Development of Toxicity Values for GenX Chemicals and PFBS

Briefing #2 to States and Federal Agencies

US EPA

May 2, 2018

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Purpose of this Briefing

- Provide States and federal agencies an overview of EPA's analysis and effects characterization of toxicity values for two PFAS chemicals
  - GenX chemicals – assessment led by EPA Office of Water and Office of Pollution Prevention and Toxics
  - PFBS – assessment led by EPA Office of Research and Development

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Overall Scientific Objectives

- Provide the health effects information for the development of toxicity values (e.g., oral reference doses) including the science-based decisions supported by relevant studies, effects, and estimated point(s) of departure (POD)

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Plan for Engagement:

- States and Federal Agencies: Update # 1 – problem formulation and review of available information (3/9/2018)
- States and Federal Agencies: Update #2 – overview of analysis, including effects characterization and approaches to derivation of toxicity values
- Independent, external peer review
- States and Federal Agencies: Update #3 – Summary of external peer review comments, Agency response, and determination of toxicity values
- Public meeting to present the toxicity values and discuss risk communication

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Document Structure

- Background
- Nature of the stressor including occurrence, chemical and physical properties and toxicokinetics
- Problem Formulation, including conceptual model and analysis plan
- Study Synthesis
- Summary of Hazard
- Dose response assessment including modeling, uncertainty factors and derivation of Reference Value(s)
- Characterization of Uncertainties

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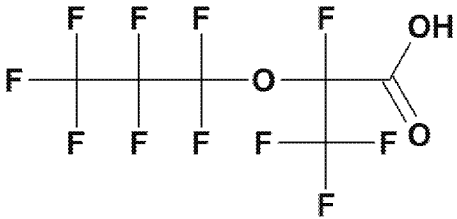
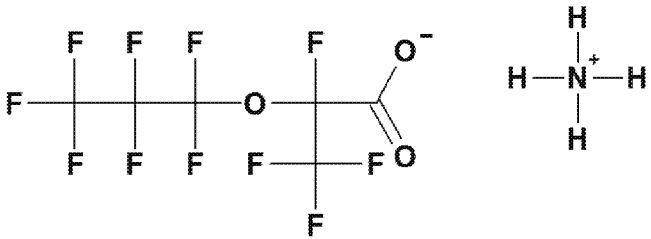
GenX Chemicals



HFPO dimer acid
CASRN 13252-13-6



HFPO dimer acid, ammonium salt
CASRN 62037-80-3

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Environmental Fate

- GenX chemicals are stable to photolysis, hydrolysis and biodegradation and are persistent in air, water, soil and sediments.
- Highly soluble
- Low sorption to sediment and soil
  - Potential to rapidly leach to groundwater from soil and landfills.
- Partitioning from surface water to the vapor phase may occur.
  - They may undergo long range atmospheric transport in the vapor phase and be associated with particulate matter.
  - Removal from air may occur by scavenging by water droplets and attachment to particulates followed by precipitation and settling.
- They are not expected to be removed during wastewater treatment or conventional drinking water treatment.
- They have low potential to bioaccumulate in fish.

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Occurrence

- Monitoring for GenX chemicals is limited.
  - GenX chemicals were first identified in North Carolina's Cape Fear River and its tributaries in the summer of 2012.
  - Sun et al. (2016) reported detections of GenX chemicals in three drinking water treatment plants treating surface water from the Cape Fear River watershed.
  - Subsequent monitoring by NCDEQ reported GenX chemicals in surface water, groundwater, and finished drinking water in the Cape Fear Watershed close to the Chemours facility where the chemicals were used and 100 miles downstream.
  - GenX chemicals have also been detected in three on-site production wells and one on-site drinking water well at Chemours' Washington Works facility in Parkersburg, West Virginia.
  - GenX chemicals were found in rainwater samples collected between February 28-March 2, 2018 up to 7 miles from the North Carolina plant.
  - EPA's ORD is providing monitoring assistance to North Carolina and New Jersey.

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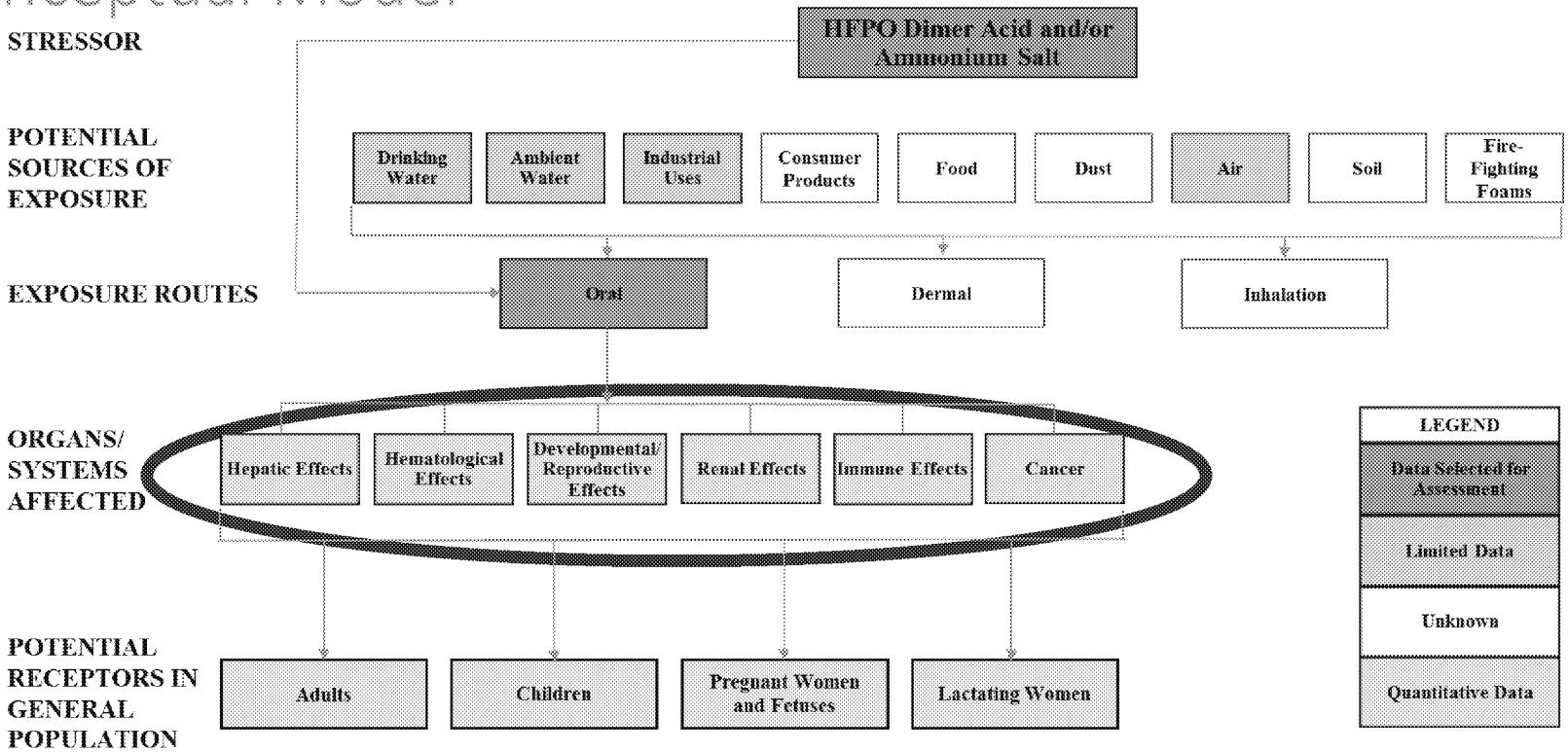
Problem Formulation

