

**BEFORE THE UNITED STATES
ENVIRONMENTAL PROTECTION AGENCY**

Petition for Rulemaking Under the)
Clean Water Act)
)
Toxic Pollutant List Designated Under)
Section 307(a)(1) in 40 C.F.R. § 401.15;)
Priority Pollutant List Published at)
40 C.F.R. Part 423, Appendix A;)
Pretreatment Determinations Pursuant to)
Section 307(b)(1).)

I. INTRODUCTION

For the reasons detailed below, Northwest Environmental Advocates (“NWEA”) and the Center for Biological Diversity (“Center”) hereby petition the U.S. Environmental Protection Agency (“EPA”) to update two lists of toxic pollutants that are key to Clean Water Act (“CWA”) regulatory programs intended to protect human health, aquatic life, and aquatic-dependent wildlife from toxic contaminants, and to take other related actions necessary to performing its statutory duties. EPA’s failure to update these two lists of toxic contaminants—to which no pollutants have been added for 47 years—cripples effective implementation of the Clean Water Act by undermining both the technology-based and the water quality-based approaches to toxics pollution control established by the law and carried out by the states and EPA. EPA’s failure to update these lists of toxic pollutants significantly impairs EPA and states’ regulation of toxic pollutants in the nation’s waters, causing human mortality, morbidity, and suffering; environmental injustice; and adverse impacts to aquatic and aquatic-dependent life, including threatened, endangered, candidate, and other species.

This petition seeks to ensure that EPA takes actions necessary to protect U.S. waters from the effects of toxic contaminants—as required by the Clean Water Act—by updating the Toxic

Pollutants and Priority Pollutant lists,¹ an action that perforce will result in EPA's strengthening both the technology-based and water quality-based controls on the discharge of those newly-listed pollutants.

As this petition demonstrates, EPA has failed to keep all aspects of its CWA regulatory program current, given identification of additional toxic pollutants; advancements in scientific understanding of how toxic pollutants affect human health and the environment; and advancements in the treatment technology available to ensure that discharges of toxic pollutants are reduced, and eventually eliminated, from the nation's waters. In the CWA, particularly for toxics, Congress repeatedly exhorted EPA and the states to move swiftly to improve and carry out these regulatory programs. Instead, the program to control toxic contaminants in the nation's waters has become obsolete, languishing for decades and in many instances without any improvements. Adding chemicals to the Toxic Pollutants Lists is an essential first step to enable EPA to meet its statutory duties to update and adopt new technology- and water quality-based requirements to control the discharge of these pollutants. It is EPA's responsibility to systematically evaluate all relevant monitoring data and scientific information to determine which new pollutants to list. This Petition only includes examples that suggest the large number of pollutants that must be listed or further evaluated for listing.

The Toxic Pollutants Lists are key to protecting human health, responding to environmental injustice, and protecting threatened and endangered species as the following examples illustrate:

- EPA's recent surge of efforts to regulate PFAS "forever" chemicals—"an urgent public

¹ Throughout the Petition, we will refer to the lists together as the "Toxic Pollutants Lists." Where we are only referring to one list or the other, we will specifically indicate as such.

health and environmental issue facing communities across the United States”²—spotlight the importance of the Toxic Pollutants Lists. EPA will not be able to complete its proposed regulatory actions during the 2021–2024 Biden-Harris Administration. Yet, by not including PFAS chemicals on the Toxic Pollutants Lists, EPA jeopardizes much of its work. As this petition explains, pursuant to the CWA, only such listed toxic pollutants are subject to mandatory actions by EPA and the states. Therefore, placing PFAS on the Toxic Pollutants Lists will trigger future regulatory action.

- Likewise, EPA cannot meet its environmental justice goals of protecting public health for all people who drink water and consume fish from the nation’s waters without bringing the Toxic Pollutants Lists into the twenty-first century.³ The agency’s focus on toxic PFAS pollutants, highlighted in its 2022 Equity Action Plan,⁴ is a good start but pales in the context of how many more toxic pollutants require the regulation that is triggered by placement on the Toxic Pollutants Lists.
- EPA’s inactions also jeopardize threatened and endangered species. For example, the 74-member population of the endangered Southern Resident killer whales⁵ is known as among the most toxic contaminated marine mammals in the world. A 2023 study not only found the chemical 4-nonylphenol⁶ predominated the orcas’ toxic burden but identified it as having the highest transfer rates from mothers to fetuses, as high as 95 percent.⁷ However, while EPA’s concern over nonylphenol production drove its 2005 publication of recommended water quality criteria for this chemical,⁸ by not adding it to the Toxic Pollutants Lists, EPA

² EPA, *PFAS Strategic Roadmap: EPA’s Commitments to Action 2021–2024* (Oct. 2021) at 2, available at https://www.epa.gov/system/files/documents/2021-10/pfas-roadmap_final-508.pdf (hereinafter “EPA PFAS Roadmap”).

³ See EPA, *EJ 2020 Action Agenda, the U.S. EPA’s Environmental Justice Strategic Plan for 2016-2020* (undated), available at https://www.epa.gov/sites/default/files/2016-05/documents/052216_ej_2020_strategic_plan_final_0.pdf (peculiarly, the word “fish” does not appear in this document and the word “toxic” appears only four times). The word “toxic” appears in three footnotes in EPA’s more recent action plan: EPA, *E.O. 13985 Equity Action Plan: U.S. Environmental Protection Agency* (April 2022), available at https://www.epa.gov/system/files/documents/2022-04/epa_equityactionplan_april2022_508.pdf.

⁴ *Id.* at 26.

⁵ Orca Network, *Southern Resident Orca Community Demographics, Composition of Pods, Births and Deaths since 1998* (updated June 30, 2023), available at <https://www.orcanetwork.org/births-and-deaths> (“As of June 30, 2023, the Southern Resident Killer Whale (Orca) population was comprised of 75 individuals (74 including *Lolita/Tokitae*, the *L pod orca* housed at the Miami Seaquarium).”).

⁶ See Kiah Lee, et al., *Emerging Contaminants and New POPs (PFAS and HBCDD) in Endangered Southern Resident and Bigg’s (Transient) Killer Whales (Orcinus orca): In Utero Maternal Transfer and Pollution Management Implications*, 57 *Environ. Sci. Technol.* 360-374 (2023) (hereinafter “CECs and Killer Whales”) at 360. The PFAS—7:3-fluorotelomer carboxylic acid (“7:3 FTCA”)—is not one EPA is currently seeking to regulate.

⁷ *Id.*

⁸ EPA, *Aquatic Life Ambient Water Quality Criteria – Nonylphenol* (Dec. 2005) (hereinafter “2005 Nonylphenol Criteria”) at 1. Note that while EPA’s recommended criteria publication states it only applies to CAS nos. 84852-15-3 and 25154-52-3, thereby only including *branched* 4-nonylphenol and

failed to make state adoption of nonylphenol criteria mandatory.

This petition is brought pursuant to the Administrative Procedure Act, 5 U.S.C. §§ 553(e) and 555(e), to request EPA take the following actions: (1) pursuant to CWA Section 307(a)(1) add the toxic pollutants identified herein to the Toxic Pollutant List, codified at 40 C.F.R. § 401.15, and the Priority Pollutant List, codified at 40 C.F.R., Part 423, Appendix A; (2) establish by rule a method by which EPA will propose changes to and accept public input on the Toxic Pollutant List and Priority Pollutant List every three years pursuant to its authority under CWA Section 307(a)(1); (3) establish by rule a commitment to revise the lists upon completion of such a triennial review; (4) establish by rule that EPA will make determinations pursuant to CWA Section 307(b)(1) to identify newly-listed toxic pollutants that are not susceptible to treatment by publicly owned treatment works and are therefore likely to pass through such facilities, or to interfere with the operation of such treatment works; and (5) for the pollutants identified in this petition, make determinations pursuant to CWA Section 307(b)(1) to identify those pollutants not susceptible to treatment by publicly owned treatment works and are therefore likely to pass through such facilities, or to interfere with the operation of such treatment works.

EPA has a heightened responsibility to remedy the deficiencies in its regulatory program for control of toxic contamination in the nation's waters because they are so long-standing. As the Congressional Research Service ("CRS") observed in 1993—29 years ago—"[m]any of EPA's criteria documents are outdated and need to be revised, and some do not include data needed to set standards for coastal waters and lakes. Further, there are many toxic pollutants for

omitting linear 4-nonylphenol (CAS No. 104-40-5), it concluded the studies it relied on were likely for branched because much nonylphenol is mislabeled. Correspondence with EPA on file with author.

which no criteria have been developed.”⁹

But no better proof of EPA’s failure to act is needed beyond the agency’s own words: “Portions of both lists are outdated.”¹⁰ In the absence of EPA action on its own acknowledged failure to maintain updated Toxic Pollutants Lists, it must grant this petition.

⁹ CRS, Claudia Copeland, *Toxic Pollutants and the Clean Water Act: Current Issues* (Sept. 21, 1993).

¹⁰ EPA, Effluent Guidelines, *Toxic and Priority Pollutants Under the Clean Water Act*, <https://www.epa.gov/eg/toxic-and-priority-pollutants-under-clean-water-act> (hereinafter “EPA Toxic Lists Website”).

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II. JURISDICTION, AUTHORITY, AND STATUTORY DUTIES OF THE ENVIRONMENTAL PROTECTION AGENCY

The stated objective of the 1972 Clean Water Act “is to restore and maintain the chemical, physical, and biological integrity of the Nation’s waters.”¹¹ As one of the main ways to achieve this objective, the Act establishes a “national goal that the discharge of pollutants into the navigable waters be eliminated by 1985,”¹² a goal that is carried out in part through Section 402, which establishes a program of National Pollutant Discharge Elimination System (“NPDES”) permits for pollution sources that discharge to the nation’s surface waters. Consistent with the Act’s stated objective, the Act further establishes that “it is the national policy that the discharge of toxic pollutants¹³ in toxic amounts be prohibited.”¹⁴ To attain these aims, the Act requires all NPDES permits (or pretreatment permits for indirect industrial discharges into public sewage treatment plants) to incorporate technology-based effluent limitations for all point source discharges of toxic and other pollutants.¹⁵ Those limits must require the elimination of pollutant discharges wherever feasible, and where not feasible, minimization of discharges based on various statutory tests.¹⁶ EPA is required to adopt effluent

¹¹ CWA § 101(a).

¹² CWA § 101(a)(1).

¹³ The term “toxic pollutant” is defined in CWA § 502(13) to mean “those pollutants, or combinations of pollutants, including disease-causing agents, which after discharge and upon exposure, ingestion, inhalation or assimilation into any organism, either directly from the environment or indirectly by ingestion through food chains, will, on the basis of information available to the Administrator, cause death, disease, behavioral abnormalities, cancer, genetic mutations, physiological malfunctions (including malfunctions in reproduction) or physical deformations, in such organisms or their offspring.”)

¹⁴ CWA § 101(a)(3).

¹⁵ Throughout the Petition, we will refer to national effluent guidelines (“ELG”) and new source performance standards (“NSPS”) together as “ELGs.” Where we are only referring to one requirement or the other, we will specifically indicate as such. Likewise we will refer to pretreatment standards for existing sources (“PSES”) and pretreatment Standards for new sources (“PSNS”) together as “pretreatment standards.”

¹⁶ CWA §§ 302(a); 307(a)(2), (3), (4); 307(b), (c).

limitations guidelines governing those discharges and permits.¹⁷

However, prior to the elimination of discharges, the Act calls for an “interim goal of water quality which provides for the protection and propagation of fish, shellfish, and wildlife and provides for recreation in an on the water be achieved by July 1, 1983.”¹⁸ The water quality-based framework to carry out this interim goal is found in the Act’s Sections 301, 302, 303, and 304. Section 304 requires EPA to adopt water quality criteria guidance for toxic and other pollutants. Section 303 requires states or EPA to establish water quality standards, identify waters that fail to meet those standards, and the develop and implement clean-up plans for those identified waters. Section 301 requires NPDES permits to include water quality-based effluent limitations wherever technology-based limits will not result in attainment of water quality standards.

This section explains the EPA’s authority to undertake the rulemaking we are requesting in this petition as well as its importance. Sub-section A highlights the key role of the Toxic Pollutants Lists in controlling toxic pollution in the nation’s waters. Sub-section B discusses how these two lists of toxic pollutants guide EPA’s development of the key implementation method to the technology-based approach. Sub-section C explains why it is critical that states have clear, updated water quality standards to carry out the CWA’s water quality-based approach to regulating toxic pollution. Sub-section D presents an overview of the 1987 amendments to the CWA, which pointedly required improved EPA and state regulatory actions to control toxic pollution. Sub-section E focuses specifically on the important role that narrative criteria play to protect against toxic effects on designated uses from cumulative and synergistic effects of toxic

¹⁷ *Id.*

¹⁸ CWA § 101(a)(2).

pollutants, or other toxicity effects that cannot be controlled through individual numeric criteria. Sub-section F focuses on the statutory requirement to protect the designated use of aquatic dependent wildlife—birds, mammals, amphibians. Finally, sub-section G highlights the requirement for sediment criteria to protect designated uses from the effects of toxic contaminants.

A. The Clean Water Act Requires EPA’s Identification of Toxic Pollutants That Triggers Technology-Based and Water Quality-Based Regulation

CWA Section 307(a) codified a statutory list of toxic pollutants and authorized EPA to revise that list by regulation.¹⁹ EPA may “[f]rom time to time” revise the list of toxic pollutants or combination of pollutants, and in doing so “shall take into account the toxicity of the pollutant, its persistence, degradability, the usual or potential presence of the affected organisms in any waters, the importance of the affected organisms and the nature and extent of the effect of the toxic pollutant on such organisms.”²⁰ The Toxic Pollutant List consists of both individual pollutants and broad categories or families of pollutants. For this reason, EPA subsequently developed the Priority Pollutant List of individual pollutants in 1977 “to make implementation more practical for water testing and regulatory purposes.”²¹

The initial list was intended to be used by EPA and states as a *starting point* to ensure that NPDES permits addressed toxic pollution in the nation’s waters through technology-based effluent limitations (“TBELs”) and water quality-based effluent limitations (“WQBELs”). In the ensuing 47 years, however, EPA has not revised the lists to keep pace with a massive number of new or newly discovered toxic pollutants and families of pollutants. This impairs or, in many

¹⁹ Pub. L. 95-217 (Dec. 27, 1977).

²⁰ CWA § 307(a)(1).

²¹ EPA Toxic Lists Website, *see supra* n. 10.

cases, makes it impossible for EPA and states to fulfill their statutory duties to control toxic water pollution through the technology-based and water-quality based programs, and to implement statutory programs to control nonpoint sources.

1. History of the Toxic Pollutant List

The Toxic Pollutant List was negotiated among parties to a settlement agreement in *Natural Resource Defense Council v. Train*.²² This list of 65 chemicals and chemical groups was identified as Appendix A to the settlement decree. The lawsuit challenged the EPA Administrator’s September 7, 1973 action in publishing an initial list of a mere nine (9) toxic pollutants—aldrin/dieldrin, benzidine, cadmium, cyanide, DDT (DDE, DDD), endrin, mercury, polychlorinated biphenyls (“PCBs”), and toxaphene—as the Toxic Pollutant List and his December 27, 1973 action of proposing, but not finalizing, recommended CWA Section 304(a) recommended water quality criteria for these pollutants. Under the terms of the 1976 *Train* settlement, EPA agreed to “propose standards pursuant to § 307(a) of the Act for aldrin/dieldrin, DDT (DDD, DDE), endrin, and toxaphene on or before May 31, 1976; and for benzidine on or before June 22, 1976; and for polychlorinated biphenyls (PCB’s) on or before July 14, 1976” and to finalize each set within six months.²³ In addition,

[n]ot later than June 30, 1978, after opportunity for public comment, the Administrator shall publish under § 304(a) of the Act water quality criteria accurately reflecting the latest scientific knowledge on the kind and extent of all identifiable effects on aquatic organisms and human health of each of the pollutants listed in Appendix A. Such water quality criteria shall state, inter alia, for each of the pollutants listed in Appendix A, the recommended maximum permissible concentrations (including where appropriate zero) consistent with the

²² 6 ELR 20588, Civ. A. No. 2153-73 (D.D.C. June 9, 1976) (hereinafter “*NRDC v. Train* Settlement”).

²³ *Id.* at ¶ 14.

protection of aquatic organisms, human health, and recreational activities.^{24, 25}

Congress subsequently ratified the 1976 *Train Settlement Agreement* and its Toxic Pollutant List in Appendix A when it amended the CWA in 1977.²⁶ The list was then published in the Federal Register.²⁷ In 1979, EPA published the list again as a regulation.²⁸

2. History of the Priority Pollutant List

The Priority Pollutant List is a list of 126 individual pollutants that mirrors the chemicals and chemical groups on the Toxic Pollutant List.²⁹ EPA describes its creation of the Priority Pollutant List as follows:

Key features of the Priority Pollutant List and its relationship to the Toxic Pollutant List:

The Priority Pollutants are a set of chemical pollutants EPA regulates, and for which EPA has published analytical test methods.

The Priority Pollutant List makes the list of toxic pollutants more usable, in a practical way, for the purposes assigned to EPA by the Clean Water Act. For example, the Priority Pollutant list is more practical for testing and for regulation in that chemicals are described by their individual chemical names. The list of

²⁴ *Id.* at ¶ 11.

²⁵ A further part of the agreement required the following: “In addition to those pollutants to which regulations must be established pursuant to subsection (a) of this paragraph 4, the Administrator shall also identify the categories or category of point sources which are discharging into navigable waters or introducing into treatment works (as defined in § 212 of the Act) which are publicly owned the pollutants listed in Appendix C to this Agreement.” The toxic pollutants in Appendix C include the following: Acetone, n-alkanes (C[10]-C[30]), Biphenyl, Chlorine, Dialkyl ethers, Dibenzofuran, Diphenyl ether, Methylene Ketone, Nitrites, Secondary amines, Styrene, and Terpenes.

²⁶ Pub. L. 95-217, December 27, 1977.

²⁷ 43 Fed. Reg. 4108 (Jan. 31, 1978).

²⁸ 40 C.F.R. § 401.15; 44 Fed. Reg. 44501 (July 31, 1979).

²⁹ Originally 129 pollutants, EPA has taken only one action, in 1981, to change the Priority Pollutant List, an action in which it removed three pollutants after determining that their chemical properties did not justify their inclusion. Dichlorodifluoromethane and trichlorofluoromethane were de-listed at the request of E.I. duPont de Nemours and Co. because of low solubility in water and high volatility combined with low human and mammalian toxicity. 46 Fed. Reg. 2266 (Jan. 8, 1981). Bis(chloromethyl) ether was de-listed based on data that indicated a half-life in water of 38 seconds at 20°C. 46 Fed. Reg. 10723 (Feb. 4, 1981). De-listing these three pollutants did not change the 65 entries on the Toxics Pollutant List because they were specific compounds within entries for the groups of pollutants, Halomethanes (list entry 38) and Haloethers (list entry 37).

toxic pollutants, in contrast, contains open-ended groups of pollutants, such as “chlorinated benzenes.” That group contains hundreds of compounds; there is no test for the group as a whole, nor is it practical to regulate or test for all of these compounds.³⁰

According to EPA, the list was derived from the Toxic Pollutant List by the following method:

Starting with the list of toxic pollutants, EPA used four criteria to select and prioritize specific pollutants:

- We included all pollutants specifically named on the list of toxic pollutants;
- There had to be a chemical standard available for the pollutant, so that testing for the pollutant could be performed;
- The pollutant had to have been reported as found in water with a frequency of occurrence of at least 2.5 percent, and
- The pollutant had to have been produced in significant quantities, as reported in Stanford Research Institute’s “1976 Directory of Chemical Producers, USA.”³¹

B. National Effluent Limitations Guidelines and Pretreatment Standards Implement the CWA’s Technology-Based Approach to Controlling the Discharge of Toxic Pollution

In the CWA, Congress instructed EPA to require point source dischargers to reduce and eliminate releases of all pollutants on the Toxic Pollutants Lists through the establishment of national effluent limitation guidelines (“ELG”) applied to individual dischargers through NPDES and pretreatment permits. The statutory goal is the elimination of all water pollution.³² To achieve this goal, the CWA prohibits the “discharge of any pollutant” from a “point source”—defined as “any discernible, confined and discrete conveyance”—to navigable waters “except in compliance with law.”³³ The primary way to achieve compliance with the CWA’s pollutant

³⁰ EPA Toxic Lists Website, *supra* n. 10.

³¹ *Id.*

³² *Waterkeeper Alliance, Inc. v. U.S. Env’tl. Prot. Agency*, 399 F.3d 486, 490 (2d Cir. 2005), citing 33 U.S.C. § 1251(a)(1), CWA § 101(a)(1).

³³ CWA §§ 301, 502(14) (definition of “point source”).

discharge prohibition is to obtain and comply with an NPDES permit.³⁴ Every NPDES permit must establish “effluent limitations” for the pollutants being discharged.³⁵ These effluent limitations are first based on technology-based effluent limitations (“TBELs”) and then, if necessary, additional water quality-based effluent limitations (“WQBELs”).³⁶ Both technology-based and water quality-based limitations are intended to be “technology forcing.”

For this reason, TBELs are based on “a series of increasingly stringent technology-based standards,” depending on the type of pollutant being discharged.³⁷ Most of the technology-based standards used in NPDES permits are based on national ELGs, which are national wastewater discharge standards developed by EPA on an industry-by-industry basis. In establishing these national effluent limitations guidelines, EPA describes its role as to “identif[y] the best available technology that is economically achievable for that industry and set[] regulatory requirements based on the performance of that technology.”³⁸ When ELGs are not available, EPA instructs permit writers to use best professional judgment (“BPJ”) in determining the TBELs on a case-by-

³⁴ CWA §§ 301(a), 402.

³⁵ *Waterkeeper Alliance*, 399 F.3d at 491 (citing *S. Fla. Water Mgmt. Dist. v. Miccosukee Tribe of Indians*, 541 U.S. 95, 102 (2004)).

³⁶ CWA § 301(b).

³⁷ *NRDC v. U.S. E.P.A.*, 822 F.2d 104, 123–24 (D.C. Cir. 1987); see also *Entergy Corp. v. Riverkeeper, Inc.*, 556 U.S. 208, 219–21 (2009). These technology-based standards are designed to be “technology-forcing.” See *NRDC v. U.S. E.P.A.*, 822 F.2d at 123 (“[T]he most salient characteristic of this statutory scheme, articulated time and again by its architects and embedded in the statutory language, is that it is technology-forcing.”). In 1987, the D.C. Circuit emphasized that the CWA seeks “not only to stimulate but to press development of new, more efficient and effective technologies,” which is the “essential purpose of this series of progressively more demanding technology-based standards.” *Id.* at 124. Underscoring this point, in 2009, the U.S. Supreme Court explained that “Congress wished to mandate the greatest feasible reduction in water pollution” with the BAT standard because the “plain language” of the CWA “requires the EPA to set ‘effluent limitations [which] shall require the elimination of discharges of all pollutants if the Administrator finds . . . that such elimination is technologically and economically achievable[.]’” *Entergy Corp.*, 556 U.S. at 219 (quoting 33 U.S.C. § 1311(b)(2)(A)).

³⁸ EPA, Effluent Guidelines, *Learn about Effluent Guidelines*, available at <https://www.epa.gov/eg/learn-about-effluent-guidelines>. As EPA points out, “[t]he Effluent Guidelines do not require facilities to install the particular technology identified by EPA; however, the regulations do require facilities to achieve the regulatory standards which were developed based on a particular model technology.” *Id.*

case basis.³⁹ The following graphic illustrates the relationships between the different technology-based standards:⁴⁰

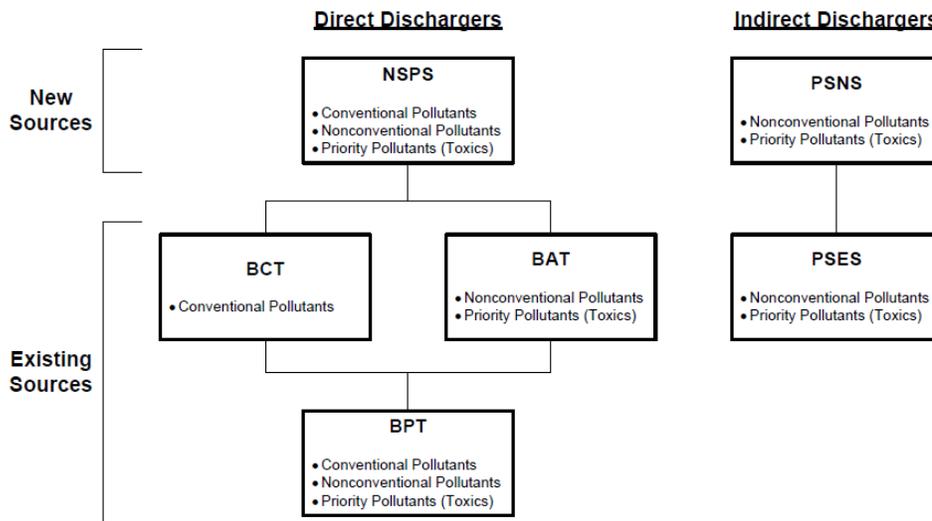


Figure 2-5. Regulations of Direct and Indirect Wastewater Discharges

1. National Effluent Limitation Guidelines and New Source Performance Standards

The CWA requires EPA to establish national ELGs for the discharge of all toxic pollutants on the Toxic Pollutant List.⁴¹ The goals of the ELGs and the standards required by the ELGs underscore why it is essential for EPA to revise the Toxic Pollutant lists and the ELGs regularly. The least stringent of the ELGs applicable to toxic pollutants is Best Practicable Control Technology Currently Available (“BPT”).⁴² The next most stringent technology-based

³⁹ See 40 C.F.R. § 125.3(c)(2) (technology-based treatment requirements may be imposed in a permit “on a case-by-case basis under section 402(a)(1) of the Act, to the extent that EPA-promulgated effluent limitations are inapplicable.”); see also EPA, *NPDES Permit Writers’ Manual* (Sept. 2010) (hereinafter “Permit Writers’ Manual”), available at https://www.epa.gov/sites/default/files/2015-09/documents/pwm_2010.pdf at 5-45.

⁴⁰ EPA, *Final 2012 and Preliminary 2014 Effluent Guidelines Program Plans* (Sept. 2014) (hereinafter “2014 ELG Plan”) at 2-11, available at https://19january2021snapshot.epa.gov/sites/static/files/2016-10/documents/final-2012-and-preliminary-2014-effluent-guidelines-program-plans_sept-2014_508.pdf.

