not complete. UCMR 3 monitoring occurred through December 2015, and data are expected to be reported to EPA through the summer of 2016.
- Data are added and possibly removed or updated over the course of this reporting cycle. These results are subject to change following further review by the analytical laboratory, the public water system, the State and EPA.
- Data are presented as method-specific text files (UCMR3_200_8.txt, UCMR3_218_7.txt, UCMR3_300_1.txt, UCMR3_522.txt, UCMR3_524_3.txt, UCMR3_537.txt, UCMR3_539.txt, EPA_1615A, EPA_1615B, EPA_1615C, EPA_1615D, EPA_1615E, SM_9223B, ASTM_D6503_99, SM_9218, EPA_1602), one text file containing disinfectant residual type (UCMR3_DRT.txt), one text file containing the U.S. Postal Service Zip Code(s) for all areas served by a PWS (UCMR3_ZipCodes.txt) and one text file containing all UCMR 3 data to date (UCMR3_All.txt).
- These text files are tab delimited and have no text qualifier. Field names are included in the first row of each file.
- If you wish to perform additional data analyses, EPA suggests you import each field into your choice of software as text. Some of the IDs can be misinterpreted as long integer field types when they actually contain alpha characters.
- Samples collected at the maximum residence time in the distribution system (MR) are required to be analyzed for metals (including chromium-6) and chlorate.
- Water systems monitoring for Method 300.1 (chlorate) report disinfectant types.
- Population categories are based on retail population as indicated by the Safe Drinking Water Information System (Federal) (SDWIS/FED) as of December 31, 2010.
- In addition to reporting occurrence data for UCMR 3 target analytes, EPA tasked its small-system contract-support laboratories with reporting results for sec-butylbenzene, n-propylbenzene, tellurium, germanium and manganese. These additional unregulated analytes are within the scope of the methods already being performed for the UCMR analytes.

UCMR 3 Data Field Names and Definitions

Field Name	Definition
PWSID	Public Water System Identification Code, 9-character identification code (Begins with the standard 2-character postal State abbreviation or Region code, and the remaining seven numbers are unique to each PWS in the state)
PWSName	Name of the Public Water System (PWS)
Size	Size category of the PWS for UCMR, based on retail population as of December 31, 2010
	S: ≤ 10,000
	L: > 10,000
FacilityID	Public Water System Facility Identification Code, 5-digit identification code
FacilityName	Name of the facility at the PWS
FacilityWaterType	Source of water at the facility
	SW: Surface water
	GW: Ground water
	GU: Ground water under the direct influence of surface water
	MX: Any combination of: SW, GW and GU
SamplePointID	Identification code for each sample point location in the PWS
SamplePointName	Name of the sample point for every sample point ID at a PWS
SamplePointType	Sampling Point Type Code
	EP: Entry point to the distribution system
	MR: Distribution system at maximum residence time
AssociatedFacilityID	The facility ID of the associated DS/MRT
AssociatedSamplePointID	The sample point ID of the associated DS/MRT

Field Name	Definition
Disinfectant Type	CLGA: Gaseous Chlorine
	CLOF: Offsite Generated Hypochlorite (stored as liquid)
	CLON: Onsite Generated Hypochlorite (no storage)
	CAGC: Chloramine (formed from gaseous chlorine)
	CAOF: Chloramine (formed from offsite hypochlorite)
	CAON: Chloramine (formed from onsite hypochlorite)
	CLDO: Chlorine Dioxide
	OZON: Ozone
	ULVL: Ultraviolet Light
	OTHD: All other types of disinfectant
	NODU: No Disinfectant Used
CollectionDate	Date of sample collection (month, day, year)
SampleID	Identification code for each sample, as defined by the laboratory
Contaminant	Unregulated contaminant being analyzed in UCMR 3
MRL	Minimum Reporting Level defined by UCMR 3
MethodID	Identification code of the analytical method
AnalyticalResultsSign	Less than (<) the minimum reporting level (MRL) or equal to (=) a numeric value at or above the MRL
AnalyticalResultValue	Numeric value of the analytical result, null values represent less than MRL
SampleEventCode	Identification code for each sample event. Includes sample event one (SE1), sample event two (SE2), sample event three (SE3), and sample event four (SE4).

Field Name	Definition
MonitoringRequirement	AM: Assessment Monitoring (List 1)
	SS: Screening Survey (List 2)
	PST: Pre-Screen Testing (List 3)
Region	EPA Region: States
	1: CT, ME, MA, NH, RI, VT
	2: NJ, NY, PR (Puerto Rico), VI (Virgin Islands)
	3: DE, DC, MD, PA, VA, WV
	4: AL, FL, GA, KY, MS, NC, SC, TN
	5: IL, IN, MI, MN, OH, WI
	6: AR, LA, NM, OK, TX
	7: IA, KS, MO, NE
	8: CO, MT, ND, SD, UT, WY
	9: AZ, CA, HI, NV, AS (American Samoa), GU (Guam), MP (Northern Marianas Islands), NN (Navajo Nation)
	10: AK, ID, OR, WA
State	State abbreviation
ZipCode	U.S. Postal Service zip code(s) for all areas being served water by a PWS

UCMR 3 Chemical Contaminants and Methods

Contaminant	Contaminant Full Name	CAS[1] Number	Method ID	Method Name	Monitoring Requirement
1,2,3-trichloropropane	1,2,3-trichloropropane	96-18-4	524.3	Volatile Organic Compounds	AM
1,3-butadiene	1,3-butadiene	106-99-0	524.3	Volatile Organic Compounds	AM
Chloromethane	methyl chloride	74-87-3	524.3	Volatile Organic Compounds	AM
1,1-dichloroethane	1,1-dichloroethane	75-34-3	524.3	Volatile Organic Compounds	AM
Bromomethane	methyl bromide	74-83-9	524.3	Volatile Organic Compounds	AM
HCFC-22	chlorodifluoromethane	75-45-6	524.3	Volatile Organic Compounds	AM
Halon 1011	bromochloromethane	74-97-5	524.3	Volatile Organic Compounds	AM
1,4-dioxane	1,4-dioxane	123-91-1	522	Synthetic Organic Compound	AM
Vanadium	vanadium	7440-62-2	200.8	Metals	AM
Molybdenum	molybdenum	7439-98-7	200.8	Metals	AM
Cobalt	Cobalt	7440-48-4	200.8	Metals	AM
Strontium	Strontium	7440-24-6	200.8	Metals	AM
Chromium	total chromium	N/A	200.8	Metals	AM
Chromium-6	chromium-6	18540-29-9	218.7	Chromium-6	AM
Chlorate	Chlorate	14866-68-3	300.1	Oxyhalide Anion	AM
PFOS	perfluorooctanesulfonic acid	1763-23-1	537	Perfluorinated Compounds	AM
PFOA	perfluorooctanoic acid	335-67-1	537	Perfluorinated Compounds	AM
PFNA	perfluorononanoic acid	375-95-1	537	Perfluorinated Compounds	AM
PFHxS	perfluorohexanesulfonic acid	355-46-4	537	Perfluorinated Compounds	AM
PFHpA	perfluoroheptanoic acid	375-85-9	537	Perfluorinated Compounds	AM
PFBS	perfluorobutanesulfonic acid	375-73-5	537	Perfluorinated Compounds	AM
17β-estradiol	estradiol	50-28-2	539	Hormones	SS
17α-ethynylestradiol	ethinyl estradiol	57-63-6	539	Hormones	SS
Estriol	16-α-hydroxyestradiol	50-27-1	539	Hormones	SS
Equilin	Equilin	474-86-2	539	Hormones	SS
Estrone	Estrone	53-16-7	539	Hormones	SS
Testosterone	testosterone	58-22-0	539	Hormones	SS
4-androstene-3,17-dione	4-androstene-3,17-dione	63-05-8	539	Hormones	SS

[1]Chemical Abstract Service

UCMR 3 Microbiological Contaminants and Methods

Contaminant	Method ID	Method Name	Monitoring Requirement
Enteroviruses	EPA 1615A	Enterovirus cell culture	PST
Enteroviruses	EPA 1615B	Enterovirus RT-qPCR	PST
Noroviruses	EPA 1615C	Norovirus genogroup I with RT-qPCR primer set A	PST
Noroviruses	EPA 1615D	Norovirus genogroup I with RT-qPCR primer set B	PST
Noroviruses	EPA 1615E	Noroviruses genogroup II	PST
Total coliforms	SM 9223B	Colilert®	PST
E.coli	SM 9223B	Colilert®	PST
Enterococci	ASTM D6503-99	Enterolert®	PST
Aerobic spores	SM 9218	Aerobic endospores	PST
Somatic phage	EPA 1602	Bacteriophage	PST
Male specific phage	EPA 1602	Bacteriophage	PST

UCMR 3 Reference Concentrations for Chemical Contaminants

Under the current cycle of the Unregulated Contaminant Monitoring Rule (UCMR 3) chemicals are being studied at levels that are often significantly below those in prior UCMR cycles. Importantly, UCMR 3 minimum reporting levels (MRLs) were established based on the capability of the analytical method, not based on a level established as "significant" or "harmful." In fact, the UCMR 3 MRLs are often below current "health reference levels" (to the extent that HRLs have been established).

Results of UCMR 3 measurements should be interpreted accordingly. The detection of a UCMR 3 contaminant above the MRL does not represent cause for concern, in and of itself. Rather, the implications of the detection should be judged considering health effects information (which is often still under development or being refined for unregulated contaminants).

The intent of the following table is to identify draft UCMR reference concentrations, where possible, to provide context around the detection of a particular UCMR contaminant above the MRL. The draft reference concentration does not represent an "action level" (EPA requires no particular action[1,2] based simply on the fact that UCMR monitoring results exceed draft reference concentrations), nor should the draft reference concentration be interpreted as any indication of an Agency intent to establish a future drinking water regulation for the contaminant at this or any other level. Decisions as to whether or not to regulate the contaminant in drinking water will continue to be made following the Agency's Regulatory Determination process: [http://water.epa.gov/scitech/drinkingwater/dws/ccl/index.cfm].