Conceptual Model



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Study Evaluation for GenX Chemicals

- Many of the available studies were conducted by industry to support new uses and Pre-Manufacturing Notifications and were submitted to the Agency for review.
  - These studies are available through the HERO database: https://hero.epa.gov/hero/index.cfm/project/page/project_id/2627
- Studies were designed and implemented according to OECD Test Guidelines and followed Principles of Good Laboratory Practices.
- EPA evaluated the studies based on Agency Guidelines and criteria to determine if the studies:
  - Adequately describe study protocol and methods
  - Evaluate appropriate endpoints
    - Toxicity depends on the amount, duration, timing and pattern of exposure, and could range from frank effects (e.g., mortality) to subtler biochemical, physiological, pathological or functional changes in multiple organs and tissues.
  - Use appropriate statistical procedures to determine an effect
  - Establish a dose-response relationship (i.e., NOAEL) and/or lowest observed adverse effect level (LOAEL)
  - Have data to identify a POD for a change in the effect considered to be adverse (out of the range of normal biological variability).

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Available Studies

Published Peer Reviewed Literature

- 28 day oral toxicity study evaluating hepatotoxic effects in mice (Wang et al., 2016)
- 28 day oral toxicity study evaluating immunomodulatory effects in mice (Rushing et al., 2017)
- 2 studies that are published versions of DuPont/Chemours data:
  - The OECD 453 combined chronic toxicity/oncogenicity study (2 year) in rats (Rae et al., 2015)
  - An oral, single dose pharmacokinetic study describing absorption, distribution, metabolism, and elimination in rats, mice and cynomolgus monkeys (Gannon et al., 2016)

DuPont/Chemours Studies

- Acute oral, dermal, and inhalation toxicity studies
- Toxicokinetic studies
- Genotoxicity studies (in vivo and in vitro)
- Repeated-dose metabolism and pharmacokinetics in rats and mice (OPPTS 870.7485)
- 28 day oral toxicity study in mice and rats (OECD TG 407)
- 90-day toxicity study (OPPTS 870.3100; OECD 408)
- Chronic toxicity/carcinogenicity study in rats (OPPTS 870.4300; OECD 408)
- One-generation reproduction study in mice (OECD 421, modified)

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EFFECTS CHARACTERIZATION

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28-Day Oral Toxicity Studies (Chemours)

OECD Guideline 407

EPA

Mouse

- DuPont 24459
- Dose (gavage):
  - 0, 0, 0.1, 3 and 30 mg/kg/day
- Effects:
  - Liver effects (↑ relative liver weight in both sexes and ↑ hepatocellular hypertrophy in both sexes and single cell necrosis in males)
  - Hematological effects (↓ hemoglobin and hematocrit in males)
  - Immune effects (↓ globulin in females and ↑ A/G ratio in both sexes)
- NOAEL = 0.1 mg/kg/day

Rat

- DuPont 24447
- Dose (gavage):
  - 0, 0.3, 3 and 30 mg/kg/day (males)
  - 0, 3, 30 and 300 mg/kg/day (females)
- Effects:
  - Liver effects (↑ relative liver weight and hepatocellular hypertrophy in males)
  - Hematological effects (↓ erythrocyte count, hemoglobin, and hematocrit in males)
  - Immune effects (↓ globulin and ↑ A/G ratio in males)
- NOAEL = 0.3 mg/kg/day