⁴¹ CWA § 301(b)(2)(A), (C), (D).

⁴² CWA § 304(b)(1)(B).

standard is known as “best available technology economically achievable” (“BAT”),⁴³ which requires “implementation of pollution controls to the full extent of the best technology which would become available.”⁴⁴ As EPA explains,

BAT is intended to reflect the highest performance in the industry, and it may reflect a higher level of performance than is currently being achieved based on technology transferred from a different subcategory or category, bench scale or pilot studies, or foreign facilities. *Am. Paper Inst. v. Train*, 543 F.2d 328, 353 (D.C. Cir. 1976); *Am. Frozen Food Inst. v. Train*, 539 F.2d 107, 132 (D.C. Cir. 1976). BAT may be based upon process changes or internal controls, even when these technologies are not common industry practice. *See Am. Frozen Food Inst.*, 539 F.2d at 132, 140; *Reynolds Metals Co. v. EPA*, 760 F.2d 549, 562 (4th Cir. 1985); *Cal. & Hawaiian Sugar Co. v. EPA*, 553 F.2d 280, 285–88 (2nd Cir. 1977).⁴⁵

Finally, new facilities are subject to even more stringent effluent limitations through use of Best Available Demonstrated Control Technology (“BADCT”) used in New Source Performance Standards (“NSPS”). NSPS reflect “the greatest degree of effluent reduction” that is achievable based on BADCT, “including, where practicable, a standard permitting no discharge of pollutants.”⁴⁶

In addition to its definition of BAT and NSPS, another way in which Congress sought to make ELGs technology-forcing, and to move steadily toward the statutory zero-discharge goal, is by requiring EPA to review and revise them regularly. First, the CWA makes clear in two sections that EPA is required to “revise, if appropriate” its ELGs annually after publication.⁴⁷ EPA can only determine if revision is appropriate if it first reviews the ELGs annually. After establishing swift timelines for the development of ELGs in CWA Section 301(b), the statute

⁴³ CWA § 301(b)(2)(A). CWA § 307(a)(4) also requires an “ample margin of safety.”

⁴⁴ *NRDC v. U.S. E.P.A.*, 822 F.2d at 123–24.

⁴⁵ EPA, *Clean Water Act Effluent Limitations Guidelines and Standards for the Organic Chemicals, Plastics and Synthetic Fibers Point Source Category ANPRM*, 86 Fed. Reg. 14562 (March 17, 2021).

⁴⁶ CWA § 306(a)(1).

⁴⁷ CWA §§ 304(b), (m)(1)(A).

requires EPA to review the ELGs every five years and make revisions if appropriate, specifically for priority toxic pollutants.⁴⁸ Finally, Congress required EPA to incorporate these timelines into a biennially published schedule for the annual review and revision of ELGs for priority pollutants that also includes EPA's identification of categories of sources discharging "toxic or nonconventional pollutants" for which ELGs have not yet been published, and to establish a schedule for completing ELGs for these sources that is within three years of the first identification of those source categories.⁴⁹

Not only does the CWA require EPA to move with alacrity in establishing and revising ELGs, but it requires permittees to swiftly comply with the ELGs established for toxic pollutants listed on the Toxic Pollutant List as expeditiously as possible but not later than three years after ELGs' publication and in no case later than March 31, 1989.⁵⁰ For toxic pollutants not on the Toxic Pollutant List but placed on that list subsequently by EPA pursuant to CWA Section 304(a)(1), the Act requires permittees' compliance with effluent limitations based on ELGs within the same time frame.⁵¹

For EPA, the Act requires the establishment of these ELGs or a prohibition on discharge,

⁴⁸ CWA § 301(d). CWA § 301(d) applies to ELGs developed under § 301(b)(2), which includes those developed for the original priority pollutants, § 301(b)(2)(C), for any toxic pollutants subsequently added to the list of priority pollutants, § 301(b)(2)(D), and for nonconventional and newly-listed toxic pollutants, § 301(b)(2)(F).

⁴⁹ CWA 304(m)(1)(A)-(C).

⁵⁰ CWA § 301(b)(2)(A), (C). *See also* CWA § 307(a)(6) ("Any effluent standard (or prohibition) established pursuant to this section shall take effect on such date or dates as specified in the order promulgating such standard, but in no case, more than one year from the date of such promulgation. If the Administrator determines that compliance within one year from the date of promulgation is technologically infeasible for a category of sources, the Administrator may establish the effective date of the effluent standard (or prohibition) for such category at the earliest date upon which compliance can be feasibly attained by sources within such category, but in no event more than three years after the date of such promulgation.").

⁵¹ CWA § 301(b)(2)(D).

for pollutants on the Toxic Pollutant List, “tak[ing] into account the toxicity of the pollutant, its persistence, degradability, the usual or potential presence of the affected organisms in any waters, the importance of the affected organisms and the nature and extent of the effect of the toxic pollutant on such organisms, and the extent to which effective control is being or may be achieved under other regulatory authority”⁵² without delay.⁵³ Such ELGs are required for “every toxic pollutant” on the Toxic Pollutant List “as soon as practicable after December 27, 1977, but no later than July 1, 1980.”⁵⁴ For toxic pollutants placed on the Toxic Pollutant List by EPA subsequently, “effluent standards (or prohibitions) shall be established . . . as soon as practicable after it is so listed.”⁵⁵ Not only are the ELGs intended to be timely promulgated and timely implemented, and may include prohibitions on discharge, but Congress mandated that “[a]ny effluent standard promulgated under this section shall be at that level which the Administrator determines provides an ample margin of safety.”⁵⁶ The purpose of this entire scheme is to reduce all pollutant discharges, and to eliminate them wherever possible. EPA cannot accomplish that goal fully unless it continuously identifies and adds new or newly discovered toxic pollutants to the Toxic Pollutants Lists, and then adopts or revises ELGs to address those pollutants.

2. Pretreatment Standards

Not all industries discharge directly to receiving waters but, instead, discharge through publicly owned treatment works, as explained in sub-section V.B, *infra*. For all pollutants—not

⁵² CWA § 307(a)(2).

⁵³ *Id.* (“Such promulgation by the Administrator shall be made within two hundred and seventy days after publication of proposed standard (or prohibition).”)

⁵⁴ *Id.*

⁵⁵ *Id.*

⁵⁶ CWA § 307(a)(4).

limited to priority pollutants—that EPA determines “not to be susceptible to treatment by such treatment works or which would interfere with the operation of such treatment works,” EPA is required to develop Pretreatment Standards for Existing Sources (“PSES”).⁵⁷ Categorical pretreatment standards are technology-based and are analogous to BPT and BAT effluent limitations guidelines that apply to direct dischargers. Congress established urgency in the development and use of pretreatment standards: the CWA requires EPA to swiftly publish these pretreatment standards for the identified pollutants⁵⁸ and such standards “shall specify a time for compliance not to exceed three years from the date of promulgation.”⁵⁹ Indirect dischargers must quickly come into compliance with published pretreatment standards.⁶⁰ The statute requires that pretreatment standards be established to “prevent the discharge of any pollutant through treatment works . . . which are publicly owned, which pollutant interferes with, passes through, or otherwise is incompatible with such works.”⁶¹ EPA is also required to continue to publish pretreatment standards “from time to time thereafter.”⁶² In addition, EPA is required to simultaneously promulgate Pretreatment Standards for New Sources (“PSNS”) for new indirect dischargers whenever it promulgates NSPS for new direct dischargers.⁶³ Such PSNS must “prevent the discharge of any pollutant . . . which pollutant may interfere with, pass through, or otherwise be incompatible with such works.”⁶⁴ As with the ELGs, it is impossible for EPA to

⁵⁷ CWA 307(b)(1).

⁵⁸ CWA § 307(b)(1) (the timelines are swift: (1) proposed pretreatment standards are required within 180 days after October 18, 1972; and (2) not later than 90 days after EPA proposes the standards, EPA shall promulgate them).

⁵⁹ CWA 307(b)(1).

⁶⁰ CWA § 307(d).

⁶¹ CWA § 307(b)(1).

⁶² *Id.*

⁶³ CWA § 307(c).

⁶⁴ *Id.*

meet these statutory obligations fully, and to accomplish the statutory goals, unless it identifies all relevant toxic pollutants by reviewing and revising the Toxic Pollutants Lists, and then by reviewing and revising the pretreatment standards regularly to address those new or newly identified pollutants.

C. The Water Quality-Based Approach Relies on Adequate Water Quality Standards for All Pollutants Entering the Nation’s Waters

In the CWA, Congress required states to adopt numeric criteria in their water quality standards for all chemicals on the Toxic Pollutants Lists for which EPA has also published recommended criteria pursuant to Section 304(a).⁶⁵ Water quality standards are key to ensuring protection of water where technology-based pollution limits are not sufficient to protect water quality. They apply to all pollutants from all pollution sources, including nonpoint sources, and to all waters within a state’s boundaries.⁶⁶ Just as the CWA requires EPA to publish ELGs and pretreatment standards to carry out the Act’s technology-based approach, it requires EPA to publish recommended numeric criteria to support the adoption of state water quality standards and information to support states’ adopting other methods of assessing toxicity.⁶⁷

The CWA requires that states or, if states fail, EPA adopt water quality standards. Such standards must consist of three components: the designated uses, the water quality criteria (numeric and narrative) for waters based upon such uses, and antidegradation requirements.⁶⁸

⁶⁵ CWA § 303(c)(2)(B).

⁶⁶ See *Pronsolino v. Nastri*, 291 F.3d 1123, 1127 (9th Cir. 2002) (“[S]tates are required to set water quality standards for all waters within their boundaries regardless of the sources of the pollution entering waters.”), 1126 (“The precise statutory question before us is whether the phrase “are not stringent enough” triggers the identification requirement both for waters as to which effluent limitations apply but do not suffice to attain water quality standards and for waters as to which effluent limitations do not apply at all to the pollution sources impairing the water. We answer this question in the affirmative[.]”).

⁶⁷ CWA § 304(a)(1), (8).

⁶⁸ 33 U.S.C. § 1313(c)(2)(A). See also 40 C.F.R. §§ 131.2, 131.3(i), 131.6.

The standards must protect the public health or welfare, enhance the quality of water and wherever attainable, provide water quality for the protection and propagation of fish, shellfish, and wildlife and for recreation in and on the water, taking into consideration their use and value for public water supplies, and agricultural, industrial, and other purposes including navigation.⁶⁹

Water quality criteria must protect the designated uses.⁷⁰ Water quality criteria are expressed as constituent concentrations, levels, and/or narrative statements, representing a quality of water that supports designated uses.⁷¹ Such criteria must be based on sound scientific rationale and must contain sufficient parameters or constituents to protect the designated use.⁷² For waters with multiple use designations, the criteria must support the most sensitive use.⁷³

The adoption of criteria for the protection of human health is required for water bodies designated for public water supply and where fish ingestion is considered an important activity included in a designated use.⁷⁴ Criteria for protection of aquatic life generally consist of both chronic and acute measures of acceptable toxicity, for both fresh and marine water, if needed. The CWA requires that state toxic criteria be numerical if EPA has published recommended criteria. EPA's policy allows states to adopt statewide numeric criteria regardless of whether the pollutants are known to be present in navigable waters within the state, or as necessary where such pollutants are discharged or are present in the affected waters and could reasonably be

⁶⁹ 33 U.S.C. § 1313(c)(2)(A).

⁷⁰ 40 C.F.R. § 131.11(a)(1).

⁷¹ 40 C.F.R. § 131.3(b).

⁷² 40 C.F.R. § 131.11(a)(1).

⁷³ *Id.*

⁷⁴ EPA, *Water Quality Standards Handbook: Second Edition*, EPA-823-B-12-002 (March 2012), Chapter 3.1, available at <http://water.epa.gov/scitech/swguidance/standards/handbook/chapter03.cfm#section1> (web version last updated 2017).

expected to interfere with designated uses.⁷⁵ If a state selects the latter alternative, water quality data and information on discharges must be reviewed to identify specific water bodies where toxic pollutants may be adversely affecting water quality or the attainment of the designated water use. States must adopt criteria for such toxic pollutants applicable to the waterbody sufficient to protect the designated use. EPA expects similar determinations to occur during each triennial review of water quality standards as required by CWA Section 303(c)(2)(B).⁷⁶

Similar to technology-based standards, the water quality-based approach is intended to be “technology-forcing.” As the D.C. Circuit held in 1988, “Section 301(b)(1)(C) requires satisfaction of ‘*any more stringent* limitation . . . necessary to meet water quality standards.’ The import of this language is made clear by the legislative history[.]”⁷⁷ Quoting the legislative history, the court concluded:

This evidence strongly supports EPA’s position that Congress did not intend to tie compliance with water quality-based limitations to the capabilities of any given level of technology. A technology-based standard discards its fundamental premise when it ignores the limits inherent in the technology. By contrast, a water quality-based permit limit begins with the premise that a certain level of water quality will be maintained, come what may, and places upon the permittee the responsibility for realizing that goal.⁷⁸

Thus, “[n]othing in the Act indicates suitably that individual technological failures may excuse violations of water quality-based limitations.”⁷⁹

⁷⁵ *Id.* at *State Options*, available at

<http://water.epa.gov/scitech/swguidance/standards/handbook/chapter03.cfm#section4>.

⁷⁶ *Id.*

⁷⁷ *Natural Resources Defense Council v. U.S. E.P.A.*, 859 F.2d 156, 208 (D.C. Cir. 1988).

⁷⁸ *Id.* (footnotes omitted). See also *Riverkeeper, Inc. v. U.S. E.P.A.*, 475 F.3d 83, 108 (2d Cir. 2007) (Sotomayor, J.) (referencing the Act’s “technology-forcing imperative”), *rev’d sub nom by Entergy Corp*, 556 U.S. 208.

⁷⁹ *Id.*

Water quality standards are also the method by which state control of nonpoint source polluted runoff is judged for its adequacy. Absent listing of all relevant pollutants on the Toxic Pollutants Lists, and subsequent EPA development of 304(a) recommended criteria and state or EPA adoption of water quality standards for those pollutants, many toxic pollutants reaching the nation's waters from agricultural, silvacultural, and other nonpoint sources will remain unaddressed.

As the Ninth Circuit explained in *Pronsolino*, Section 319 of the CWA “encourages the states to institute an approach to the elimination of nonpoint source pollution similar to the federally-mandated effluent controls contained in the CWA, while [the impaired listing and TMDL processes of CWA Section] 303 encompass[] a water quality based approach applicable to all sources of water pollution.”⁸⁰ In order for TMDLs to be effective in achieving their goal of controlling all sources of pollution to meet water quality standards, they make allocations—set pollution limits—for both point and nonpoint sources.⁸¹ Moreover, EPA TMDL regulations specifically require these limits, known as “wasteload allocations,” for point sources to take into account likely pollution reductions by nonpoint source controls, a concept referred to as “reasonable assurance.”⁸²

⁸⁰ *Pronsolino v. Nastri*, 291 F.3d 1139.

⁸¹ CWA § 303(d)(1)(C) (“Such [total maximum daily] load shall be established at a level necessary to implement the applicable water quality standards[.]”); 40 C.F.R. § 130.3(i) (defining TMDLs as the “sum of the individual [wasteload allocations] WLA for point sources and [load allocations] LAs for nonpoint sources and natural background.”); *id.* at 130.7(c)(1) (mirroring the statutory requirement to “attain and maintain the applicable narrative and numerical WQS”).

⁸² *Id.* (“If Best Management Practices (BMPs) or other nonpoint source pollution controls make more stringent load allocations practicable, then wasteload allocations can be made less stringent. Thus, the TMDL process provides for nonpoint source control tradeoffs.”). CWA Section 301(b)(1)(C) and EPA’s permitting regulations provide additional support for including “reasonable assurance” in TMDLs. *See* 40 C.F.R. § 122.44(d)(1)(vii)(A), (B) (effluent limits must be derived from water quality standards and be “consistent with the assumption and requirements of any available wasteload allocation for the discharge”).

A complete listing of all relevant pollutants on the Toxic Pollutant Lists is key to ensuring states adequately control nonpoint sources. For example, CWA Section 319 requires states, among other tasks, to: (1) identify all navigable waters that cannot attain water quality standards or the goals of the Act absent additional nonpoint source controls;⁸³ (2) identify nonpoint sources or categories of sources contributing to that pollution;⁸⁴ and (3) describe processes and programs to develop best management practices (“BMPs”) and other controls for those pollutant sources.⁸⁵ Likewise, in state nonpoint source management programs, states are required to develop and assess implementation and the degree of success of BMPs to control those pollution sources or categories of sources.⁸⁶ Given that, as shown in Section III of this petition, large amounts of currently unregulated toxic pollutants are reaching the nation’s waters from nonpoint sources, it is difficult or impossible—and practically unlikely—for states to implement these requirements adequately absent identification of toxic pollutants on the Toxic Pollutants Lists and the subsequent actions that such listing will trigger.

For coastal states, Congress has made clear through the Coastal Zone Act Reauthorization Amendments (“CZARA”) that these nonpoint source controls must be sufficient to achieve and maintain water quality standards⁸⁷ and that the Coastal Nonpoint Pollution Control Program it requires must be “coordinated closely” with both CWA Section 319 and Section 303.⁸⁸ In fact, CZARA programs “shall serve as an update and expansion of the State nonpoint source management program developed under [Section 319.]” Thus, for coastal states,

⁸³ 33 U.S.C. § 1329(a)(1)(A).

⁸⁴ *Id.* § 1329(a)(1)(B).

⁸⁵ *Id.* § 1329(a)(1)(C), (D).

⁸⁶ *Id.* § 1329(b)(2).

⁸⁷ 16 U.S.C. § 1455b(b)(3).

⁸⁸ *Id.* at (a)(2).

nonpoint source controls are required to meet water quality standards even in the absence of a TMDL.

This entire water quality-based process is only effective for toxic pollutants for which states have adopted criteria. To be sure, nothing in the CWA expressly limits EPA's development of 304(a) recommended criteria or state adoption of water quality standards to pollutants on the Toxics Pollutants Lists. Historically, however, neither EPA nor states have done so for the vast majority of the many pollutants that are the focus of this petition. Nor have states historically monitored for most pollutants that are not covered by 304(a) criteria or state criteria, meaning they have no way to ascertain whether those pollutants are present in, or interfering with designated uses in, their waters. Thus, states cannot fully and adequately administer the water quality-based approach to toxic pollutant control unless EPA expands the Toxic Pollutants Lists to address the full range of toxic pollutants plaguing the Nation's Waters.

D. The 1987 Amendments to the Clean Water Act Focus on the Need for Greater Control of Toxics Through Multiple Approaches

When Congress amended the CWA in 1987, it added provisions to address EPA and states' failed efforts to control toxic pollution, one that EPA can substantially remedy by adding pollutants to the Toxic Pollutants Lists. During the 1970s, the water quality standards program was a relatively low priority for EPA in comparison with other approaches established by the CWA.⁸⁹ By the early 1980s, however, it became clear to Congress that effective protection and enhancement of the nation's waters must include a greater focus on water quality-based pollution control.⁹⁰ One issue that particularly concerned Congress was states' heavy reliance on vague

⁸⁹ EPA, *Water Quality Standards History*, available at <http://water.epa.gov/scitech/swguidance/standards/history.cfm>, last updated April 3, 2012.

⁹⁰ *Id.*

narrative criteria in their control of toxics, such as “no toxics in toxic amounts.”⁹¹ To rectify this problem, among other actions, Congress adopted CWA amendments that require states, when reviewing their water quality standards, to “adopt criteria for all toxic pollutants listed pursuant to section 1317(a)(1) [307(a)] of this title for which criteria have been published under section 1314(a) [304(a)] of this title, the discharge of which in the affected waters could reasonably be expected to interfere with those designated uses adopted by the State.”⁹² If available as recommended 304(a) numeric criteria from EPA, the criteria adopted by the states must be “specific numerical criteria for such toxic pollutants.”⁹³

As EPA described in promulgating the subsequent National Toxics Rule (“NTR”), discussed *infra* sub-section VII.H, the agency had attempted to use its own authority to obtain state action to adopt “appropriate toxics criteria” by “vigorously pursu[ing] the alternative approach of EPA issuance of scientific water quality criteria documents which States could use to adopt enforceable water quality standards” and in 1983 “amend[ing] the water quality standards regulation to explicitly address toxic criteria requirements in State standards.”⁹⁴ But EPA described a very disappointing state response to these new regulations:

State response to EPA’s criteria publication and toxics initiative was disappointing. A few States adopted large numbers of numeric toxics criteria, although primarily for the protection of aquatic life. Most other States adopted few or no water quality criteria for priority toxic pollutants. Some relied on a narrative “free from toxicity” criterion, and so-called “action levels” for toxic pollutants or occasionally calculated site-specific criteria. Few States addressed the protection of human health by adopting numeric human health criteria.⁹⁵

⁹¹ *Id.*

⁹² CWA § 303(c)(2)(B).

⁹³ *Id.*

⁹⁴ Water Quality Standards; Establishment of Numeric Criteria for Priority Toxic Pollutants; States’ Compliance (hereinafter “NTR Rules”), 57 Fed. Reg. 60848, 60851 (Dec. 22, 1992), citing Water Quality Standards Regulation, 48 Fed. Reg. 51400 (Nov. 8, 1983) (codified at 40 C.F.R. Part 131).

⁹⁵ *Id.*

EPA also noted that “[s]tate practices of developing case-by-case effluent limits using procedures that were not standardized in State regulations made it difficult to ascertain whether such procedures were consistently applied.”⁹⁶ EPA describes how it pressed on, issuing guidance, including the Water Quality Standards Handbook in 1983 and the Technical Support Document for Water Quality Based Toxics Control in 1985, which included “needed information to convert chemical specific and biologically based criteria into water quality standards for ambient receiving waters and permit limits for discharges to those waters” along with bioassay testing of effluent “to implement the ‘free from toxicity’ narrative standards in State water quality standards.”⁹⁷ Despite these EPA efforts, “by the time of Congressional consideration and action on the CWA reauthorization, most States had adopted few, if any, water quality standards for priority toxic pollutants.”⁹⁸

In addition to the provisions added in Section 303(c), in the 1987 Amendments, Congress strengthened other failed aspects of the statute’s regulation of toxic pollution in the nation’s waters with an emphasis on swift pollution control actions.⁹⁹ Notably, it added Section 304(l) that mirrored the requirements of Section 303(d) insofar as it required identification of waters with unsafe levels of toxic pollutants,¹⁰⁰ with a specific focus on those waters affected by point sources¹⁰¹ and, in some instances identification of the point sources causing or contributing to those levels,¹⁰² along with a revised NPDES permit to:

⁹⁶ *Id.*

⁹⁷ *Id.*

⁹⁸ *Id.*

⁹⁹ In addition to the three-year provision of CWA § 304(l)(1)(D), the amendments gave EPA only nine months in which to publish guidance for the states on how to carry out the requirements of 304(l). CWA § 304(a)(7).

¹⁰⁰ CWA § 304(l)(1)(A).

¹⁰¹ CWA § 304(l)(1)(B).

¹⁰² CWA § 304(l)(1)(C).

produce a reduction in the discharge of toxic pollutants from point sources identified by the State under this paragraph through the establishment of effluent limitations under section 402 of this Act and water quality standards under section 303(c)(2)(B) of this Act, which reduction is sufficient, in combination with existing controls on point and nonpoint sources of pollution, to achieve the applicable water quality standard as soon as possible, but not later than 3 years after the date of the establishment of such strategy.¹⁰³

Consistent with its emphasis on using the water quality-based approach to control toxics, particularly from NPDES-permitted sources, the 1987 Amendments also addressed the importance of improving the timely implementation of the technology-based approach. To address EPA's slow progress in establishing national effluent limitations guidelines, Congress called for EPA to publish a schedule for reviewing these guidelines and, specifically focused on the importance of using ELGs to control toxics by requiring EPA to:

(B) identify categories of sources discharging toxic or nonconventional pollutants for which guidelines under subsection (b)(2) of this section and section 306 have not previously been published; and

(C) establish a schedule for promulgation of effluent guidelines for categories identified in subparagraph (B), under which promulgation of such guidelines shall be no later than 4 years after such date of enactment for categories identified in the first published plan or 3 years after the publication of the plan for categories identified in later published plans.¹⁰⁴

It also required EPA to report on the pretreatment program in four years and to make recommendations for improving the effectiveness of the pretreatment program to Congress.¹⁰⁵

This study was required to address the following aspects of the pretreatment program:

- (1) the adequacy of data on environmental impacts of toxic industrial pollutants from publicly owned treatment works;
- (2) the extent to which secondary treatment at publicly owned treatment works removes toxic pollutants;

¹⁰³ CWA § 304(l)(1)(D).

¹⁰⁴ CWA § 304(m)(1).

¹⁰⁵ CWA § 519(b); Pub. L 100-4, Title V, § 519, Feb. 4, 1987, 100 Stat. 87.

- (3) the capability of publicly owned treatment works to revise pretreatment requirements under section 307(b)(1) of the Federal Water Pollution Control Act;
- (4) possible alternative regulatory strategies for protecting the operations of publicly owned treatment works from industrial discharges, and shall evaluate the extent to which each such strategy identified may be expected to achieve the goals of this Act;
- (5) for each such alternative regulatory strategy, the extent to which removal of toxic pollutants by publicly owned treatment works results in contamination of sewage sludge and the extent to which pretreatment requirements may prevent such contamination or improve the ability of publicly owned treatment works to comply with sewage sludge criteria developed under section 405 of the Federal Water Pollution Control Act; and
- (6) the adequacy of Federal, State, and local resources to establish, implement, and enforce multiple pretreatment limits for toxic pollutants for each such alternative strategy.¹⁰⁶

Finally, in the 1987 Amendments, Congress highlighted the importance of toxics in specific waterbodies, to address pollutant loading of chlorine and other “toxic pollutants, including organic chemicals and heavy metals” in estuaries,¹⁰⁷ to meet a goal of “reducing or eliminating the input of chemical contaminants from all controllable sources to levels that result in no toxic or bioaccumulative impact on the living resources of the Chesapeake Bay ecosystem or on human health,”¹⁰⁸ and to monitor and meet the goals for protection of the Great Lakes “with specific emphasis on the monitoring of toxic pollutants”¹⁰⁹ and “with particular emphasis on [meeting] goals related to toxic pollutants,”¹¹⁰ and for the control and removal of toxic

¹⁰⁶ EPA, *National Pretreatment Program Report to Congress* (July 1991) at ES-1.

¹⁰⁷ CWA § 320(j)(1)(C).

¹⁰⁸ CWA § 117(g)(1)(C).

