EAST WATERWAY OPERABLE UNIT SUPPLEMENTAL REMEDIAL INVESTIGATION/ FEASIBILITY STUDY APPENDIX B: BASELINE HUMAN HEALTH RISK ASSESSMENT

ADDENDUM: cPAH TEQ UPDATES

For submittal to:

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Region 10
Seattle, WA

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Acronyms

API	Asian and Pacific Islander
BHC	benzene hexachloride
CDI	chronic daily intake
COC	contaminant of concern
COPC	contaminant of potential concern
cPAH	carcinogenic polycyclic aromatic hydrocarbon
CT	central tendency
DDT	dichlorodiphenyltrichloroethane
EPA	US Environmental Protection Agency
EPC	exposure point concentration
EW	East Waterway
HHRA	human health risk assessment
HI	hazard index
HQ	hazard quotient
PAH	polycyclic aromatic hydrocarbon
PCB	polychlorinated biphenyl
PEF	potency equivalency factor
RfD	reference dose
RL	reporting limit
RME	reasonable maximum exposure
TBT	tributyltin
TEQ	toxic equivalent

1 Introduction

In January 2017, the US Environmental Protection Agency (EPA) published updated toxicity values for benzo(a)pyrene (BaP). EPA updated the slope factor for BaP, which is used to estimate excess lifetime cancer risk from exposures to carcinogenic polycyclic aromatic hydrocarbons (cPAHs). EPA also published a reference dose (RfD) for BaP for non-cancer based on the developmental endpoint (neurobehavioral changes). Both of these toxicity values were published on EPA's Integrated Risk Information System website (EPA 2019).

cPAH risks are estimated for a cPAH toxic equivalent (TEQ). TEQs are commonly used to estimate total exposure concentrations for certain groups of chemicals, such as cPAHs. The new BaP toxicity values do not change how the TEQ is calculated using potency equivalency factors (PEFs), which relate the toxicity of six other cPAH compounds to that of BaP. However, because the cPAH TEQ is multiplied by the BaP slope factor to estimate excess cancer risk, changes to the BaP slope factor can have a large effect on the cPAH TEQ risk.

This addendum to the East Waterway (EW) human health risk assessment (HHRA) presents the updated risk calculations for cPAH TEQ and evaluates updates to the status of cPAHs as a contaminant of concern (COC) and risk driver for the EW.

This addendum is structured similarly to the EW HHRA, with updates made to the following sections as required (Table 1). With the exception of updating the toxicological profile for cPAHs to incorporate the revised toxicity values (see Attachment 1), no updates to the other EW HHRA appendices were needed.

Table 1. Summary of updates based on new BaP toxicity values

Section	Section Title	Summary of Updates
Section B.2	Data Evaluation	No updates were needed.
Section B.3	Exposure Assessment	No updates were needed.
Section B.4	Toxicity Assessment	Updated toxicity values are provided in Section 2 of this addendum.
Section B.5	Risk Characterization	Updated risk estimates for cPAH TEQ (and new risk totals for each scenario) are provided in Section 3 of this addendum.
Section B.6	Uncertainty Analysis	No updates were needed.
Section B.7	Identification of Risk Drivers	An updated determination of the status of cPAHs as a risk driver is provided in Section 4 of this addendum.
Section B.8	Conclusions	Updated conclusions are provided in Section 5 of this addendum.

BaP – benzo(a)pyrene
cPAH – carcinogenic polycyclic aromatic hydrocarbon
TEQ – toxic equivalent

2 Toxicity Value

Following the format used in the EW HHRA, details regarding the RfD and slope factor used to calculate the updated risk estimates for cPAH TEQ are presented in Table 2. The RfD and slope factor for benzo(a) pyrene were taken from EPA's IRIS database (EPA 2019), which is considered a Tier I source in EPA's hierarchy of toxicity values (Section 4.1, Appendix B). An updated toxicity profile for cPAHs is attached to this addendum (see Attachment 1).

Table 2. Applicable toxicity values for BaP

Parameter	Value
Non-cancer	
Oral RfD	3×10^{-4} mg/kg-day
Endpoint (critical effect)	developmental system (based on neurobehavioral changes)
Uncertainty factor	300
Date updated	January 19, 2017
Cancer[a]	
Oral slope factor	1 $(mg/kg\text{-}day)^{-1}$
Cancer description guideline	carcinogenic to humans
Date updated	January 19, 2017

[a] As described in the EW HHRA, there are additional considerations for calculating risks to children associated with cPAH TEQ, which has a mutagenic mode of action. Risks to children were calculated in accordance with EPA guidance (EPA 2005), which provides adjustments for chemicals that have a mutagenic mode of action.