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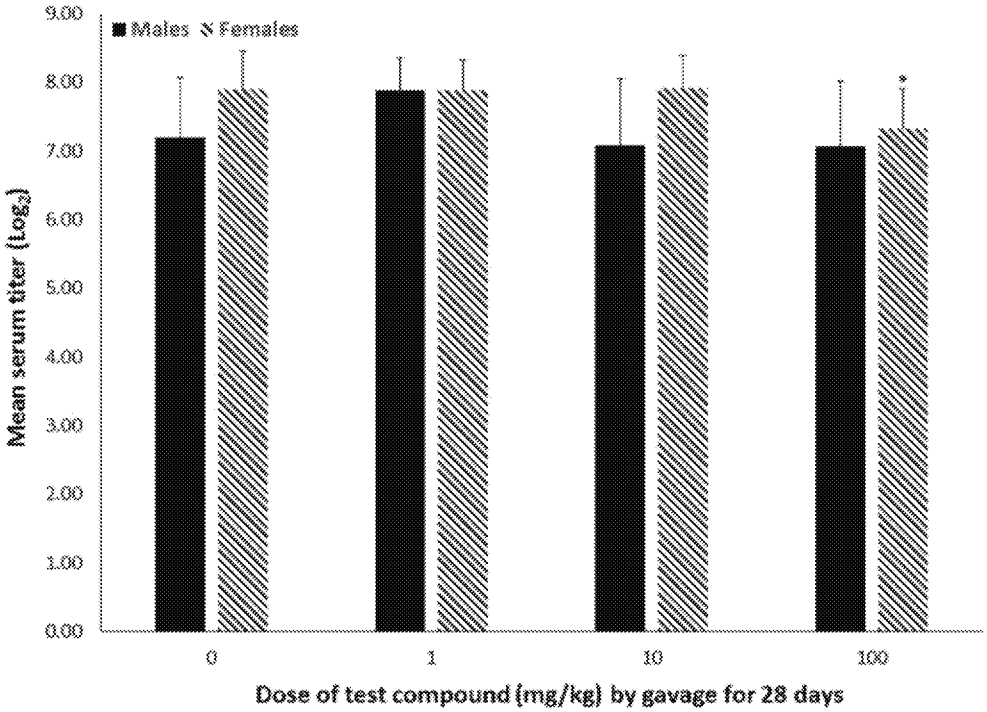
28-Day Oral Immunotoxicity Study

Rushing et al., 2017

- C57BL/6 mice
- 0, 1, 10, and 100 mg/kg/day HFPO dimer acid
- Effects:
  - TDAR suppression in females
  - ↑ lymphocytes in males
- NOAEL = 10 mg/kg/day



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90-Day Oral Toxicity Studies (Chemours)

OECD Guideline 408

Mouse

- DuPont 18405-1307
- 0, 0.1, 0.5, and 5 mg/kg/day
- Effects:
  - Liver enzyme level changes (↑aspartate aminotransferase, alanine aminotransferase, and alkaline phosphatase) in both sexes
  - ↑ relative liver weight in both sexes
  - ↑ hepatocellular hypertrophy and single cell necrosis in males
- NOAEL = 0.5 mg/kg/day

Rat

- DuPont 17751-1026
- 0, 0.1, 10 and 100 mg/kg/day (males) and 0, 10, 100, and 1000 mg/kg/day (females)
- Effects:
  - ↓ erythrocyte count, hemoglobin, and hematocrit in males
- NOAEL = 0.1 mg/kg/day

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2-Year Oral Toxicity/Carcinogenicity Study

OECD Guideline 453

- DuPont 18405-1238
- Crl:CD(SD) rats
- 0, 0.1, 1, and 50 mg/kg/day (males) and 0, 1, 50, and 500 mg/kg/day (females)
- Effects:
  - ↑ liver enzyme levels (alkaline phosphatase, ALT, and SDH) in males
  - ↑ centrilobular hepatocellular hypertrophy and cystic focal degeneration in males
  - ↑ centrilobular necrosis in both sexes
- NOAEL = 1 mg/kg/day

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Oral Reproductive/Developmental Toxicity Study

Modified OECD Guideline 421

- DuPont 18405-1037
- Crl:CD1(ICR) mice
- 0, 0.1, 0.5, and 5 mg/kg/day
- Effects:
  - F0- ↑ relative liver weight in both sexes and single cell necrosis in males
  - Offspring- ↓ pup weights and delays in the attainment of balanopreputial separation and vaginal patency
- NOAEL = 0.1 mg/kg/day (F0) and 0.5 mg/kg/day (offspring)

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Oral Prenatal and Developmental Screening Study

OECD Guideline 414

- DuPont 18405-841
- 0, 10, 100, and 1000 mg/kg/day
- Effects:
  - ↑ early deliveries and↓ gravid uterine weight
  - ↓fetal weights in both sexes
- NOAEL = 10 mg/kg/day (maternal and offspring)

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Study	NOAEL (mg/kg/day)	Effects
DuPont 24447: 28-Day Oral (Gavage) Toxicity Study in Rats	NOAEL = 0.3	• Liver effects • Hematological effects • Immune effects
DuPont 24459: 28-Day Oral (Gavage) Toxicity Study in Mice	NOAEL = 0.1	• Liver effects • Hematological effects • Immune effects
Rushing et al. (2017): 28-day Oral (Gavage) Immunotoxicity Study in Mice	NOAEL = 10	• Immune effects
DuPont 17751-1026: 90-Day Oral (Gavage) Toxicity Study in Rats	NOAEL = 0.1	• Hematological effects
DuPont 18405-1307: 90-Day Oral (Gavage) Toxicity Study in Mice	NOAEL = 0.5	• Liver effects
DuPont 18405-1238: Combined Chronic Toxicity/ Oncogenicity Study in Rats	NOAEL = 1	• Liver effects
DuPont 18405-1037 Oral (Gavage) Reproduction/ Developmental Toxicity Screening Study in Mice	NOAEL (F0) = 0.1 NOAEL (offspring) = 0.5	• Liver effects • Developmental effects
DuPont 18405-841 Prenatal and Developmental Toxicity Study in Rats	NOAEL (maternal and offspring) = 10	• Developmental effects

Weight of Evidence for Hazard

- Adverse effects are observed in the liver, developing fetus, and hematological and immune systems.
- The single cancer bioassay show increased liver tumors (females) and combined adenomas and carcinomas pancreatic acinar (males) in rats at the high doses only.
  - There was an increased incidence of testicular interstitial cell adenoma in males, but this increase was not statistically significant.
  - There are no studies measuring cancer endpoints in mice.
- Liver is primary target of toxicity. Effects are observed in both male and female mice and rats at varying durations of exposures and doses and are the endpoints that are observed at the lowest doses of exposure to these chemicals.
  - Use of Hall et al. (2012) criteria for adversity of liver endpoints.
    - Hepatocellular hypertrophy and an increased liver weight are common findings in rodents, but are often considered non-adverse if there is evidence for PPARα activation.
    - These effects were considered adverse when accompanied by necrosis, fibrosis, inflammation, and/or steatosis.