¹⁰⁹ CWA § 118(c)(1)(B); *see also* CWA § 118(c)(10)(B) (EPA to issue a report that “describes the progress made in such preceding fiscal year in implementing the system of surveillance of the water quality in the Great Lakes System, including the monitoring of groundwater and sediment, with particular reference to toxic pollutants.”).

¹¹⁰ CWA § 118(a)(1)(B).

sediments.^{111, 112}

1. In the 1987 Amendments, Congress Highlighted the Key Importance of Numeric Criteria

As EPA noted in the preamble to the National Toxics Rule, in which it promulgated numeric criteria for states that failed to take the basic actions now required by the 1987 Amendments, the legislative history underscores Congressional concern about states' failure to address toxics and EPA's failure to use its oversight role to push states to more swift action. EPA cited the statements of Senator Robert T. Stafford, first chairman and then ranking minority member of the authorizing committee, who noted:

An important problem in this regard is that few States have numeric ambient criteria for toxic pollutants. The lack of ambient criteria [for toxic pollutants] makes it impossible to calculate additional discharge limitations for toxics It

¹¹¹ CWA § 118(c)(7)(A) (EPA to carry out a five-year study and demonstration projects “with emphasis on the removal of toxic pollutants from bottom sediments.”).

¹¹² Other provisions of the 1987 Amendments focused on toxics include the Clean Lakes Program, CWA § 314(a)(1)(F) (states to assess lakes “particularly with respect to toxic pollution”); National Estuary Program, CWA § 320(b)(2) (to assess “data on toxics, nutrients, and natural resources within the estuarine zone”), CWA § 320(j)(C) (to carry out a “comprehensive water quality sampling program for the continuous monitoring of nutrients, chlorine, acid precipitation dissolved oxygen, and potentially toxic pollutants (including organic chemicals and metals) in estuarine zones”); Sewage Sludge Program, CWA § 405(d)(2)(A)(i) (in which EPA shall “identify those toxic pollutants which, on the basis of available information on their toxicity, persistence, concentration, mobility, or potential for exposure, may be present in sewage sludge in concentrations which may adversely affect public health or the environment, and propose regulations specifying acceptable management practices for sewage sludge containing each such toxic pollutant and establishing numerical limitations for each such pollutant for each use identified under paragraph (1)(A).”), CWA § 405(d)(2)(B)(i) (“Not later than July 31, 1987, the Administrator shall identify those toxic pollutants not identified under subparagraph (A)(i) which may be present in sewage sludge in concentrations which may adversely affect public health or the environment, and propose regulations specifying acceptable management practices for sewage sludge containing each such toxic pollutant and establishing numerical limitations for each pollutant for each such use identified under paragraph (1)(A).”), CWA § 405(d)(2)(C) (“From time to time, but not less often than every 2 years, the Administrator shall review the regulations promulgated under this paragraph for the purpose of identifying additional toxic pollutants and promulgating regulations for such pollutants consistent with the requirements of this paragraph.”), CWA § 405(d)(4) (“Prior to the promulgation of the regulations required by paragraph (2), the Administrator shall impose conditions in permits issued to publicly owned treatment works under section 402 of this Act or take such other measures as the Administrator deems appropriate to protect public health and the environment from any adverse effects which may occur from toxic pollutants in sewage sludge.”).

is vitally important that the water quality standards program operate in such a way that it supports the objectives of the Clean Water Act to restore and maintain the integrity of the Nation's Waters.¹¹³

EPA pointed out the driver for the 1987 Amendments was “the Congressional perception that the States were failing to aggressively address toxics and that EPA was not using its oversight role to push the States to move more quickly and comprehensively.”¹¹⁴ This was described by Sen.

John H. Chafee:

A cornerstone of the bill's new toxic pollution control requirements is the so called beyond-BAT program Adopting the beyond BAT provisions will assure that EPA continues to move forward rapidly on the program [and] if we are going to repair the damage to those water bodies that have become highly degraded as a result of toxic substances, we are going to have to move forward expeditiously on this beyond-BAT program. The Nation cannot tolerate endless delays and negotiations between EPA and States on this program. Both entities must move aggressively in taking the necessary steps to make this program work within the time frame established by this Bill[.]¹¹⁵

In EPA's own words, “[t]his Congressional impatience with the pace of State and EPA progress and an appreciation that the lack of State standards for toxics undermined the effectiveness of the entire CWA-based scheme, resulted in the 1987 adoption of stringent new water quality standard provisions in the Water Quality Act amendments.”¹¹⁶ Put another way, “for the first time in the history of the Clean Water Act, Congress took the unusual action of explicitly mandating that States adopt numeric criteria for specific toxic pollutants.”¹¹⁷ It also specifically emphasized a new two-year requirement to establish “specific numerical limits to protect health, aquatic life, and wildlife from the bioaccumulation of toxins” pertaining to

¹¹³ NTR Rules, *supra* n. 94 at 60851. *See also* U.S. Government Printing Office, *A Legislative History of the Water Quality Act of 1987* (Pub. L. 100-4), Senate Print 100-144 (Nov. 1988) (hereinafter “1987 Legislative History”) at 1324.

¹¹⁴ NTR Rules, *supra* n. 94 at 60852.

¹¹⁵ 1987 Legislative History, *supra* n. 113 at 1309.

¹¹⁶ NTR Rules, *supra* n. 94 at 60852.

¹¹⁷ *Id.*

contaminants in Great Lakes' sediment.¹¹⁸

2. In the 1987 Amendments, Congress Also Sought to Strengthen Implementation of States' Narrative Criteria

Where EPA had not completed recommended 304(a) criteria for use by the states, Congress also required the use of biological monitoring and assessment to ensure that state-adopted narrative criteria would be meaningful in regulatory efforts to reduce toxic pollution:

Where such numerical criteria are not available, whenever a State reviews water quality standards pursuant to paragraph (1), or revises or adopts new standards pursuant to this paragraph, such State shall adopt criteria based on biological monitoring or assessment methods consistent with information published pursuant to section 304(a)(8). Nothing in this section shall be construed to limit or delay the use of effluent limitations or other permit conditions based on or involving biological monitoring or assessment methods or previously adopted numerical criteria.¹¹⁹

To support this requirement, Congress instructed EPA to first address the importance of protecting beneficial uses from bioaccumulating toxic pollutants by research:

In carrying out the provisions of section 104(a) of the Federal Water Pollution Control Act, the Administrator shall conduct research on the harmful effects on the health and welfare of persons caused by pollutants in water, in conjunction with the United States Fish and Wildlife Service, the National Oceanic and Atmospheric Administration, and other Federal, State, and interstate agencies carrying on such research. Such research shall include, and shall place special emphasis on, the effect that bioaccumulation of these pollutants in aquatic species has upon reducing the value of aquatic commercial and sport industries. Such research shall further study methods to reduce and remove these pollutants from the relevant affected aquatic species so as to restore and enhance these valuable resources.¹²⁰

And, second, to swiftly move this research into the regulatory arena, Congress instructed EPA to “develop and publish information on methods for establishing and measuring water quality

¹¹⁸ CWA § 118(c)(7)(C).

¹¹⁹ CWA § 303(c)(2)(B).

¹²⁰ 33 U.S.C. § 1254a (emphasis added).

criteria for toxic pollutants on other bases than pollutant-by-pollutant criteria, including biological monitoring and assessment methods.”¹²¹

E. Narrative Criteria Are an Essential Gap-Filler to Protect Against Adverse Toxic Effects to Designated Uses

Water quality standards consist of designated uses for waters and water quality criteria—both numeric and narrative—necessary to protect those uses, as well as an antidegradation policy.¹²² The CWA requires numeric criteria adopted in water quality standards to protect the “most sensitive use.”¹²³ However, since it is not always possible to adopt numeric criteria, and because numeric criteria for individual pollutants do not fully address cumulative and synergistic effects of multiple pollutants within a discharge or a water body, the task of evaluating whether water quality standards have been met also requires an assessment of the impacts to designated beneficial uses through narrative criteria, including but absolutely not limited to whole effluent toxicity (“WET”) criteria. In 1994, the U.S. Supreme Court underscored the importance of protecting beneficial uses as a “complementary requirement” that “enables the States to ensure that each activity—even if not foreseen by the criteria—will be consistent with the specific uses and attributes of a particular body of water.”¹²⁴ As the Supreme Court explained, numeric criteria “cannot reasonably be expected to anticipate all of the water quality issues arising from every activity which can affect the State’s hundreds of individual water bodies”¹²⁵ and thus “the Act permits enforcement of broad, narrative criteria based on, for example, ‘aesthetics.’”¹²⁶

¹²¹ CWA § 304(a)(8).

¹²² 33 U.S.C. § 1313(c)(2)(a); 40 C.F.R. § 131.6.

¹²³ 40 C.F.R. § 131.11(a)(1).

¹²⁴ *PUD No. 1 of Jefferson County v. Washington Department of Ecology*, 511 U.S. 700, 717 (1994).

¹²⁵ *Id.*

¹²⁶ *Id.* at 716.

For this reason, the Supreme Court cited EPA’s interpretation of the statute:

EPA has not interpreted § 303 to require the States to protect designated uses exclusively through enforcement of numerical criteria. In its regulations governing state water quality standards, EPA defines criteria as “*elements* of State water quality standards, expressed as constituent concentrations, levels, or narrative statements, representing a quality of water that supports a particular use.” 40 CFR § 131.3(b) (1993) (emphasis added). The regulations further provide that “[w]hen criteria are met, water quality will *generally* protect the designated use.” *Ibid.* (emphasis added). Thus, the EPA regulations implicitly recognize that in some circumstances, criteria alone are insufficient to protect a designated use.¹²⁷

The decision specifically called out the role of narrative criteria for control of toxic pollutants as explained by the federal government:

As the Solicitor General points out, even “criteria” are often expressed in broad, narrative terms, such as “there shall be no discharge of toxic pollutants in toxic amounts.” Brief for United States as *Amicus Curiae* 18. See *American Paper Institute, Inc. v. EPA*, 996 F. 2d 346, 349 (CADC 1993). In fact, under the Clean Water Act, only one class of criteria, those governing “toxic pollutants listed pursuant to section 1317(a)(1),” need be rendered in numerical form. See 33 U. S. C. § 1313(c)(2)(B); 40 CFR § 131.11(b)(2) (1993).¹²⁸

As cited by the Solicitor General, the importance of narrative criteria is set out in EPA’s water quality standards regulations themselves regarding the development and use of narrative criteria for toxic pollutants:

Where a State adopts narrative criteria for toxic pollutants to protect designated uses, the State must provide information identifying the method by which the State intends to regulate point source discharges of toxic pollutants on water quality limited segments based on such criteria. Such information may be included as part of the standards or may be included in documents generated by the State in response to the Water Quality Planning and Management Regulations (40 CFR part 35).¹²⁹

In addition, these regulations require that States must “[e]stablish narrative criteria or criteria

¹²⁷ *Id.* at 715.

¹²⁸ *Id.* at 716.

¹²⁹ 40 C.F.R. § 131.11(a)(2).

based upon biomonitoring methods where numerical criteria cannot be established or to supplement numerical criteria.”¹³⁰

Leading up to the Supreme Court’s decision, EPA’s policy of “independent applicability” reinforced the importance of usable narrative criteria. As explained by EPA:

In 1991, EPA established its policy on independent application (U.S. EPA, transmittal memorandum of final policy on biological assessment and criteria from Tudor Davies to Regions, June 19, 1991). EPA’s independent application policy speaks to how assessments based on these three kinds of criteria are to be integrated into all forms of water quality management decision-making. EPA’s independent application policy and the ensuing discussion here address the issue of how the three different kinds of assessments are interpreted only in the context of protection of aquatic life and aquatic life uses and not in the context of protection of human health or wildlife.

* * *

Independent application states that where different types of monitoring data are available for assessment of whether a water body is attaining aquatic life uses or for identifying the potential of pollution sources to cause or contribute to non attainment of aquatic life uses, any one assessment is sufficient to identify an existing or potential impact/ impairment, and no one assessment can be used to override a finding of existing or potential impact or impairment based on another assessment. The independent application policy takes into account that each assessment provides unique insights into the integrity and health of an aquatic system. In addition, each assessment approach has differing strengths and limitations, and assesses different stressors and their effects, or potential effects, on aquatic systems. For example, while biological assessments can provide information in determining the cumulative effect of past or current impacts from multiple stressors, these assessments may be limited in their ability to predict, and therefore prevent, impacts. While chemical-specific assessments are useful to evaluate and predict ecosystem impacts from single pollutants, chemical-specific methods are unable to assess the combined interactions of pollutants (e.g., additivity). Similar to biological assessments, toxicity testing provides a means of evaluating the aggregate toxic effects of pollutants, and like chemical assessments, can also be used when testing effluent to predict single chemical impacts. One of the limitations of toxicity testing, however, is that the identification of pollutants causing toxicity is not always possible or cost effective. Each of these three assessment approaches relies on different kinds of water quality data, measures different endpoints and, in practice, will be interpreted in the context of implementing a water quality management program that includes assessment and pollution control. EPA’s policy on independent

¹³⁰ 40 C.F.R. § 131.11(b)(2).

application is based on the premise that any valid, representative data indicating an actual or projected water quality impairment must not be ignored when determining the appropriate action to be taken. Independent application recognizes the strengths and limitations of all three assessment approaches.¹³¹

EPA regulations implementing section 303(d) of the CWA also reflect the importance of each independent component of a state's water quality standards:

For the purposes of listing waters under §130.7(b), the term “water quality standard applicable to such waters” and “applicable water quality standards” refer to those water quality standards established under section 303 of the Act, including numeric criteria, narrative criteria, waterbody uses, and antidegradation requirements.¹³²

When EPA adopted these regulations, the preamble to the rule clearly set out its expectations of states:

[I]n today's final action the term “applicable standard” for the purposes of listing waters under section 303(d) is defined in § 130.7(b)(3) as those water quality standards established under section 303 of the Act, including numeric criteria, narrative criteria, waterbody uses and antidegradation requirements. In the case of a pollutant for which a numeric criterion has not been developed, a State should interpret its narrative criteria by applying a proposed state numeric criterion, an explicit State policy or regulation (such as applying a translator procedure developed pursuant to section 303(c)(2)(B) to derive numeric criteria for priority toxic pollutants), EPA national water quality criteria guidance developed under section 304(a) of the Act and supplemented with other relevant information, or by otherwise calculating on a case-by-case basis the ambient concentration of the pollutant that corresponds to attainment of the narrative criterion. Today's definition is consistent with EPA's Water Quality Standards regulation at 40 CFR part 131. EPA may disapprove a [303(d)] list that is based on a State interpretation of a narrative criterion that EPA finds unacceptable.¹³³

EPA's NPDES permitting regulations also mirror its rules on developing approvable 303(d) lists based on narrative criteria, making abundantly clear that NPDES permits must

¹³¹ EPA, *Water Quality Standards Regulation ANPRM*, 63 Fed. Reg. 36794-36795 (July 7, 1998). (hereinafter “Standards ANPRM”).

¹³² 40 C.F.R. § 130.7(b)(3).

¹³³ Surface Water Toxics Control Program and Water Quality Planning and Management Program, 57 Fed. Reg. 33040, 33046 (July 24, 1992).

comply with narrative criteria: NPDES effluent limitations must control all pollutants that are or may be discharged at a level “which will cause, have the reasonable potential to cause, or contribute to an excursion above any State water quality standard, including State narrative criteria for water quality.”¹³⁴ In fact, numerous aspects of these NPDES permitting regulations specifically cite to the requirement to meet narrative criteria.¹³⁵ Likewise, courts have upheld both the requirement and the importance of issuing permits that meet narrative criteria. As the D.C. Circuit Court found, when it upheld EPA’s permitting regulations pertaining to narrative criteria, faced with the conundrum of narrative criteria “some permit writers threw up their hands and, *contrary to the Act*, simply ignored water quality standards including narrative criteria altogether when deciding upon permit limitations.”¹³⁶ The First Circuit agreed: “When issuing NPDES permits for states that employ narrative criteria, the EPA must translate those criteria into a ‘calculated numeric water quality criterion.’”¹³⁷

Moreover, the NPDES regulations do not permit agencies to avoid imposing limits on pollution discharges in the face of uncertainty surrounding the application of narrative criteria.

As the Second Circuit articulated,

Even if determining the proper standard is difficult, EPA cannot simply give up and refuse to issue more specific guidelines. *See Am. Paper Inst., Inc. v. EPA*, 996 F.2d 346, 350 (D.C. Cir. 1993) (articulating that, even if creating permit

¹³⁴ 40 C.F.R. § 122.44(d)(1)(i) (emphasis added).

¹³⁵ *See* 40 C.F.R. §§ 122.44(d)(1) (limits must be included to “[a]chieve water quality standards established under section 303 of the CWA, including State narrative criteria for water quality”); 122.44(d)(1)(i) (limitations must include all parameters “including State narrative criteria for water quality”); 122.44(d)(1)(ii) (reasonable potential must be evaluated for “in-stream excursion above a narrative or numeric criteria”); 122.44(d)(1)(v) (WET tests required where reasonable potential exists to cause or contribute to a narrative criterion excursion unless chemical-specific pollutants are “sufficient to attain and maintain applicable numeric and narrative State water quality standards”); 122.44(d)(1)(vi) (options for establishing limitations where reasonable potential exists for a discharge to cause or contribute to an excursion above a narrative criterion) (emphases added).

¹³⁶ *American Paper Inst., Inc. v. U.S. E.P.A.*, 996 F.2d 346, 350 (D.C. Cir. 1993) (emphasis added).

¹³⁷ *City of Taunton, Massachusetts v. U.S. Env’tl. Protection Agency*, 895 F. 3d 120, 133 (1st Cir. 2018).

limits is difficult, permit writers cannot just “thr[o]w up their hands and, contrary to the Act, simply ignore[] water quality standards including narrative criteria altogether when deciding upon permit limitations”). Scientific uncertainty does not allow EPA to avoid responsibility for regulating discharges. *See Massachusetts v. EPA*, 549 U.S. 497, 534 (2007) (“EPA [cannot] avoid its statutory obligation by noting the uncertainty surrounding various features of climate change and concluding that it would therefore be better not to regulate at this time.”).¹³⁸

Both the First Circuit¹³⁹ and the EPA’s Environmental Appeals Board¹⁴⁰ have agreed that uncertainty does not excuse the permit writer from its obligation to set permit limits. For this reason, it is imperative that states and EPA know in advance of the issuance of NPDES permits how the agencies will interpret and apply their narrative criteria that protect designated uses against the toxic effects of toxic pollutants, as required by 40 C.F.R. § 131.11(a)(2).

The availability of narrative criteria and methods to apply them, however, does not imply any lesser need for EPA to identify and list additional individual toxic pollutants and pollutant families, as requested in this Petition. Despite such listing, EPA will encounter significant delays in developing numeric criteria for such pollutants; narrative criteria will have to be used in the interim. However, narrative criteria cannot be used to avoid the need to list additional pollutants and to develop numeric criteria for them where feasible. Indeed, identification and listing of additional toxic pollutants will help states to monitor for additional pollutants that may be adversely affecting their waters, which will help with implementation of both numeric and narrative criteria, thus strengthening implementation of the entire water quality-based system to control toxic pollution.

¹³⁸ *Nat. Res. Defense Council v. U.S. Envtl. Protection Agency*, 808 F.3d 556, 578 (2d Cir. 2015).

¹³⁹ *Upper Blackstone Water Pollution Abatement Dist. v. U.S. Envtl. Protection Agency*, 690 F.3d 9, 24–25 (1st Cir. 2012), *cert. denied*, 133 S. Ct. 2382 (2013).

¹⁴⁰ *In re: City of Taunton Dept. of Public Works*, NPDES Appeal No. 15-08, 17 E.A.D. 105, 2016 WL 3352212, *42 (EAB May 3, 2016).

F. The Clean Water Act Requires the Protection of Designated and Existing Uses of Wildlife from Toxics in Water Quality Standards

In identifying and listing toxic pollutants on the Toxic Pollutants Lists, EPA must consider impacts on aquatic and aquatic-dependent wildlife as well as impacts on fish and shellfish. It is the interim goal of the CWA to provide, wherever attainable, “water quality which provides for the protection and propagation of . . . shellfish, and wildlife . . . by July 1, 1983.”¹⁴¹ This protection for wildlife as a designated use is buttressed by another fundamental statutory goal: “it is the national policy that the discharge of toxic pollutants in toxic amounts be prohibited.”¹⁴² The CWA explicitly contemplates EPA’s taking the initial steps to ensure the protection of wildlife from such toxic effects, including bioaccumulation of toxic pollutants:

The Administrator . . . shall develop and publish . . . criteria for water quality accurately reflecting the latest scientific knowledge (A) on the kind and extent of all identifiable effects on health and welfare including, but not limited to, . . . shellfish, wildlife . . . which may be expected from the presence of pollutants in any body of water[.] . . . ; (B) on the concentration and dispersal of pollutants, or their byproducts, through biological, physical, and chemical process[.]¹⁴³

Based on the statute, EPA regulations for water quality standards require states to designate shellfish and wildlife uses.¹⁴⁴ Next, the regulations require states to adopt

criteria that protect the designated use. Such criteria must be based on sound scientific rationale and must contain sufficient parameters or constituents to protect the designated use. For waters with multiple use designations, the criteria shall support the most sensitive use.¹⁴⁵

¹⁴¹ CWA § 101(A)(2).

¹⁴² CWA § 101(A)(3).

¹⁴³ CWA § 304(a)(1)(A), (B) (emphasis added). *See also id.* § 304(a)(2) (“EPA “shall develop and publish within one year after October 18, 1972 (and from time to time thereafter revise) information (A) on the factors necessary to restore and maintain the chemical, physical, and biological integrity of all navigable waters . . . (B) on the factors necessary for the protection and propagation of shellfish, fish, and wildlife for classes and categories of receiving water[.]” (emphasis added).

¹⁴⁴ 40 C.F.R. § 131.10(a).

¹⁴⁵ *Id.*

EPA has noted that “[w]hile the [1985] Guidelines [for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses] remain the primary instrument the Agency uses to meet its broad objectives for the development of [aquatic life criteria], there have been many advances in aquatic sciences, aquatic and wildlife toxicology, population modeling, and ecological risk assessment that are relevant to deriving [aquatic life criteria].¹⁴⁶

G. Congress Authorized and Required EPA to Develop Recommended 304(a) Sediment Quality Criteria

The CWA requires EPA to develop recommended 304(a) sediment quality criteria and ensure that states adopt such criteria, as well as to use narrative criteria to preclude adverse toxic effects caused by the accumulation of toxic chemicals in sediments. Like wildlife impacts, evidence of the adverse impacts of pollutants in sediment, including the tendency of those pollutants to bioaccumulate and bio-magnify due to their persistence in sediments, warrants listing of sediment pollutants independent of other water body impacts. As discussed *supra*, EPA is directed to develop and publish recommended criteria for the states,

accurately reflecting the latest scientific knowledge (A) on the kind and extent of all identifiable effects on health and welfare including, but not limited to, plankton, fish, shellfish, wildlife, plant life, shorelines, beaches, esthetics, and recreation which may be expected from the presence of pollutants in any body of water . . .; (B) on the concentration and dispersal of pollutants, or their byproducts, through biological, physical, and chemical processes; and (C) on the effects of pollutants on biological community diversity, productivity, and stability, including information on the rates of organic and inorganic sedimentation for varying types of receiving waters.¹⁴⁷

¹⁴⁶ EPA, *White Paper Aquatic Life Criteria for Contaminants of Emerging Concern, Part I, General Challenges and Recommendations, Prepared by the OW/ORD Emerging Contaminants Workgroup* (June 03, 2008) (hereinafter “EPA CEC White Paper”) at 1 (emphasis added).

¹⁴⁷ CWA § 304(a)(1) (emphasis added).

Some toxic pollutants are expected to disperse and then concentrate in sediments of depositional areas of water bodies wherein they enter the food web and cause effects to the designated uses of flora and fauna. EPA is, therefore, required to develop sediment quality criteria to protect against these processes insofar as they harm designated uses. In turn, states are required to adopt criteria for which EPA has published 304(a) recommended criteria but specifically for pollutants on the Toxic Pollutants Lists.¹⁴⁸

The need for sediment quality criteria is explicitly recognized regarding sediments contaminated with toxic chemicals in the Great Lakes. There, Congress directed EPA to study and conduct a demonstration project “relating to the control and removal of toxic pollutants in the Great Lakes, with emphasis on the removal of toxic pollutants from bottom sediments.”¹⁴⁹ In conducting the assessments of the locations chosen for these projects, EPA was required to announce “the numerical standard of protection intended to be achieved at each location.”¹⁵⁰ Finally, EPA was required, by November 1992, to

publish information concerning the public health and environmental consequences of contaminants in Great Lakes sediment. Information published pursuant to this subparagraph shall include specific numerical limits to protect health, aquatic life, and wildlife from the bioaccumulation of toxics.¹⁵¹

The Great Lakes Initiative (“GLI”) regulations, codified at 40 C.F.R. Part 132, address Great Lakes sediment contamination in two ways, to account for it in developing biota-sediment accumulation factors,¹⁵² and in the development of TMDLs for toxic contaminants that

¹⁴⁸ CWA § 303(c)(2)(B) (This requirement applies to pollutants “the discharge or presence of which in the affected waters could reasonably be expected to interfere with those designated uses adopted by the State, as necessary to support such designated uses.”)

¹⁴⁹ CWA § 118(c)(7)(A).

¹⁵⁰ *Id.* § 118(c)(7)(B)(ii).

¹⁵¹ *Id.* § 118(c)(7)(C).

¹⁵² *See, e.g.* Final Water Quality Guidance for the Great Lakes System, 60 Fed. Reg. 15366, 15374 (March 23, 1995).

shall reflect, where appropriate and where sufficient data are available, contributions to the water column from sediments inside and outside of any applicable mixing zones. TMDLs shall be sufficiently stringent so as to prevent accumulation of the pollutant of concern in sediments to levels injurious to designated or existing uses, human health, wildlife and aquatic life.¹⁵³

EPA does not appear to have developed the required sediment criteria.

III. TOXIC CONTAMINATION PLAGUES THE NATION'S WATERS

EPA is charged with the responsibility to identify toxic pollutants that warrant inclusion on the Toxic Pollutants Lists based on the wide range of data and scientific information available from multiple sources, including state and federal water quality monitoring and assessment, discharge monitoring data, and academic and independent research, among other sources. The following documents the type and range of available information demonstrating that toxic pollution is a significant water quality problem that justifies a long overdue expansion of the Toxic Pollutant Lists. It is not, however, intended to be exhaustive. Rather, it illustrates the kind of analysis EPA should undertake on an ongoing basis.