BaP – benzo(a)pyrene
cPAH – carcinogenic polycyclic aromatic hydrocarbon
EPA – US Environmental Protection Agency
EW – East Waterway
HHRA – human health risk assessment
RfD – reference dose
TEQ – toxic equivalent

3 Updated Risk Calculations

This section presents the updated risk calculations for cPAH TEQ. As described, EPA's slope factor for BaP has decreased from 7.3 to 1 $(mg/kg\text{-}day)^{-1}$. Estimated excess cancer risks associated with cPAH TEQ are expected to decrease proportionally (i.e., also decrease by a factor of 7.3). However, because excess cancer risks are presented with one significant figure, their factor of decrease may not be exactly proportional to the factor of decrease of the slope factor.

In addition, non-cancer HQs were calculated for cPAH TEQ for all scenarios and were less than the threshold of one for all scenarios. As discussed in the memorandum *USEPA Updates to Human Health Toxicity Values for Benzo(a)pyrene and Potential Effects on Cleanup Levels and Remedial Action Levels in Portland Harbor*, included in the *Proposed Explanation of Significant Differences for the Portland Harbor Superfund Site* (EPA 2018), it is not clear whether EPA intended the new RfD to be applied to cPAH TEQ or only to BaP. In this HHRA addendum, the new BaP RfD was applied to cPAH TEQ. Because BaP is only one component of cPAH TEQ, risks associated with cPAH TEQ are greater than risks associated with BaP alone. This is a health-protective approach, such that if cPAH TEQ does not present an unacceptable risk (i.e., the hazard quotient [HQ] associated with cPAH TEQ is less than the acceptable risk threshold), neither will BaP present an unacceptable risk.

Excess cancer risks and non-cancer HQs were evaluated for seafood consumption and direct sediment exposure scenarios. cPAH TEQ was not a contaminant of potential concern (COPC) for the surface water exposure scenarios, so it was not necessary to calculate updated risk estimates for these scenarios. Details regarding changes to risk estimates are described in Table 3.

Table 3. Overview of risk tables in the EW HHRA and cPAH addendum

Scenario and Risk Type	Final EW HHRA Table Reference	Addendum Table	Notes Regarding Updated Risk Calculations
Seafood consumption scenarios			
Cancer risks	Table B.5-47	Table 4	Table presents updated cPAH TEQ excess cancer risks and updated total risks (green-shaded rows).
Non-cancer HQs	Table B.5-48; no cPAH TEQ HQs because no RfD was available for BaP at the time the HHRA was finalized	Table 5	Table presents non-cancer HQs for cPAH TEQ and the updated hazard index (HI)[a] for the developmental endpoint (which includes cPAH TEQ) (green-shaded rows).
Direct sediment contact scenarios			
Cancer risks	Table B.5-49	Table 6	Table presents updated cPAH TEQ excess cancer risks and updated total risks (green-shaded rows).

Scenario and Risk Type	Final EW HHRA Table Reference	Addendum Table	Notes Regarding Updated Risk Calculations
Non-cancer HQs	No table (all HQs < 1); no cPAH TEQ HQs because no RfD was available for BaP at the time the HHRA was finalized	none	As in the EW HHRA, no table is presented for these scenarios because all HQs for these scenarios (i.e., for cPAH TEQ and all other COPCs) are less than the threshold of one.

[a] Chemicals that affect the same organ or physiological function (called "toxicity endpoints") may have additive effects. For those chemicals, the HQs for the same endpoint may be summed as a hazard index (HI).

BaP – benzo(a)pyrene
COPC – contaminant of potential concern
cPAH – carcinogenic polycyclic aromatic hydrocarbon
EW – East Waterway
HHRA – human health risk assessment
HQ – hazard quotient
RfD – reference dose
TEQ – toxic equivalent

Table 4. Updated summary of estimated excess cancer risks for the seafood consumption scenarios