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APPROACH FOR DERIVATION OF REFERENCE DOSE

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Approach for Dose-Response Assessment

- Follow the general guidelines for risk assessment set forth by the National Research Council (1983) and EPA's *Framework for Human Health Risk Assessment to Inform Decision Making* (2014)
- EPA's *A Review of the Reference Dose and Reference Concentration* (2002) document describes a multi-step approach to dose–response assessment including analysis in the range of observation followed by extrapolation to lower levels.

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Selection of Critical Study and Effect

- Studies were evaluated based on duration of exposure, use of a control and two or more doses, and provision of NOAEL and/or LOAEL values.
  - Given the availability of subchronic, chronic and reproductive and developmental toxicity studies indicating effects at lower doses, the 28-day studies were not considered quantitatively.
- From the available subchronic (90 day), chronic (2-year cancer bioassay) and reproductive and developmental toxicity studies, the studies that observed adverse effects at the lowest doses tested are considered in the selection of the critical study for derivation of the RfD.
  - NOAELs for liver effects range from 0.1-1 mg/kg/day
  - NOAEL for hematological effects is 0.1 mg/kg/day

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Determination of Point of Departure

Benchmark Dose Modeling

- Use of EPA's *Benchmark Dose Technical Guidance Document* (2012).
  - No biologically based dose-response models are available
- Considerations influencing selection of BMD model endpoints include: available data with dose-response, percent change from controls, adversity of effect, and consistency in effect observed across studies.

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Determination of Point of Departure

Allometric scaling

- Use *Recommended Use of Body Weight$^{3/4}$ as the Default Method in Derivation of the Oral Reference Dose* (2011) when applicable.
  - Use of a body weight scaling applied to extrapolate toxicologically equivalent doses of oral doses from adult laboratory animals to adult humans.
  - Addresses some aspects of cross-species extrapolation of toxicokinetic and toxicodynamic processes and affects interspecies uncertainty factor.

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Characterization of Uncertainty

Uncertainty factors will be selected in accordance with EPA guidelines considering the following:

- Variations in sensitivity among humans (UF_H)
  - No information to is available to characterize interindividual and age-related variability in the toxicokinetics or toxicodynamics.
- Differences between animals and humans (UF_A)
  - Use of allometric scaling will address some of the toxicokinetic and toxicodynamic aspects
- Duration of exposure in the key study compared to a lifetime of the species studied (UF_S)
- Extrapolation from a LOAEL to a NOAEL (UF_L)
  - When the POD type is a BMDL, the current approach is to address this factor as one of the considerations in selecting a BMR for BMD modeling.

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Characterization of Uncertainty (Cont'd)

EPA

Completeness of the toxicology database (UF_D)

- There are no data from epidemiological studies in the general population or worker cohorts available for use in evaluating human health effects.
- The database available to EPA assesses numerous endpoints: acute toxicity, metabolism and toxicokinetics, genotoxicity, and systemic toxicity in mice and rats with dosing durations of up to 2 years.
  - Deficiencies in the database include limited developmental toxicity testing and immune studies.

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Derivation of RfDs

EPA will use the information described above to derive both a subchronic and a chronic toxicity value, or RfD:

$$\text{Subchronic RfD} = \frac{POD_{HED}}{Total\ UF}$$

$$\text{Chronic RfD} = \frac{POD_{HED}}{Total\ UF}$$

Total Uncertainty Factor ($Total\ UF$) will be different for subchronic and chronic RfD calculations

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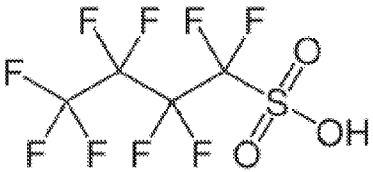
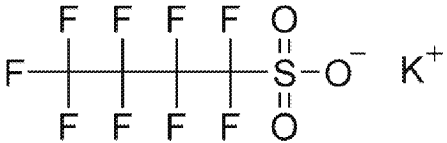
Perfluorobutane sulfonate (PFBS)

1-Perfluorobutanesulfonic Acid



Potassium Perfluorobutane Sulfonate



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PFBS-Occurrence

- EPA's UCMR data for public drinking water utilities in 2013-2015 showed levels of PFBS above the Minimum Reporting Level (>0.09µg/L) in water systems serving Georgia, Colorado, Alabama, Pennsylvania and the Northern Mariana Islands (EPA 2017).
- Measurements from 37 surface water bodies in the Northeastern United States (metropolitan New York area and Rhode Island) collected in 2014 showed an 85% site detection rate (Zhang 2016).
- PFBS has also been identified in surface waters in North Carolina, Georgia, New Jersey and the Upper Mississippi River Basin (Lasier 2011; Nakayama 2007; Nakayama 2010; Post 2013).
- It has been detected in wastewater treatment plant effluent, seawater, soil and biosolids (Houtz 2016; Sepulvado 2011; Zhao 2012).