In 1992—30 years ago—in explaining its decision to promulgate toxic criteria for certain states that had failed to do so on their own, EPA wrote about the urgent problem of toxic pollution in the nation's waters:

[C]ontrol of toxic pollutants in surface waters is an important priority to achieve the Clean Water Act's goals and objectives. The most recent National Water Quality Inventory indicates that one-third of monitored river miles, lake acres, and coastal waters have elevated levels of toxics. Forty-seven States and Territories have reported elevated levels of toxic pollutants in fish tissues. States have issued a total of 586 fishing advisories and 135 bans, attributed mostly to industrial discharges and land disposal.¹⁵⁴

¹⁵³ *Id.* at 15417.

¹⁵⁴ NTR Rules, 57 Fed Reg 60848 (Dec. 22, 1992).

The next year, in 1993, Dr. Theo Colburn and her colleagues wrote about the mounting evidence that numerous chemicals were causing endocrine disruption, an endpoint not considered by regulators such as EPA:

The deleterious effects of endocrine-disrupting chemicals in the environment on the reproductive success of wildlife populations have been documented; this is not an isolated problem, and today many wildlife populations are at risk. At present, no coherent policy has been articulated to remedy this problem. This is due in part to the lack of knowledge concerning which of the many chemicals present in the environment are responsible for endocrine-disrupting effects. Regulatory agencies should recognize that the current endpoints of most test to assess the risk of pesticides and other pollutants (carcinogenicity, acute toxicity, and immediate mutagenicity) have led to the misconception that these chemicals do not pose a threat to the health of wildlife, domestic animals, or humans.¹⁵⁵

Although Congress amended the CWA in 1987 to add additional requirements to address the failure of EPA and states to adequately control toxic pollution, these new provisions worked only to a limited extent. Since then, toxic pollution has continued to contaminate water and sediments, and to enter, bioaccumulate, and recirculate in food webs nationwide. As a result of numerous loopholes in the water quality-based aspects of the NPDES permitting program,¹⁵⁶ EPA's decades-long failure to update technology-based controls,¹⁵⁷ the agency's failure to keep up with scientific understanding of what should be on the list of toxic and priority pollutants,¹⁵⁸ and the general failure of state nonpoint source control programs, toxics continue to plague the nation's waters. As one example, despite major flaws in the methodology used to generate listings of waters impaired by toxics in the states' CWA Section 303(d) lists, *see infra* section VI, EPA's database of 303(d) listed waters impaired by toxic contaminants nationwide yields

¹⁵⁵ Theo Colburn, *et al.*, *Developmental Effects of Endocrine-Disrupting Chemicals in Wildlife and Humans*, 101 *Environmental Health Perspectives* at 378 (Oct. 1993).

¹⁵⁶ *See infra*, section VI.

¹⁵⁷ *See infra*, section V.

¹⁵⁸ *See infra*, section VII.

86,166 segment/parameters identified based on current monitoring, current state and EPA listing methodologies, and current water quality standards, all of which severely limit the waters and pollutants that are placed on this list. Updating the Toxic Pollutants Lists to reflect the new or newly identified pollutants plaguing the nation's waters will improve implementation of each of these programs.

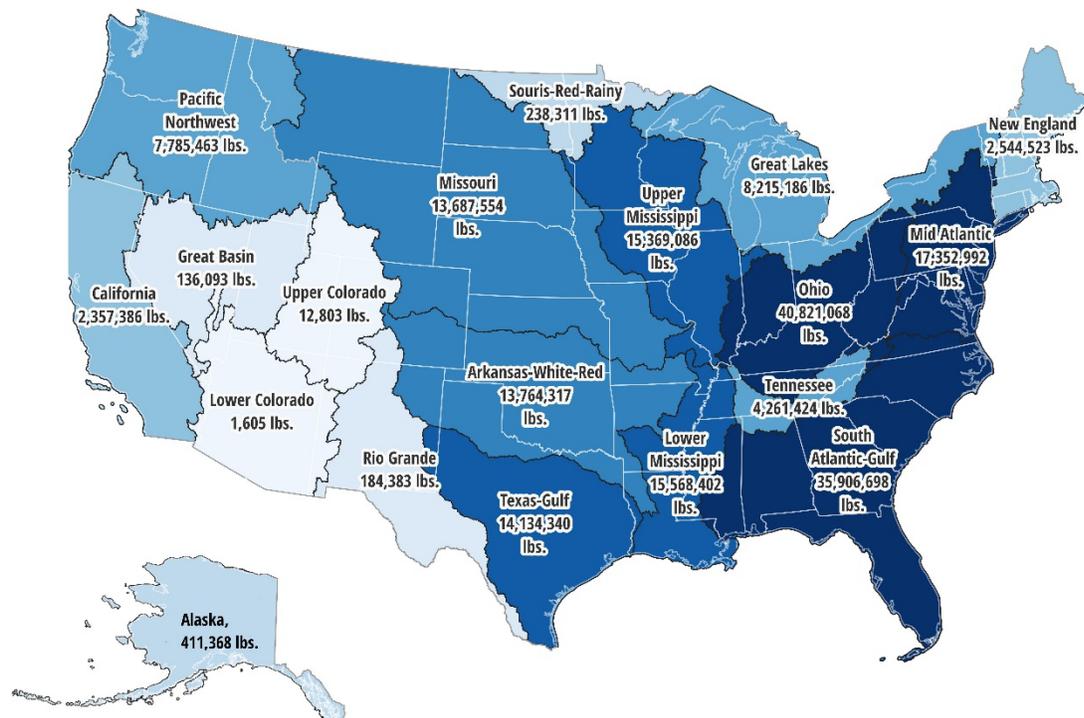
In this section we capture just a tiny fraction of the information available on toxic contaminants in the nation's waters, aquatic food web, and sediments and their effects on human health and species. In sub-section A, we look at the information about toxic loading to U.S. waters and we provide the results of studies looking at national water quality as well as more focused studies on the Lower Columbia River, Puget Sound, and the Piedmont region in the nation's southeastern states (presented as examples for other regions of the country as well). Sub-section B looks at some of the adverse effects of toxic contamination in the nation's waters including ecosystem exposures to contaminant mixtures, contaminants that cause intersex conditions in aquatic species, sublethal effects to salmonids, and adverse impacts to marine mammals and other aquatic-dependent species, again as examples of the kinds of impacts caused by toxic water pollutants.

A. The Quality of the Nation's Waters as Measured by Toxic Loading and Monitoring

1. Toxic Releases to Waters of the United States

While often not reflected in the states' and EPA's 303(d) lists of impaired waters, reporting of toxic chemicals discharged to waterbodies in the U.S. provides an indication of what those lists are missing. A report issued on the 50th anniversary of the passage of the CWA used data from EPA's Toxics Release Inventory ("TRI") to demonstrate that industrial facilities

(excluding exempt sources such as oil and gas extraction facilities) released at least 193.6 million pounds of toxic substances into U.S. waterways in 2020, 90 percent of which are nitrate compounds.¹⁵⁹ While these discharges are not equally distributed across the country, as the following map demonstrates, neither are toxic discharges merely a regional problem:¹⁶⁰



Similarly, as not all toxic chemicals are equally toxic, use of EPA’s Risk-Screening Environmental Indicators (“RSEI”) tool—which assigns weights to chemical releases based on their toxicity to humans—demonstrates a highly unequal distribution of toxic effects of these discharged chemicals, highlighting the potential of this pattern to perpetuate environmental injustice on communities of people who depend on local waters for drinking water, fish and shellfish, and recreation, as discussed in section IV of this petition:¹⁶¹

¹⁵⁹ Environment America *et al.*, *Wasting our Waterways: Toxic pollution and the unfulfilled promise of the Clean Water Act* (Sept. 2022) at 1.

¹⁶⁰ *Id.* at 2 (Fig. ES-1, Toxic releases to watershed regions nation, 2020).

¹⁶¹ *Id.* at 9.

TABLE 2. TOP 10 STATES BY TOXICITY-WEIGHTED CHEMICALS RELEASED, 2020

State or territory	Toxicity-weighted chemicals released (lbs. eq.)
Wisconsin	45,122,237,956
Texas	39,673,055,922
Virginia	31,982,111,294
Louisiana	10,853,487,483
Indiana	7,319,010,165
West Virginia	4,832,813,087
Ohio	3,441,960,029
South Carolina	1,856,799,384
Pennsylvania	1,670,004,499
Alabama	1,667,496,165

Even at the watershed level, the distribution is highly skewed, again highlighting its potential for perpetuating environmental injustice:¹⁶²

TABLE 5. TOP 10 WATERSHEDS BY TOXICITY-WEIGHTED CHEMICALS RELEASED, 2020

Receiving watershed	State(s) containing watershed	Toxicity-weighted chemicals released (lbs. eq.)
Manitowoc-Sheboygan	WI	45,021,201,876
Austin-Oyster	TX	38,320,027,272
Upper New	NC, TN, VA	31,725,710,405
Lake Maurepas	LA	8,889,410,342
Middle Wabash-Little Vermillion	IL, IN	6,188,334,032
Raccoon-Symmes	KY, OH, WV	4,442,261,040
Upper Ohio-Wheeling	OH, PA, WV	1,684,294,112
Jordan	UT	1,466,638,353
Cooper	SC	1,463,567,312
Upper Ocmulgee	GA	1,020,772,866

¹⁶² *Id.* at 11.

Likewise, the distribution of toxic chemicals in water that have human health effects of cancer, reproductive problems, and developmental effects are similarly concentrated:¹⁶³

TABLE 7. TOP 10 STATES BY CANCER-CAUSING TOXIC CHEMICAL RELEASES, 2020

State or territory	Cancer-causing chemicals released (lbs.)
South Carolina	130,579
Texas	123,257
Alabama	106,122
Louisiana	71,252
West Virginia	53,861
Indiana	52,207
Georgia	40,777
North Carolina	40,080
Florida	36,992
Tennessee	35,872

TABLE 9. TOP 10 STATES BY RELEASES OF REPRODUCTIVE TOXICS, 2020

State or territory	Reproductive toxics released (lbs.)
Texas	28,333
Indiana	27,088
Pennsylvania	22,621
Louisiana	18,545
Alabama	12,194
Tennessee	10,723
Illinois	8,888
Kentucky	6,834
West Virginia	6,823
Virginia	6,785

¹⁶³ *Id.* at 16, 17, 18.

TABLE 11. TOP 10 STATES BY DEVELOPMENTAL TOXICS RELEASED, 2020

State or territory	Developmental toxics released (lbs.)
North Carolina	602,927
Wisconsin	595,112
Alabama	406,729
Washington	387,509
Mississippi	345,666
South Carolina	325,355
Kentucky	319,661
Texas	298,076
Louisiana	266,377
Georgia	189,610

In addition to direct discharges of toxic contaminants captured by these data is polluted runoff from nonpoint sources, such as farming and logging that use large amounts of pesticides and other chemicals, and other human activities that are largely unregulated. Failure to include all of the pollutants captured by these data in the Toxic Pollutants Lists makes it difficult or impossible for EPA and the states to meet the stated goals of the CWA to eliminate toxic chemicals in toxic amounts.

2. Water Quality of the Nation’s Waters as Measured in Ambient Water and Animal Tissue

In the first nationwide reconnaissance of pharmaceuticals, hormones, and other organic wastewater contaminants in water resources, in 1999–2000, the U.S. Geological Survey (“USGS”) found the compounds in 80 percent of 139 streams across 30 states.¹⁶⁴ Eight-two of the 95 compounds evaluated were found during the study, representing a “wide range of

¹⁶⁴ Dana W. Kolpin, *et al.*, *Pharmaceuticals, Hormones, and Other Organic Wastewater Contaminants in U.S. Streams, 1999–2000: A National Reconnaissance*, 36 *Environ. Sci. Technol.* 1202-1222 (2002) (hereinafter “Pharmaceuticals National Reconnaissance”), available at <https://pubs.acs.org/doi/pdf/10.1021/es011055j>.

residential, industrial, and agricultural origins.” The most prevalent were: coprostanol, cholesterol, N,N-diethyltoluamide, caffeine, triclosan, tri(2-chloroethyl) phosphate, and 4-nonylphenol.¹⁶⁵ Seventy-five percent of the streams sampled had more than one compound identified, suggesting “the toxicity of the target compounds should include not only the individual [organic wastewater contaminants] OWCs but also mixtures of these compounds,” including the potential for additive or synergistic effects.¹⁶⁶ USGS also pointed out the limits of its focus on water column concentrations and specific compounds:

Select OWCs may be hydrophobic and thus may be more likely to be present in stream sediments than in streamwater. For example, the low frequency of detection for the tetracycline (chlortetracycline, doxycycline, oxytetracycline, tetracycline) and quinolone (ciprofloxacin, enrofloxacin, norfloxacin, sarafloxacin) antibiotics is not unexpected given their apparent affinity for sorption to sediment. In addition, select OWCs may be degrading into new, more persistent compounds that could be transported into the environment instead of (or in addition to) their associated parent compound.¹⁶⁷

The report documented that “[m]any compounds . . . do not have such [criteria] guidelines established.”¹⁶⁸

A subsequent nationwide survey of 38 streams conducted by USGS and EPA in 2012–2014 looked at chemical-mixture exposures to aquatic life.¹⁶⁹ The study concluded that the results present “aquatic health concerns,” as described in its abstract:

Surface water from 38 streams nationwide was assessed using 14 target-organic methods (719 compounds). Designed-bioactive anthropogenic contaminants (biocides, pharmaceuticals) comprised 57% of 406 organics detected at least once. The 10 most-frequently detected anthropogenic-organics included eight pesticides (desulfinylfipronil, AMPA, chlorpyrifos, dieldrin, metolachlor, atrazine, CIAT,

¹⁶⁵ *Id.* at 1202.

¹⁶⁶ *Id.* at 1210.

¹⁶⁷ *Id.* at 1210 (internal citations omitted).

¹⁶⁸ *Id.* at 1204–1205 (Table 1), 1208.

¹⁶⁹ Paul M. Bradley, *et al.*, *Expanded Target-Chemical Analysis Reveals Extensive Mixed-Organic-Contaminant Exposure in U.S. Streams*, 51 *Environ. Sci. Technol.* 4792-4802 (2017) (hereinafter “EPA/USGS 2017 National Study”) (internal citations omitted).

glyphosate) and two pharmaceuticals (caffeine, metformin) with detection frequencies ranging 66–84% of all sites. Detected contaminant concentrations varied from less than 1 ng L⁻¹ to greater than 10 µg L⁻¹, with 77 and 278 having median detected concentrations greater than 100 ng L⁻¹ and 10 ng L⁻¹, respectively. Cumulative detections and concentrations ranged 4–161 compounds (median 70) and 8.5–102 847 ng L⁻¹, respectively, and correlated significantly with wastewater discharge, watershed development, and toxic release inventory metrics. Log 10 concentrations of widely monitored HHCB, triclosan, and carbamazepine explained 71–82% of the variability in the total number of compounds detected (linear regression; *p*-values: < 0.001–0.012), providing a statistical inference tool for unmonitored contaminants. Due to multiple modes of action, high bioactivity, bioresistance, and direct environment application (pesticides), designed-bioactive organics (median 41 per site at µg L⁻¹ cumulative concentrations) in developed watersheds present aquatic health concerns, given their acknowledged potential for sublethal effects to sensitive species and lifecycle stages at low ng L⁻¹.

A summary of these results is shown in this map:

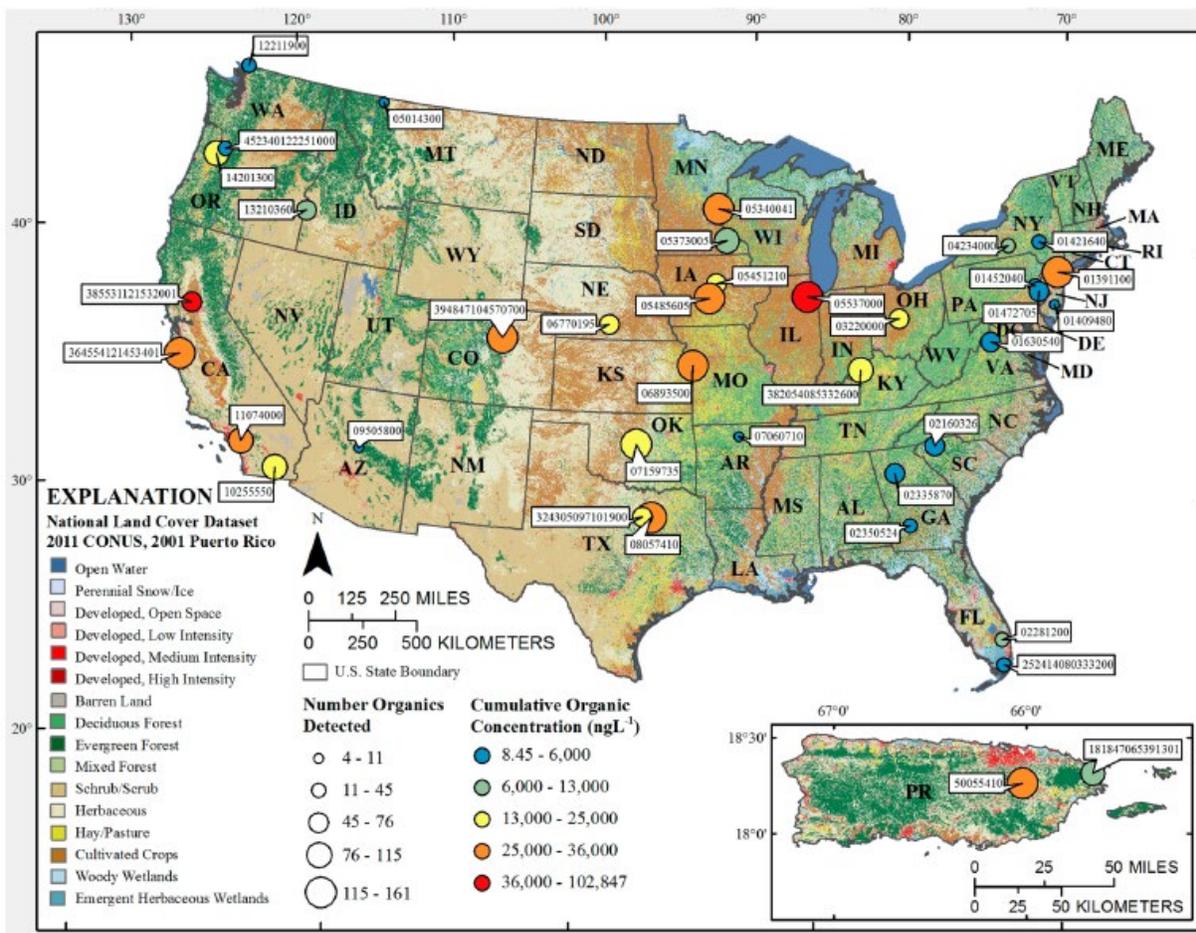


Figure 1. Total numbers and cumulative concentrations (ng L^{-1}) of organic analytes detected in water during 2012–2014 sampling of 38 stream sites (SI Table S1) across the nation. Numeric labels indicate USGS NWIS⁹² stream station IDs.

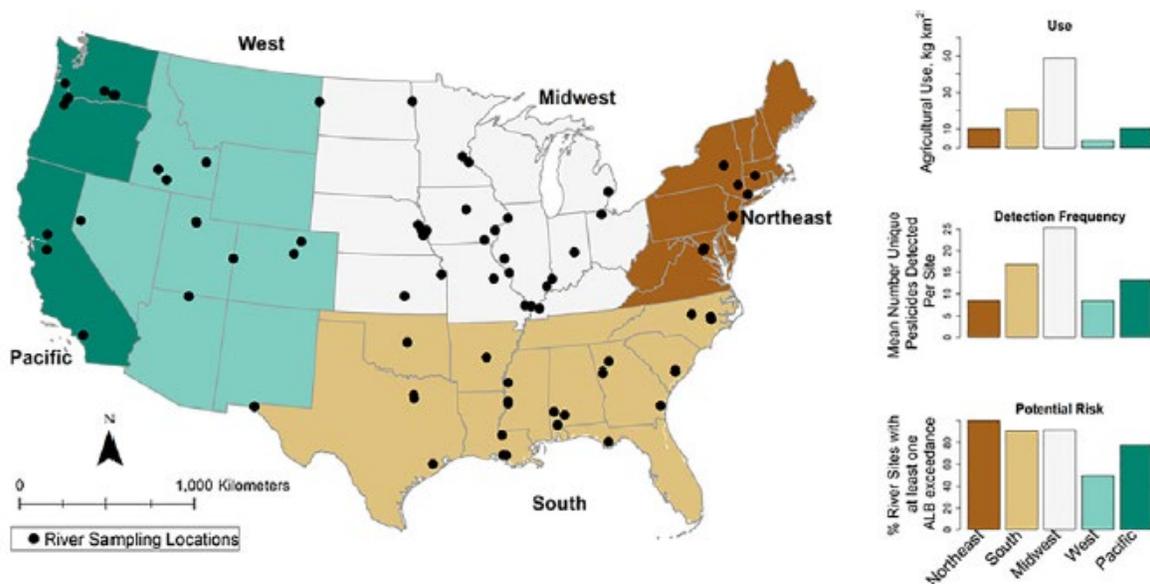
Another recently published nationwide evaluation of toxics by the USGS focused on pesticides, using water samples from 74 sites and quantifying 221 pesticide concentrations, concluded that “1) pesticides persist in environments beyond the site of application and expected period of use, and 2) the potential toxicity of pesticides to aquatic life is pervasive in surface waters.”¹⁷⁰ Detection of parent and degradate compounds was “widespread” with

[a]t least one pesticide . . . detected at 71 of the 74 sites, on average 17 unique pesticides were detected at every site, and 105 of the 221 study pesticides were detected at least once. 75% of the detected pesticides had not been measured in

¹⁷⁰ Sarah M. Stackpoole, *et al.*, *Pesticides in US Rivers: Regional differences in use, occurrence, and environmental toxicity, 2013 to 2017*, 787 *Science of the Total Environment* 147147 (2021) at 9.

previous USGS national-scale assessments (Stone et al., 2014a). This analytical method, which enabled the assessment of more pesticides with increased sensitivity, has improved our knowledge about the breadth of compounds that contribute to surface water contamination in the CONUS.¹⁷¹

At a regional level pesticide use was markedly different, as illustrated by the following graphic:¹⁷²



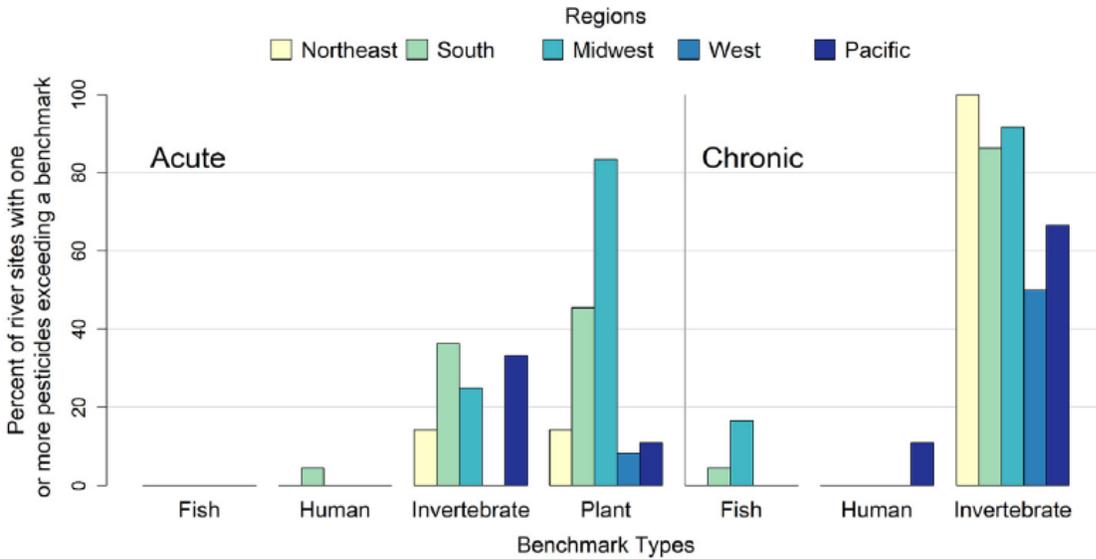
While the USGS found that agricultural pesticide use was a “key driver of surface water pesticide concentrations,” they cautioned that for insecticides, contributions from urban areas are of concern, noting that “[e]levated surface water concentrations, particularly of insecticides like fipronil, diazinon and carbaryl have been documented in rivers draining watersheds <80 km² or smaller wadeable streams draining primarily urban settings[.]”¹⁷³ Moreover, at least 50 percent of sites within a region had exceedances of at least one chronic aquatic life benchmark:¹⁷⁴

¹⁷¹ *Id.* at 4–5. “Five of the 17 pesticides with exceedances, including chlorimuronethyl, diuron, halosulfuronmethyl, dichlorvos, and imidacloprid, were new additions to the USGS analytical method for the time period 2013 to 2017.” *Id.* at 6.

¹⁷² *Id.* at 1.

¹⁷³ *Id.* at 6.

¹⁷⁴ *Id.* at 6, 7. Nine herbicides and eight insecticides contributed to exceedances of benchmarks. *Id.* at 6.



The authors explained that the results reflect some herbicides with numerous benchmark exceedances (e.g., acetochlor, atrazine, and metolachlor) were heavily used, found at elevated concentrations, and also exceeded benchmarks across a range of taxa. Insecticides (e.g., dichlorvos, fipronil, and imidacloprid), on the other hand, were less used, found at lower concentrations, but are toxic to invertebrates at very low concentrations.¹⁷⁵

In contrast to the USGS evaluation, one of EPA’s most recent national surveys shows the agency’s evaluation of toxic contaminants in surface water to be focused only on human health.¹⁷⁶ The results demonstrated 24 percent 24% (25,119 miles) of the sampled river miles (105,989 miles) had fish above a mercury benchmark; 40 percent (24,583 miles) of river miles (61,305 miles) had fish exceeding a PCB cancer benchmark, and three percent (3,490 miles) of river miles (102,652 miles) had fish exceeding a PFOS benchmark.¹⁷⁷

a. Columbia River Basin

¹⁷⁵ *Id.* at 7.

¹⁷⁶ EPA, *National Rivers and Streams Assessment 2013–2014: A Collaborative Survey* (Dec. 2020) at 14 (contaminants in fish tissue as a human health indicator only), 31 (fish samples were fillets only).