COPC	Estimated Excess Cancer Risk											
	Adult Tribal RME (Tulalip Data)	Adult Tribal CT (Tulalip Data)	Child Tribal RME (Tulalip Data)	Child Tribal CT (Tulalip Data)	Adult Tribal (Suquamish Data)	Adult API RME	Adult API CT	Adult One Meal per Month				
								Benthic Fish	Clam	Crab	Pelagic Fish, Rockfish	Pelagic Fish, Perch
Arsenic[b]	2×10^{-4}	1×10^{-5}	4×10^{-5}	4×10^{-6}	2×10^{-3}	8×10^{-5}	2×10^{-6}	3×10^{-7}[c]	1×10^{-5}	2×10^{-6}	7×10^{-7}	2×10^{-6}
cPAH TEQ	1×10^{-5}	6×10^{-7}	1×10^{-5}	1×10^{-6}	1×10^{-4}	7×10^{-6}	1×10^{-7}	2×10^{-8}	1×10^{-6}	5×10^{-8}	1×10^{-8}	7×10^{-8}[c]
1,4-Dichlorobenzene	1×10^{-6}[d]	7×10^{-8}[d]	2×10^{-7}[d]	3×10^{-8}[d]	7×10^{-6}[d]	4×10^{-7}[d]	8×10^{-9}[d]	4×10^{-8}[c]	4×10^{-8}[c]	4×10^{-8}[c]	4×10^{-8}[c]	2×10^{-7}[c]
Pentachlorophenol	2×10^{-6}[d]	4×10^{-8}[d]	4×10^{-7}[d]	2×10^{-8}[d]	2×10^{-5}[d]	3×10^{-7}	4×10^{-9}	1×10^{-8}[c]	4×10^{-8}	1×10^{-8}[c]	1×10^{-8}[c]	3×10^{-8}[c]
Total PCBs	1×10^{-3}	5×10^{-5}	2×10^{-4}	2×10^{-5}	9×10^{-3}	4×10^{-4}	7×10^{-6}	2×10^{-4}	6×10^{-6}	1×10^{-5}	4×10^{-4}	1×10^{-4}
PCB TEQ[e]	7×10^{-4}	4×10^{-5}	1×10^{-4}	2×10^{-5}	6×10^{-3}	3×10^{-4}	8×10^{-6}	1×10^{-4}	5×10^{-6}	1×10^{-5}	3×10^{-4}	9×10^{-5}
Total DDTs	1×10^{-6}	9×10^{-8}	2×10^{-7}	4×10^{-8}	1×10^{-5}	6×10^{-7}	1×10^{-8}	2×10^{-7}	2×10^{-8}	2×10^{-8}[c]	5×10^{-7}	2×10^{-7}
alpha-BHC	4×10^{-6}[d]	2×10^{-7}[d]	7×10^{-7}[d]	1×10^{-7}[d]	2×10^{-5}[d]	9×10^{-7}[d]	3×10^{-8}[d]	1×10^{-7}[c]	1×10^{-7}[c]	1×10^{-7}[c]	2×10^{-7}	1×10^{-7}[c]
beta-BHC	1×10^{-6}[d]	7×10^{-8}[d]	2×10^{-7}[d]	3×10^{-8}[d]	7×10^{-6}[d]	3×10^{-7}[d]	8×10^{-9}[d]	4×10^{-8}[c]	4×10^{-8}[c]	3×10^{-8}[c]	4×10^{-8}[c]	3×10^{-8}[c]
Dieldrin	8×10^{-6}[d]	5×10^{-7}[d]	1×10^{-6}[d]	2×10^{-7}[d]	5×10^{-5}[d]	2×10^{-6}[d]	7×10^{-8}[d]	2×10^{-7}	3×10^{-7}[c]	3×10^{-7}[c]	4×10^{-7}	5×10^{-7}
Total chlordane	2×10^{-6}	9×10^{-8}	3×10^{-7}	4×10^{-8}	1×10^{-5}	7×10^{-7}	1×10^{-8}	4×10^{-8}	8×10^{-8}	2×10^{-8}[c]	1×10^{-7}	5×10^{-8}
Heptachlor	1×10^{-6}[d]	7×10^{-8}[d]	2×10^{-7}[d]	3×10^{-8}[d]	7×10^{-6}[d]	3×10^{-7}[d]	1×10^{-8}[d]	4×10^{-8}[c]	4×10^{-8}[c]	4×10^{-8}[c]	5×10^{-8}[c]	4×10^{-8}[c]
Heptachlor epoxide	2×10^{-6}[d]	2×10^{-7}[d]	4×10^{-7}[d]	7×10^{-8}[d]	1×10^{-5}[d]	7×10^{-7}[d]	2×10^{-8}[d]	9×10^{-8}[c]	9×10^{-8}[c]	9×10^{-8}[c]	1×10^{-7}	9×10^{-8}[c]
Mirex	4×10^{-6}[d]	3×10^{-7}[d]	8×10^{-7}[d]	1×10^{-7}[d]	3×10^{-5}[d]	1×10^{-6}[d]	4×10^{-8}[d]	2×10^{-7}[c]	2×10^{-7}[c]	2×10^{-7}[c]	4×10^{-7}	2×10^{-7}[c]
Dioxin/furan TEQ[e]	1×10^{-4}	6×10^{-6}	2×10^{-5}	3×10^{-6}	7×10^{-4}	4×10^{-5}	1×10^{-6}	5×10^{-6}	3×10^{-6}	3×10^{-6}	2×10^{-5}	9×10^{-6}
Total TEQ excess cancer risk for dioxins/furans and coplanar PCBs	**8×10^{-4}**	**5×10^{-5}**	**1×10^{-4}**	**2×10^{-5}**	**7×10^{-3}**	**3×10^{-4}**	**9×10^{-6}**	**1×10^{-4}**	**8×10^{-6}**	**1×10^{-5}**	**3×10^{-4}**	**1×10^{-4}**
Total excess cancer risk (excluding PCB TEQ)[f]	**1×10^{-3}**	**7×10^{-5}**	**3×10^{-4}**	**3×10^{-5}**	**1×10^{-2}**	**5×10^{-4}**	**1×10^{-5}**	**2×10^{-4}**	**2×10^{-5}**	**2×10^{-5}**	**4×10^{-4}**	**1×10^{-4}**
Total excess cancer risk (excluding total PCBs)[f]	**1×10^{-3}**	**6×10^{-5}**	**2×10^{-4}**	**3×10^{-5}**	**9×10^{-3}**	**4×10^{-4}**	**1×10^{-5}**	**1×10^{-4}**	**2×10^{-5}**	**2×10^{-5}**	**3×10^{-4}**	**1×10^{-4}**