The following key principles guided the development of the table:

(1) The reference concentrations are based on publically-available health information found in the following EPA resources: 2012 Drinking Water Standards and Health Advisories, the CCL 3 Contaminant Information Sheets, the Human Health Benchmark for Pesticides (HHBPs), the Integrated Information Risk System (IRIS), or the 2014 Preliminary Regulatory Determinations for Contaminants on CCL 3. The primary/secondary sources of health information vary with respect to scientific rigor from health assessment to single studies and are cited in the table.
(2) If health information was available from more than one of the EPA resources listed above, the most recent health information was used for the draft reference concentrations.
(3) Where both cancer and non-cancer draft reference concentrations existed, the lower (more conservative) of the two concentrations was used. For chemicals with reference concentrations based on a cancer endpoint, the table presents a range of values associated with 10^{-6} to 10^{-4} cancer risk. For chemicals with reference concentrations based on a non-cancer endpoint, the duration of exposure (short-term, intermediate/long-term, chronic) of the toxicity factor (e.g. Reference Dose) used as the basis for the reference concentration is shown.

Recognizing that additional health effects information will become available over time, EPA will periodically update the following table. Those attempting to assess UCMR occurrence data are encouraged to visit EPA's website for the most recent information.

[1] Consumer Confidence Report (CCR) and Public Notification (PN) reporting requirements (see 40 CFR 141.153(d) and 141.207, respectively) apply to public water systems; CCR requires particular reporting based on measurements relative to the UCMR method reporting limits (MRLs) defined in 40 CFR 141.40.
[2]States may establish requirements for drinking water contaminants not yet regulated by EPA, and those requirements may be based on State-established levels that differ from EPA's reference concentrations. Public Water Systems are responsible for being aware of and complying with their State's requirements, if any.

Contaminant	***MRL (µg/L)***	***Reference Concentration (µg/L)***	***Reference Concentration based on a Cancer Endpoint (Y/N)***	***EPA Reference(s)***
Cobalt[1]	1	70	N (intermediate exposure)	CCL 3 Contaminant Information Sheets
Molybdenum[2]	1	40	N (chronic exposure)	2012 Edition of the Health Advisories Table
Strontium[3]	0.3	1,500	N (chronic exposure)	Federal Register Notice for the Preliminary Regulatory Determinations for Contaminants on CCL 3
Vanadium[1,4]	0.2	21	N (intermediate exposure)	CCL 3 Contaminant Information Sheets
Chromium (Total)	0.2	100	N (chronic exposure)	The MCL for the National Primary Drinking Water Regulation
Chromium-6[1]	0.03	NA		
Chlorate	20	210	N (chronic exposure)	CCL 3 Contaminant Information Sheets
1,4-dioxane[5]	0.07	0.35 to 35	Y	2012 Edition of the Health Advisories Table
1,1-dichloroethane[5]	0.03	6.14 to 614	Y	CCL 3 Contaminant Information Sheets
1,2,3-trichloropropane[5,6,7]	0.03	0.0004 to 0.04	Y	2009 IRIS Assessment
1,3-butadiene[5,6]	0.1	0.0103 to 1.03	Y	CCL 3 Contaminant Information Sheets

[1] The contaminant is on the IRIS 20[illegible]2 Agenda for either a new assessment or an updated assessment (Federal Register Notice May 7, 20[illegible]2).
[2] The 20[illegible]2 Edition of the Health Advisories Table and the CCL 3 Contaminant Information Sheets (35 µg/L) have slightly different numbers due to rounding.
[3] The reference concentration has been updated based on the HRL cited in the preliminary regulatory determination for strontium [Docket No. EPAHQ-OW-20[illegible]2-0[illegible]55].
[4] The ATSDR, [illegible]992 used for the CCL 3 Contaminant Information Sheets is no longer publically available and has been replaced by a new assessment (ATSDR, 20[illegible]3).
The minimum risk level (RfD equivalent) was 0.003 mg/kg/day for minor renal effects in an animal study (ATSDR, [illegible]992) compared to 0.0[illegible] mg/kg/day for lack of minor effects in blood pressure, body weight, and hematological parameters in a human study with a [illegible]2 weeks exposure (ATSDR, 20[illegible]3).
[5] Reference Concentration range based on cancer risk of [illegible]$0^{-[illegible]}$ to [illegible]0^{-4}.
[illegible] [illegible]$0^{-[illegible]}$ cancer risk < MRL < [illegible]0^{-4} cancer risk.
[7] To derive the reference concentration, age dependent adjustment factors were applied to the IRIS oral slope factor of 30 per mg/kg-day (calculated using adult exposure data) to address presumed early-life susceptibility for this chemical (per www.epa.gov/cancerguidelines/).

Contaminant	***MRL (µg/L)***	***Reference Concentration (µg/L)***	***Reference Concentration based on a Cancer Endpoint (Y/N)***	***EPA Reference(s)***
HCFC-22 (chlorodifluoromethane)[8]	0.08	NA		
Chloromethane (methyl chloride)[5]	0.2	2.69 to 269	Y	CCL 3 Contaminant Information Sheets
Halon 1011 (bromochloromethane)[9]	0.06	90	N (chronic exposure)	2012 Edition of the Health Advisories Table
Bromomethane (methyl bromide)	0.2	140	N (chronic exposure)	Human Health Benchmark for Pesticides (HHBPs)
PFBS	0.09	NA		
PFHpA	0.01	NA		
PFHxS	0.03	NA		
PFNA	0.02	NA		
PFOS	0.04	0.2	N (short-term exposure)	2012 Edition of the Health Advisories Table
PFOA	0.02	0.4	N (short-term exposure)	2012 Edition of the Health Advisories Table
17α-ethynylestradiol (ethinyl estradiol)[10]	0.0009	0.035	N (chronic exposure)	CCL 3 Contaminant Information Sheets
17β-estradiol (estradiol)[5]	0.0004	0.0009 to 0.09	Y	CCL 3 Contaminant Information Sheets
Equilin	0.004	0.35	N (chronic exposure)	CCL 3 Contaminant Information Sheets
Estriol (16-α-hydroxyestradiol)	0.0008	0.35	N (chronic exposure)	CCL 3 Contaminant Information Sheets
Estrone	0.002	0.35	N (chronic exposure)	CCL 3 Contaminant Information Sheets
4-androstene-3,17-dione	0.0003	NA		
Testosterone	0.0001	NA		

[8] The CCL 3 Contaminant Information Sheets provide a reference level of 3[illegible].5 µg/L; the number is based on a single LOAEL from a [illegible]983 study.

[9] The 20[illegible]2 Edition of the Health Advisories Table and the CCL 3 Contaminant Information Sheets (70 µg/L) have slightly different numbers due to rounding.

[10] This corrects the CCL 3 Contaminant Information Sheets reference level (originally listed as 0.28 µg/L).

Terms

a) UCMR Draft Reference Concentration = The reference concentrations are based on publically-available health information found in the following EPA resources: 2012 Drinking Water Standards and Health Advisories, the CCL 3 Contaminant Information Sheets, the Human Health Benchmark for Pesticides (HHBPs), or the 2014 Preliminary Regulatory Determinations for Contaminants on CCL 3. The primary/secondary sources of health information vary with respect to scientific rigor from health assessment to single studies. Many of the contaminants are currently under regulatory review or development and are subject to change as new health assessments are completed.

b) MRL = UCMR Minimum Reporting Level. *[Note that the Agency for Toxic Substances & Disease Registry (ATSDR) uses the term "MRL" for a different purpose (i.e., to describe "Minimal Risk Levels"). The UCMR term and the ATSDR term have no relationship to each other.]*

c) HRLs = Health Reference Levels. HRLs are not final determinations about the level of a contaminant in drinking water that is necessary to protect any particular population and are derived prior to development of a complete exposure assessment. HRLs are risk derived concentrations against which to evaluate the occurrence data to determine if contaminants occur at levels of potential public health concern.

d) MCL = Maximum Contaminant Level. The highest level of a contaminant allowed in drinking water. MCLs are enforceable standards.

e) Cancer Risk of 10^{-6} to 10^{-4} = the concentration of a contaminant in drinking water corresponding to an excess estimated lifetime cancer risk of one-in-a-million ($1x\ 10^{-6}$) to one-in-ten-thousand (1×10^{-4}). The 2012 Drinking Water Standards and Health Advisories provide the cancer risk at 1×10^{-4}. The CCL 3 Contaminant Information Sheets provide the cancer risk at $1x\ 10^{-6}$.

f) LOAEL = Lowest Observed Adverse Effect Level

g) NA = Not Available

h) Short-term = Typically refers to animal toxicological studies with an exposure duration of days to weeks.

i) Intermediate/Longer-term = Typically refers to animal toxicological studies with an exposure duration of weeks to months.

j) Chronic = Typically refers to animal toxicological studies with an exposure duration of months to years; representing a lifetime exposure in humans.

References

k) 2012 Drinking Water Standards and Health Advisories
l) CCL 3 Contaminant Information Sheets
m) Human Health Benchmark for Pesticides (HHBPs)
n) Announcement of Preliminary Regulatory Determinations for Contaminants on the Third Drinking Water Contaminant Candidate List
o) Integrated Risk Information System (IRIS)