¹⁷⁷ *Id.* at 33, fig. 4.4.

More targeted regional studies have demonstrated similar results. In the Lower Columbia River, extensive studies beginning in 1989—over 30 years ago— “demonstrated that water and sediment in the lower Columbia and its tributaries have levels of toxic contamination that are harmful to fish and wildlife.”¹⁷⁸ Its findings included that dioxins, furans, metals, PCBs, PAHs, and pesticides—including both the banned DDT and its metabolites and current use pesticides (simazine, atrazine, chlorpyrifos, metalochlor, diazinon, carbaryl)—exceeded acceptable levels and that some bioaccumulative contaminants were causing reproductive abnormalities in river otter and mink, impaired reproduction in bald eagles, and posing a human health concern.¹⁷⁹ Subsequent studies confirmed these findings and identified copper, cadmium, zinc, dioxins, furans, PCBs, dieldrin, lindane, chlordane, DDT, and PAHs in bed and suspended sediment.¹⁸⁰ Banned pesticides were demonstrated to be moving up the food chain to fish predators such as osprey.¹⁸¹ Later, in 2000, PBDEs “were detected in mountain whitefish in the upper Columbia River Basin at concentrations of up to 72 parts per billion—12 times the concentrations measured in 1992[.]”¹⁸²

In 2007, a new multi-media study looking at more pesticides, PPCPs, and wastewater compounds in the Lower Columbia River was published to correlate water and sediment quality and salmon sampling data.¹⁸³ The most frequently detected pesticides in filtered water were:

¹⁷⁸ Lower Columbia River Estuary Partnership, *Lower Columbia River and Estuary Ecosystem Monitoring, Water Quality and Salmon Sampling Report* (2007) (hereinafter “2007 Columbia Sampling”), available at https://www.estuarypartnership.org/sites/default/files/resource_files/WaterSalmonReport.pdf at 1, citing Tetra Tech, *The Health of the River 1990 – 1996 Integrated Technical Report* (May 20, 1996) (water samples taken from 20 sites, fish samples from 90 sites, and sediment samples from 300 sites).

¹⁷⁹ 2007 Columbia Sampling, *supra* n. 178 at 5, 6, 7.

¹⁸⁰ *Id.* at 6.

¹⁸¹ *Id.* at 7.

¹⁸² *Id.* at 7.

¹⁸³ *Id.* at 32.

atrazine, simazine, metochlor, CIAT (deethylatrazine), EPTC, DCPA, and diuron.¹⁸⁴ The most frequently detected pharmaceuticals in filtered water were: ahhydro-erythromycin, trimethoprim, acetaminophen, diphenhydramine, tylosin.¹⁸⁵ Other wastewater compounds included: caffeine, HHCB, bisphenol A, anthraquinone, DEET, and tri(2-chloroethyl)phosphate.¹⁸⁶ In suspended sediment, PCBs, and PBDEs were frequently detected.¹⁸⁷ Toxic contaminants found in semi-permeable membrane devices included PCBs, PAHs, and PBDEs.¹⁸⁸

This study underscored the cumulative effect of toxic contamination in a river basin. It concluded that locations “downstream of the lower river’s major population centers . . . are affected by releases of toxic contaminants associated with urban and industrial facilities.”¹⁸⁹ More important, the concentrations of toxics in juvenile salmon from upstream waters increased as those stocks moved downstream: “In the end, juveniles from Snake River and Middle Columbia stocks had some of the highest levels of PCBs and PBDEs, respectively. . . . [T]his study suggests the lower river and estuary may be a significant source of toxic contaminants for juveniles from upriver stocks.”¹⁹⁰ Moreover, and not surprisingly, “DDT was measured at relatively high levels in salmon samples (both stomach contents and body tissue) but was detected during water quality sampling only a few times and at very low concentrations.”¹⁹¹ The authors concluded “that DDT concentrations in predators such as salmon can be orders of

¹⁸⁴ *Id.* at 36.

¹⁸⁵ *Id.* at 37.

¹⁸⁶ *Id.* at 37.

¹⁸⁷ *Id.* at 38.

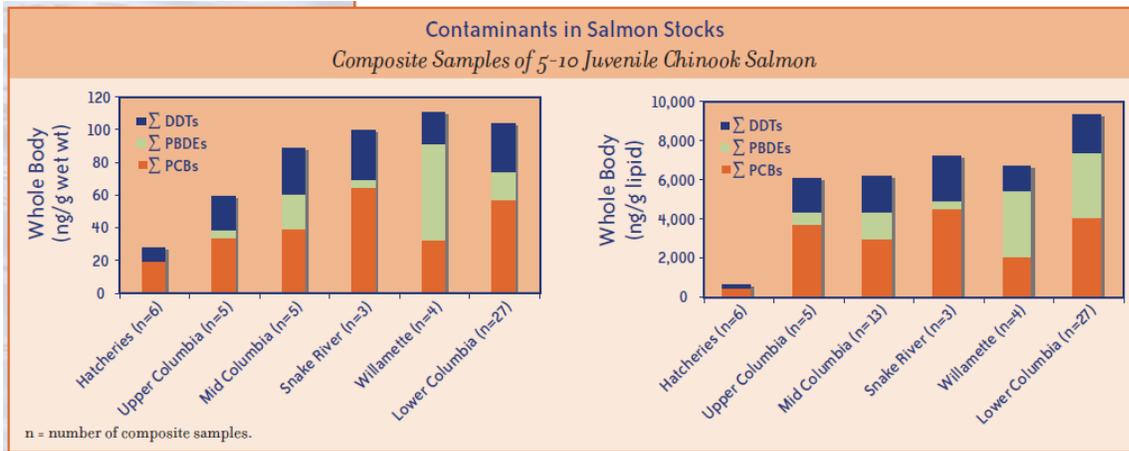
¹⁸⁸ *Id.* at 39.

¹⁸⁹ *Id.* at 53.

¹⁹⁰ *Id.*

¹⁹¹ *Id.*

magnitude higher than concentrations in the surrounding environment.”¹⁹² The effect on whole body tissue and lipid content is illustrated by the following graph:¹⁹³



A subsequent study published in 2012 by the USGS to assess contaminant loading to the Columbia River from sewage treatment plants and urban stormwater looked at anthropogenic organic compounds, pharmaceuticals, PCBs, PBDEs, organochlorine or legacy compounds, currently used pesticides, mercury, and estrogenicity.¹⁹⁴ Of 210 compounds analyzed from nine sewage treatment plant effluents, 112 (53 percent) were detected and the detection rate for most compound classes exceeded 80 percent.¹⁹⁵ A complex mixture of compounds was detected in stormwater runoff, with detections of 114 (58 percent) of the 195 compounds analyzed.¹⁹⁶ The USGS concluded:

Although there are variations in the individual composition of the samples for each plant, there are many similarities in the frequency of detections across the plants. For example, the detection frequency for flame retardants at all plants was

¹⁹² *Id.* at 54.

¹⁹³ *Id.*

¹⁹⁴ Jennifer L. Morace, *Reconnaissance of Contaminants in Selected Wastewater-Treatment-Plant Effluent and Stormwater Runoff Entering the Columbia River, Columbia River Basin, Washington and Oregon, 2008–10*, Scientific Investigations Report 2012–5068 (2012) (hereinafter “Columbia Reconnaissance”) at 1.

¹⁹⁵ *Id.*

¹⁹⁶ *Id.*

65–82 percent. Similarly, personal care products, pesticides, steroids, pharmaceuticals, and miscellaneous compounds showed similar detection frequencies amongst the plants. These similarities illustrate that although there are differences between the plants based on location, population, treatment type, and plant size, many of the results are similar.¹⁹⁷

The results are illustrated by the following graph:¹⁹⁸

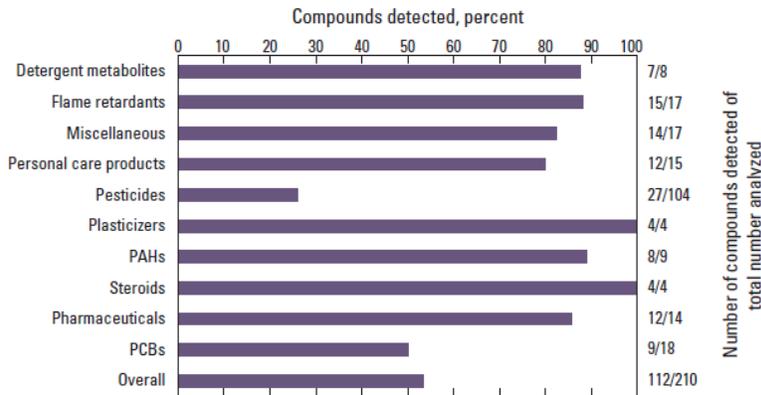


Figure 3. Percentage of compounds detected in wastewater-treatment-plant effluent, Columbia River Basin, Washington and Oregon, 2008–09.

b. Puget Sound

The Washington Department of Ecology has highlighted high levels of lead, cadmium, tributyl tins, copper, mercury, arsenic, PCBs, PAHs, dioxins and furans, pesticides, phthalate esters, polybrominated diphenyl ethers (PBDEs), hormone disrupting chemicals (Bisphenol A), petroleum & petroleum by-products, and pharmaceuticals in Puget Sound waters.¹⁹⁹ Not only is the scope of toxic chemicals in Washington’s waters sweeping but the levels of these chemicals demonstrate the high body burdens in Puget Sound as compared to other locations of salmonids in the region. For example, Ecology reports that “Puget Sound Chinook salmon fillets are almost three times more contaminated than fillets of Chinook salmon from other Pacific West Coast

¹⁹⁷ *Id.* at 24.

¹⁹⁸ *Id.* at 27.

¹⁹⁹ Ecology, *Fish Consumption Rates Technical Support Document: A Review of Data and Information about Fish Consumption in Washington, Version 2.0 Final C-11*(Jan. 2013) (hereinafter “Final FCR Report”) available at <https://fortress.wa.gov/ecy/publications/publications/1209058.pdf>.

areas.”²⁰⁰

In 2011, the Washington Department of Ecology and King County, Washington, published an assessment of the sources and loads of a limited number of toxic chemicals in the marine waters of Puget Sound.²⁰¹ The study’s hazard evaluation identified the following chemicals as the most likely to be found at “concentrations where effects are documented or at levels above criteria used to protect aquatic organisms and consumers of aquatic organisms”:

copper, mercury, PCBs, dioxins and furans, DDT and its metabolites, PAHs, and bis(2-ethylhexyl) phthalate (DEHP).²⁰² The study determined that sewage treatment plants are a major source of PBDEs to Puget Sound, as shown by this graph:²⁰³

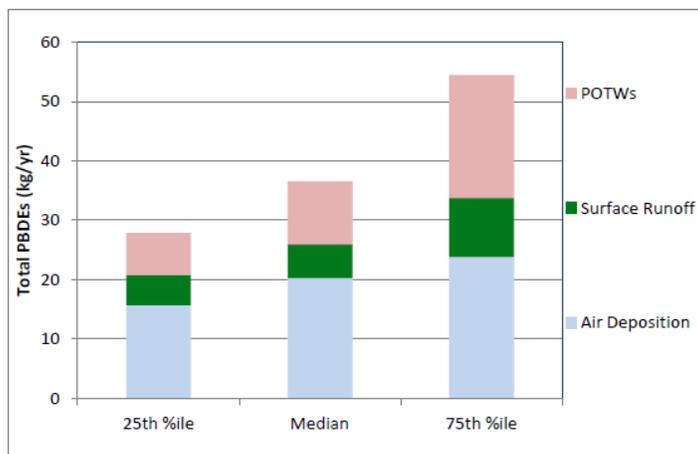


Figure 27. Total PBDE Loads to Puget Sound as the Sum of Major Delivery Pathways.

This should not have been a surprise given the much earlier work by state and federal fish and wildlife agencies. For example, in 2004, the Washington Department of Fish and Wildlife

²⁰⁰ *Id.*

²⁰¹ Washington Department of Ecology/King County, *Assessment of Selected Toxic Chemicals in the Puget Sound Basin, 2007-2011* (Nov. 2011) (hereinafter “Puget Sound Assessment”). (The study noted that “[w]hile there is general consensus that a much larger number of potentially harmful chemicals are released to Puget Sound,” studying them was beyond the scope of the project. *Id.* at 12.)

²⁰² *Id.* at 9.

²⁰³ *Id.* at 104.

(“WDFW”) and National Marine Fisheries Service (“NMFS”) showed that PBDEs were bioaccumulating in predator fish, in highest concentrations in urban areas, and that “the longer chinook reside in Puget Sound, the higher their PBDE concentration.”²⁰⁴

In 2008, WDFW reflected on 30 years of monitoring persistent bioaccumulative toxics in Puget Sound, focused on PCBs.²⁰⁵ The results indicated some decline in PCB exposure to English sole in urban areas but no decline in the 15-year period from 1990 to 2005, leading to the hypothesis that PCBs are being biotically recycled in the food web.²⁰⁶ Another paper concluded that the PCB body burdens of Chinook salmon were accumulated primarily in the marine habitats of Puget Sound.²⁰⁷ A subsequent study of juvenile Chinook salmon—evaluating whole bodies for persistent organic pollutants (“POPs”), stomach contents for PAHs, and gills for metals in fish from estuaries, nearshore marine shorelines and offshore habitats—supported the “hypothesis that salmon residing and feeding in the more urbanized and industrialized environments are exposed to higher concentrations of contaminants than those in less developed habitats.”²⁰⁸ However, “[a]s juvenile Chinook salmon migrated from river systems to offshore waters of Puget Sound, all fish continued to accumulate substantial amounts of POPs . . . and after four months of feeding in the offshore habitats, fish from all basins had uniform concentrations of POPs[.]”²⁰⁹ WDFW scientists determined that the evidence indicated “the

²⁰⁴ Sandra O’Neill, *et al.*, *Concentrations of Polybrominated Diphenyl Ethers (PBDEs) in Fish from Puget Sound, WA, USA* (2004).

²⁰⁵ James West and Sandra O’Neill, *Thirty years of Persistent Bioaccumulative Toxics in Puget Sound: time trends of PCBs and PBDE flame retardants in three fish species* (2008).

²⁰⁶ *Id.* at 5.

²⁰⁷ Sandra O’Neill and James West, *Marine Distribution, Life History Traits, and the Accumulation of Polychlorinated Biphenyls in Chinook Salmon from Puget Sound, Washington*, 138 *Transactions of the American Fisheries Society* 616-632 (2009) at 1.

²⁰⁸ Sandra O’Neill, *et al.*, *Toxic contaminants in juvenile Chinook salmon (*Oncorhynchus tshawytscha*) migrating through estuary, nearshore and offshore habitats of Puget Sound* (Oct. 2015).

²⁰⁹ *Id.* at xiv-xv.

offshore was more contaminated than the undeveloped river systems habitats but less contaminated than the developed river systems habitats.”²¹⁰ Copper and lead were also elevated in gill tissues of fish from the more developed nearshore marine habitats.²¹¹ The study concluded:

A significant proportion of Puget Sound Chinook salmon are at risk for some type of health impairment due to contaminant exposure. Approximately one third of the juvenile Chinook salmon sampled from Puget Sound, regardless of the degree of development, had contaminant concentrations associated with adverse effects. Levels of TPCBs, Σ_{11} PBDEs in whole body tissue samples of salmon from the Snohomish, Green/Duwamish and Hylebos/Puyallup river systems, and TPCBs in fish from the offshore habitat of the Whidbey and Central Basins were high enough to potentially cause adverse effects, including reduction in growth, disease resistance, and altered hormone and protein levels. Additionally, Σ_{42} PAHs in stomach contents were elevated in salmon from the nearshore habitats of the Snohomish and Green/Duwamish systems, at concentrations high enough to potentially affect growth and alter plasma chemistry and lipid class profiles. Elevated concentrations of copper and lead were also measured in gills tissue of salmon from developed nearshore marine habitat, however, the potential effects on salmon health are unknown.

* * *

These findings suggest that controlling the initial release of contaminants to the environment may be necessary to protect offshore habitats and their associated pelagic species, including Chinook salmon.²¹²

These results are illustrated by the graphs below:^{213, 214}

²¹⁰ *Id.* at xv.

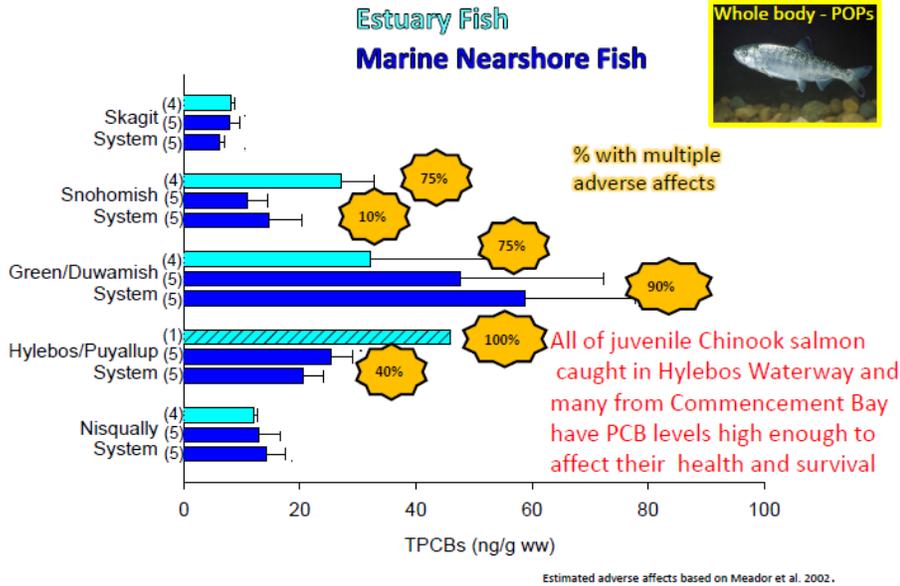
²¹¹ *Id.* at xv.

²¹² *Id.* at 75–76.

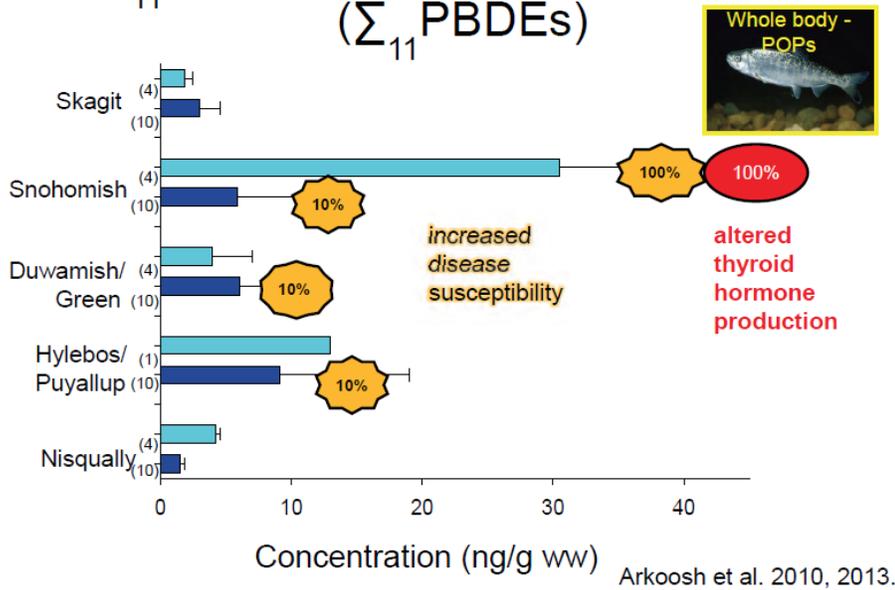
²¹³ Sandra O’Neill, *et al.*, *Contaminants in seaward migrating juvenile salmon from the Puyallup River: potential impacts on their early marine survival* (2019) (hereinafter “Puyallup River Salmon”) at 13.

²¹⁴ Sandra O’Neill, *et al.*, *Assessing the threat of toxic contaminants to early marine survival of Chinook salmon in the Salish Sea* (May 2014) at 17, available at <https://cedar.wvu.edu/cgi/viewcontent.cgi?article=1366&context=ssec>.

Total Polychlorinated Biphenyls (PCBs)



Σ_{11} Polybrominated diphenyl ethers (Σ_{11} PBDEs)



Other Puget Sound studies have demonstrated that while some pollutants disperse, others do not. For example, using mussel tissue contaminant concentrations of organic pollutants and metals, the WDFW concluded that positive correlations between PAHs, PCBs, PBDEs, DDTs, lead and zinc and the percent of impervious surface in adjacent watersheds is “evidence that this

characteristic of urbanization provides a transport pathway for toxic chemicals from terrestrial to aquatic habitats.” However, it also demonstrated variability between contaminants:

- “Similar to the 2015/16 survey results, all metals were frequently detected in mussels at all sites.”²¹⁵
- The majority of Puget Sound urban growth area shorelines have relatively low concentrations of PAHs, PBDEs, and DDTs and “only a few sites have much higher concentrations, perhaps from locally high non-point sources, or site specific point sources.”²¹⁶
- The pattern for PCBs had “a more gradual contaminant accumulation as the shoreline length increased, suggesting sources of this contaminant is more widely dispersed within the Puget Sound [urban growth areas].”²¹⁷
- The patterns for most metals (arsenic, cadmium, lead, mercury, and zinc) had “a more gradual contaminant accumulation as the shoreline increased, suggesting these contaminants are more widely dispersed within the Puget Sound [urban growth] shoreline.”²¹⁸
- Copper was skewed to lower concentrations, with only a few sites having much higher concentrations.²¹⁹
- The highest concentrations of organic contaminants and metals were located mainly in the more urbanized and industrialized areas of Puget Sound. “However, low metal concentration sites occurred within the same urban south-central basin; a pattern not observed with the organic contaminants where all the sites had high or intermediate concentrations within the south-central basin.”²²⁰

A recent study on contamination patterns in Dungeness crab of Puget Sound

demonstrated different outcomes:

- PCBs were the most abundant contaminant measured in both Dungeness crab (followed by PAHs, PBDEs and DDTs) and measured in spot prawn (followed by PAHs and DDTs). PBDEs were rarely detected in spot prawn.
- Highest concentrations for both species and all pollutants were observed in urban areas.
- Mercury is the only metal with a strong (positive) correlation with urban areas, and only in Dungeness crab.

²¹⁵ Mariko Langness and James West, WDFW, *Stormwater Action Monitoring 2017/18 Mussel Monitoring Survey Final Report* (Oct. 2020) at 1.

²¹⁶ *Id.* at 1

²¹⁷ *Id.*

²¹⁸ *Id.*

²¹⁹ *Id.*

²²⁰ *Id.*

- Arsenic, copper, and zinc in both species, and mercury in spot prawn were distributed more equally.
- In general, contaminant concentrations in the hepatopancreas of Dungeness crab and head tissue of spot prawn were consistently higher (as much as 36 times higher) than the concentrations in the corresponding muscle for each species.²²¹

A 2020 report evaluating PBDEs in the Nisqually river system that feeds into Puget

Sound noted that previous studies showed:

contaminant exposure in outmigrating steelhead trout (*Oncorhynchus mykiss*) from in-river and the estuary habitats of Skagit, Green/Duwamish and Nisqually rivers and their associated nearshore marine habitats documented that polybrominated diphenyl ethers (PBDEs) were highest in the Nisqually River system. Moreover, PBDEs concentrations in steelhead trout were above critical body residues (CBRs) for increased disease susceptibility throughout the Nisqually river system: 33% of fish in-river at the smolt traps, 33% of fish caught in the estuary and 50% of fish in the associated marine basin. Subsequent sampling of steelhead trout at the Nisqually River smolt trap in 2015 also confirmed that approximately one third of the fish had PBDE at levels known to increase disease susceptibility in salmonids.²²²

Further evaluation of how juvenile Chinook salmon are being exposed to persistent organic pollutants was reported that same year using multiple chemical tracers.²²³ The study “reveal[ed] that wastewaters discharging into the [Snohomish] river was the likely source of these POPs [PBDEs and to a lesser extent PCBs].”²²⁴ It concluded:

reductions in PBDE exposure should improve Chinook salmon health and enhance their marine survival. The Snohomish River is the second largest contributor of Chinook salmon to the Puget Sound evolutionarily significant unit (Jonathan Carey, National Marine Fisheries Service, Personal communication); consequently, reductions in salmon survival due to wastewater-contaminant exposure could affect the recovery of the ESA-listed

²²¹ Andrea Carey, et al., *Toxic Contaminants in Dungeness crab (Metacarcinus magister) and Spot Prawn (Pandalus platyceros) from Puget Sound, Washington, USA* (March 2014) at 66.

²²² Sandra O’Neill et al., *Persistent Organic Pollutant Sources and Pathways to Juvenile Steelhead Trout in the Nisqually River* (April 2020) (hereinafter “Nisqually Sources and Pathways”).

²²³ Sandra O’Neill et al., *Chemical tracers guide identification of the location and source of persistent organic pollutants in juvenile Chinook salmon (Oncorhynchus tshawytscha), migrating seaward through an estuary with multiple contaminant inputs*, 712 *Science of the Total Environment* 135516 (2020).

²²⁴ *Id.* at 14.

Chinook salmon from Puget Sound. Furthermore, exposure to contaminants in wastewater may thwart substantial habitat remediation efforts underway throughout the US Pacific Northwest to improve survival of natural-origin salmon. For example, between 2005 and 2017 approximately \$ 90,000,000 US has been spent to improve the freshwater, estuarine and nearshore marine rearing habitat for natural-origin Chinook salmon originating from the Snohomish River (Snohomish Basin Salmon Recovery Forum, 2019), with the ultimate goal of improving their overall survival. The efficacy of this effort could be reduced if juvenile salmon have increased susceptibility to disease because of exposure to wastewater-derived contaminants. More broadly, Chinook and other salmon species are at risk in much of the southern part of their North American range (Gustafson et al., 2007), where interactions with many anthropogenic factors affect them, including contaminants (Lundin et al., 2019; Meador, 2014).²²⁵

In 2009, the agency described evidence that persistent organic pollutants were related to sediment contamination and the pelagic food web:

[S]tudies support the hypothesis that benthic (bottom dwelling) species reflect contaminant conditions in sediments. However, assessments of pelagic (open water) species, such as Pacific herring (*Clupea pallasii*), suggest that the pelagic food web is more directly linked to POPs that occur in Puget Sound's waters and pelagic biota (rather than sediments). Pacific herring hold unusually high tissue burdens of bioaccumulative POPs (e.g., polychlorinated biphenyls (PCBs), an observation that is not typically predicted from sediment-as-source models. In addition, other research indicates that PCBs and polybrominated diphenyl ethers (PBDEs) have biomagnified in Puget Sound's harbor seals (*Phoca vitulina*) and killer whales (*Orcinus orca*) to levels that have impaired their health (Hickie, 2007; Ross, et al., 2000; Ross, et al., 2004).²²⁶

WDFW's stated concern was that while PCBs, PBDEs, and PAHs were all present in "high enough doses to impair the[] health. . . . [of] high-level predators such as salmon, harbor seals, killer whales, seabirds, and humans," not understanding the pathways made it "difficult to prioritize management actions aimed at reducing loading of toxicants, remediating contaminated habitats, or reducing exposure of biota to toxicants."²²⁷ Given the varied outcome of studies

²²⁵ *Id.*

²²⁶ WDFW, *Quality Assurance Project Plan Persistent Organic Pollutants in Three Guilds of Pelagic Marine Species from the Puget Sound* (Dec. 14, 2009) at 6.