Note: Green-shaded rows have been updated based on the new toxicity values for BaP. All other parts of this table are unchanged from the final EW HHRA (Table B.5-47).

[a] The adult one-meal-per-month scenarios are presented for informational purposes only and are not used by EPA for risk management decisions.

[b] Arsenic EPCs and risk estimates are based on inorganic arsenic.

[c] There were no detected values of this COPC for this seafood category. Risk estimate is based on one-half the maximum RL.

[d] More than 50% of the risk associated with this COPC was derived from seafood categories (e.g., benthic fish, crab, or clams) with no detected values.

[e] No mussel data were available for this COPC. When the CDI and risk values were calculated, the portion of seafood consumption that had been assigned to mussels was divided proportionally among the remaining consumption categories.

[f] Total risk values include the risks associated with all COPCs.

API – Asian and Pacific Islander
BaP – benzo(a)pyrene
BHC – benzene hexachloride
CDI – chronic daily intake
COPC – contaminant of potential concern
cPAH – carcinogenic polycyclic aromatic hydrocarbon
CT – central tendency
DDT – dichlorodiphenyltrichloroethane
EPA – US Environmental Protection Agency
EPC – exposure point concentration
EW – East Waterway
HHRA – human health risk assessment
PCB – polychlorinated biphenyl
RL – reporting limit
RME – reasonable maximum exposure
TEQ – toxic equivalent

Table 5. Summary of estimated non-cancer hazards for the seafood consumption scenarios