January 2016 UCMR 3 Data Summary for Chemical Contaminants

Contaminant	MRL (µg/L)	Reference Concentration (µg/L)	Total number of results	Number of results ≥MRL	Number of results >Reference Concentration	% of total results >Reference Concentration	Total number of PWSs with results	Number of PWSs with results ≥MRL	Number of PWSs with results >Reference Concentration	% of PWSs with results >Reference Concentration
1,2,3-trichloropropane	0.03	0.0004 / 0.04[1]	34,749	243	243 / 186[1]	0.7% / 0.5%[1]	4,775	64	64 / 53[1]	1.3% / 1.1%[1]
1,3-butadiene	0.1	0.0103 / 1.03[1]	34,749	1	1 / 0[1]	0.003% / 0%[1]	4,775	1	1 / 0[1]	0.02% / 0%[1]
Chloromethane	0.2	2.69 / 269[1]	34,747	261	18 / 0[1]	0.05% / 0%[1]	4,775	130	7 / 0[1]	0.1% / 0%[1]
1,1-dichloroethane	0.03	6.14 / 614[1]	34,747	804	1 / 0[1]	0.003% / 0%[1]	4,775	237	1 / 0[1]	0.02% / 0%[1]
Bromomethane	0.2	140	34,748	110	0	0%	4,775	47	0	0%
HCFC-22	0.08	NA	34,749	799	- -	- -	4,775	278	- -	- -
Halon 1011	0.06	90	34,749	619	0	0%	4,775	297	0	0%
1,4-dioxane	0.07	0.35 / 35[1]	34,684	4,035	1,054 / 0[1]	3% / 0%[1]	4,773	1,041	333/ 0[1]	7% / 0%[1]
Vanadium	0.2	21	59,602	35,776	1,640	2.8%	4,789	3,526	159	3.3%
Molybdenum	1	40	59,607	24,308	135	0.2%	4,789	2,468	35	0.7%
Cobalt	1	70	59,590	809	3	0.005%	4,789	236	3	0.06%
Strontium	0.3	1,500	59,526	59,328	1,643	2.8%	4,789	4,789	275	5.7%
Chromium	0.2	100	59,504	30,171	1	0.002%	4,789	3,558	1	0.02%
Chromium-6	0.03	NA	59,442	44,884	- -	- -	4,787	4,276	- -	- -
Chlorate	20	210	59,357	32,746	9,248	15.6%	4,776	3,281	1,803	37.8%
PFOS	0.04	0.2	35,060	273	33	0.09%	4,788	91	17	0.4%
PFOA	0.02	0.4	35,060	345	0	0%	4,788	107	0	0%
PFNA	0.02	NA	35,060	19	- -	- -	4,788	14	- -	- -
PFHxS	0.03	NA	35,060	195	- -	- -	4,788	54	- -	- -
PFHpA	0.01	NA	35,060	225	- -	- -	4,788	82	- -	- -
PFBS	0.09	NA	35,060	14	- -	- -	4,788	6	- -	- -
17β-estradiol	0.0004	0.0009 / 0.09[1]	10,729	3	1 / 0[1]	0.009% / 0%[1]	1,146	1	1 / 0[1]	0.09% / 0%[1]
17α-ethynylestradiol	0.0009	0.035	10,730	4	0	0%	1,146	4	0	0%
Estriol	0.0008	0.35	10,729	1	0	0%	1,146	1	0	0%
Equilin	0.004	0.35	10,730	0	0	0%	1,146	0	0	0%
Estrone	0.002	0.35	10,730	0	0	0%	1,146	0	0	0%
Testosterone	0.0001	NA	10,729	58	- -	- -	1,146	52	- -	- -
4-androstene-3,17-dione	0.0003	NA	10,730	89	- -	- -	1,146	69	- -	- -

[1]Where two reference concentrations are listed, the first number is associated with a 10^{-6} cancer risk; the second number a 10^{-4} cancer risk.
Where two results are presented the first number is associated with the first reference concentration; the second number is associated with the second reference concentration.

January 2016 UCMR 3 Data Summary for Microbiological Contaminants

Contaminant	MRL	Unit	Total number of results	Number of results ≥MRL	Total number of PWSs with results	Number of PWSs with results ≥MRL
Aerobic spores	1	SFO[1]/100 mL[2]	794	219	772	216
E. coli	1	MPN[3]/100 mL	792	4	770	4
Enterococci	1	MPN/100 mL	791	40	769	40
Enteroviruses (cell culture)	0.002	MPN/L[4]	790	2	768	2
Enteroviruses (RT-qPCR[5])	0.398	GC[6]/L	790	6	768	6
Male specific phage	1	PFU[7]/100 mL	776	11	754	11
Noroviruses GIA[8]	0.398	GC/L	790	2	768	2
Noroviruses GIB[9]	0.398	GC/L	790	1	768	1
Noroviruses GII[10]	0.398	GC/L	790	4	768	4
Somatic phage	1	PFU/100 mL	776	5	754	5
Total coliforms	1	MPN/100 mL	792	41	770	40

[1]SFO = Spore Forming Units
[2]mL = milliliters
[3]MPN = Most Probable Number
[4]L = liters
[5]RT-qPCR = Reverse Transcription-Polymerase Chain Reaction
[6]GC = Genomic Copies
[7]PFU = Plaque Forming Units
[8]Noroviruses GIA = qPCR analysis of Norovirus genogroup I with RT-qPCR primer set A
[9]Noroviruses GIB = qPCR analysis of Norovirus genogroup I with RT-qPCR primer set B
[10]Noroviruses GII = qPCR analysis of Norovirus genogroup II

UCMR 3 Minimum Reporting Levels for Microbiological Contaminants

Under UCMR 3 microbe analytical results are reported as “below”, “at” or “above” MRL. UCMR 3 MRLs were established based on the capability of the analytical method.

It is important to note that microbial contamination can be transient in nature and microbial detections under UCMR 3 should be interpreted in the context of the time samples were collected. However, the presence of any UCMR 3 microbe indicates a potential vulnerability of the PWS to contamination.

United States Environmental Protection Agency

The Third Unregulated Contaminant Monitoring Rule (UCMR 3): Data Summary

October 2015

EPA uses the Unregulated Contaminant Monitoring Rule (UCMR) program to collect data for contaminants suspected to be present in drinking water, but that do not have health-based standards set under the Safe Drinking Water Act (SDWA). Every five years EPA develops a new list of UCMR contaminants, largely based on the Contaminant Candidate List (CCL). The SDWA Amendments of 1996 provide for:

- Monitoring no more than 30 contaminants per 5-year cycle
- Monitoring only a representative sample of public water systems serving less than or equal to 10,000 people
- Storing analytical results in a National Contaminant Occurrence Database (NCOD)

This dataset represents the eighth NCOD release of analytical results for UCMR 3. Updates will occur approximately quarterly and EPA anticipates that additional reference material will be made available to assist with the assessment of the UCMR 3 data. For more information about UCMR 3, please visit our website: http://water.epa.gov/lawsregs/rulesregs/sdwa/ucmr/ucmr3/index.cfm. Information regarding many of the UCMR 3 contaminants (including a description of their use) may also be found at the CCL website: http://water.epa.gov/scitech/drinkingwater/dws/ccl/ccl3.cfm#chemical.

UCMR 3 Data Considerations

- This dataset is not complete. UCMR 3 monitoring occurs through December 2015, and data are expected to be reported to EPA through the summer of 2016.
- Data are added and possibly removed or updated over the course of this reporting cycle. These results are subject to change following further review by the analytical laboratory, the public water system, the State and EPA.
- Data are presented as method-specific text files (UCMR3_200_8.txt, UCMR3_218_7.txt, UCMR3_300_1.txt, UCMR3_522.txt, UCMR3_524_3.txt, UCMR3_537.txt, UCMR3_539.txt, EPA_1615A, EPA_1615B, EPA_1615C, EPA_1615D, EPA_1615E, SM_9223B, ASTM_D6503_99, SM_9218, EPA_1602), one text file containing disinfectant residual type (UCMR3_DRT.txt), one text file containing the U.S. Postal Service Zip Code(s) for all areas served by a PWS (UCMR3_ZipCodes.txt) and one text file containing all UCMR 3 data to date (UCMR3_All.txt).
- These text files are tab delimited and have no text qualifier. Field names are included in the first row of each file.
- If you wish to perform additional data analyses, EPA suggests you import each field into your choice of software as text. Some of the IDs can be misinterpreted as long integer field types when they actually contain alpha characters.
- Samples collected at the maximum residence time in the distribution system (MR) are required to be analyzed for metals (including chromium-6) and chlorate.
- Water systems monitoring for Method 300.1 (chlorate) report disinfectant types.
- Population categories are based on retail population as indicated by the Safe Drinking Water Information System (Federal) (SDWIS/FED) as of December 31, 2010.
- In addition to reporting occurrence data for UCMR 3 target analytes, EPA tasked its small-system contract-support laboratories with reporting results for sec-butylbenzene, n-propylbenzene, tellurium, germanium and manganese. These additional unregulated analytes are within the scope of the methods already being performed for the UCMR analytes.

UCMR 3 Data Field Names and Definitions

Field Name	Definition
PWSID	Public Water System Identification Code, 9-character identification code (Begins with the standard 2-character postal State abbreviation or Region code, and the remaining seven numbers are unique to each PWS in the state)
PWSName	Name of the Public Water System (PWS)
Size	Size category of the PWS for UCMR, based on retail population as of December 31, 2010
	S: ≤ 10,000
	L: > 10,000
FacilityID	Public Water System Facility Identification Code, 5-digit identification code
FacilityName	Name of the facility at the PWS
FacilityWaterType	Source of water at the facility
	SW: Surface water
	GW: Ground water
	GU: Ground water under the direct influence of surface water
	MX: Any combination of: SW, GW and GU
SamplePointID	Identification code for each sample point location in the PWS
SamplePointName	Name of the sample point for every sample point ID at a PWS
SamplePointType	Sampling Point Type Code
	EP: Entry point to the distribution system
	MR: Distribution system at maximum residence time
AssociatedFacilityID	The facility ID of the associated DS/MRT
AssociatedSamplePointID	The sample point ID of the associated DS/MRT

Field Name	Definition
Disinfectant Type	CLGA: Gaseous Chlorine
	CLOF: Offsite Generated Hypochlorite (stored as liquid)
	CLON: Onsite Generated Hypochlorite (no storage)
	CAGC: Chloramine (formed from gaseous chlorine)
	CAOF: Chloramine (formed from offsite hypochlorite)
	CAON: Chloramine (formed from onsite hypochlorite)
	CLDO: Chlorine Dioxide
	OZON: Ozone
	ULVL: Ultraviolet Light
	OTHD: All other types of disinfectant
	NODU: No Disinfectant Used
CollectionDate	Date of sample collection (month, day, year)
SampleID	Identification code for each sample, as defined by the laboratory
Contaminant	Unregulated contaminant being analyzed in UCMR 3
MRL	Minimum Reporting Level defined by UCMR 3
MethodID	Identification code of the analytical method
AnalyticalResultsSign	Less than (<) the minimum reporting level (MRL) or equal to (=) a numeric value at or above the MRL
AnalyticalResultValue	Numeric value of the analytical result, null values represent less than MRL
SampleEventCode	Identification code for each sample event. Includes sample event one (SE1), sample event two (SE2), sample event three (SE3), and sample event four (SE4).