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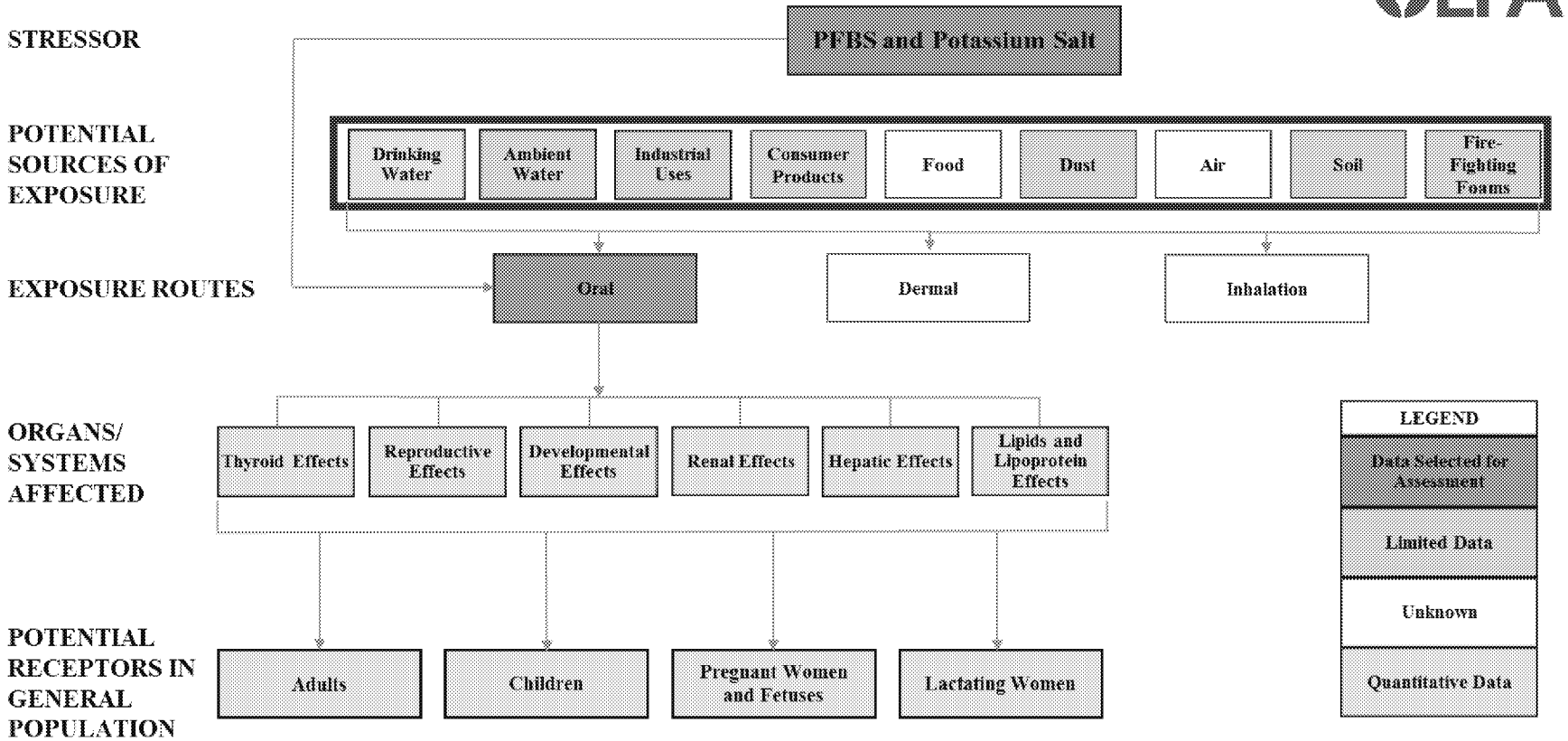
Problem Formulation-PFBS



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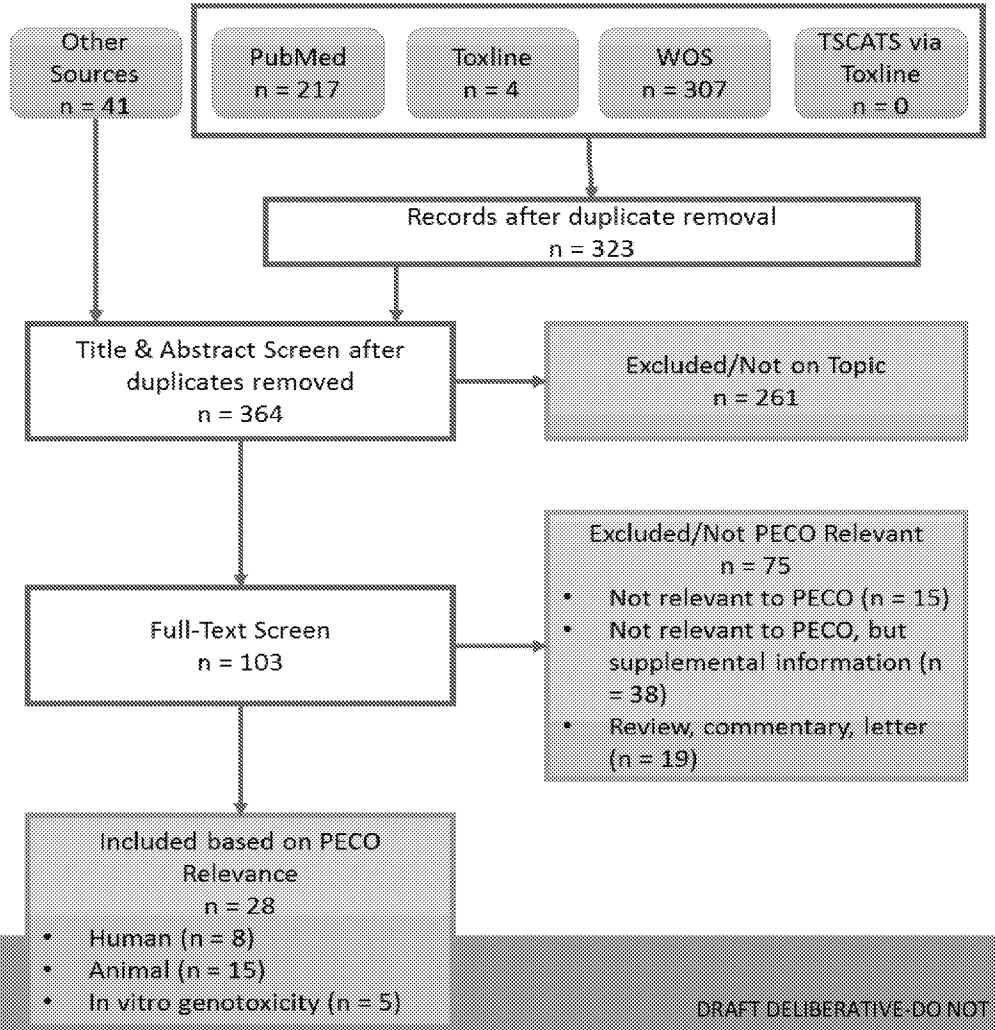