COPC	Estimated Non-Cancer Hazard											
	Adult Tribal RME (Tulalip Data)	Adult Tribal CT (Tulalip Data)	Child Tribal RME (Tulalip Data)	Child Tribal CT (Tulalip Data)	Adult Tribal (Suquamish Data)	Adult API RME	Adult API CT	Adult One Meal per Month[a]				
								Benthic Fish	Clam	Crab	Pelagic Fish, Rockfish	Pelagic Fish, Perch
Arsenic[b]	0.4	0.05	0.9	0.1	4	0.4	0.03	0.002	0.08	0.01	0.004	0.009
Cadmium	0.7	0.08	2	0.2	2	0.4	0.03	0.01	0.01	0.09	0.004	0.004
Cobalt	0.6	0.07	1	0.2	4	0.5	0.04	0.01	0.07	0.05	0.02	0.02
cPAH TEQ	0.05	0.004	0.1	0.009	0.5	0.06	0.003	0.0001	0.009	0.0004	0.0001	0.0006
Mercury	0.6	0.07	1	0.2	3	0.4	0.04	0.05	0.02	0.09	0.2	0.04
TBT as ion	0.3	0.03	0.7	0.07	4	0.4	0.03	0.007	0.05	0.003	0.2	0.04
Total PCBs[c]	27	3	58	6	214	24	1	13	0.4	0.8	21	8
Total PCBs[d]	8	0.8	17	2	61	7	0.4	4	0.1	0.2	6	2
PCB TEQ	7	0.9	14	2	58	7	0.6	2	0.1	0.3	6	2
Dioxin/furan TEQ	1	0.1	2	0.3	7	0.9	0.07	0.1	0.06	0.07	0.4	0.2
HIs by endpoint[e]												
HI for hematological endpoint[f]	**0.3**	**0.05**	**0.8**	**0.1**	**2**	**0.2**	**0.02**	**0.01**	**0.02**	**0.04**	**0.03**	**0.02**
HI for immunological endpoint[g]	**27**	**3**	**59**	**6**	**218**	**24**	**1**	**13**	**0.5**	**0.8**	**21**	**8**
HI for kidney endpoint[h]	**0.8**	**0.1**	**2**	**0.2**	**3**	**0.5**	**0.04**	**0.02**	**0.02**	**0.1**	**0.01**	**0.01**
HI for liver endpoint[i]	**0.06**	**0.008**	**0.1**	**0.02**	**0.3**	**0.04**	**0.003**	**0.007**	**0.006**	**0.004**	**0.01**	**0.008**
HI for neurological endpoint[j]	**28**	**3**	**59**	**6**	**218**	**25**	**1**	**13**	**0.4**	**0.9**	**21**	**8**
HI for endocrine endpoint[k]	**0.6**	**0.08**	**1**	**0.2**	**4**	**0.5**	**0.04**	**0.01**	**0.08**	**0.05**	**0.02**	**0.02**
HI for integumentary endpoint[l]	**28**	**3**	**59**	**6**	**219**	**25**	**1**	**13**	**0.5**	**0.8**	**21**	**8**
HI for digestive system endpoint[m]	**0.5**	**0.06**	**1**	**0.1**	**2**	**0.3**	**0.03**	**0.005**	**0.04**	**0.04**	**0.02**	**0.02**
HI for developmental endpoint[n]	**10**	**1**	**20**	**3**	**72**	**8**	**0.7**	**4**	**0.2**	**0.5**	**7**	**2**

Note: With the exception of cPAH TEQ, only those COPCs with HQs greater than one for one or more scenario are included in this table. Green-shaded rows have been updated based on the new toxicity values for BaP. All other parts of this table are unchanged from the final EW HHRA (Table B.5-48).

[a] The adult one-meal-per-month scenarios are presented for informational purposes only and are not used by EPA for risk management decisions.

[b] Arsenic EPCs and risk estimates are based on inorganic arsenic.

[c] HQ was used for the calculation of the immunological, integumentary, and neurological endpoint HIs (Table B.4-1 of the EW HHRA).

[d] HQ was used for the calculation of the developmental endpoint HI (Table B.4-1 of the EW HHRA).

[e] Total risk values include the risks associated with all COPCs. However, only those COPCs with HQs greater than one for at least one scenario are listed in this table.

[f] Hematological endpoint includes the following COPCs: antimony, selenium, and zinc.

[g] Immunological endpoint includes the following COPCs: dibutyltin, total PCBs, and TBT.

[h] Kidney endpoint includes the following COPCs: cadmium, molybdenum, and pentachlorophenol.

[i] Liver endpoint includes the following COPCs: 1,4-dichlorobenzene, alpha-BHC, total chlordane, total DDTs, dieldrin, heptachlor, heptachlor epoxide, mirex, and pentachlorophenol.

[j] Neurological endpoint includes the following COPCs: mercury, total PCBs, and selenium. Neurological effects associated with exposure to lead are discussed in Section B.5.4 of the EW HHRA.

[k] Endocrine endpoint includes the following COPCs: antimony and cobalt.

[l] Integumentary endpoint includes the following COPCs: arsenic, total PCBs, selenium, and vanadium.

[m] Digestive system endpoint includes the following COPCs: chromium and copper.

[n] Developmental endpoint includes the following COPCs: cPAH TEQ, mercury, PCBs (the higher of either the total PCB HQ based on the developmental endpoint or the PCB TEQ HQ), and dioxin/furan TEQ.