Field Name	Definition
MonitoringRequirement	AM: Assessment Monitoring (List 1)
	SS: Screening Survey (List 2)
	PST: Pre-Screen Testing (List 3)
Region	EPA Region: States
	1: CT, ME, MA, NH, RI, VT
	2: NJ, NY, PR (Puerto Rico), VI (Virgin Islands)
	3: DE, DC, MD, PA, VA, WV
	4: AL, FL, GA, KY, MS, NC, SC, TN
	5: IL, IN, MI, MN, OH, WI
	6: AR, LA, NM, OK, TX
	7: IA, KS, MO, NE
	8: CO, MT, ND, SD, UT, WY
	9: AZ, CA, HI, NV, AS (American Samoa), GU (Guam), MP (Northern Marianas Islands), NN (Navajo Nation)
	10: AK, ID, OR, WA
State	State abbreviation
ZipCode	U.S. Postal Service zip code(s) for all areas being served water by a PWS

UCMR 3 Chemical Contaminants and Methods

Contaminant	Contaminant Full Name	CAS[1] Number	Method ID	Method Name	Monitoring Requirement
1,2,3-trichloropropane	1,2,3-trichloropropane	96-18-4	524.3	Volatile Organic Compounds	AM
1,3-butadiene	1,3-butadiene	106-99-0	524.3	Volatile Organic Compounds	AM
Chloromethane	methyl chloride	74-87-3	524.3	Volatile Organic Compounds	AM
1,1-dichloroethane	1,1-dichloroethane	75-34-3	524.3	Volatile Organic Compounds	AM
Bromomethane	methyl bromide	74-83-9	524.3	Volatile Organic Compounds	AM
HCFC-22	chlorodifluoromethane	75-45-6	524.3	Volatile Organic Compounds	AM
Halon 1011	bromochloromethane	74-97-5	524.3	Volatile Organic Compounds	AM
1,4-dioxane	1,4-dioxane	123-91-1	522	Synthetic Organic Compound	AM
Vanadium	vanadium	7440-62-2	200.8	Metals	AM
Molybdenum	molybdenum	7439-98-7	200.8	Metals	AM
Cobalt	Cobalt	7440-48-4	200.8	Metals	AM
Strontium	Strontium	7440-24-6	200.8	Metals	AM
Chromium	total chromium	N/A	200.8	Metals	AM
Chromium-6	chromium-6	18540-29-9	218.7	Chromium-6	AM
Chlorate	Chlorate	14866-68-3	300.1	Oxyhalide Anion	AM
PFOS	perfluorooctanesulfonic acid	1763-23-1	537	Perfluorinated Compounds	AM
PFOA	perfluorooctanoic acid	335-67-1	537	Perfluorinated Compounds	AM
PFNA	perfluorononanoic acid	375-95-1	537	Perfluorinated Compounds	AM
PFHxS	perfluorohexanesulfonic acid	355-46-4	537	Perfluorinated Compounds	AM
PFHpA	perfluoroheptanoic acid	375-85-9	537	Perfluorinated Compounds	AM
PFBS	perfluorobutanesulfonic acid	375-73-5	537	Perfluorinated Compounds	AM
17β-estradiol	estradiol	50-28-2	539	Hormones	SS
17α-ethynylestradiol	ethinyl estradiol	57-63-6	539	Hormones	SS
Estriol	16-α-hydroxyestradiol	50-27-1	539	Hormones	SS
Equilin	Equilin	474-86-2	539	Hormones	SS
Estrone	Estrone	53-16-7	539	Hormones	SS
Testosterone	testosterone	58-22-0	539	Hormones	SS
4-androstene-3,17-dione	4-androstene-3,17-dione	63-05-8	539	Hormones	SS

[1]Chemical Abstract Service

UCMR 3 Microbiological Contaminants and Methods

Contaminant	Method ID	Method Name	Monitoring Requirement
Enteroviruses	EPA 1615A	Enterovirus cell culture	PST
Enteroviruses	EPA 1615B	Enterovirus RT-qPCR	PST
Noroviruses	EPA 1615C	Norovirus genogroup I with RT-qPCR primer set A	PST
Noroviruses	EPA 1615D	Norovirus genogroup I with RT-qPCR primer set B	PST
Noroviruses	EPA 1615E	Noroviruses genogroup II	PST
Total coliforms	SM 9223B	Colilert®	PST
E.coli	SM 9223B	Colilert®	PST
Enterococci	ASTM D6503-99	Enterolert®	PST
Aerobic spores	SM 9218	Aerobic endospores	PST
Somatic phage	EPA 1602	Bacteriophage	PST
Male specific phage	EPA 1602	Bacteriophage	PST

UCMR 3 Reference Concentrations for Chemical Contaminants

Under the current cycle of the Unregulated Contaminant Monitoring Rule (UCMR 3) chemicals are being studied at levels that are often significantly below those in prior UCMR cycles. Importantly, UCMR 3 minimum reporting levels (MRLs) were established based on the capability of the analytical method, not based on a level established as "significant" or "harmful." In fact, the UCMR 3 MRLs are often below current "health reference levels" (to the extent that HRLs have been established).

Results of UCMR 3 measurements should be interpreted accordingly. The detection of a UCMR 3 contaminant above the MRL does not represent cause for concern, in and of itself. Rather, the implications of the detection should be judged considering health effects information (which is often still under development or being refined for unregulated contaminants).

The intent of the following table is to identify draft UCMR reference concentrations, where possible, to provide context around the detection of a particular UCMR contaminant above the MRL. The draft reference concentration does not represent an "action level" (EPA requires no particular action[1,2] based simply on the fact that UCMR monitoring results exceed draft reference concentrations), nor should the draft reference concentration be interpreted as any indication of an Agency intent to establish a future drinking water regulation for the contaminant at this or any other level. Decisions as to whether or not to regulate the contaminant in drinking water will continue to be made following the Agency's Regulatory Determination process: [http://water.epa.gov/scitech/drinkingwater/dws/ccl/index.cfm].

The following key principles guided the development of the table:

(1) The reference concentrations are based on publically-available health information found in the following EPA resources: 2012 Drinking Water Standards and Health Advisories, the CCL 3 Contaminant Information Sheets, the Human Health Benchmark for Pesticides (HHBPs), the Integrated Information Risk System (IRIS), or the 2014 Preliminary Regulatory Determinations for Contaminants on CCL 3. The primary/secondary sources of health information vary with respect to scientific rigor from health assessment to single studies and are cited in the table.
(2) If health information was available from more than one of the EPA resources listed above, the most recent health information was used for the draft reference concentrations.
(3) Where both cancer and non-cancer draft reference concentrations existed, the lower (more conservative) of the two concentrations was used. For chemicals with reference concentrations based on a cancer endpoint, the table presents a range of values associated with 10^{-6} to 10^{-4} cancer risk. For chemicals with reference concentrations based on a non-cancer endpoint, the duration of exposure (short-term, intermediate/long-term, chronic) of the toxicity factor (e.g. Reference Dose) used as the basis for the reference concentration is shown.

Recognizing that additional health effects information will become available over time, EPA will periodically update the following table. Those attempting to assess UCMR occurrence data are encouraged to visit EPA's website for the most recent information.

[1] Consumer Confidence Report (CCR) and Public Notification (PN) reporting requirements (see 40 CFR 141.153(d) and 141.207, respectively) apply to public water systems; CCR requires particular reporting based on measurements relative to the UCMR method reporting limits (MRLs) defined in 40 CFR 141.40.
[2] States may establish requirements for drinking water contaminants not yet regulated by EPA, and those requirements may be based on State-established levels that differ from EPA's reference concentrations. Public Water Systems are responsible for being aware of and complying with their State's requirements, if any.

Contaminant	***MRL (µg/L)***	***Reference Concentration (µg/L)***	***Reference Concentration based on a Cancer Endpoint (Y/N)***	***EPA Reference(s)***
Cobalt[1]	1	70	N (intermediate exposure)	CCL 3 Contaminant Information Sheets
Molybdenum[2]	1	40	N (chronic exposure)	2012 Edition of the Health Advisories Table
Strontium[3]	0.3	1,500	N (chronic exposure)	Federal Register Notice for the Preliminary Regulatory Determinations for Contaminants on CCL 3
Vanadium[1,4]	0.2	21	N (intermediate exposure)	CCL 3 Contaminant Information Sheets
Chromium (Total)	0.2	100	N (chronic exposure)	The MCL for the National Primary Drinking Water Regulation
Chromium-6[1]	0.03	NA		
Chlorate	20	210	N (chronic exposure)	CCL 3 Contaminant Information Sheets
1,4-dioxane[5]	0.07	0.35 to 35	Y	2012 Edition of the Health Advisories Table
1,1-dichloroethane[5]	0.03	6.14 to 614	Y	CCL 3 Contaminant Information Sheets
1,2,3-trichloropropane[5,6,7]	0.03	0.0004 to 0.04	Y	2009 IRIS Assessment
1,3-butadiene[5,6]	0.1	0.0103 to 1.03	Y	CCL 3 Contaminant Information Sheets

[1] The contaminant is on the IRIS 2012 Agenda for either a new assessment or an updated assessment (Federal Register Notice May 7, 2012).
[2] The 2012 Edition of the Health Advisories Table and the CCL 3 Contaminant Information Sheets (35 µg/L) have slightly different numbers due to rounding.
[3] The reference concentration has been updated based on the HRL cited in the preliminary regulatory determination for strontium [Docket No. EPA-HQ-OW-2012-0155].
[4] The ATSDR, 1992 used for the CCL 3 Contaminant Information Sheets is no longer publically available and has been replaced by a new assessment (ATSDR, 2013).
The minimum risk level (RfD equivalent) was 0.003 mg/kg/day for minor renal effects in an animal study (ATSDR, 1992) compared to 0.01 mg/kg/day for lack of minor effects in blood pressure, body weight, and hematological parameters in a human study with a 12 weeks exposure (ATSDR, 2013).
[5] Reference Concentration range based on cancer risk of 10^{-6} to 10^{-4}.
[6] 10^{-6} cancer risk < MRL < 10^{-4} cancer risk.
[7] To derive the reference concentration, age dependent adjustment factors were applied to the IRIS oral slope factor of 30 per mg/kg-day (calculated using adult exposure data) to address presumed early-life susceptibility for this chemical (per www.epa.gov/cancerguidelines/).

Contaminant	***MRL (µg/L)***	***Reference Concentration (µg/L)***	***Reference Concentration based on a Cancer Endpoint (Y/N)***	***EPA Reference(s)***
HCFC-22 (chlorodifluoromethane) [8]	0.08	NA		
Chloromethane (methyl chloride)[5]	0.2	2.69 to 269	Y	CCL 3 Contaminant Information Sheets
Halon 1011 (bromochloromethane)[9]	0.06	90	N (chronic exposure)	2012 Edition of the Health Advisories Table
Bromomethane (methyl bromide)	0.2	140	N (chronic exposure)	Human Health Benchmark for Pesticides (HHBPs)
PFBS	0.09	NA		
PFHpA	0.01	NA		
PFHxS	0.03	NA		
PFNA	0.02	NA		
PFOS	0.04	0.2	N (short-term exposure)	2012 Edition of the Health Advisories Table
PFOA	0.02	0.4	N (short-term exposure)	2012 Edition of the Health Advisories Table
17α-ethynylestradiol (ethinyl estradiol)[10]	0.0009	0.035	N (chronic exposure)	CCL 3 Contaminant Information Sheets
17β-estradiol (estradiol)[5]	0.0004	0.0009 to 0.09	Y	CCL 3 Contaminant Information Sheets
Equilin	0.004	0.35	N (chronic exposure)	CCL 3 Contaminant Information Sheets
Estriol (16-α-hydroxyestradiol)	0.0008	0.35	N (chronic exposure)	CCL 3 Contaminant Information Sheets
Estrone	0.002	0.35	N (chronic exposure)	CCL 3 Contaminant Information Sheets
4-androstene-3,17-dione	0.0003	NA		
Testosterone	0.0001	NA		

[8] The CCL 3 Contaminant Information Sheets provide a reference level of 31.5 µg/L; the number is based on a single LOAEL from a 1983 study.
[9] The 2012 Edition of the Health Advisories Table and the CCL 3 Contaminant Information Sheets (70 µg/L) have slightly different numbers due to rounding.
[10] This corrects the CCL 3 Contaminant Information Sheets reference level (originally listed as 0.28 µg/L).