²²⁷ *Id.* at 6

done on different toxic contaminants and different species, it is not clear that any one approach will be the ideal “silver bullet” to solve the bioaccumulation of toxic contaminants in Puget Sound species. Moreover, efforts to reduce PCB contamination have not been highly successful.²²⁸

WDFW scientists also investigated contaminants of emerging concern (“CECs”) other than PBDEs in the Puget Sound region.²²⁹ A 2016 study identified the following conclusions about estrogenic chemicals in Puget Sound:

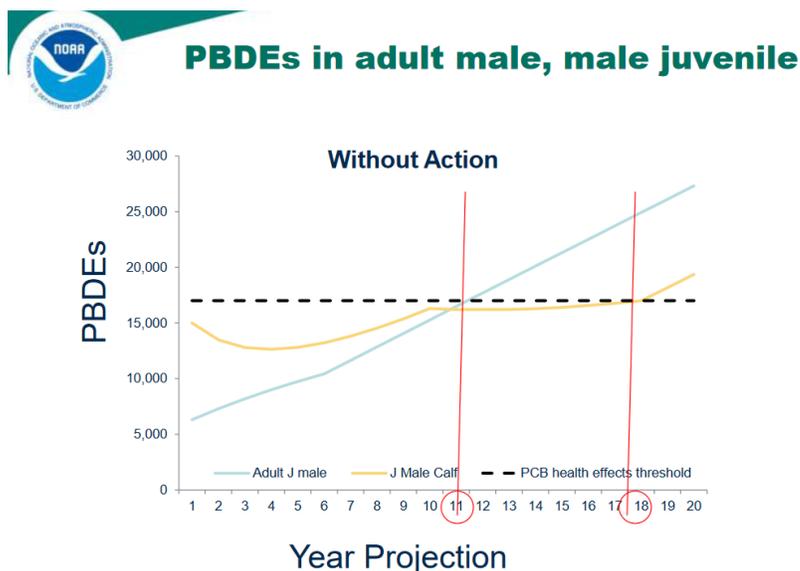
- continued altered reproductive timing in female fish from Seattle Waterfront in Elliott Bay, likely from exposure to [estrogenic chemicals] ECs,
- relatively high concentrations of EC in sole from highly-developed urbanized habitats, especially Seattle Waterfront and Sinclair Inlet,
- widespread vitellogenin induction in male sole, with highest values primarily observed in highly developed urbanized habitats, especially Tacoma Waterway and Seattle Waterfront,
- little or no recent exposure of English sole to [selective serotonin reuptake inhibitors] SSRIs, likely because sole did not occur near enough to, or forage long enough near, putative SSRI sources (such as wastewater treatment plants).²³⁰

²²⁸ See James West, *An Evaluation of the Efficacy of Remedial Actions Implemented in the Commencement Bay Nearshore and Tidelands [CB/NT] Superfund Site to Reduce PCB Contamination: 1984-2019* (Aug. 2022). While this study concluded that PCB levels in English sole have “declined sufficiently since 1984 to achieve EPA’s target tissue cleanup objective for human health,” it also concluded that “substantial areas of contamination in the CB/NT remained,” including that “English sole from two of the largest CB/NT waterways, Hylebos and Thea Foss, exhibited significantly greater PCB tissue concentrations than the [Carr Inlet Reference Area] CIRA, at concentrations among the highest PCB levels measured in English sole on a Puget Sound-wide scale[.] Moreover, long-term [WDFW] monitoring of PCB levels in English sole from the Thea Foss Waterway has shown no evidence of a declining PCB trend over the past 30 years.”

²²⁹ Sandra O’Neill, *et al.*, *Contaminants of Emerging Concern in Puget Sound English sole (Parophrys vetulus): Exposure to and Effects of Selected Estrogenic Chemicals and Pharmaceuticals* (June, 2016); see also Puyallup River Salmon, *supra* n. 213 at 20 (48 CECs detected in Puget Sound river systems), 21 (“Doses of Zoloft and Prozac are high enough to cause potential adverse effects in Puyallup juvenile Chinook.”) (percent of samples containing anti-depressants: Citalopram (Celexa) – 40%; Sertraline (Zoloft) – 27%; Fluoxetine (Prozac) – 20%; Norfluoxetine (Prozac metabolite) – 7%; Amitriptyline – 7%).

²³⁰ *Id.* at 50.

NMFS too, has identified PBDEs as a threat to Southern Resident killer whales, calculating for two scenarios (an adult male and male calf) that both will “continue to increase their PBDE body burdens in the next 20 years” and PBDE levels “will surpass the PCB health-effects thresholds within the individual’s life span,” for the adult male in 10 years and for the calf between 15 and 18 years, the shorter timeframe if the evaluated discharge did not occur (“without action” in the following graph):²³¹



c. Piedmont Region, Southeast United States

In 2014, a study focused on pharmaceuticals in the waters of the Piedmont region of Alabama, Georgia, South Carolina, North Carolina, Virginia, West Virginia, Tennessee, and the District of Columbia concluded that “[h]uman-use pharmaceuticals were ubiquitous in the 59 wadeable streams sampled throughout the study region[.]”²³² The authors concluded that

²³¹ NMFS, *NMFS’ Preliminary Analysis of Effects on Southern Resident Killer Whales* (Dec. 13, 2012) (date is incorrectly printed as 2022) at 13; based on Theresa Mongillo, NMFS, *PBDE accumulation in Southern Resident Killer Whales: Incremental Increase Model* (2012, undated).

²³² Bradley *et al.*, *Metformin and Other Pharmaceuticals Widespread in Wadeable Streams of the Southeastern United States*, 3 *Environ. Sci. Technol. Lett.* 243 (2016) at 246.

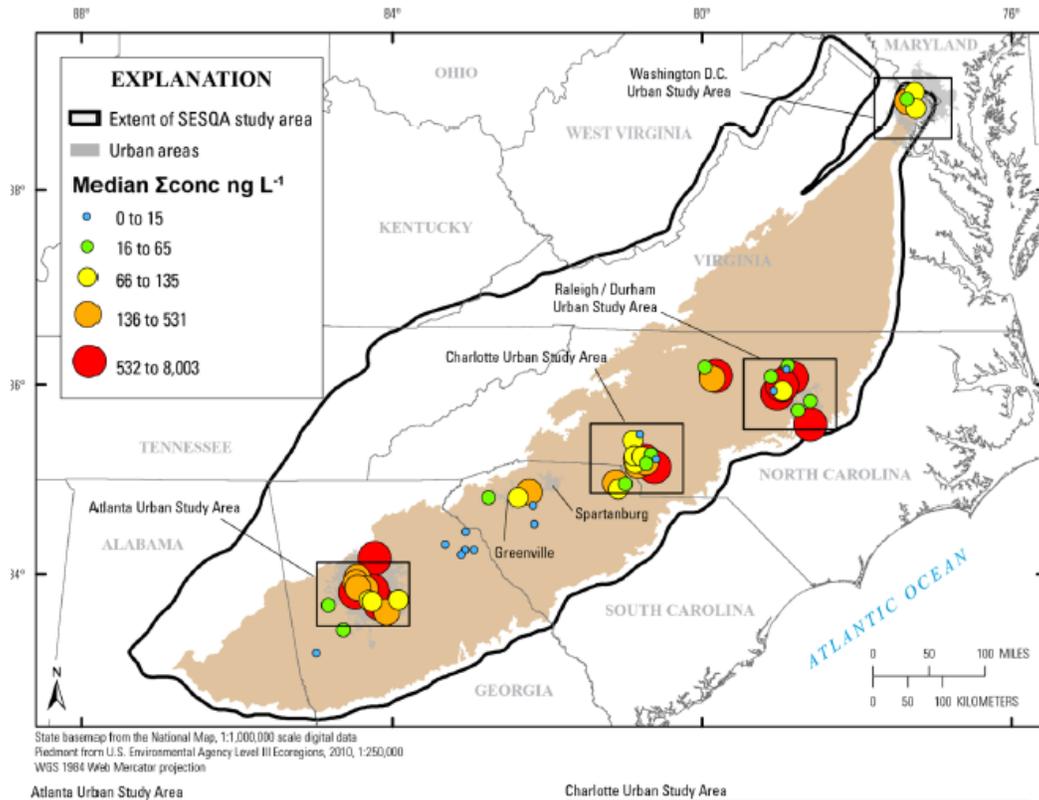
“pharmaceutical contaminants are substantial environmental health concerns in wadeable streams throughout the southeastern Piedmont ecoregion, irrespective of NPDES-wastewater discharge,” noting that the 108 pharmaceuticals studied was but a small subset:

[The study encompasses] a fraction of the pharmaceutical universe, with more than 4000 pharmaceutical parent compounds in current use and an incalculable chemical space of potential daughter products. Thus, actual pharmaceutical occurrence and concentrations undoubtedly substantially exceed current observations. Nevertheless, the nanograms per liter to micrograms per liter concentrations of individual contaminants and multiple pharmaceutical detections per site (median of 6) at cumulative concentrations of up to more than $16 \mu\text{g L}^{-1}$ are substantial concerns in their own right, because adverse environmental impacts have been documented for single pharmaceutical contaminants at low nanograms per liter concentrations.²³³

This concern is illustrated by the following map of the results:²³⁴

²³³ *Id.* at 247.

²³⁴ *Id.* at 244, partial reproductions of Fig. 1 (Cumulative median concentrations of human-use pharmaceuticals detected during the June 2014 synoptic sampling of 59 wadeable streams in the Piedmont region.)



3. Toxic Contamination of Sediment

In addition to water column and animal tissue contamination levels, EPA has long expressed concern about the accumulation of toxic contaminants in sediment of rivers, lakes, estuaries, and oceans.²³⁵ As EPA said in 1998—24 years ago—“chemicals released to surface waters from industrial and municipal discharges, and polluted runoff from urban and agricultural areas, continue to accumulate to environmentally harmful levels in sediment.”²³⁶ In terms of sheer volume, at that time, EPA estimated that

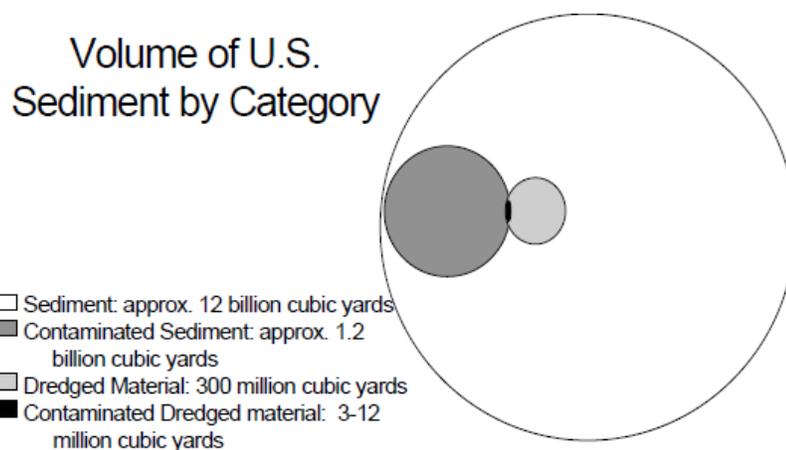
approximately 10 percent of the sediment underlying our nation’s surface water is sufficiently contaminated with toxic pollutants to pose potential risks to fish and to humans and wildlife who eat fish. This represents about 1.2 billion cubic yards of contaminated sediment out of the approximately 12 billion cubic yards of total surface sediments (upper five centimeters) where many bottom dwelling

²³⁵ See, e.g., EPA, *EPA’s Contaminated Sediment Management Strategy* (April 1998).

²³⁶ *Id.* at Foreword.

organisms live, and where the primary exchange processes between the sediment and overlying surface water occur. Approximately 300 million cubic yards of sediments are dredged from harbors and shipping channels annually to maintain commerce, and about 3-12 million cubic yards of those are sufficiently contaminated to require special handling and disposal.²³⁷

EPA provided the following illustration of the scope of the problem of sediments contaminated with toxic chemicals.²³⁸



In New York alone, EPA cited that 20 percent of all river miles had contaminated sediments and 30,000 acres of lakes had fish consumption advisories.²³⁹

The agency described the threat to human health and the environment from this contaminated sediment as follows:

Contaminated sediment poses ecological and human health risks in many watersheds throughout the United States. In these watersheds, sediment serves as a contaminant reservoir from which fish and bottom-dwelling organisms can accumulate toxic compounds and pass them up the food chain. Sediment contaminants can be passed to fish, birds, and mammals until they accumulate to levels that may be toxic. Such toxic effects may include neurological, developmental, and reproductive impacts. Toxic chemicals come from discharges from industrial waste and sewage; storm water runoff from waste dumps, city streets, and farms; air pollutants contained in rainwater; contaminants in ground

²³⁷ *Id.* at Foreword.

²³⁸ *Id.*

²³⁹ *Id.* at 93-94.

water; discharges to surface water; and from natural sources. The magnitude of the sediment contamination problem in the United States is evidenced in more than 2,100 State advisories that have been issued against consuming fish. Sediments were identified as a potential source of contamination at many of the sites where consumption of fish may pose health risks. EPA has studied sediment quality data from 1,372 of the 2,111 watersheds in the continental United States. Of these, EPA has identified 96 watersheds that contain “areas of probable concern” where potential adverse effects of sediment contamination are more likely to be found.²⁴⁰

EPA went on to describe its having ranked toxic contamination of sediment as the 11th most significant problem of 32 environmental problems facing the nation in a 1987 report, a ranking supported by its Science Advisory Board (“SAB”).²⁴¹ EPA also noted the threat to human health, and the significant increase in fish consumption advisories from contaminated sediment, particularly in the context of environmental justice concerns:

In this report, SAB indicated that cancer and noncancer illnesses can be caused by bioaccumulation of toxic chemicals from sediments in fish and shellfish which are then consumed by humans. Both EPA and SAB gave contaminated sediments a medium risk score as a causative agent of non cancer illnesses. SAB judged that consumption of contaminated fish posed a low cancer risk, but noted that bioaccumulation in fish of chemicals in contaminated sediments was the primary route of human exposure to carcinogens in surface waters.

In comparative risk analyses performed by EPA Regions 1, 2, 3, 5, and 10, sediment contamination was given a medium-high score for cancer risks to consumers of fish and shellfish (U.S. EPA, 1989c). Since actual risks may be higher for certain ethnic groups due to fish consumption patterns, environmental justice concerns have been raised in certain parts of the country. In 1996, there were 2,193 waterbodies with fish consumption advisories in the United States, with sediments identified as a potential source of contamination at many sites. This number of advisories is a 26 percent increase from 1995 and a 72 percent increase since 1993.²⁴²

EPA’s Contaminated Sediment Strategy included case studies on human health risks,

²⁴⁰ *Id.* at i.

²⁴¹ *Id.* at 90, citing EPA, *Unfinished Business: A Comparative Assessment of Environmental Problems* (1987) and SAB, *Reducing Risk: Setting Priorities and Strategies for Environmental Protection* (1990).

²⁴² *Id.* at 90–91.

such as for Quincy Bay where EPA found the regular consumption of tomalley from local lobsters posed a “high cancer risk” and noted that despite thousands of acres of shellfish being closed for harvest in the New Bedford Harbor Superfund site, “[m]any individuals regularly consumed seafood from the area before the extent of contamination was known, however, and some residents still harvest both finfish and shellfish for personal consumption.”²⁴³ PCB levels in shellfish and fish there produced unacceptable lifetime cancer risks as high as 1×10^{-2} for weekly consumption of edible lobster tissue.²⁴⁴ Similarly, EPA reported unacceptable cancer risks for people consuming animal and plant material from Puget Sound in Washington State:

A high background incidence of cancer was observed and it was determined that 25 percent of the individuals in the Puget Sound region would develop cancer during their lifetimes. The health risk assessment predicted that two additional cases of cancer would be added to the 2,500 cases expected per 10,000 individuals consuming an average quantity of seafood (a risk level of 2×10^{-4}), and 40 additional cases of cancer would be added to the 2,500 expected per 10,000 individuals consuming a large quantity of seafood (a risk level of 4×10^{-3}). The principal carcinogens identified in this study were PCBs in fish and polycyclic aromatic hydrocarbons in seaweed.²⁴⁵

And, EPA identified unacceptable risks from contaminated sediments in the Los Angeles-Long Beach Harbor in California:

Following a risk assessment analysis of toxic contaminants in fish, the California Department of Health Services issued a health advisory concerning the consumption of local sport fish from the Santa Monica Bay, Palos Verdes Peninsula, and Los Angeles-Long Beach Harbor areas (Gossett et al., 1989). Sediments in these areas are contaminated with PCBs, DDT, and DDT metabolites which were discharged in the 1960s and early 1970s. Analysis showed that the bottom-feeding white croaker was particularly contaminated, and cancer risks to the population consuming white croaker were significantly higher than levels generally considered to be acceptable. (Cancer risk levels on the order of 10^{-3} to 10^{-4} were calculated.) In the Los Angeles area, significantly higher levels of DDT and its metabolites were found in the blood serum of local and

²⁴³ *Id.* at 90-91.

²⁴⁴ *Id.* at 92.

²⁴⁵ *Id.* at 92.

sport fishermen who ate their catch than in the blood serum of nonconsumers.²⁴⁶

EPA called out the impacts of contaminated sediments in Lake Michigan on mothers and children:

A study of mothers and their newborn infants showed that as the period of time over which fish was consumed from the lake increased, so did the mothers' body burdens of PCBs (Swain, 1988). Exposed mothers were found to have increased levels of PCBs in whole blood serum and breast milk. The higher the PCB body burdens, the more intense were the effects exhibited by the infants (Fein et al., 1984; Jacobsen and Fein, 1985). Infants of highly exposed mothers were born at reduced birth rates and reduced gestational ages, had smaller head circumferences, and exhibited neuro-motor effects. A study published in the New England Journal of Medicine showed that children of these mothers had learning and reading difficulties as well as lowered IQ scores (Jacobsen, 1996).²⁴⁷

EPA cited a 1991 public health directive to not eat any fish or shellfish in inner Pago Pago Harbor, in American Samoa, identifying the following greatest risks:

1) Potential brain damage. If lead contamination alone were considered, lead concentrations in fish could reach levels that would cause 70 percent to 80 percent of children who regularly eat 3 to 4 fish meals per week to suffer a permanent reduction in intelligence. 2) Increased cancer risk. Consuming fish from the inner harbor at a rate of 3 to 4 fish meals per week over a lifetime would significantly increase the risk of cancer due to arsenic contamination. 3) Increased non-cancer health risks. Using a hazard index in which non-cancer health risks occur at levels greater than a value of "1," EPA Region 9 calculated the hazard index at 1-3 for adults consuming inner harbor fish and at 2-3 for children consuming inner harbor fish (Baker, 1993).²⁴⁸

EPA also provided case studies on ecological risks from contaminated sediment, citing studies demonstrating that the extent of contaminated sediments in the Elizabeth River, a sub-estuary of the Chesapeake Bay, is correlated to the frequency and intensity of neoplasms, cataracts, enzyme induction, finrot, and other lesions observed in fish populations.²⁴⁹ The

²⁴⁶ *Id.* at 92-93.

²⁴⁷ *Id.* at 93.

²⁴⁸ *Id.* at 94.

²⁴⁹ *Id.* at 95.

Contaminated Sediment Strategy pointed to PAH contamination of sediments' being linked to increased incidence of skin, liver, and lip tumors in certain fish in the Great Lakes and the occurrence of liver cancer in native fish populations, as well as reproductive problems in Forster's tern and to reproductive failure and mortality in mink, concluding about the latter two examples that they "are indicative of the risks to fish-eating birds and mammals posed by a PCB-contaminated food chain, and may provide clues to explain why certain fish-eating birds and mammals may have disappeared or become rare in ranges where they were historically found."²⁵⁰

B. Adverse Impacts of Toxic Contaminants on Designated Uses

Toxic contaminants, by definition, cause a wide range of toxic effects on designated uses of human health, fish, wildlife, and ecological health. For example, the 2011 Puget Sound report discussed above went on to discuss regionally important biological effects data for PBDEs including the following discussion that cites numerous studies on a pollutant for which EPA has not developed recommended 304(a) criteria nor placed on the Toxic Pollutants Lists:

PBDEs were detected in outmigrant Chinook salmon tissue and their stomach contents from four sites in Puget Sound (Sloan et al., 2010). Levels in wild outmigrant juveniles were higher than in hatchery fish, ranging from 67 to 13,000 ug/kg lipid, generally comparable to those measured in the Lower Columbia River and Estuary. Sloan et al. (2010) conclude that PBDEs may be contributing to reduced health and fitness in outmigrant juvenile Chinook salmon. PBDEs were detected in adult Chinook salmon returning to the Duwamish River and were not detected in adult Chinook returning to the Johnstone Strait, Lower Fraser River, or Deschutes River (Cullon et al., 2009).

Lema et al. (2008) demonstrated that dietary exposures to certain PBDEs by adult fathead minnows can alter thyroid status and thyroid hormone-regulated gene transcription. Arkoosh et al. (2010) found that juvenile Chinook salmon exposed to moderate doses of PBDEs in their diet may be at increased risk of disease relative to those exposed to higher or lower doses of PBDEs in their diet. PBDE levels were found to be about four to five times higher in a mixture of fishes designed to represent the diet of Puget Sound harbor seals than in a similar

²⁵⁰ *Id.* at 96-97.

mixture of fish designed to represent the diet of harbor seals from the Strait of Georgia (Cullon et al., 2005).

Very few studies have been conducted examining effects of PBDEs on birds. The studies reviewed indicate that PBDEs impact the reproduction and endocrine system similarly to PCBs. Exposure to BDE-71 for 75 days adversely impacted courtship and mating behavior of American kestrels (*Falco sparverius*) (Ferne et al., 2008). These birds also displayed significant delays in clutch initiation and smaller eggs (Ferne et al., 2009). Eggshell thinning and reduced hatching success also resulted. A study of species sensitivity to PBDEs (PBDE-71) observed that pentabrominated diphenyl ether (Penta BDE) exposure to eggs at 0.01 to 20 mg/kg caused decreased pipping and hatching success in American kestrels but not chickens (*Gallus gallus*) or Mallard ducks (*Anas platyrhynchos*) (McKernan et al., 2009). Species sensitivity was concluded to be Mallard ducks <chickens <American kestrels.

Total PBDE concentrations in osprey eggs and nestling plasma are significantly lower in the Lower Duwamish River (eggs: 321 ug/kg ww; plasma: 6 ug/kg ww) compared to those from the upper Willamette River (eggs: 897 pb ww; plasma: 22 ppb ww) (Johnson et al., 2009). Total PBDE concentrations in the osprey eggs did not change significantly between 2003 and 2007. Reproductive failure was observed in four of nine nests in the Lower Duwamish area. A small dataset from this study suggests that some nestlings may have experienced immunosuppression. However, the results were inconclusive due to the small sample size.

Compared to birds, a larger but still limited number of publications exist on the effects of PBDEs in mammals. Rodent exposure studies have demonstrated thyroid hormone disruption (Hallgren et al., 2001; Zhou et al., 2002) and developmental neurotoxic and behavioral effects (Ericksson et al., 2001; Viberg et al., 2003a; Viberg et al., 2003b). A study of grey seal pups and juveniles observed a relationship between circulating thyroid hormones, transport proteins, and PBDE uptake (Hall et al., 2003).

Similar to PCBs, there is evidence of bioaccumulation of PBDEs in marine mammals at high concentrations in blubber. However, absolute concentrations of total PBDEs appear to be lower than total PCBs. Cullon et al. (2005) measured PBDE concentrations five times higher in harbor seal prey from Puget Sound than the Strait of Georgia, but the mean PBDE concentration was five times lower than that measured for PCBs. Krahn et al. (2009) and Rayne et al. (2004) found the same pattern of killer whale blubber concentrations as found for PCBs in males, mothers, and calves. Krahn et al. (2009) measured total PBDE concentrations ranged from 680 to 15,000 ug/kg lipid. Mean PBDE concentrations in northern male killer whale blubber have been found to be significantly lower (203 ug/kg

lipid) than those of southern resident (942 ug/kg lipid) and transient males (1,015 ug/kg lipid).

Although a quantitative effects assessment was not conducted for PBDE exposure to marine mammals, published research demonstrates that PBDEs are bioaccumulating to high concentrations in Puget Sound killer whales. This coupled with the growing evidence that PBDE exposure can cause thyroid and developmental effects in mammals strongly suggest that PBDEs are an important contaminant to monitor.²⁵¹

Startlingly, even after drawing its conclusion that “a growing body of evidence suggests environmental concentrations [of PBDEs] may cause adverse effects to humans, marine animals, and birds,”²⁵² the study made no priority assessment of PBDEs because “the paucity of documented effects, standards, or guidelines for PBDEs consistent with those used for other [contaminants of concern] COCs preclude the assignment of a Priority 1 or Priority 2 level of concern[.]”²⁵³ And, notwithstanding the study’s conclusion that “evidence in the available literature to suggest this COC [PBDE] may pose a hazard at observed concentrations”²⁵⁴—including the Southern Resident killer whales, a species with a population so small that it is classified as “endangered”—and the fact that sewage treatment plants were the only source of PBDEs identified as primarily from NPDES-regulated sources, the agencies did not include in their recommendations any reference to the need to collect data on PBDE discharges from sewage treatment plants in order to regulate them.²⁵⁵

The adverse effects of toxic contaminants are many and varied. In the sections below, we discuss four aspects of toxic effects including in sub-section 1, the effects of mixtures of toxic

²⁵¹ Puget Sound Assessment, *supra* n. 201 at 106–107.

²⁵² *Id.* at 168 (internal citations omitted).

²⁵³ *Id.* at 105.

²⁵⁴ *Id.* at 174.