API – Asian and Pacific Islanders
BaP – benzo(a)pyrene
BHC – benzene hexachloride
COPC – contaminant of potential concern
cPAH – carcinogenic polycyclic aromatic hydrocarbon
CT – central tendency
DDT – dichlorodiphenyltrichloroethane
EPA – US Environmental Protection Agency
EPC – exposure point concentration
EW – East Waterway
HHRA – human health risk assessment
HI – hazard index
HQ – hazard quotient
PCB – polychlorinated biphenyl
RME – reasonable maximum exposure
TBT – tributyltin
TEQ – toxic equivalent

Table 6. Summary of estimated excess cancer risks for direct sediment exposure scenarios

COPC	Estimated Excess Cancer Risk					
	Netfishing		Habitat Restoration Worker	Clamming		
	RME	CT		Tribal – 183 Days per Year	Tribal RME	7 Days per Year
Arsenic	3×10^{-6}	7×10^{-7}	5×10^{-7}	2×10^{-5}	1×10^{-5}	4×10^{-7}
cPAH TEQ	3×10^{-7}	2×10^{-8}	1×10^{-7}	3×10^{-6}	2×10^{-6}	8×10^{-8}
Total PCBs	6×10^{-7}	6×10^{-8}	2×10^{-7}	6×10^{-6}	3×10^{-6}	1×10^{-7}
PCB TEQ	3×10^{-7}	4×10^{-8}	5×10^{-8}	2×10^{-6}	1×10^{-6}	3×10^{-8}
Dioxin/furan TEQ	6×10^{-7}	1×10^{-7}	na	2×10^{-6}	1×10^{-6}	4×10^{-8}
Total TEQ excess cancer risk for dioxins/furans and coplanar PCBs	**9×10^{-7}**	**1×10^{-7}**	**na**	**4×10^{-6}**	**2×10^{-6}**	**7×10^{-8}**
Total excess cancer risk (excluding PCB TEQ)[a]	**5×10^{-6}**	**9×10^{-7}**	**8×10^{-7}**	**3×10^{-5}**	**2×10^{-5}**	**6×10^{-7}**
Total excess cancer risk (excluding total PCBs)[a]	**4×10^{-6}**	**9×10^{-7}**	**7×10^{-7}**	**3×10^{-5}**	**1×10^{-5}**	**6×10^{-7}**

Note: Green-shaded rows have been updated based on the new toxicity values for BaP. All other parts of this table are unchanged from the final EW HHRA (Table B.5-49)..

[a] Total risk values include the risks associated with all COPCs. However, only those COPCs with excess cancer risks greater than 1×10^{-6} for at least one scenario are listed in this table.

BaP – benzo(a)pyrene
COPC – contaminant of potential concern
cPAH – carcinogenic polycyclic aromatic hydrocarbon
CT – central tendency
EW – East Waterway
HHRA – human health risk assessment
na – not applicable (not a COPC)
PCB – polychlorinated biphenyl
RME – reasonable maximum exposure
TEQ – toxic equivalent

In general, the total excess cancer risks and non-cancer HIs for the seafood consumption scenarios (Tables 4 and 5) did not change significantly from the risks calculated in the EW HHRA, because cPAH TEQ contributes a small percent of the total seafood consumption risks as compared with total PCBs and arsenic. However, cPAH TEQ was a larger contributor to the total risks associated with the direct sediment exposure scenarios (Table 6); therefore, there were larger changes in the total risks for these scenarios.

The updated risk estimates presented in Tables 4 through 6 were used to determine whether cPAH TEQ is a COC for each of the reasonable maximum exposure (RME) scenarios used in the HHRA. Table 7 presents a summary of the updated cPAH TEQ excess cancer risks for the five RME scenarios, as well as a determination of whether cPAH TEQ is a COC for each of those scenarios. COPCs were identified as a COC for a scenario if the associated estimated risk was greater than the risk threshold (i.e., excess cancer risk was greater than 1×10^{-6} or the HQ was greater than 1).

Table 7. Summary of RME scenarios for which cPAH TEQ is a COC

Scenario	cPAH TEQ Excess Cancer Risk	cPAH TEQ HQ	Identified as a COC?
Seafood consumption scenarios			
Adult tribal RME (Tulalip data)	**1×10^{-5}**	0.05	yes
Child tribal RME (Tulalip data)	**1×10^{-5}**	0.1	yes
Adult API RME	**7×10^{-6}**	0.06	yes
Direct contact scenarios			
Netfishing RME	3×10^{-7}	0.002	no
Tribal clamming RME	**2×10^{-6}**	0.008	yes

Note: **Bold text** indicates that risk estimate is above applicable threshold.