Terms

a) UCMR Draft Reference Concentration = The reference concentrations are based on publically-available health information found in the following EPA resources: 2012 Drinking Water Standards and Health Advisories, the CCL 3 Contaminant Information Sheets, the Human Health Benchmark for Pesticides (HHBPs), or the 2014 Preliminary Regulatory Determinations for Contaminants on CCL 3. The primary/secondary sources of health information vary with respect to scientific rigor from health assessment to single studies. Many of the contaminants are currently under regulatory review or development and are subject to change as new health assessments are completed.
b) MRL = UCMR Minimum Reporting Level. *[Note that the Agency for Toxic Substances & Disease Registry (ATSDR) uses the term "MRL" for a different purpose (i.e., to describe "Minimal Risk Levels"). The UCMR term and the ATSDR term have no relationship to each other.]*
c) HRLs = Health Reference Levels. HRLs are not final determinations about the level of a contaminant in drinking water that is necessary to protect any particular population and are derived prior to development of a complete exposure assessment. HRLs are risk derived concentrations against which to evaluate the occurrence data to determine if contaminants occur at levels of potential public health concern.
d) MCL = Maximum Contaminant Level. The highest level of a contaminant allowed in drinking water. MCLs are enforceable standards.
e) Cancer Risk of 10^{-6} to 10^{-4} = the concentration of a contaminant in drinking water corresponding to an excess estimated lifetime cancer risk of one-in-a-million ($1x\ 10^{-6}$) to one-in-ten-thousand (1×10^{-4}). The 2012 Drinking Water Standards and Health Advisories provide the cancer risk at 1×10^{-4}. The CCL 3 Contaminant Information Sheets provide the cancer risk at $1x\ 10^{-6}$.
f) LOAEL = Lowest Observed Adverse Effect Level
g) NA = Not Available
h) Short-term = Typically refers to animal toxicological studies with an exposure duration of days to weeks.
i) Intermediate/Longer-term = Typically refers to animal toxicological studies with an exposure duration of weeks to months.
j) Chronic = Typically refers to animal toxicological studies with an exposure duration of months to years; representing a lifetime exposure in humans.

References

k) 2012 Drinking Water Standards and Health Advisories
l) CCL 3 Contaminant Information Sheets
m) Human Health Benchmark for Pesticides (HHBPs)
n) Announcement of Preliminary Regulatory Determinations for Contaminants on the Third Drinking Water Contaminant Candidate List
o) Integrated Risk Information System (IRIS)

October 2015 UCMR 3 Data Summary for Chemical Contaminants

Contaminant	MRL (µg/L)	Reference Concentration (µg/L)	Total number of results	Number of results ≥MRL	Number of results >Reference Concentration	% of total results >Reference Concentration	Total number of PWSs with results	Number of PWSs with results ≥MRL	Number of PWSs with results >Reference Concentration	% of PWSs with results >Reference Concentration
1,2,3-trichloropropane	0.03	0.0004 / 0.04[1]	32,893	228	228 / 174[1]	0.7% / 0.5%[1]	4,750	62	62 / 52[1]	1.3% / 1.1%[1]
1,3-butadiene	0.1	0.0103 / 1.03[1]	32,893	1	1 / 0[1]	0.003% / 0%[1]	4,750	1	1 / 0[1]	0.02% / 0%[1]
Chloromethane	0.2	2.69 / 269[1]	32,891	251	18 / 0[1]	0.05% / 0%[1]	4,750	127	7 / 0[1]	0.1% / 0%[1]
1,1-dichloroethane	0.03	6.14 / 614[1]	32,891	764	1 / 0[1]	0.003% / 0%[1]	4,749	230	1 / 0[1]	0.02% / 0%[1]
Bromomethane	0.2	140	32,892	109	0	0%	4,750	46	0	0%
HCFC-22	0.08	NA	32,893	742	- -	- -	4,750	266	- -	- -
Halon 1011	0.06	90	32,893	596	0	0%	4,750	286	0	0%
1,4-dioxane	0.07	0.35 / 35[1]	32,740	3,796	989 / 0[1]	3% / 0%[1]	4,746	1,025	325/ 0[1]	6.8% / 0%[1]
Vanadium	0.2	21	56,298	33,595	1,563	2.8%	4,756	3,454	159	3.3%
Molybdenum	1	40	56,307	23,089	133	0.2%	4,756	2,438	34	0.7%
Cobalt	1	70	56,290	772	3	0.005%	4,756	226	3	0.06%
Strontium	0.3	1,500	56,227	56,038	1,546	2.7%	4,756	4,755	272	5.7%
Chromium	0.2	100	56,161	28,378	1	0.002%	4,756	3,517	1	0.02%
Chromium-6	0.03	NA	56,039	42,151	- -	- -	4,756	4,229	- -	- -
Chlorate	20	210	55,990	30,991	8,608	15.4%	4,749	3,236	1,744	36.7%
PFOS	0.04	0.2	33,178	260	33	0.1%	4,764	90	17	0.4%
PFOA	0.02	0.4	33,178	324	0	0%	4,764	103	0	0%
PFNA	0.02	NA	33,178	19	- -	- -	4,764	14	- -	- -
PFHxS	0.03	NA	33,178	192	- -	- -	4,764	54	- -	- -
PFHpA	0.01	NA	33,178	216	- -	- -	4,764	80	- -	- -
PFBS	0.09	NA	33,178	13	- -	- -	4,764	6	- -	- -
17β-estradiol	0.0004	0.0009 / 0.09[1]	10,234	3	1 / 0[1]	0.01% / 0%[1]	1,145	1	1 / 0[1]	0.09% / 0%[1]
17α-ethynylestradiol	0.0009	0.035	10,235	4	0	0%	1,145	4	0	0%
Estriol	0.0008	0.35	10,234	1	0	0%	1,145	1	0	0%
Equilin	0.004	0.35	10,235	0	0	0%	1,145	0	0	0%
Estrone	0.002	0.35	10,235	0	0	0%	1,145	0	0	0%
Testosterone	0.0001	NA	10,234	57	- -	- -	1,145	51	- -	- -
4-androstene-3,17-dione	0.0003	NA	10,235	84	- -	- -	1,145	64	- -	- -

[1]Where two reference concentrations are listed, the first number is associated with a 10^{-6} cancer risk; the second number a 10^{-4} cancer risk.
Where two results are presented the first number is associated with the first reference concentration; the second number is associated with the second reference concentration.

October 2015 UCMR 3 Data Summary for Microbiological Contaminants

Contaminant	MRL	Unit	Total number of results	Number of results ≥MRL	Total number of PWSs with results	Number of PWSs with results ≥MRL
Aerobic spores	1	SFO[1]/100 mL[2]	732	201	710	198
E. coli	1	MPN[3]/100 mL	730	3	708	3
Enterococci	1	MPN/100 mL	729	37	707	37
Enteroviruses (cell culture)	0.002	MPN/L[4]	728	2	706	2
Enteroviruses (RT-qPCR[5])	0.398	GC[6]/L	728	6	706	6
Male specific phage	1	PFU[7]/100 mL	714	10	692	10
Noroviruses GIA[8]	0.398	GC/L	728	0	706	0
Noroviruses GIB[9]	0.398	GC/L	728	1	706	1
Noroviruses GII[10]	0.398	GC/L	728	4	706	4
Somatic phage	1	PFU/100 mL	714	3	692	3
Total coliforms	1	MPN/100 mL	730	33	708	32

[1]SFO = Spore Forming Units
[2]mL = milliliters
[3]MPN = Most Probable Number
[4]L = liters
[5]RT-qPCR = Reverse Transcription-Polymerase Chain Reaction
[6]GC = Genomic Copies
[7]PFU = Plaque Forming Units
[8]Noroviruses GIA = qPCR analysis of Norovirus genogroup I with RT-qPCR primer set A
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Under UCMR 3 microbe analytical results are reported as “below”, “at” or “above” MRL. UCMR 3 MRLs were established based on the capability of the analytical method.

It is important to note that microbial contamination can be transient in nature and microbial detections under UCMR 3 should be interpreted in the context of the time samples were collected. However, the presence of any UCMR 3 microbe indicates a potential vulnerability of the PWS to contamination.

NEW YORK STATE OF OPPORTUNITY. | Department of Environmental Conservation | Department of Health

January 14, 2016

The Honorable Gina McCarthy
Administrator
USEPA Headquarters
William Jefferson Clinton Building
1200 Pennsylvania Avenue, N. W.
Mail Code: 1101A
Washington, DC 20460

Dear Administrator McCarthy:

We write to you to request that EPA take vigorous action to address the presence of perfluorooctanoic acid (PFOA) in drinking water and groundwater. Respectfully, we ask that EPA:

- lower its provisional health advisory of 400 parts per trillion (ppt) for PFOA drinking water to take into account the most current scientific evidence;
- act expeditiously to adopt a protective maximum contaminant level for PFOA;
- expeditiously list PFOA as a hazardous substance under the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA) to facilitate the cleanup of contaminated groundwater and other media; and
- review the remaining uses of PFOA under the Toxic Substances Control Act and curtail it whenever less toxic alternatives are available.

The New York State Department of Health has been working with the Village of Hoosick Falls and the Town of Hoosick for more than a year to address PFOA contamination of drinking water. PFOA in the Village of Hoosick Falls public water supply exceeds the provisional EPA health advisory of 400 ppt. Private wells in the Town of Hoosick have also shown signs of contamination, but at lower levels than in the municipal supply. The Department of Health, the Village, and Saint-Gobain Performance Plastics have collaborated to evaluate treatment options for the Village water supply, implement a bottled water program, and design and order a temporary treatment system to be installed in the coming weeks on the Village water supply. This temporary treatment system will remain in place until a planned permanent treatment system is operational later this year.