- Four online scientific databases (PubMed, Web of Science, Toxline, and TSCATS via Toxline) were searched.
- In addition, studies were identified by our colleagues in EPA/OPPT (Other Sources).
- Two screeners independently conducted a title and abstract screen.
- Studies that met the Population, Exposure, Comparator, and Outcome (PECO) criteria were then full-text reviewed and moved on to data study/data evaluation and extraction.

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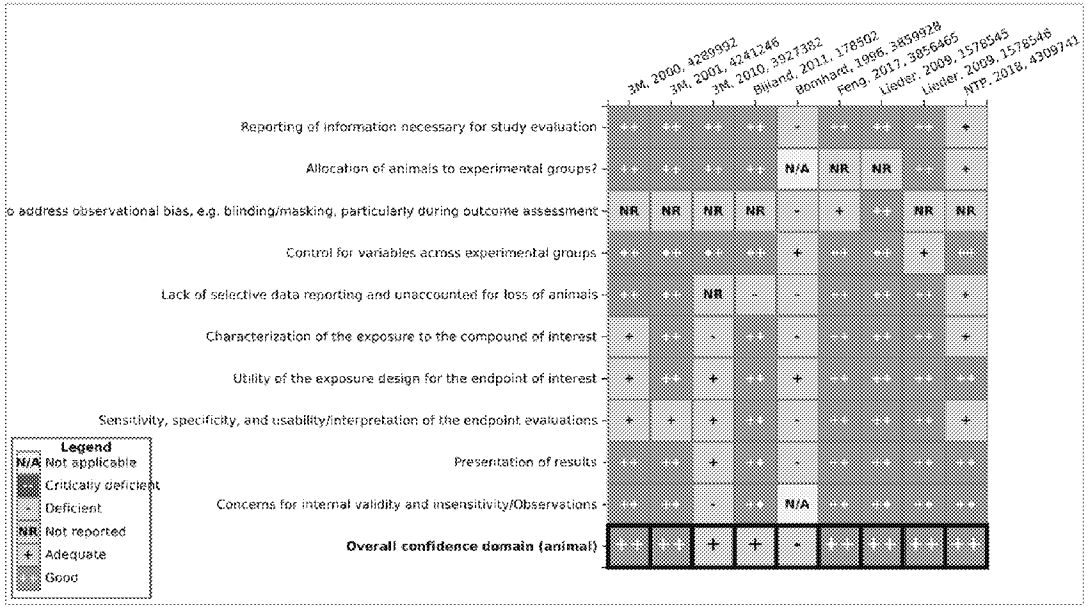
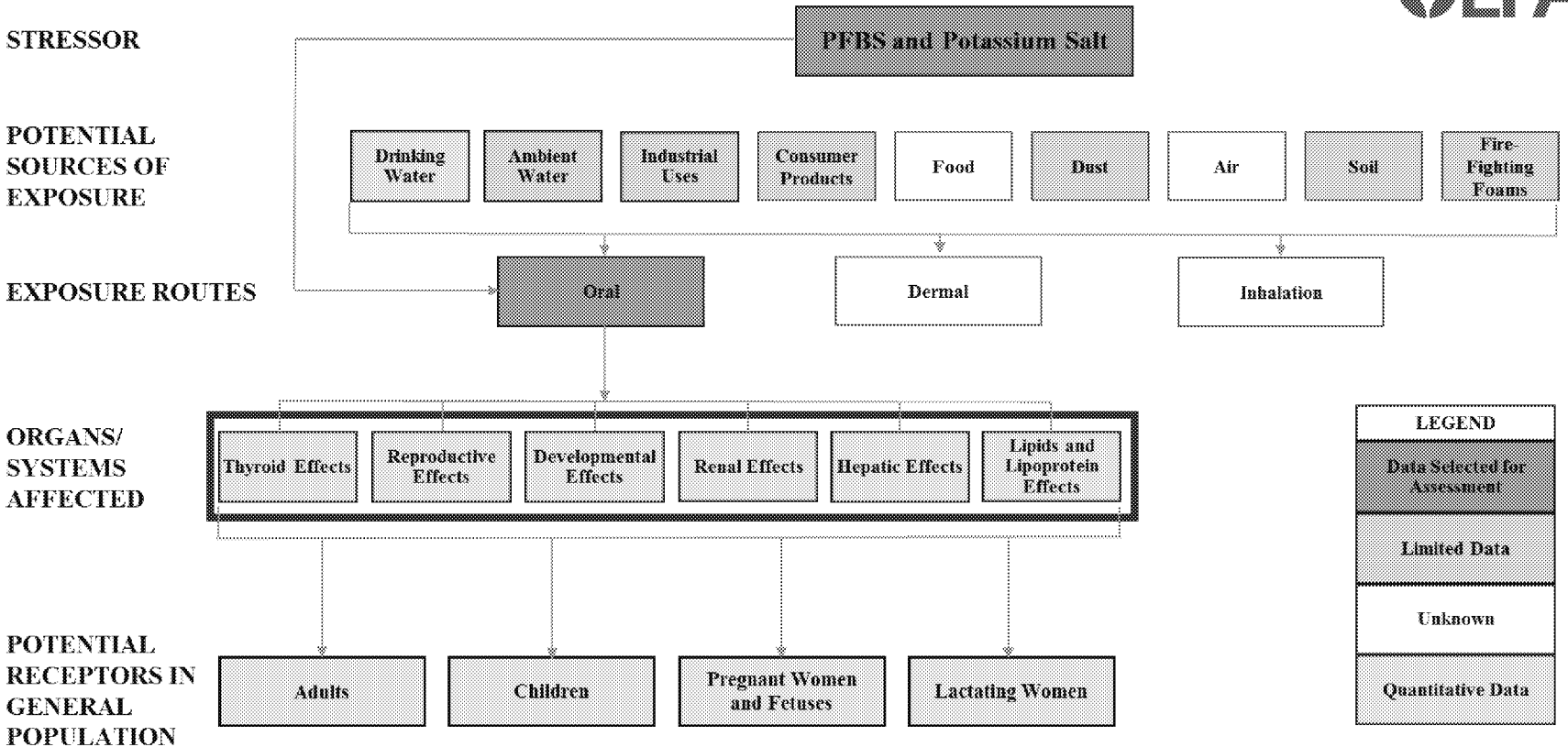
Study Quality Evaluation