API – Asian and Pacific Islanders
COC – contaminant of concern
cPAH – carcinogenic polycyclic aromatic hydrocarbon
HQ – hazard quotient
RME – reasonable maximum exposure
TEQ – toxic equivalent

In the EW HHRA, cPAH TEQ was identified as a COC for all five of the RME scenarios listed in Table 7. Based on the updated excess cancer risks, cPAH TEQ is still a COC for the three seafood consumption RME scenarios and the tribal clamming RME scenario. However, cPAH TEQ is no longer a COC for the netfishing RME scenario because the excess cancer risk for cPAH TEQ is less than 1×10^{-6}. The non-cancer HQs for cPAH TEQ are significantly less than the risk threshold (i.e., HQ of one) for all scenarios, so these HQs did not factor into the COC determination.

4 Identification of Risk Drivers

This section presents an updated discussion of the identification of cPAH TEQ as a risk driver (EPA 1999) based on estimated human health risks in the EW. As described in the EW HHRA, the following criteria were used to identify risk drivers:

- Relative percentage that the COC contributes to the total human health risk
- Absolute magnitude of the risk associated with the COC (including a consideration of background concentrations, if applicable)
- Frequency of detection of the COC
- Level of uncertainty in the risk estimate

Based on these criteria, Table 8 presents the determination of whether cPAH TEQ is a risk driver for each of the five RME scenarios. Based on the magnitude of the excess cancer risks and the greater toxicity of cPAH TEQ to children because of the mutagenic modes of action for such chemicals, cPAH TEQ was identified as a risk driver for the seafood consumption RME scenarios. Because cPAH TEQ risks were only slightly greater than the risk threshold in the tribal clamming RME scenario, cPAH TEQ was not identified as a risk driver for this scenario.

Table 8. Selection of cPAH TEQ as a risk driver by scenario

RME Scenario	cPAH TEQ Excess Cancer Risk (% of Total)	Risk Driver?	Rationale
Seafood consumption scenarios			
Adult tribal RME (Tulalip data)	1×10^{-5} (1%)	yes	risks within EPA's acceptable risk range (up to 1×10^{-5}) and high detection frequency across tissue types (71%)
Child tribal RME (Tulalip data)	1×10^{-5} (5%)		
Adult API RME	7×10^{-6} (1%)		
Direct contact scenarios			
Netfishing RME	3×10^{-7} (7%)	na	not a COC
Tribal clamming RME	2×10^{-6} (13%)	no	risks only slightly greater than the 1×10^{-6} threshold

Note: All cPAH TEQ HQs calculated for the RME scenarios were less than the risk threshold (i.e., 1).

API – Asian and Pacific Islanders
COC – contaminant of concern
cPAH – carcinogenic polycyclic aromatic hydrocarbon
EPA – US Environmental Protection Agency
HQ – hazard quotient
na – not applicable
RME – reasonable maximum exposure
TEQ – toxic equivalent

5 Conclusions

The following summarizes the key conclusions based on the updated cPAH TEQ risk calculations as presented in this addendum:

- **Non-cancer HQs –** Based on the new RfD for BaP, HQs were calculated for cPAH TEQ (no HQs were calculated in the final EW HHRA). All HQs were less than the risk threshold of one.
- **Excess cancer risks –** Excess cancer risks from cPAH TEQ were calculated using the updated slope factor for BaP. Based on the updated cancer risks (which decreased by approximately a factor of 7.3), cPAH TEQ remains a COC for the three seafood consumption RME scenarios and the tribal clamming RME scenario. cPAH TEQ is no longer a COC for the netfishing RME scenario.
- **Risk driver determinatio**n –
  - **Seafood consumption scenarios –** The cPAH TEQ excess cancer risks represent a small percentage (1 to 5%) of the total excess cancer risks associated with the seafood consumption RME scenarios. However, cPAH TEQ remains a risk driver for the seafood consumption RME scenarios based on the magnitude of risk posed by this contaminant.
  - **Direct sediment exposure scenarios –** cPAH TEQ is no longer a risk driver for the any of the direct sediment exposure scenarios. This is because it is not a COC for the netfishing RME scenario, and the excess cancer risks for the tribal clamming RME scenario were only slightly greater than the COC risk threshold.

6 References

EPA. 1999. A guide to preparing Superfund proposed plans, records of decision, and other remedy selection decision documents. EPA 540-R-98-031; OSWER 9200.1-23P; PB98-963241. Office of Solid Waste and Emergency Response, US Environmental Protection Agency, Washington, DC.