We write to you because this is not just a local issue. The presence of PFOA in drinking water is an emerging nation-wide issue.

NEW YORK STATE OF OPPORTUNITY. | Department of Environmental Conservation | Department of Health

As reported in the New York Times Magazine on January 10, 2016, several studies have asserted that the presence of PFOA in drinking water and groundwater may be more pervasive than originally thought and may subject people across the country to PFOA exposure since EPA first began working on this issue in 2001.

It is imperative that the federal government step forward and use the authority it already holds under federal law to comprehensively address this national issue. The State of New York stands ready to assist EPA in any way we can in this important effort to protect public health and the environment from PFOA.

Sincerely,

Howard Zucker M.D.

Dr. Howard Zucker
Commissioner
DOH

Basil Seggos
Acting Commissioner
DEC

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION 2
290 BROADWAY
NEW YORK, NY 10007-1866

NOV 2 5 2015

Honorable Mayor David B. Borge
Municipal Building
24 Main Street
Hoosick Falls, NY 12090

Dear Mayor Borge:

I am writing regarding the perfluorooctanoic acid (PFOA) water contamination that has been discovered in groundwater and drinking water in the Village of Hoosick Falls, NY. On October 15, 2015, I was contacted about the problem with the Hoosick Falls public water supply and was asked if funding from the U.S. Environmental Protection Agency (EPA) was available to address this drinking water problem.

EPA does not have a funding stream to which the Village could apply in this situation. A more detailed response will be provided shortly. In summary, EPA provides Safe Drinking Water Act State Revolving Funds to New York State to address drinking water needs. EPA's Safe Drinking Water Act State Revolving Fund Program is implemented by the New York State Department of Health (DOH).

Because of PFOA's extreme persistence in the environment and its toxicity, mobility and bioaccumulation potential, which pose potential adverse effects to human health and the environment, EPA has been gathering information regarding the Hoosick Falls PFOA contamination and has been discussing this matter with DOH and the New York State Department of Environmental Conservation (DEC). While EPA has not, to date, promulgated an enforceable drinking water standard for PFOA under the Safe Drinking Water Act, in 2009, EPA's Office of Water established a provisional health advisory of 400 nanograms per liter, that is, 400 parts per trillion (ppt), for PFOA.[1]

Provisional health advisories reflect reasonable, health-based hazard concentrations above which action should be taken to reduce exposure to unregulated contaminants in drinking water. In 2014, EPA stated that its provisional health advisory for PFOA, if exceeded, suggests the need for discontinuing use of the water for drinking or cooking; and that the advisory reflects an amount of PFOA that may cause adverse health effects in the short term (weeks to months).

[1] See http://water.epa.gov/action/advisories/drinking/upload/2009_01_15_criteria_drinking_pha-PFOA_PFOS.pdf.

[2] An additional EPA fact sheet about PFOA can be found at http://www2.epa.gov/sites/production/files/2014-04/documents/factsheet_contaminant_pfos_pfoa_march2014.pdf and is enclosed. Please note that EPA is currently reviewing the state of the science on PFOA and other perfluorinated compounds, which may lead to further advisories, including an advisory addressing long-term health effects.

As you know, four samples collected from the public water supply in Hoosick Falls on June 4, 2015 were found to contain more than 600 ppt of PFOA. Additionally, 2015 groundwater sampling at the Saint-Gobain Performance Plastics facility on McCaffrey Street in Hoosick Falls found levels as high as 18,000 ppt. Certain private wells in the area have also shown the presence of PFOA, though not at levels above 400 ppt, as far as EPA is aware.

Based on the presence of PFOA above 400 ppt in Hoosick Falls public drinking water supply wells, it is recommended that an alternate drinking water source (*e.g.*, bottled water) be provided to the users of the Hoosick Falls public water supply, until such time as PFOA concentrations in drinking water are brought consistently below the 400 ppt level. EPA also recommends that during this period, drinking water from the public water supply not be used for cooking (*e.g.*, boiling pasta, making soup, steaming vegetables, etc.). Boiling the water does not diminish the potential health risk associated with the PFOA.

EPA recommends that the Village of Hoosick Falls' web site be updated to conform with the information provided above, and that any information provided to the residents by the Village relating to the water contamination issue be consistent with this information as well. While the Village's web site does mention an EPA "guideline" of 400 ppt for PFOA, we recommend that the information that I have provided above regarding EPA's 400 ppt provisional health advisory, and the significance of that advisory, along with the above Internet links, be included in the web site's discussion of the water contamination issue.

In addition, EPA recommends the following corrections to the Village's web site:

- The web site incorrectly cites a 200 ppt guideline for PFOA. The 200 ppt provisional health advisory that EPA issued is for a different compound ("PFOS"), not PFOA.

- The web site contains the statement, "The EPA is in the data collection phase only." We recommend that sentence be deleted. While EPA is gathering data about PFOA in public water systems, EPA has also issued a provisional health advisory, as discussed above.

- The following sentences on the Village web site should be deleted: "Village residents should be aware that the numbers in each sample represent parts per trillion and the EPA guidelines are based on parts per billion. As yet, there is no standard or benchmark to determine how these numbers are to be interpreted for their impact on public health." As discussed above, EPA's provisional health advisory for PFOA is 400 parts per trillion.

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[2] See http://water.epa.gov/drink/standards/upload/Peer-Review-of-Health-Effects-Documents-for-PFOA-and-PFOS-Factsheet-February-2014.pdf.

In addition to ensuring the accuracy of the information provided to the public, appropriate measures in groundwater and drinking water contamination situations such as this one may include, among other things, some or all of the following actions, whether such measures are taken by the Village, by an entity that is the source of the contamination, or by some other party:

Public and private drinking water supplies

- As a temporary measure until a more permanent, safe drinking water supply is provided, provision of bottled water to residents in the impacted and potentially impacted area;
- Encourage the private well owners to identify themselves and sign up for well sampling to determine whether contamination is present;
- After obtaining approval from the Department of Health, installation, operation and maintenance of a treatment system on the public water supply that will effectively treat PFOA (granular activated carbon filtration is one such system);
- Appropriate training for the public water system operators to ensure that the system is working at optimum capacity and effectively and efficiently removing the contaminant in the water source;
- Ensuring the financial capability to support the cost of the granular activated carbon system maintenance requirements going forward;
- A contingency plan to ensure that safe drinking water will continue to be provided to the public even if and when the primary treatment system is taken offline;
- If a private well is confirmed to have PFOA contamination at a level of 400 ppt or above, then bottled water should be provided to that residence, followed by a more permanent solution – *i.e.*, either an individual treatment system (such as a Point of Entry Treatment Systems) or connection to the public water supply;
- Regular monitoring of both the treated and untreated water at the public water supply;

- Regular monitoring of the impacted private wells, and based on a groundwater plume delineation or other appropriate information, sampling of certain additional private wells. (To date, DOH has done some essential, limited sampling of private drinking water wells in the area to determine whether the wells are impacted by PFOA at levels of concern. There needs to be a commitment to do substantially more private well sampling.)

In addition, EPA will work with DEC and DOH with respect to the need for the following efforts, which would be directly overseen by EPA or the State of New York, based on future discussions with the State:

- Investigation of the nature and extent of contamination and identification of the source(s) of the contamination
    - Such an investigation may include a hydrogeological study (including installation and sampling of groundwater monitoring wells), soil, sediment and surface water sampling, review of historical records and databases, and other investigations and analyses; and
    - Modeling of air deposition from PFOA air emissions.
- Identification and implementation of one or more early interim measures
    - Such interim measures may involve addressing the potential source(s) and/or containing the groundwater contaminant plume.
- Feasibility Study and Remedial Action
    - Identification and analysis of potential alternatives to remediate the groundwater contamination, the source(s) of the contamination, and any other contaminated areas; and
    - Selection and implementation of a remedial alternative(s).
- Sampling in Hoosick River, including fish

I understand that Saint-Gobain Performance Plastics has agreed to pay for the provision of bottled water to residents and the installation of a carbon filtration system on the public drinking water supply. As indicated above, there are additional important measures that need to be addressed.

If you have any questions or would like additional information about any of the matters discussed above, please contact me at 212-637-5000 or Pat Evangelista at 212-637-4447 or evangelista.pat@epa.gov.

Thank you.

Sincerely yours,

Judith A. Enck

Judith A. Enck
Regional Administrator

Enclosure

cc: Nathan Graber, NYSDOH
Basil Seggos, NYSDEC
Honorable Kathy Jimino, Rensselaer County Executive

United States Environmental Protection Agency

Emerging Contaminants – Perfluorooctane Sulfonate (PFOS) and Perfluorooctanoic Acid (PFOA)

March 2014

EMERGING CONTAMINANTS FACT SHEET – PFOS and PFOA

At a Glance

- Fully fluorinated compounds that are human-made substances and are not naturally found in the environment
- Used as a surface-active agent and in a variety of products, such as firefighting foams, coating additives and cleaning products
- Do not hydrolyze, photolyze or biodegrade under typical environmental conditions and are extremely persistent in the environment
- Studies have shown they have the potential to bioaccumulate and biomagnify in wildlife
- Readily absorbed after oral exposure and accumulate primarily in the serum, kidney and liver
- Toxicological studies on animals indicate potential developmental, reproductive and systemic effects
- Health-based advisories or screening levels for PFOS and PFOA have been developed by the EPA and state agencies
- Standard detection methods include high-performance liquid chromatography and tandem mass spectrometry
- Common ex situ water treatment technologies include activated carbon filters and reverse osmosis units

Introduction

An "emerging contaminant" is a chemical or material that is characterized by a perceived, potential, or real threat to human health or the environment or by a lack of published health standards. A contaminant may also be "emerging" because a new source or a new pathway to humans has been discovered or a new detection method or treatment technology has been developed (DoD 2011). This fact sheet, developed by the U.S. Environmental Protection Agency (EPA) Federal Facilities Restoration and Reuse Office (FFRRO), provides a summary of the emerging contaminants perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA), including physical and chemical properties; environmental and health impacts; existing federal and state guidelines; detection and treatment methods; and additional sources of information. This fact sheet is intended for use by site managers who may address PFOS and PFOA at cleanup sites or in drinking water supplies and for those in a position to consider whether these chemicals should be added to the analytical suite for site investigations.