- Studies were evaluated based on predefined criteria to assess the potential for bias and insensitivity.
- Overall judgments for each study were determined to define confidence in the reliability of the results.



PFBS-Effects



Human Studies

- Six epidemiologic studies in eight publications were identified.
- Outcomes assessed in human populations include female and male reproduction, kidney function (e.g., filtration), serum lipids, and asthma.
- The ability to draw conclusions about associations was limited due to the small number of studies per outcome. Thus, the evidence was considered inadequate for hazard characterization and not considered further for derivation of toxicity values.

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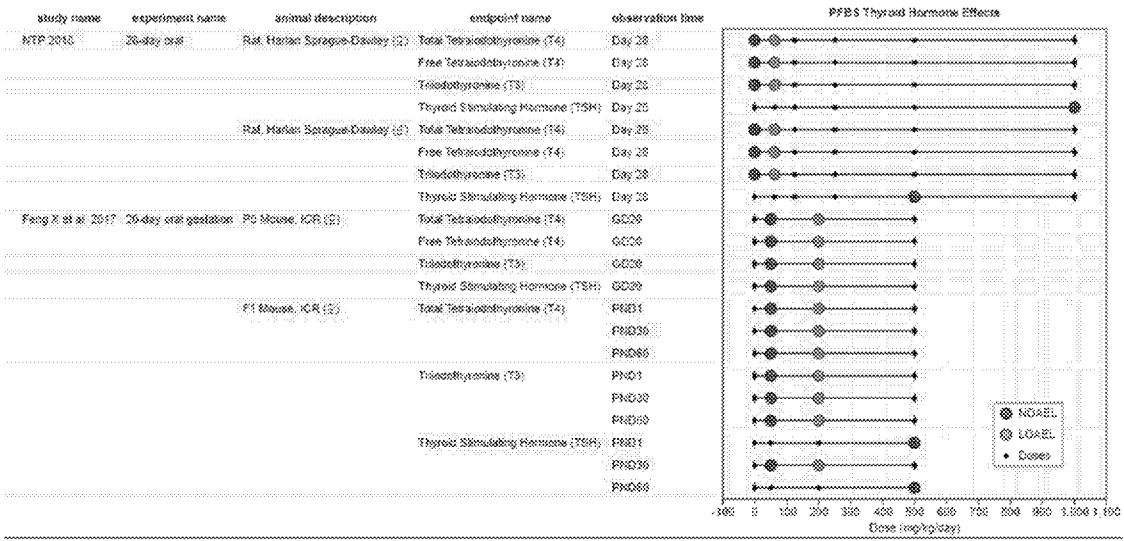
Study	NOAEL (mg/kg/day)	LOAEL (mg/kg-day)	Effects at LOAEL
3M (2000): 10-Day Oral (Gavage) Toxicity Study in Rats	300	1000	• Liver effects
3M (2001): 28-Day Oral (Gavage) Toxicity Study in Rats	300	900	• Liver effects • Renal effects
NTP (2018): 28-day Oral (Gavage) Toxicity Study in Rats	Not Determined	62.6	• Thyroid effects • Liver effects • Renal effects • Reproductive effects
Lieder et al. (2009); York (2003): 90-Day Oral (Gavage) Toxicity Study in Rats	200	600	• Renal effects
Bijland et al. (2011); 3M (2010): 4-6 Week Oral (Diet) Lipid/Lipoprotein Metabolism Study in Mice	Not Determined	Not Determined	• Lipid effects of uncertain biological significance
Feng et al. (2017): GD 1-20 Developmental Study in Mice	50	200	• Thyroid effects • Developmental effects • Reproductive effects
York (2003) GD 6-20 Developmental Toxicity Screening Study in Rats	Not Determined	Not Determined	• Maternal and pup body weight changes of uncertain significance
York (2002) GD 6-20 Developmental Toxicity Study in Rats	Not Determined	Not Determined	• Maternal and pup body weight changes of uncertain significance
Lieder et al., (2009); York (2003) 2-Generation Reproductive Toxicity Study in Rats	100 (F0 and F1) 1,000 (F2)	300 (F0 and F1) ND (F2)	• Renal effects

Thyroid

- Two high confidence studies evaluated the effects of PFBS exposure on thyroid, specifically thyroid hormone levels, thyroid histopathology, and thyroid weight.
- PFBS-induced perturbation of the thyroid was consistently observed across two species, sexes, lifestages, and varied exposure durations.
- Similar patterns of decreases in total T3, total T4, and free T4 were observed in PFBS-exposed pregnant and non-pregnant female mice, adult male rats from a 28-d study, and gestationally exposed female mouse offspring.
- The available evidence supports thyroid as a potential hazard.