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ATTACHMENT 1. UPDATED cPAH TEQ TOXICOLOGICAL PROFILE

Updated cPAH TEQ Toxicological Profile

Polycyclic aromatic hydrocarbons (PAHs) are a group of organic chemicals that have a fused ring structure of two or more benzene rings that are formed during the incomplete combustion of organic materials. Industrial activities that produce PAHs include: coal coking; production of carbon blacks, creosote, and coal tar; petroleum refining; synfuel production from coal; and the use of Soderberg electrodes in aluminum smelters and ferrosilicum in iron works (EPA 2000). Domestic activities that produce PAHs include: cigarette smoking, burning wood and fossil fuels, waste incineration, broiling and smoking foods, and using combustion engines. Benzo(a)pyrene is the PAH with the most available health effects data.

PHARMACOKINETICS

PAHs can be absorbed through the lungs, the stomach, or the skin. Oral absorption is greater with more lipophilic PAHs or when oil is present in the gastrointestinal tract. Upon inhalation or oral/dermal exposure among animals, the greatest levels of PAHs were found in highly perfused tissues, such as the lung, liver, gastrointestinal tract, and kidneys. It has been demonstrated that PAHs metabolize to reactive intermediates by enzyme systems, which then covalently bind to cellular macromolecules, leading to mutation and tumor development (EPA 2000).

ACUTE TOXICITY

There is relatively little data describing the acute toxicity of PAHs after inhalation or oral or dermal exposure among humans or animals. However, benzo(a)pyrene is fatal to mice following ingestion, and the liver and skin have been identified as target organs in animals after oral or dermal exposure, respectively (ATSDR 1995). The intraperitoneal LD50[1] values (injected dose that kills half of the animals being tested) in mice for pyrene, anthracene, and benzo(a)pyrene are 514, > 430, and 232 mg/kg, respectively.

CHRONIC TOXICITY

PAHs have a high chronic exposure toxicity characterized by chronic dermatitis[2] and hyperkeratosis (ATSDR 1995). Chronic studies of animals exposed to PAHs via ingestion, intratracheal installation, or skin-painting have not yet identified any adverse health effects other than cancer.

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[1] LD50 is the lethal dose at 50% (i.e., the amount of a chemical or other toxic substance that is sufficient to kill 50% of a population of test animals).

[2] Contact dermatitis is an inflammation of the skin caused by direct contact with an irritating or allergy-causing substance.

The US Environmental Protection Agency (EPA) has derived a **reference dose (RfD) of 0.0003 mg/kg-day** for benzo(a)pyrene[3] based on effects on the developmental system (neurobehavioral changes). This RfD is used to evaluate non-cancer hazards caused by cPAHs in this baseline human health risk assessment (HHRA).

CARCINOGENICITY

Occupational studies of workers exposed to mixtures containing PAHs have shown that such mixtures are carcinogenic to humans. Cancer associated with exposure to PAH-containing mixtures among humans occurs mainly in the lungs and skin following inhalation and dermal exposure.

EPA and California EPA guidance describe the cancer-causing ability of individual cPAHs relative to the cancer-causing ability of a reference compound, benzo(a)pyrene (EPA 1993; California EPA 1994). This approach is described in greater detail in Sections B.2.2.4 and B.4.2 of the baseline HHRA. **The oral cancer slope factor developed by EPA for the carcinogenicity of benzo(a)pyrene is 1 per mg/kg-day** (IRIS; updated January 17, 2017). EPA has classified benzo(a)pyrene as being "carcinogenic to humans" based on "strong and consistent evidence in animals and humans. The evidence includes an extensive number of studies demonstrating carcinogenicity in multiple animal species exposed via all routes of administration and increased cancer risks, particularly in the lung and skin, in humans exposed to different PAH mixtures containing benzo[a]pyrene" (IRIS). The oral cancer potency factor was applied to the sum of cPAHs, using the TEFs described in Section B.2.2.4 of the baseline HHRA.

[3] Source: Integrated Risk Information System (IRIS).

References

ATSDR. 1995. Toxicological profile for polycyclic aromatic hydrocarbons (PAHs). Agency for Toxic Substances and Disease Registry, Atlanta, GA.

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EPA. 1993. Provisional guidance for quantitative risk assessment of polycyclic aromatic hydrocarbons. EPA-600/R-93/089. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, US Environmental Protection Agency, Cincinnati, OH.

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