²⁵⁵ *Id.* at 176.

contaminants; in sub-section 2, some aspects of endocrine disruption, focused on the development of intersex conditions; in sub-section 3, an example of sublethal effects of toxics, in salmonids; and finally, in sub-section 4, a short survey of toxic effects to marine mammals and aquatic-dependent species.

1. Effects of Ecosystem Exposures to Chemical Contaminant Mixtures

In the real world, few chemicals are found by themselves and few act upon human health and aquatic species in the absence of yet other chemicals and conventional pollutants, leading to increasing concerns about the effects of chemical mixtures.²⁵⁶ The 2017 EPA/USGS nationwide survey of chemical mixtures concluded the results of 70 median detections per site at cumulative concentrations up to more than 102 $\mu\text{g L}^{-1}$ “are substantial concerns . . . because adverse environmental impacts have been documented for individual designed-bioactive contaminants at low ng L^{-1} concentrations[.]”²⁵⁷ The authors point out that the 719 organic compounds evaluated in the study are but “a fraction of the contaminant universe, estimated at more than 80000 parent compounds in current use and an incalculable chemical-space of potential metabolites and degradates; logically, actual surface water contaminant complexity and concentrations may substantially exceed the current observations.”²⁵⁸ They also noted some specific concerns:

Detection of 17 pharmaceuticals (11 antibiotics, 4 antivirals, 2 antifungals), 9 fungicides, and triclosan (bacteriostat with recognized antibiotic selection potential) in this study with individual detection frequencies up to 53% and maximum concentrations up to 1.8 $\mu\text{g L}^{-1}$ strongly suggests impacts at the

²⁵⁶ Note that even the use of WET testing of chemical mixtures does not solve the problem of translating evidence of toxicity into individual chemicals and their sources. Moreover, WET testing does not address bioaccumulation concerns. More pervasive use of WET testing, while desirable, would not obviate the need to add pollutants to the Toxic Pollutants Lists.

²⁵⁷ EPA/USGS 2017 National Study, *supra* n. 169 at 4798.

²⁵⁸ *Id.*

microbial base of the aquatic foodweb in streams nationwide. Antibiotic-contaminant concentrations as low as $0.5 \mu\text{g L}^{-1}$ have been shown to affect the structure and composition of aquatic microbial communities.

* * *

sublethal selection of antibiotic-resistant bacteria has been documented at ciprofloxacin concentrations as low as 100 ng L^{-1} . Ciprofloxacin was detected in this study at 26% of the sites, with maximum and median detected concentrations of 400 ng L^{-1} and 135 ng L^{-1} , respectively.

* * *

Invertebrates comprise most animal biomass in aquatic ecosystems and the current results suggest substantial potential for adverse contaminant impacts.

* * *

[neonicotinoid insecticides] imidacloprid and clothianidin were detected at 37% and 24% of sites, respectively, with maximum and median concentrations ranging $175\text{--}475 \text{ ng L}^{-1}$ and $66\text{--}143 \text{ ng L}^{-1}$, respectively. Acute and chronic ecological health thresholds below 200 ng L^{-1} and 35 ng L^{-1} , respectively, have been recommended to protect aquatic invertebrate communities.

* * *

exposure to approximately $2 \mu\text{g L}^{-1}$ fexofenadine has been shown to impair survival behavior (flight response) in damselfly (*Zygoptera*) species and result in bioconcentration up to 2000 times the dissolved concentration. Multiple (fexofenadine, diphenhydramine, loratadine, hydroxyzine) antihistamines were detected in this study (16–42% of sites) at concentrations up to approximately $4 \mu\text{g L}^{-1}$. Fexofenadine was detected at 42% of sites, with maximum and median concentrations of 2047 ng L^{-1} and 576 ng L^{-1} , respectively; two or more antihistamines were detected at 13 sites (34%).

* * *

Eighty-four pharmaceuticals were detected across all sites, with detection frequencies for individual compounds ranging 3–74% of sites (median 24%). Frequent detection of metformin [type II diabetes medicine] (66% of sites) at median concentrations greater than 400 ng L^{-1} , including seven sites with concentrations in the $\mu\text{g L}^{-1}$ range, is noteworthy because metformin is a designed endocrine-active compound and effluent-equivalent metformin exposures in the $\mu\text{g L}^{-1}$ range induce up-regulation of vitellogenin mRNA and male intersex in fathead minnow (*Pimephales*).²⁵⁹

The concern about chemical mixtures and, specifically, whether EPA's Section 304(a) recommended numeric criteria and states' adoption of them in their water quality standards is not

²⁵⁹ *Id.* at 4799 (internal citations omitted) (emphasis added).

new. As the National Marine Fisheries Service pointed out, toxic effects are likely more severe in the wild as opposed to in a laboratory:

In field conditions, organisms never experience exposure to a single pollutant; rather, ambient waters typically have low concentrations of numerous chemicals. The toxic effects of chemicals in mixture can be less than those of the same chemicals singly, greater than, or have no appreciable difference. The best known case of one toxicant reducing the effects of another is probably Se and Hg (e.g., Belzile *et al.* 2006). However, strongly antagonistic responses are probably uncommon, and much more common are situations where chemical mixtures have greater toxicity than each singly or little obvious interaction (e.g., Norwood *et al.* 2003; Borgert 2004; Playle 2004; Scholz *et al.* 2006; Laetz *et al.* 2009). In general, it seems prudent to assume that if more than one toxicant were jointly elevated it is likely that lower concentrations of chemicals would be required to produce a given magnitude of effect than would be predicted from their actions separately. However, the magnitude or increased effects at environmentally relevant concentrations is uncertain and for some combinations may be slight or imperceptible.²⁶⁰

NMFS cited a 1986 laboratory test that demonstrated the toxic hazards of multiple metals:

[S]ome studies have shown significant additive toxicity. For instance, Spehar and Fiandt (1986) exposed rainbow trout and *Ceriodaphnia dubia* simultaneously to a mixture of five metals and arsenic, each at their acute CMC, which by definition were intended to be protective. There were no survivors. In chronic tests, adverse effects were observed at mixture concentrations of one-half to one-third the approximate chronic toxicity threshold of fathead minnows and daphnids, respectively, suggesting that components of mixtures at or below no effect concentrations may contribute significantly to the toxicity of a mixture on a chronic basis (Spehar and Fiandt 1986).²⁶¹

The agency also noted that organic compounds are “mixtures of a large number of congeners with differing levels of toxicity and modes of action,” pointing out that “are a mixture of over 200 separate congeners, and toxaphene, which is a combination of over 600 isomers.”²⁶² It noted

²⁶⁰ National Marine Fisheries Service, *Final Endangered Species Act Section 7 Formal Consultation and Magnuson-Stevens Fishery Conservation and Management Act Essential Fish Habitat Consultation for Water Quality Toxics Standards for Idaho* (May 7, 2014) (hereinafter “NMFS Idaho BiOp”) at 69.

²⁶¹ *Id.* at 88.

²⁶² *Id.* at 204.

that for PCBs, “methods such as the calculation of a toxicity equivalence factor (TEF) for those congeners with dioxin-like activity can provide a measure of the overall toxicity of mixtures containing these congeners[.]”²⁶³ And it concluded that for neither PCBs nor toxaphene “are these issues dealt with in existing water quality standards.”²⁶⁴

NMFS also cited other examples of the toxicity of mixtures of pesticides, such as dieldrin’s interacting synergistically with the pesticide carbaryl, DDT, and the aflatoxin Aflatoxin B1²⁶⁵; chlordane’s additive and synergistic effects with the pesticide furadan²⁶⁶; and toxaphene with other pesticides and contaminants.²⁶⁷ NMFS was sufficiently concerned about the potential for mixture toxicity in discharges that it directed EPA to minimize that potential by summing the cumulative criterion units and requiring provisions for Whole Effluent Toxicity (“WET”) testing and biomonitoring in NPDES permits.²⁶⁸ It concluded that with a regulatory recommendation:

[F]ield assessments may be one of the few practical means for addressing the issue of interactions, mixture effects and multiple stressors. However, there has been little implementation of bioassessment into permitting decisions. . . . Bioassessment of receiving waters has been required as a monitoring element for receiving waters in NPDES permits issued by EPA in Idaho; however, to our knowledge, the data collected has never been a factor in determining the adequacy of permit limits in renewal applications. NMFS recommends that EPA develop an approach to effectively use bioassessment data in permitting decisions.²⁶⁹

²⁶³ *Id.*

²⁶⁴ *Id.*

²⁶⁵ *Id.* at 215.

²⁶⁶ *Id.* at 222.

²⁶⁷ *Id.* at 259.

²⁶⁸ *Id.* at 292. Improved and more pervasive use of WET testing methods is an obvious response to the multiplicity of chemicals that are not on the Toxic Pollutants Lists. However, WET testing does not reflect bioaccumulation of many toxic pollutants and, in addition, is only a means to identifying the individual chemicals that are causing the toxicity. The more of these that are on the Toxic Pollutants Lists, the better able permit writers and permittees will be to translate evidence of toxicity to possible sources.

²⁶⁹ *Id.* at 295.

Finally, NMFS pointed out that there are synergistic interactions between toxic chemicals and conventional pollutants. For example, “[a]t temperatures well above optimal ranges, increased toxicity from chemicals often results from increased metabolic rates (Sprague 1985).”²⁷⁰ Specifically, NMFS pointed out that “with cyanide, temperature has a strong influence on toxicity.”²⁷¹ It also concluded that “[t]he acute criterion cannot be considered to be reliably protective when water temperatures drop to about 6°C or lower.” For salmonids in the Pacific Northwest, temperatures “routinely drop below 6°C” and temperatures are often well above optimal ranges, as demonstrated by the states’ 303(d) lists of impaired waters that are dominated by temperature impairments.²⁷² Temperature interactions were also cited for mercury,²⁷³ selenium,²⁷⁴ zinc,²⁷⁵ chromium VI,²⁷⁶ lead,²⁷⁷ chlordane,²⁷⁸ DDT,²⁷⁹ endosulfan,²⁸⁰ endrin,²⁸¹ and lindane.²⁸² Another study has shown significant increased toxicity from malathion with increasing temperature.²⁸³ Despite long-standing knowledge about how conventional

²⁷⁰ *Id.* at 67.

²⁷¹ *Id.* at 139.

²⁷² Washington Department of Ecology, *Washington State Water Quality Assessment* (hereinafter “Washington Assessment Database”), available at <https://apps.ecology.wa.gov/ApprovedWQA/ApprovedPages/ApprovedSearch.aspx> (Washington waters impaired by temperature (Categories 5, 4a, 4b): total 2,321 segments); Oregon Department of Environmental Quality, *EPA Approved Integrated Report*, available at <https://www.oregon.gov/deq/wq/Documents/IR2022-303dImpWaters-TMDL.xlsx> (Oregon waters impaired by temperature (Category 5): total 3,717 segments).

²⁷³ NMFS Idaho BiOp, *supra* n. 260 at 149.

²⁷⁴ *Id.* at 168.

²⁷⁵ *Id.* at 186.

²⁷⁶ *Id.* at 196.

²⁷⁷ *Id.* at 197.

²⁷⁸ *Id.* at 222.

²⁷⁹ *Id.* at 230.

²⁸⁰ *Id.* at 234.

²⁸¹ *Id.* at 240.

²⁸² *Id.* at 249.

²⁸³ Joseph P. Dietrich, *et al.*, *The impact of temperature stress and pesticide exposure on mortality and disease susceptibility of endangered Pacific salmon*, 108 *Chemosphere* 353–359 (2014), available at

pollutants both increase toxicity and adversely affect metabolism rendering aquatic life more susceptible to toxic effects, EPA has only incorporated the impacts of conventional parameters other than pH and hardness in a few of its CWA Section 304(a) recommended criteria, namely: copper (temperature, pH, dissolved organic carbon (DOC), calcium, magnesium, sodium, potassium, sulfate, chloride, and alkalinity)²⁸⁴; ammonia (pH, temperature)²⁸⁵; and aluminum (ph, hardness, DOC).²⁸⁶

2. Chemical Contaminants Causing Intersex Conditions and Other Endocrine Disruption in Fish

One form of adverse impact of chemical contaminants, many of which remain unlisted, is the development of intersex conditions, one form of endocrine disruption. As explained by the USGS, “[i]ntersex conditions occur when exposure to chemicals disrupts the hormonal systems of an animal, leading to the presence of both male and female characteristics in an animal that should exhibit the characteristics of just one sex in its lifetime.”²⁸⁷ In the case of the smallmouth bass, the first species in which intersex conditions were observed, “male intersex fish are found with immature eggs in their testes, which indicates exposure to estrogenic and anti-androgenic chemicals.”²⁸⁸ The USGS has conducted numerous studies since the first observation of this

<https://www.sciencedirect.com/science/article/abs/pii/S0045653514001702?via%3Dihub> (mortality increased 11.2 percent at elevated temperatures as compared to optimal temperatures).

²⁸⁴ EPA, *Aquatic Life Ambient Freshwater Quality Criteria – Copper, 2007 Revision* (Feb. 2007) available at <https://www.epa.gov/sites/default/files/2019-02/documents/al-freshwater-copper-2007-revision.pdf>.

²⁸⁵ EPA, Water Quality Criteria, *Aquatic Life Criteria – Ammonia* (hereinafter “Ammonia Criteria website”), available at <https://www.epa.gov/wqc/aquatic-life-criteria-ammonia>.

²⁸⁶ EPA, *Final Aquatic Life Ambient Water Quality Criteria for Aluminum 2018* (Dec. 2018) (hereinafter “2018 Aluminum Recommended Criteria”), available at <https://www.epa.gov/sites/default/files/2018-12/documents/aluminum-final-national-recommended-awqc.pdf>.

²⁸⁷ Chesapeake Bay Program, Chesapeake Bay Activities, *Intersex Fish*, available at <https://www.usgs.gov/centers/chesapeake-bay-activities/science/intersex-fish>.

²⁸⁸ Chesapeake Bay Program, Discover the Chesapeake, *Bay 101: Intersex Fish*, available at <https://www.chesapeakebay.net/discover/videos/bay-101-intersex-fish>.

effect.²⁸⁹

In 2007, USGS investigated fish exhibiting external lesions, incidences of intersex, and increasing regularity of fish kills in the Shenandoah and James River Basins, finding “measurable estrogenicity in each of the site samples.”²⁹⁰ It called out specific chemicals of concern:

The fragrance components, galaxolide, indole, and tonalide, were the predominant waste indicator chemicals detected. Caffeine, the caffeine metabolite 1,7-dimethylxanthine, the nicotine metabolite cotinine, and the prescription pharmaceuticals carbamazepine, venlafaxine, and trimethoprim were detected at several sites. Natural and synthetic hormones were detected at a few sites with 17 α -ethynylestradiol concentrations estimated up to 8.1 nanograms per liter. Screening of the POCIS extracts for estrogenic chemicals by using the yeast estrogen screen revealed estrogenicity similar to levels reported for rural areas with minor effect from wastewater effluents.²⁹¹

The USGS continued to assess the degree to which chemicals are causing intersex conditions in fish.

- In 2009, USGS published a paper on intersex occurrence in fish from nine river basins, finding the condition in three percent of fish collected. The intersex condition was observed in four of the 16 species examined (25 percent) and in fish from 34 of 111 sites (31 percent). Intersex was not found in multiple species from the same site but was most prevalent in largemouth bass (*Micropterus salmoides*; 18% of males) and smallmouth bass (*M. dolomieu*; 33% of males). The percentage of intersex fish per site was 8–91% for largemouth bass and 14–73% for smallmouth bass.²⁹²
- Also in 2009, USGS published a paper that investigated the reproductive health of bass species from both up- and downstream of sewage treatment plants on the Potomac River, Maryland, concluding that “proximity to effluent from WWTPs may influence the reproductive health of bass in the Potomac watershed, but inputs from other sources likely

²⁸⁹ USGS, *Explore Search: intersex fish*, available at <https://www.usgs.gov/search?keywords=intersex+fish>.

²⁹⁰ USGS, *Reconnaissance of Persistent and Emerging Contaminants in the Shenandoah and James River Basins, Virginia, During Spring of 2007*, Open-File Report 2008–1231 (2008),

²⁹¹ *Id.* at 1.

²⁹² Jo Ellen Hinck, et al., *Widespread occurrence of intersex in black basses (Micropterus spp.) from U.S. rivers, 1995-2004* (2009)

contribute to the widespread, high incidence of testicular oocytes.”²⁹³ A high prevalence of intersex conditions, in the range of 82–100 percent, was identified in male smallmouth bass at all sites. Water quality sampling found wastewater-associated chemicals—celestolide, tonalide, galaxolide, prometon, and tri(2-chloroethyl)phosphate—downstream with atrazine and its metabolites upstream.²⁹⁴

- In 2014, USGS published a paper on the smallmouth bass in Pennsylvania’s Susquehanna, Delaware and Ohio river basins exhibiting the effects of exposure to endocrine-disrupting chemicals. The researchers concluded: the prevalence and severity of the immature eggs in smallmouth bass corresponded with the percent of agricultural land use in the watershed above the collection sites and “[c]hemical compounds associated with estrogenic endocrine disruption, in particular estrone, a natural estrogen, were also associated with the extent and severity of these effects in bass.”²⁹⁵ “There was no significant relationship between the number of waste water treatment plants and the prevalence of immature eggs in male fish, though results did indicate that the severity of intersex characteristics of male small mouth bass generally increased at downstream sites from waste water treatment plants.”²⁹⁶
- In 2018, USGS measured endocrine active compounds and their impacts on intersex occurrence and severity in in Yadkin-Pee Dee River of North Carolina and South Carolina. Polycyclic aromatic hydrocarbons (PAHs), ethinylestradiol (EE2), and heavy metals were the most prevalent contaminants that exceeded effect levels for the protection of aquatic organisms, with the PAHs most correlated with the intersex occurrence and severity in black bass and sunfish.²⁹⁷ The occurrence of the intersex condition in fish showed site-related effects, rather than increasing longitudinal trends from upstream to downstream.
- In 2020, USGS followed up a reconnaissance project that identified a high prevalence of intersex smallmouth bass, including an observation of 100 percent prevalence in

²⁹³ Luke Iwanowicz, *et al.*, *Reproductive Health of Bass in the Potomac, USA, Drainage: Part 1. Exploring the Effects of Proximity to Wastewater Treatment Plant Discharge*, 28 *Environmental Toxicology and Chemistry* 1072-1083 (2009).

²⁹⁴ *Id.* at 1072, 1079 (table 2).

²⁹⁵ USGS, *Intersex Fish Now in Three Pennsylvania River Basins* available at <https://www.usgs.gov/news/state-news-release/intersex-fish-now-three-pennsylvania-river-basins>, citing V.S. Blazer, *et al.*, *Reproductive health indicators of fishes from Pennsylvania watersheds: association with chemicals of emerging concern*, 186 *Environ Monit Assess* 6471–6491 (2014), available at <https://link.springer.com/content/pdf/10.1007/s10661-014-3868-5.pdf?pdf=button>.

²⁹⁶ *Id.*

²⁹⁷ Casey Grieshaber, *Relation of contaminants to fish intersex in riverine sport fishes*, 643 *Science of the Total Environment* 73-89 (Dec. 2018) available at <https://www.sciencedirect.com/science/article/abs/pii/S0048969718321429?via%3Dihub>.

smallmouth bass males collected from the Wallkill River, New Jersey.²⁹⁸ Correlation analysis indicated significant positive correlations between land use and surface water estrogenicity. Detectable estrogenicity was observed at 90 percent of the sites and 64 percent of the sites were above EPA’s trigger level of 1 ng/L, as shown in the map below.²⁹⁹

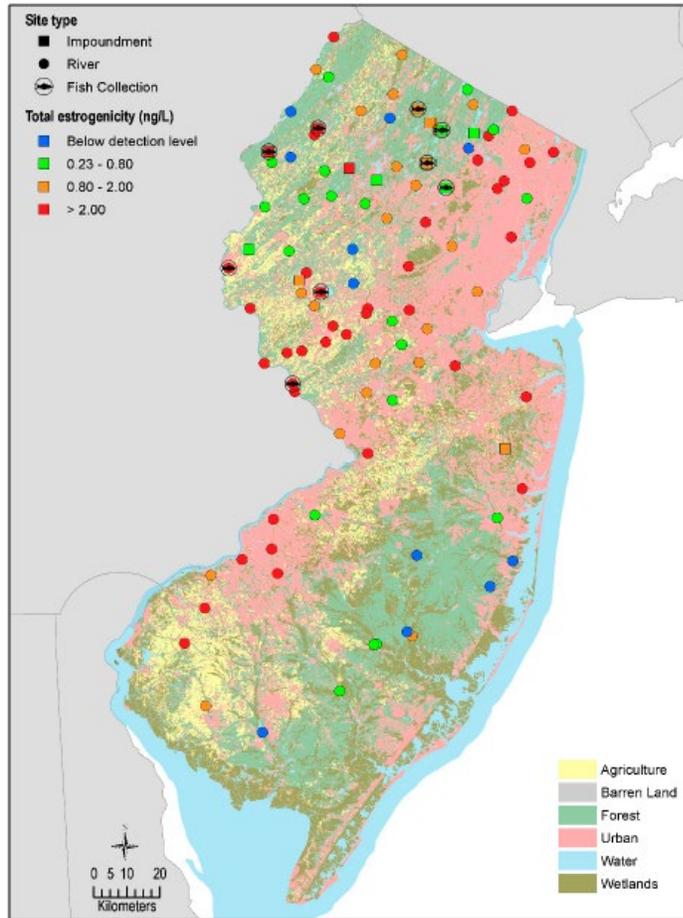


Figure 1. Surface water and fish sampling locations throughout New Jersey. Ranges of total estrogenicity (ng/L) are binned as below detection (BD), low (0.23–0.80), medium (0.80–2.00) and high (>2.00). Values are those from the fall 2016 sampling. Dominant land-use designations are colormetrically depicted across the landscape.

When USGS screened samples of wastewater from sewage treatment plants in the Columbia River basin for total estrogenicity, it found them to be “well above levels that have

²⁹⁸ Luke R. Iwanowicz et al., *Reconnaissance of Surface Water Estrogenicity and the Prevalence of Intersex in Smallmouth Bass (Micropterus Dolomieu) Inhabiting New Jersey*, 17 Int. J. Environ. Res. Public Health 2024 (2020).

²⁹⁹ *Id.* at 3.

been shown to cause effects in aquatic biota.”³⁰⁰ In its 2012 analysis, the USGS cited previous studies on estrogenic effects:

In Swiss midland rivers, brown trout showed a relationship between sites with higher EEQ values and male fish with elevated vitellogenin levels (Vermeirssen and others, 2005). Colman and others (2009) showed that short-term exposure to estrogenic compounds could alter reproductive success in male zebrafish. In their experiment, one-half of the dominant male zebrafish in waters with EEQ levels of 50 ng/L relinquished their paternal dominance. Kidd and others (2007) designed a study in which Canadian experimental lakes were dosed with varying levels of the synthetic estrogen 17 α -ethynylestradiol to study the long-term effects on fathead minnows. Chronic exposure to low concentrations (5–6 ng/L) led to the feminization of the males through the production of vitellogenin, and ultimately, the near extinction of this species from the lake.³⁰¹

The study calculated the estrogenicity near the City of Portland sewage treatment plant as consistently greater than 1 ng/L (a range of 1.1 to 1.7 over the course of a day), “a concentration that may potentially cause endocrine disruption in different aquatic species.”³⁰²

A 2016 study also looked at endocrine disruption in salmonids from compounds discharged by Puget Sound sewage treatment plants.³⁰³ When juvenile coho salmon were subjected to 72 hours of 17 α -ethynylestradiol (EE2) and 17 β -trenbolone (TREN) at 2 and 10 ng EE2 L, they had 17-fold and 215-fold higher *lhb* mRNA levels relative to control fish and hepatic *vtg* mRNA levels were “dramatically increased 6670-fold, but only in response to 10 ng EE2 L.”³⁰⁴ A second laboratory study exposed juvenile coho to effluent from eight sewage treatment plants (both with secondary and tertiary treatment) from the Puget Sound region for 72

³⁰⁰ Columbia Reconnaissance, *supra* n. 194 at 22–23.

³⁰¹ *Id.* at 23.

³⁰² *Id.* at 23; *see also id.* at 22, Table 11 (Estrogenicity in wastewater-treatment-plant effluent samples, instantaneous loadings, and calculated concentrations in the Columbia River, Columbia River Basin, Washington and Oregon, December 2008).

³⁰³ Louisa A. Harding, *et al.*, *Wastewater treatment plant effluent alters pituitary gland gonadotropin mRNA levels in juvenile coho salmon (Oncorhynchus kisutch)*, 178 *Aquatic Toxicology* 118-131 (2016).

³⁰⁴ *Id.*

hours at 20 or 100 percent effluent or control water, the result of which was that in five of the effluent exposures *lhb* mRNA levels were “significantly elevated.” The study concluded: “Mean levels of natural and synthetic estrogens in fish bile were consistent with pituitary *lhb* expression, suggesting that the observed *lhb* induction may be due to estrogenic activity of the WWTP effluents. These results suggest that *lhb* gene expression may be a sensitive index of acute exposure to estrogenic chemicals in juvenile coho salmon.”³⁰⁵

3. Sublethal Effects to Salmonids

In 2005, regarding salmonids in the Pacific Northwest, scientists observed that “[s]ublethal effects from toxic exposures have been implicated as important factors in population decline” and that “[t]he concept of delayed effects, impacts that extend beyond the life stage in which the exposure occurs . . . is not new and has been observed in salmon.”³⁰⁶ Noting that “[t]here is a growing body of evidence demonstrating that sublethal effects on individual organisms are occurring after exposure to environmentally relevant concentrations,” they cited a few examples known at that time—over 15 years ago:

Field studies by Arkoosh et al. (1998, 2001) documented significant and long-term immunosuppression in Chinook salmon with concentrations of total PAHs in stomach contents as low as 8 ppm wet weight, far lower than stomach contents of fish collected in urban estuaries that ranged up to 365 ppm wet weight (Varanasi et al. 1993). Other studies have simulated environmental conditions in a laboratory setting by exposing eggs and larvae to extracts of contaminated sediments. Heintz et al. (2000) used this method to show decreased growth and marine survival in pink salmon exposed 5.4 ppb to PAHs during development, simulating exposure to weathered crude oil. In another study, 4-d-old surf smelt exposed for 96 h to sediment extracts of 11 and 29 ppm total PAHs exhibited increased mortality, decreased length, and increased abnormal larvae (Misitano et al. 1994). Rice et al. (2000) found a severe growth reduction in English sole (*P.*

³⁰⁵ *Id.*

³⁰⁶ Julann A. Spromberg and James P. Meador, *Relating Results of Chronic Toxicity Responses to Population-Level Effects: Modeling Effects on Wild Chinook Salmon Populations*, 1 Integrated Environmental Assessment and Management 9-21 (2005) at 17.

vetulus) fed polychaete worms exposed to a 0.1% dilution of sediment from Eagle Harbor, Washington, USA. The total PAH tissue concentration in worms of 11.3 ppm (wet weight) fed at 6% body weight/d was high enough to reduce the growth rate in fish by 10- to 25-fold.