PFOS and PFOA are extremely persistent in the environment and resistant to typical environmental degradation processes. As a result, they are widely distributed across the higher trophic levels and are found in soil, air and groundwater at sites across the United States. The toxicity, mobility and bioaccumulation potential of PFOS and PFOA pose potential adverse effects for the environment and human health.

What are PFOS and PFOA?

- PFOS and PFOA are fully fluorinated, organic compounds and are the two perfluorinated chemicals (PFCs) that have been produced in the largest amounts within the United States (ATSDR 2009; EFSA 2008).
- PFOS is a perfluoralkyl sulfonate that is commonly used as a simple salt (such as potassium, sodium or ammonium) or is incorporated into larger polymers (EFSA 2008; EPA 2009c).
- PFOA is a perfluoralkyl carboxylate that is produced synthetically as a salt. Ammonium salt is the most widely produced form (EFSA 2008; EPA 2009c).

Disclaimer: The U.S. EPA prepared this fact sheet from publicly available sources; additional information can be obtained from the source documents. This fact sheet is not intended to be used as a primary source of information and is not intended, nor can it be relied on, to create any rights enforceable by any party in litigation with the United States. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

What are PFOS and PFOA? (continued)

- PFOS synonyms include 1-octanesulfonic acid, heptadecafluoro-, 1-perfluorooctanesulfonic acid, heptadecafluoro-1-octanesulfonic acid, perfluoro-n-octanesulfonic acid, perfluoroctanesulfonic acid and perfluoroctylsulfonic acid (ATSDR 2009; UNEP 2005).
- PFOA synonyms include pentadecafluoro1-octanoic acid, pentadecafluoro-n-octanoic acid, pentadecaflurooctanoic acid, perfluorocaprylic acid, perfluoroctanoic acid, perfluoroheptanecarboxylic acid and octanoic acid (ATSDR 2009).
- They are stable chemicals that include long carbon chains. Because of their unique lipid- and water-repellent characteristics, PFOS and PFOA are used as surface-active agents in various high-temperature applications and as a coating on surfaces that contact with strong acids or bases (Schultz and others 2003; UNEP 2005).
- PFCs are used in a wide variety of industrial and commercial products such as textiles and leather products, metal plating, the photographic industry, photolithography, semi-conductors, paper and packaging, coating additives, cleaning products and pesticides (ATSDR 2009; EPA 2009c; OECD 2002).
- Through 2001, PFCs were used to manufacture Aqueous Film Forming Foam (AFFF). PFOS-based AFFF is used to extinguish flammable liquid fires (for example, hydrocarbon fueled), such as fires involving gas tankers and oil refineries (EPA 2013a; DoD SERDP 2012).
- They are human-made compounds and do not occur naturally in the environment (ATSDR 2009; EPA 2009c).
- PFOS and PFOA can also be formed by environmental microbial degradation or by metabolism in larger organisms from a large group of related substances or precursor compounds (ATSDR 2009; UNEP 2006).
- The 3M Company, the primary manufacturer of PFOS, completed a voluntary phase-out of PFOS production in 2002 (ATSDR 2009; 3M 2008).

Exhibit 1: Physical and Chemical Properties of PFOS and PFOA
(ATSDR 2009; Brooke and others 2004; EFSA 2008; Environment Canada 2012; EPA 2002b; OECD 2002; UNEP 2006)

Property	PFOS (Potassium Salt)	PFOA (Free Acid)
Chemical Abstracts Service (CAS) Number	2795-39-3	335-67-1
Physical Description (physical state at room temperature and atmospheric pressure)	White powder	White powder/ waxy white solid
Molecular weight (g/mol)	538	414
Water solubility at 25°C (mg/L)	550 to 570 (purified), 370 (fresh water), 25 (filtered sea water)	9.5×10^3(purified)
Melting Point (°C)	> 400	45 to 54
Boiling point (°C)	Not measurable	188 to 192
Vapor pressure at 20 °C (mm Hg)	2.48×10^{-6}	0.017[1]
Octanol-water partition coefficient (log K_{ow})	Not measurable	Not measurable
Organic-carbon partition coefficient (log K_{oc})	2.57 (Value estimated based on anion and not the salt)	2.06
Henry's law constant (atm-m^3/mol)	3.05×10^{-9}	Not measurable
Half-Life	Atmospheric: 114 days Water: > 41 years (at 25° C)	Atmospheric: 90 days[2] Water: > 92 years (at 25° C)

Abbreviations: g/mol – grams per mole; mg/L – milligrams per liter; °C – degree Celsius; mm Hg – millimeters of mercury; atm-m^3/mol – atmosphere-cubic meters per mole.
[1] Extrapolation from measurement.
[2] The atmospheric half-life value identified for PFOA is estimated based on available data determined from short study periods.

What are PFOS and PFOA? (continued)

- PFOS chemicals are no longer manufactured in the United States; however, EPA significant new use rules (SNURs) allow for the continuation of a few, limited, highly technical applications of PFOS-related substances where no known alternatives are available. In addition, existing stocks of PFC-based chemicals that were manufactured or imported into the United States before the effective date of the SNURs (for example, PFOS-based AFFF produced before the rules took effect in 2002) can still be used (EPA 2009c, 2013a).
- PFOA as its ammonium salt is manufactured primarily for use as an aqueous dispersion agent and in the manufacture of fluoropolymers (which are used in a wide variety of mechanical and industrial components) such as electrical wire casings, fire- and chemical-resistant tubing and plumbing seal tape. They are also produced unintentionally by the degradation of some fluorotelomers (ATSDR 2009; EPA 2009c).
- As part of the EPA's PFOA stewardship program, eight companies committed to achieve the following by 2010: (1) reduce global facility emissions of PFOA to all media; (2) reduce precursor chemicals that break down to PFOA and related higher homologue chemicals; and (3) PFOA product content (95 percent). The companies also agreed to work toward eliminating these chemicals from emissions and products by 2015 (EPA 2013a).

What are the environmental impacts of PFOS and PFOA?

- During past manufacturing processes, large amounts of PFOS and PFOA were released to the air, water and soil in and around fluorochemical facilities (ATSDR 2009).
- PFOS and PFOA have been detected in a number of U.S. cities in surface water and sediments downstream of former fluorochemical production facilities and in wastewater treatment plant effluent, sewage sludge and landfill leachate (EPA 2002b; OECD 2002).
- The environmental release of PFOS-based AFFF may also occur from tank and supply line leaks, use of aircraft hangar fire suppression systems and firefighting training (DoD SERDP 2012).
- Both PFOS and PFOA are the stable end products resulting from the degradation of precursor substances through a variety of abiotic and biotic transformation pathways (Conder and others 2010).
- Because of their chemical structure, PFCs, including PFOS and PFOA, are chemically and biologically stable in the environment and resist typical environmental degradation processes, including atmospheric photooxidation, direct photolysis and hydrolysis. As a result, these chemicals are extremely persistent in the environment (OECD 2002; Schultz and others 2003).
- PFOS and PFOA have very low volatility because of their ionic nature. Therefore, they will be persistent in water and soil (3M 2000; ATSDR 2009).
- When released directly to the atmosphere, PFCs are expected to adsorb to particles and settle to the ground through wet or dry deposition (Barton and others 2007; Hurley and others 2004).
- In their anionic forms, PFOA and PFOS are water-soluble and can migrate readily from soil to groundwater, where they can be transported long distances (Davis and others 2007; Post and others 2012).
- Monitoring data from the Arctic region and at sites remote from known point sources have shown levels of PFOS and PFOA in environmental media and biota, indicating that long-range transport has occurred. For example, PFOA and PFOS have been detected in concentrations from the low- to mid- picograms per liter (pg/L) range in remote regions of the Arctic caps. In addition, PFOS concentrations detected in the liver of the Canadian Arctic polar bear range from 1,700 to more than 4,000 nanograms per gram (ng/g) (Lau and others 2007; Martin and others 2004; Young and others 2007).
- Causes of long-range PFC transport include (1) atmospheric transport of precursor compounds (such as perfluoroalkyl sulfonamides), followed by degradation to form PFCs and (2) direct, long-range transport of PFCs via ocean currents or in the form of marine aerosols (Armitage and others 2006; Post and others 2012).

What are the environmental impacts of PFOS and PFOA? (continued)

- The wide distribution of PFCs increases the potential for bioaccumulation and bioconcentration as they are transferred from low to higher trophic level organisms. Because of their persistence and long-term accumulation, higher trophic level wildlife such as fish, piscivorous birds and other biota can continue to be exposed to PFOS and PFOA (EPA 2006a; UNEP 2006).
- The bioaccumulation potential of PFCs increases with increasing carbon chain length (ATSDR 2009; Furdui and others 2007).
- PFOS is the only PFC that has been shown to accumulate to levels of concern in fish tissue. The estimated bioconcentration factor in fish ranges from 1,000 to 4,000 (EFSA 2008; MDH 2011; OECD 2002).
- As of 2013, the Superfund Information Systems Database indicates PFCs have been reported in the 5-year reviews of 14 hazardous waste sites on the EPA National Priorities List (EPA 2013b).
- Data gathered in 2008 from the DoD Knowledge Based Corporate Reporting System show that 594 DoD facilities have been categorized as Fire/Crash/Training Sites and, therefore, have the potential for PFC contamination based on historical use of AFFF (DoD 2008; DoD SERDP 2012).

What are the routes of exposure and the health effects of PFOS and PFOA?

- Studies have found PFOS and PFOA in the blood samples of the general human population and wildlife nationwide, indicating that exposure to the chemicals is widespread (ATSDR 2009; EPA 2006a).
- Reported data indicate that serum concentrations of PFOS and PFOA are higher in workers and individuals living near fluorochemical production facilities than for the general population (Calafat and others 2007; EPA 2009c).
- Potential pathways, which may lead to widespread exposure, include ingestion of food and water, use of commercial products or inhalation from long-range air transport of PFC-containing particulate matter (ATSDR 2009; EPA 2009c).
- Based on the limited information available, fish and fishery products seem to be one of the primary sources of human exposure to PFOS (EFSA 2008).
- While a federal screening level or toxicity value for the consumption of fish has not yet been established, the Dutch National Institute for Public Health and the Environment has calculated a maximum permissible concentration for PFOS of 0.65 nanograms per liter (ng/L) for fresh water (based on consumption of fish by humans as the most critical route) (Moermond and others 2010).
- Studies also indicate that continued exposure to low levels of PFOA in drinking water may result in adverse health effects (Post and others 2012).
- Toxicology studies show that PFOS and PFOA are readily absorbed after oral exposure and accumulate primarily in the serum, kidney and liver. No further metabolism is expected (EPA 2006a, 2009c).
- PFOS and PFOA have half-lives in humans ranging from 2 to 9 years, depending on the study. This half-life results in continued exposure that could increase body burdens to levels that would result in adverse outcomes (ATSDR 2009; EPA 2009c; Kärrman and others 2006; Olsen and others 2007).
- Acute- and intermediate-duration oral studies on rodents have raised concerns about potential developmental, reproductive and other systemic effects of PFOS and PFOA (Austin and others 2003; EPA 2006a).
- The ingestion of PFOA-contaminated water was found to cause adverse effects on mammary gland development in mice (Post and others 2012).
- One study indicated that exposure to PFOS can affect the neuroendocrine system in rats; however, the mechanism by which PFOS affects brain neurotransmitters is still unclear (Austin and others 2003).
- Both PFOS and PFOA have a high affinity for binding to B-lipoproteins and liver fatty acid-binding protein. Several studies on animals have shown that these compounds can interfere with fatty acid metabolism and may deregulate metabolism of lipids and lipoproteins (EFSA 2008; EPA 2009c).