Reproductive

- Reproductive outcomes were evaluated in a prenatal exposure study in mice, in two gestational exposure studies in rats, in short-term and subchronic-duration studies in rats, and in a two-generation reproductive study in rats.
- Endpoints evaluated in these studies include fertility and pregnancy outcomes, hormone levels, markers of reproductive differentiation and development, and reproductive organ weights.
- In general, PFBS exposure in adult animals has resulted in no significant alterations in male and female fertility, pregnancy outcomes, and reproductive hormones.
- Inconsistent changes in reproductive organ weights were reported across experimental animal studies regardless of duration and timing of exposure.
- Several measures of female reproductive development were affected by gestational exposure to PFBS with effects persisting into adulthood, including decreased female fertility parameters (i.e., decreased follicles and corpora lutea) and reproductive hormones (i.e., reduced serum estradiol and progesterone), delayed first estrous, altered estrous cyclicity, and potentially a delay to vaginal opening.
- The evidence indicate that the developing, but not adult, reproductive system may be a target for PFBS toxicity.

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Developmental

- Four high or medium confidence studies examined potential alterations in developmental effects following PFBS exposure, including two gestational exposure studies in rats, one in mice, and a two-generation study in rats.
- None of the studies identified significant effects in either rats or mice on measures of fetal alterations (i.e., malformations and variations) and inconsistent reports of reduced fetal body weights.
- Developmental delays in reproductive organ growth and function, thyroid development, and delayed eye opening in rats exposed to PFBS in utero (e.g., GD 1-20).
- Due to the coherence across both thyroid and developmental effects, and biological plausibility of a link between the effects, the evidence for effects in the developing offspring supports a hazard and the developing offspring is considered a target for PFBS toxicity.

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Kidney

- Kidney effects were evaluated following short-term and subchronic-duration exposure to PFBS in rats and in a two-generation reproductive study in rats.
- Endpoints evaluated in these studies include kidney weights, histopathological changes, and serum biomarkers of effect (e.g., BUN).
- Increased kidney weight has been observed in male and female rats but typically at higher PFBS doses (≥ 500 mg/kg-d).
- Histopathological alterations were observed in the kidneys of male and female rats across studies such as hyperplasia of renal medulla and papillary tubular epithelium, papillary edema, chronic progressive nephropathy, hydronephrosis, mineralization, and tubular degeneration and necrosis.
  - Many histopathological changes were not significant compared to controls or were not dose-dependent (chronic progressive nephropathy, hydronephrosis, mineralization, papillary edema, and tubular degeneration), or occurred only at the highest tested PFBS study dose (renal papillary necrosis).
  - Hyperplasia of kidney was identified as a dose-dependent effect in the 90-day subchronic-duration study (Lieder, 2009a) and the two-generation reproductive study (Lieder, 2009b).
- Consistency in kidney effects across subchronic adult rat, and, rats exposed across lifestages supports this organ as a target of PFBS toxicity.

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Liver

- Liver effects were evaluated following short-term and subchronic-duration exposure to PFBS in rats and in a two-generation reproductive study in rats.
- Endpoints evaluated in these studies include liver weights, histopathological changes, and serum biomarkers of effect.
- Absolute and/or relative liver weights were observed in male or female rats in 10- and 28-d studies but only at higher oral doses (≥ 900 mg/kg-d).
  - The magnitude of liver weight change was significantly different between sexes of rat.
  - Although NTP (2018) and Lieder et al. (2009b) observed increased liver weights in rats following 28-d or approximately 70-d of exposure, respectively, no liver weight changes in either sex of rat were observed in the 90-d subchronic-duration study (Lieder et al., 2009a).
- Liver histopathology was not consistently observed across PFBS studies. Lesions either occurred in only one sex of rat, was not dose-dependent compared to control, and/or only occurred at the highest PFBS dose tested.
- Based on the available data, the biological relevance or significance of PFBS-induced liver effects is not clear and are not considered further for dose-response analysis.

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Lipid/Lipoprotein

- Increasing evidence has revealed perturbations in lipid homeostasis as a biological effect across a variety of PFAS.
- Most PFBS studies have not particularly focused on this effect as only measures of serum cholesterol and triglyceride were included as part of a broader panel of clinical chemistry measures in rat studies of 10-, 28-, and 90-days.
- Circulating levels of cholesterol and triglycerides were either unchanged, not significantly changed compared to controls, and/or occurred in only one sex of tested species.
- A specialized study was located where transgenic (APOE*3-Leiden CETP) mice, which are highly responsive to fat and cholesterol intake consistent with human populations exposed to a western-type diet, were evaluated following 4-6 weeks of dietary PFBS exposure.
  - Most measures of circulating lipids showed decreases compared to control diet-fed mice.
  - Modest increase in hepatic lipid load with corresponding gene expression changes.
- Overall, information pertaining to PFBS mediated perturbations in lipid homeostasis is insufficient to determine significance to human health, and these effects are not considered further for dose-response analysis.

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Dose-Response Modeling

- Effects in the kidney, thyroid, and developing offspring were further considered for dose-response modeling.
- Modeling of kidney histopathology resulted in potential POD(HEDs) ranging from 13 to 44 mg/kg-day.
- Modeling of thyroid hormone alterations resulted in potential POD(HEDs) ranging from 2 to 8 mg/kg-day for dams and 2 to 9 mg/kg-day in offspring.
- Modeling of developmental effects resulted in potential POD(HEDs) ranging from 3 to 15 mg/kg-day.

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Next Steps

- Independent External Peer Review (May-June 2018)
- Meet with States and Federal Agencies to discuss peer review comments and specific implications for GenX and PFBS toxicity assessments (June 2018)
- Public Meeting (July 2018)

ED_002330A_00004855-00044

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