What are the routes of exposure and the health effects of PFOS and PFOA? (continued)

- In May 2006, the EPA Science Advisory Board suggested that PFOA cancer data are consistent with the EPA guidelines for the Carcinogen Risk Assessment descriptor "likely to be carcinogenic to humans." EPA is still evaluating this information and additional research pertaining to the carcinogenicity of PFOA (EPA 2006b, 2013a).
- The American Conference of Governmental Industrial Hygienists (ACGIH) has classified PFOA as a Group A3 carcinogen — confirmed animal carcinogen with unknown relevance to humans (ACGIH 2002).
- The chronic exposure to PFOS and PFOA can lead to the development of tumors in the liver of rats; however, more research is needed to determine if there are similar cancer risks for humans (ATSDR 2009; OECD 2002).
- In a retrospective cohort mortality study of more than 6,000 PFOA-exposed employees at one plant, results identified elevated standardized mortality ratios for kidney cancer and a statistically significant increase in diabetes mortality for male workers. The study noted that additional investigations are needed to confirm these findings (DuPont 2006; Lau and others 2007).
- Studies have shown that PFCs may induce modest effects on reactive oxygen species and deoxyribonucleic acid (DNA) damage in the cells of the human liver (Eriksen and others 2010; Reistad and others 2013).
- Analysis of U.S. National Health and Nutrition Examination Survey representative study samples indicate that higher concentrations of serum PFOA and PFOS are associated with thyroid disease in the U.S. general adult population. Further analysis is needed to identify the mechanisms underlying this association (Melzer and others 2010).
- Epidemiologic studies have shown an association between PFOS exposure and bladder cancer; however, further research and analysis are needed to understand this association (Alexander and others 2004; Lau and others 2007).

Are there any federal and state guidelines and health standards for PFOS and PFOA?

- In January 2009, the EPA's Office of Water established a provisional health advisory (PHA) of 0.2 micrograms per liter (µg/L) for PFOS and 0.4 µg/L for PFOA to assess the potential risk from short-term exposure of these chemicals through drinking water. PHAs reflect reasonable, health-based hazard concentrations above which action should be taken to reduce exposure to unregulated contaminants in drinking water (EPA 2009d, 2013a).
- EPA Region 4 calculated a residential soil screening level of 6 milligrams per kilogram (mg/kg) for PFOS and 16 mg/kg for PFOA (EPA Region 4 2009).
- Various states have established drinking water and groundwater guidelines, including the following:
  - Minnesota has established a chronic health risk limit of 0.3 µg/L for PFOS and PFOA in drinking water (MDH 2011).
  - New Jersey has established a preliminary health-based guidance value of 0.04 µg/L for PFOA in drinking water (NJDEP 2013).
  - North Carolina has established an interim maximum allowable concentration (IMAC) of 2 µg/L for PFOA in groundwater (NCDENR 2006).
  - In 2010, the North Carolina Secretary's Science Advisory Board (NCSAB) on Toxic Air Pollutants recommended that the IMAC be reduced to 1 µg/L based on a review of the toxicological literature and discussions with scientists conducting research on the health effects associated with exposure to PFOA. As of February 2014, the NCSAB's recommendation was still pending review by the North Carolina Division of Water Quality (NCSAB 2010).
- Under the Toxic Substances Control Act (TSCA), the EPA finalized two SNURs in 2002 for 88 PFOS-related substances, which require companies to notify the EPA 90 days before starting to manufacture or importing these substances for a significant new use; this pre-notification allows time to evaluate the new use (EPA 2002a, 2013a).
- In 2007, the SNURs were amended to include 183 additional PFOS-related substances (EPA 2006a, 2013a).

Are there any federal and state guidelines and health standards for PFOS and PFOA? (continued)

- On September 30, 2013, the EPA issued a final SNUR requiring companies to report 90 days in advance of all new uses of long-chain perfluoroalkyl carboxylic (LCPFAC) chemicals (defined as having perfluorinated carbon chain lengths equal to or greater than seven carbons and less than or equal to 20 carbons) for use as part of carpets or to treat carpets, including the import of new carpet containing LCPFACs. In addition, the EPA is amending the existing SNUR to add PFOS-related substances that have completed the TSCA new chemical review process but have not yet commenced production or importation, and to designate processing as a significant new use (EPA 2012, 2013a).
- The SNURs allow for continued use for a few highly technical applications of PFOS-related substances where no alternatives are available; these specialized uses are characterized by very low volume, low exposure and low releases (EPA 2009c, 2013a).
- The Agency for Toxic Substances and Disease Registry has not established a minimal risk level (MRL) for PFOS or PFOA; when the draft toxicological profile was published, human studies were insufficient to determine with a sufficient degree of certainty that the effects are either exposure-related or adverse (ATSDR 2009).
- The EPA has not derived a chronic oral reference dose (RfD) or chronic inhalation reference concentration (RfC) for PFOS or PFOA and has not classified PFOS or PFOA carcinogenicity.
- The EPA removed PFOS and PFOA from the Integrated Risk Information System (IRIS) agenda in a Federal Register notice released on October 18, 2010. At this time, EPA is not conducting an IRIS assessment for these chemicals (EPA 2010).
- PFOS and PFOA were included on the third drinking water contaminant candidate list, which is a list of unregulated contaminants that are known to, or anticipated to, occur in public water systems and may require regulation under the Safe Drinking Water Act (EPA 2009a).

What detection and site characterization methods are available for PFOS and PFOA?

- PFOS and PFOA are commonly deposited in the environment as discrete particles with strongly heterogeneous spatial distributions. Unless precautions are taken, this distribution causes highly variable soil data that can lead to confusing or contradictory conclusions about the location and degree of contamination. Proper sample collection (using an incremental field sampling approach), sample processing (which includes grinding) and incremental subsampling are required to obtain reliable soil data (EPA 2003, 2013c).
- PFOS and PFOA in anionic form can be extracted from environmental media by conventional methods using either acidification or ion pairing to obtain a neutral form of the analyte. Sample preparation methods used for PFCs have included solvent extraction, ion-pair extraction, solid-phase extraction and column-switching extraction (Flaherty and others 2005).
- Precursors and intermediate degradation products can be extracted using solvents (Dasu and others 2012; Ellington and others 2009).
- Air samples may be collected using high-volume air samplers that employ sampling modules containing glass-fiber filters and glass columns with a polyurethane foam (Jahnke and others 2007a).
- Detection methods for PFCs are primarily based on high-performance liquid chromatography (HPLC) coupled with tandem mass spectrometry (MS/MS). HPLC-MS/MS has allowed for more sensitive determinations of individual PFOS and PFOA in air, water and soil (EFSA 2008; Jahnke and others 2007b; Washington and others 2008).
- Both liquid chromatography (LC)-MS/MS and gas chromatography-mass spectrometry (GC-MS) can be used to identify the precursors of PFOS and PFOA (EFSA 2008).
- EPA Method 537, Version 1.1, is an LC-MS/MS method used to analyze selected perfluorinated alkyl acids in drinking water. While most sampling protocols for organic compounds require sample collection in glass, this method requires plastic sample bottles because PFCs are known to adhere to glass (EPA 2009b).
- The development of LC - electrospray ionization (ESI) MS and LC-MS/MS has improved the analysis of PFOS and PFOA (EFSA 2008).
- Reported sensitivities for the available detection methods include low picograms per cubic meter (pg/m^3) levels in air, high picograms per liter (pg/L) to low ng/L levels in water and high picogram per gram to low ng/g levels in soil (ATSDR 2009).

What technologies are being used to treat PFOS and PFOA?

- Because of their unique physicochemical properties (strong fluorine-carbon bond and low vapor pressure), PFOS and PFOA resist most conventional in situ treatment technologies, such as direct oxidation (Hartten 2009; Vectis and others 2009).
- Factors to consider when selecting a treatment method in all media include: (1) initial concentration of PFCs; (2) the background organic and metal concentration; (3) available degradation time; and (4) other site-specific conditions (Vectis and others 2009).
- Ex situ treatments including activated carbon filters, nanofiltration and reverse osmosis units have been shown to remove PFCs from water; however, incineration of the concentrated waste would be needed for the complete destruction of PFCs (Hartten 2009; MDH 2008; Vectis and others 2009).
- Research into a cost-effective treatment approach for PFOS and PFOA is ongoing (DoD SERDP 2012).
- Alternative technologies studied for PFOS and PFOA degradation in water, soil and solid waste include photochemical oxidation and thermally induced reduction, which have achieved some bench-scale success (Hartten 2009; Vectis and others 2009).
- Laboratory-scale studies have also evaluated sonochemical degradation (that is, ultrasonic irradiation) to treat PFOS and PFOA in groundwater and have reported a sonochemical degradation half-life less than 30 minutes for both PFOS and PFOA (Cheng and others 2008, 2010).
- Results from a laboratory-scale study suggested the promising potential of using a double-layer permeable reactive barrier (DL-PRB) system for the in situ containment of PFC-contaminated soil and groundwater. The DL-PRB system is composed of an oxidant-releasing material layer followed by a layer of quartz sands immobilized with humification enzymes. The system drives enzyme-catalyzed oxidative humification reactions to degrade PFCs in the PRB (DoD SERDP 2013).
- In situ chemical oxidation is being explored as a possible means to treat PFCs in water. Laboratory-scale study results indicate that heat-activated persulfate and permanganate can effectively degrade PFOS and PFOA in water (Liu and others 2012a, b).

Where can I find more information about PFOS and PFOA?

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