In considering the impact of these effects on salmonid populations, these scientists pointed out that at an impact level of 10 percent, “often considered acceptable in regulatory situations,” “the results of our study indicate a substantial reduction in the growth rate of the population.”³⁰⁷

A 2007 study of toxic contamination in the Lower Columbia River built on previous studies showing that toxic contamination exceeded acceptable levels for salmonids, contamination that affects the species ability to swim, smell, and respond to predators:

Reduced swimming and sensory abilities also impair feeding and some toxics inhibit the crucial weight gain that is a key predictor of salmon survival in the ocean. Exposure to toxic contaminants can suppress the immune system; disrupt hormones that influence smoltification and reproduction; alter homing behavior; and leave juveniles susceptible to infectious diseases and parasites. Finally, potential reproductive effects of toxics in adult salmon include production of fewer and smaller eggs, disruption of sperm production, less frequent spawning and egg fertilization, and reduced hatching success.

* * *

By some estimates, exposure to toxic contaminants causes delayed, disease-induced mortality of juvenile Chinook at rates of 1.5 to 9 percent, depending on how long fish reside in the estuary. These figures are for contaminant-related deaths induced by infectious disease only; if indirect mortalities related to other effects of toxic contaminants were included, such as the failure to avoid predators, the rate would be higher.³⁰⁸

This study identified the following contaminants are bioaccumulating in salmon: PCBs, copper, mercury, chromium, nickel, DDTs, dieldrin, chlordanes, and PBDEs.³⁰⁹ It also highlighted the sensitivity of salmonids to toxic contaminants that disrupt the species’ olfactory

³⁰⁷ *Id.* at 18 (internal citations omitted).

³⁰⁸ 2007 Columbia Sampling, *supra* n. 178 at 9 (omitting internal citations) (emphasis added), citing Loge, *et al.*, *Impacts of Environmental Stressors on the Dynamics of Disease Transmission*, 39 *Environmental Science & Technology* 7329-7336 (2005).

³⁰⁹ 2007 Columbia Sampling, *supra* n. 178 at 19.

system that is needed to “detect the amino acids given off by predators and prey, pheromones given off by potential mates, and chemical signals that guide migration.”³¹⁰ The report cited copper and organophosphate insecticides as sources of olfactory impairment. Summarizing other studies, this report noted the following toxic contaminants that

can mimic hormones or alter a salmon’s own hormones. PCBs, for example, lower the thyroid hormones that help trigger smoltification (the physiological process that allows anadromous fish to adapt to a saltwater environment) and govern osmoregulation (the process that maintains the proper concentration of salts and waters in a fish’s body) (LeRoy et al 2006, Brown et al. 2004, Casillas et al. 1995, Zoeller 2005). Hormone disruptors such as DDT, natural and synthetic estrogens, plasticizers, surfactants, and synthetic musks can inappropriately spur or suppress estrogenic activity, which in turn has reproductive effects—sometimes at very small doses (Melnick et al. 2002, Tapiero et al. 2002). Disruption of sperm production and changes in the sex ratio of offspring are both possible effects of exposure to estrogen-like compounds. Other reproductive effects of toxic contaminants include reduced egg production (copper) (Munnicktrick and Dizon 1989), reduced viability of sperm (chromium) (Billard and Roubaud 1985), smaller egg size, lower fertilization rates, and reduced hatching success (PCBs and PAHs) (Carls et al. 2005, Fesit et al. 2005, Incardona et al. 2005, Johnson et al. 1998, Rice et al. 2001).³¹¹

A 2007 study on the Lower Columbia identified copper concentrations of 0.7 to 3.8 µg/L as compared to “concentrations of as low as 1 to 2 µg/L have been shown to inhibit salmon olfactory function . . . and levels within the range seen in this project have been associated with hormonal and immune system changes, reduced growth, and fry mortality in trout species[.]”³¹² In addition, “[n]ickel, silver, and zinc were detected at concentrations high enough to have health effects” while chromium “which can affect salmon feeding, predator avoidance, the immune system, and reproduction” was found at the Lower Willamette River site.³¹³ Notably, the study

³¹⁰ *Id.* at 20.

³¹¹ *Id.* at 20.

³¹² *Id.* at 40.

³¹³ *Id.*

stated that “[a]lthough the concentrations found did not exceed USEPA water quality standards, levels are high enough to affect species,”³¹⁴ demonstrating the inadequacy of the EPA water quality criteria. The key findings from extensive salmon sampling— including stomach contents of juvenile salmon, juvenile tissue, vitellogenin testing, and bile—were as follows:

- Prey were determined to be a source of PCBs, PAHs, DDTs, and PBDEs in salmon.
- PCBs, PAHs, and DDTs were “above estimated threshold levels for health effects.”³¹⁵
- Vitellogenin levels indicate exposure to estrogenic compounds.
- Industrial and urban areas are major contributors of toxic loads.
- Salmon at one site had lipid profiles “similar to those of malnourished fish.
- Upstream fish appear to be “absorbing significant amounts of PCBs as they rear in the tidal freshwater portions of the lower river.”³¹⁶
- DDTs are pervasive and evenly distributed in the Lower Columbia River.
- Juvenile salmon from every site had PBDEs and upriver stocks appear to be “absorbing much of their PBDE load while rearing in the tidal freshwater areas.”³¹⁷

Because recommended water quality criteria have not been developed for most of the findings, the report used thresholds derived from other studies for PCBs in whole fish, PAHs in stomach contents, DDTs in tissue, olfactory impacts from current use pesticides, PBDEs in tissue, and vitellogenin as demonstrated in the chart below.³¹⁸

³¹⁴ *Id.* at 41.

³¹⁵ *Id.* at 43.

³¹⁶ *Id.* at 41–50.

³¹⁷ *Id.* at 52–54.

³¹⁸ *Id.* at 50–52.

Concentrations and Estimated Thresholds for Health Effects in Juvenile Salmon			
Toxic Contaminant	Threshold	Exceeded?	Concentration Range
PCBs	2,400 ng/g lipid (in whole bodies)	Yes	33 - 39,000 ng/g lipid
PAHs	~7,000 ng/g wet weight total (in diet)	Yes	34 - 10,000 ng/g wet weight
Copper	0.2 – 2 µg/L in water (for impacts on olfaction)	Yes	0.7 – 3.8 µg/L in water
DDTs	5,000 – 6,000 ng/g lipid (in whole bodies)	Yes	78 - 5,500 ng/g lipid
Pesticides in Current Use	~0.5 µg/L in water for chlorpyrifos (for impacts on olfaction)	No	0.003 – 0.27 µg/L in water column, for various pesticides
PBDEs	None established	NA	< 1 - 93 ng/g wet weight (whole bodies)
Estrogenic Compounds/ Vitellogenin	None established; varies based on contaminant	NA	0 - 20 ng/mL vitellogenin (in blood)

The USGS 2012 study on the Lower Columbia River summarized the findings of other studies on the health of river species:

Recent research has raised questions about potential effects on fish, shellfish, wildlife, and human health from even trace exposure to these contaminants, including chronic effects (Kidd and others, 2007; Ings and others, 2011), reproductive disruption (Vajda and others, 2008; Colman and others, 2009; Jenkins and others, 2009), and physiological changes (Hoy and others, 2011).³¹⁹

In Puget Sound, studies have also been done on sublethal effects on salmonids, including the effects of roughly 97,000 pounds of CECs annually discharged to the Sound.³²⁰ In 2014, the National Marine Fisheries Service Science Center demonstrated that juvenile Chinook salmon transiting contaminated estuaries of Puget Sound exhibited an overall rate of survival 45 percent lower than Chinook moving through uncontaminated estuaries.³²¹ In 2016, these scientists analyzed effluent discharged by sewage treatment plants and fish tissue in the estuaries receiving the discharge, finding a large number of CECs in both estuary water and fish tissue (the study

³¹⁹ Columbia Reconnaissance, *supra* n. 194 at 2.

³²⁰ James Meador *et al.*, *Contaminants of emerging concern in a large temperate estuary*, 213 *Environ. Pollut.* 254-267 (June 2016) (hereinafter “Meador 2016”) at 8–9.

³²¹ James P. Meador, *Do chemically contaminated river estuaries in Puget Sound (Washington, USA) affect the survival rate of hatchery-reared Chinook salmon?*, 71 *Can. J. Fish. Aquatic. Sci.* 162-180 (2014).

detected 81 analytes in effluent, 25 analytes in estuary water, and 42 analytes in fish tissue) at levels that may cause adverse impacts, some of which were found only in tissue (“indicating a high potential for bioaccumulation”).³²² In 2018, this group demonstrated that Chinook fed toxic compounds at the same level as found in the contaminated estuaries experienced reduced growth rates and metabolic disruptions, a “pattern generally consistent with starvation” that “may result in early mortality or an impaired ability to compete for limited resources.”³²³

In 2020, continuing this effort, metabolomic analyses were conducted on juvenile Chinook salmon rearing in the same two estuaries affected by sewage treatment plant effluent and the reference estuary as well as exposing hatchery fish in the lab to a mixture of just 16 CECs, while noting there are “more than 3700 approved pharmaceuticals . . . not including illicit drugs, experimental and biotech drugs, a number of over-the-counter medicines, nutraceuticals, and personal care products. . . . [and] exogenous CEC metabolites, which may be as potent or more potent than the parent compound.”³²⁴ The data show “[t]he results from this study indicate altered metabolomes for juvenile Chinook salmon after a few weeks in effluent-receiving estuaries.”³²⁵

The results of this study reveal strong differences between juvenile Chinook residing in effluent-receiving estuaries compared to similar aged fish from a reference estuary and fish from a hatchery that is up-stream of an effluent-receiving estuary. An evaluation of their metabolomes indicates substantial differences for a large number of endogenous metabolites and most of these were reduced (especially amino acids) compared to reference fish. The high degree of altered metabolomes among sites and strong group patterns in the targeted approach was confirmed with the nontargeted approach utilizing liver from different fish collected concurrently. Because the metabolomes in fish from

³²² Meador 2016, *supra* n. 320.

³²³ James Meador *et al.*, *Adverse metabolic effects in fish exposed to contaminants of emerging concern in the field and laboratory*, 236 *Environmental Pollution* 850-861 (2018).

³²⁴ *Id.* at 8.

³²⁵ *Id.* at 6 (emphasis added).

effluent-receiving waters were altered and the general health parameters (lipid content and condition factor) were not different compared to reference site fish, we can surmise that these alterations may be an early indicator of adverse effects that can potentially translate into population relevant outcomes.³²⁶

The following graph illustrates altered pathways found in Puget Sound estuaries affected by the discharge of treated sewage:³²⁷

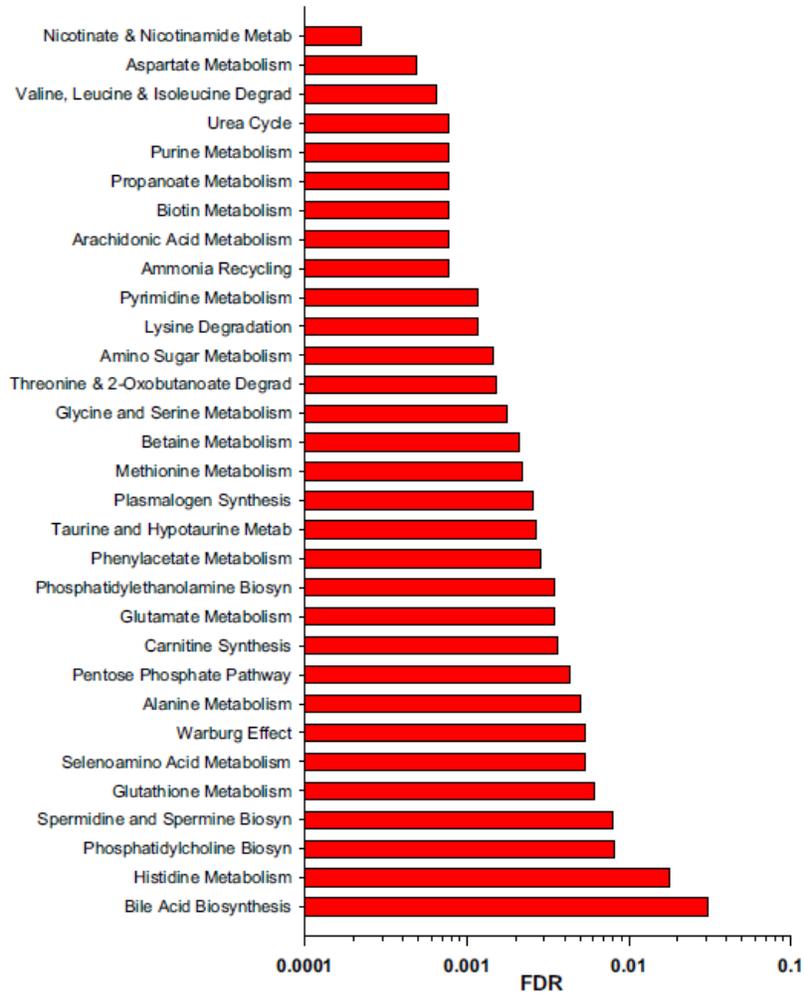


Fig. 7. Altered pathways in common between Puyallup and Sinclair fish when compared to reference site fish. All FDRs ≤ 0.05 and values are shown for fish from the Puyallup estuary compared to Nisqually estuary reference fish. Many FDRs (42%) were lower for Sinclair versus Nisqually fish.

³²⁶ James Meador *et al.*, *Metabolomic profiling for juvenile Chinook salmon exposed to Contaminants of emerging concern*, 747 *Science of the Total Environment* 141097 (2020) at 12.

³²⁷ *Id.* at 9.

Likewise, another 2020 study summarized the latest understanding of how toxic contaminants affect salmonids:

Numerous laboratory and field assessments have demonstrated the adverse effects of contaminants on the health of salmonids. Toxic contaminant exposure can directly impact the health of juvenile salmon by impairing growth, and metabolism (Varanasi et al., 1993; Meador et al., 2006), altering hormone levels (Arkoosh et al., 2010, and 2013), and disrupting reproductive development (Peck et al., 2011). Toxic contaminants can also impair immune function of salmon either alone (Arkoosh et al., 1994, 2010, 2015, and 2018), or in conjunction with other stressors (Jacobson et al., 2003), thereby increasing their susceptibility to naturally occurring infectious diseases, potentially leading to population level effects (Arkoosh et al., 1998; Loge et al., 2005; Spromberg and Meador, 2005). Impairment of immune response is particularly important for endangered and threatened salmonid species and populations because a properly functioning immune system is vital for both individual survival and population productivity (Segner et al., 2012).³²⁸

A 2022 study looked at CECs and other toxics in the effluent and receiving water of King County’s sewage treatment plants that discharge to Puget Sound.³²⁹ Up to 121 unique contaminants were detected in the effluent samples.³³⁰ The study included 10-day laboratory exposures of juvenile Chinook salmon at different dilutions,³³¹ estuarine sampling,

³²⁸ Nisqually Sources and Pathways, *supra* n. 222 at 1.

³²⁹ James Meador et al., *Academic Team Project Integration Report King County Orca Proviso, Wastewater Effluent Discharge Assessment – Impact to Marine Organisms* (Oct. 2022) (hereinafter “King County CECs”), Appendix M to King County, *Toxics in King County Wastewater Effluent, Evaluating the Presence of Toxic Elements in the Effluent of Treatment Plants* (Dec. 2022). The number of analytes organized by contaminant class and water sample type are set out in Table 2, *id.* at 13.

³³⁰ *Id.* at 118; *see also id.* (“There were 14 compounds consistently found at greater concentrations under low flow conditions, suggesting municipal sewage is their primary conveyance to wastewater effluent. These included hormones (17β-estradiol, androstenedione, estrone, and progesterone) and several medications (atorvastatin, carbamazepine, diazepam, and hydrocodone).”).

³³¹ Note that the authors pointed to the constraints of a 10-day exposure: “Importantly, it is likely that the vitellogenin response in our study underestimated the response of chronic exposure to estrogenic hormones in Puget Sound. In fish exposed for 21 days to 20 ng/L of 17α-ethinylestradiol vitellogenin continued to increase over the exposure, peaking beyond the end of the exposure with a half-life of two to four weeks among the species tested (Craft et al. 2004). Therefore, although vitellogenin was not significantly elevated at the lower WWE concentrations in our 10-day exposure, we would expect that chronic exposure would approximate the response of juvenile Chinook exposed to higher WWE concentrations in our study.” *Id.* at 118.

bioaccumulation modeling, and chemical characterization of effluent. Among its findings were the following:

In the laboratory study, juvenile Chinook exposed to [wastewater effluent] WWE showed evidence of endocrine disruption and alterations in the stress response, brain function, and metabolism. Brain function and total plasma protein were affected at low exposure concentrations, whereas other endpoints exhibited a dose response relationship with measurable differences from control evident only at the higher concentrations. However, some of the endpoints (e.g., endocrine disruption) are expected to show more pronounced effects with longer exposure durations than in the laboratory study. Higher exposure concentrations in the laboratory study may therefore be indicative of effects resulting from chronic exposures, which occur in Puget Sound.

* * *

Metabolomics analysis showed that WWE altered numerous endogenous biochemical pathways important for energy generation and utilization, lipid metabolism and biosynthesis, amino acid metabolism, growth, and oxidative stress. Pathway analysis implicated pharmaceuticals that act as antibiotics, antidepressants, antihistamines, analgesics and statins even at the lowest WWE concentrations tested (0.1% and 0.4%), although other chemicals present in WWE may have contributed.

Additional pharmaceuticals were predicted to cause harm based on a fish plasma model of bioaccumulation from tissue and water chemistry in exposed juvenile Chinook. As with metabolomics, impacts were in many cases predicted at environmentally relevant concentrations of WWE. Impacts to juvenile Chinook observed and predicted for this study are hypothesized to contribute to reduced availability as prey for [endangered Southern Resident killer whales] SRKWs. Additionally, exposure to several classes of contaminants based on bioaccumulation modeling for Chinook likely contribute to health impairments in SRKW.³³²

The report illustrates its findings on total and summed 11 congeners of PBDEs as compared to other studies on PBDEs in effluent and receiving water in the Puget Sound

³³² *Id.* at vi. Note that the bioaccumulation modeling done in this study “is limited to uptake from water via gill ventilation; however dietary uptake is also a major contributor to body burdens, especially for hydrophobic compounds.” *Id.* at 133.

region:³³³

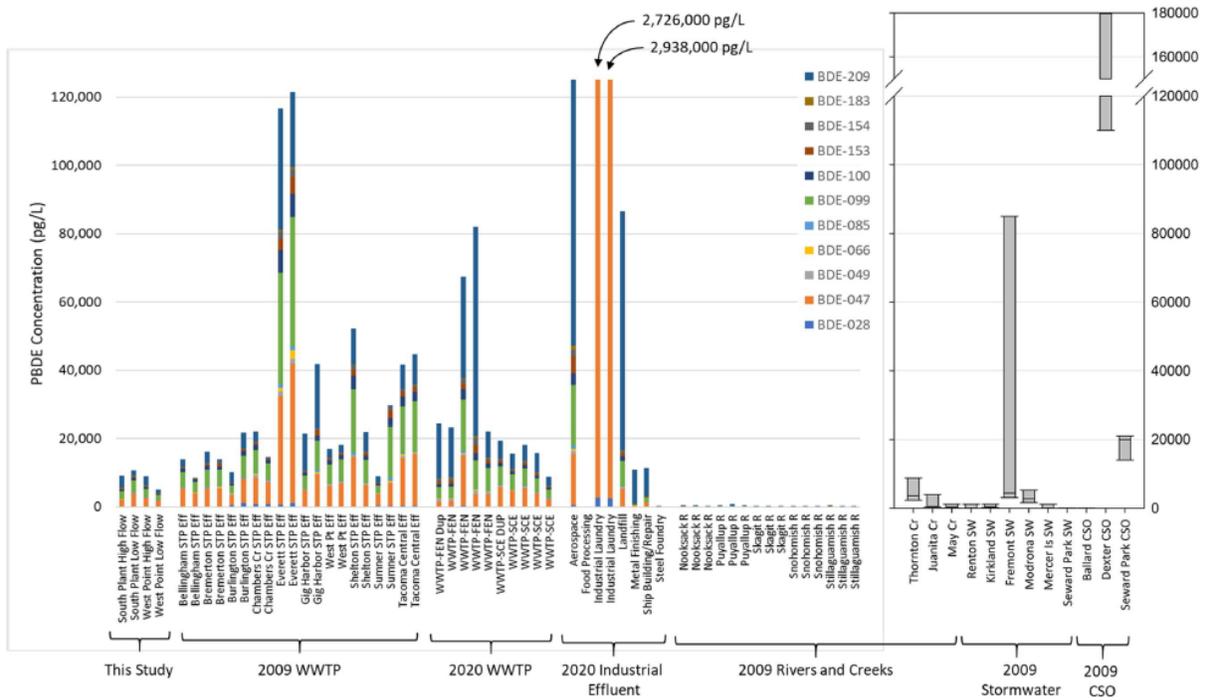


Figure 5. Σ_{11} PBDE concentration (left) and reported total PBDE concentrations (grey bars, right) for effluent water from different facilities, and surface waters in the Puget Sound. The 2009 WWTP data are from Washington State Department of Ecology (2010). 2009 rivers, creeks, and CSO data are from King County (2013b) and Washington State Department of Ecology (2011). 2020 WWTP and 2020 Industrial Effluent are from Washington State Department of Ecology.

It concluded that “several individual PBDEs and flame retardants may occur in fish tissue at concentrations able to cause adverse effects in juvenile Chinook.”³³⁴

In addition to evaluating PBDEs, the report used a toxicity screening approach to identify nine high priority compounds with “likely biological effects” based on a combination of toxicity quotients and exposure activity ratios, including: 17 β -estradiol, azithromycin, bisphenol A, diatrizoic acid, estrone, iopamidol, theophylline, triamterene, and venlafaxine.³³⁵ A further list

³³³ *Id.* at 34.

³³⁴ *Id.* at 133 (internal citations omitted).

³³⁵ *Id.* at 42.

of compounds with “potential for biological effects” was identified on a “Watch List.”³³⁶ The report noted that “there were several compounds that, based on measured concentrations in treatment system effluent, exceeded biological response thresholds by 100-1000x (venlafaxine, iopamidol, estrone, erythromycin, diatrizoic acid, bisphenol A, and 17β-estradiol). As such, a dilution of that same magnitude (100-1,000x) in estuarine waters would still leave the potential to elicit a biological response to exposed organisms.”³³⁷ The analysis also predicted a number of pharmaceuticals “to bioaccumulate at levels of 0.05 ng/g or greater, including estrone, diphenhydramine, miconazole, lopamidol, metformin, carbamazepine, diatrizoic acid, and valsartan.”³³⁸

The laboratory studies demonstrated significant effects on brain values of Na⁺/K⁺ ATPase activity levels in juvenile Chinook, as illustrated by the following graph (right side):³³⁹

³³⁶ *Id.* at 43, table 11 (“Yellow shaded cells indicate a “Watch List” compound for the given comparison.”) This list includes: 1,7-Dimethylxanthine; 10-hydroxy-amitriptyline; 2-Hydroxy-ibuprofen; Amitriptyline; Androstenedione; Atorvastatin; BEHTBP; Bisphenol F; Bisphenol S; Caffeine; Carbamazepine; Ciprofloxacin; Clarithromycin; Cloxacillin; Colchicine; Cotinine; Dehydronifedipine; Diphenhydramine; Doxycycline; EHTBB; Enrofloxacin; Erythromycin-H₂O; Fluoxetine; Fluticasone propionate; Furosemide; Glipizide; Hydrochlorothiazide; Ibuprofen; Metformin; Miconazole; Naproxen; Progesterone; Propranolol; Ranitidine; Rosuvastatin; Sertraline; Sulfamethoxazole; Testosterone; Thiabendazole; Triclocarban; Triclosan; 4-Nonylphenol diethoxylates; 4-Nonylphenol Monoethoxylates; 4-Nonylphenols; PFHxA; PFHxS; and PFOS. *Id.* at 43–47.

³³⁷ *Id.* at 48.

³³⁸ *Id.* at 83.

³³⁹ *Id.* at 55, fig. 11. *See also id.* at 119 (“Recent studies have shown that psychoactive pharmaceuticals may also cause detrimental effects on brain function in fish (Ajima et al. 2017; Lajeunesse et al. 2011; Xie et al. 2015). Pharmaceuticals are often more potent than other contaminants as they are designed to elicit specific biological effects at relatively low concentrations. Additionally, many pharmaceuticals require repeated consumption, meaning that they are continually introduced into local waterways via WWE.”); 121 (“There were also likely a large number of PPCPs in the low dose treatments that occurred below the limit of analytical detection as noted in the bioaccumulation section of this report. Some of these are potent drugs that may cause effects at these very low concentrations.”).

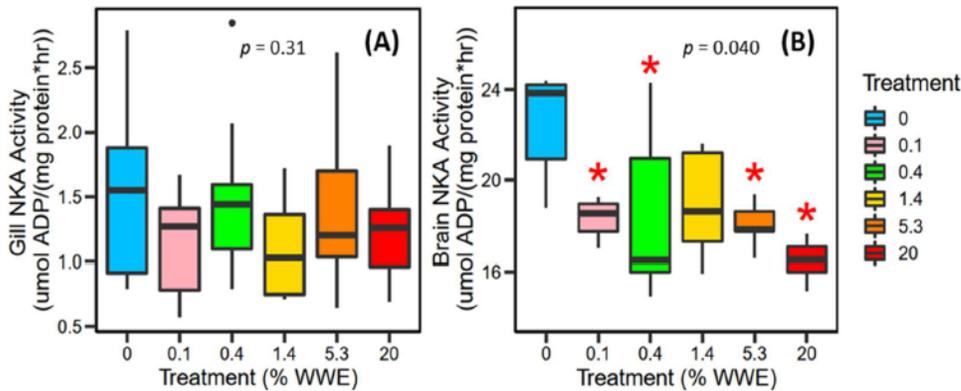


Figure 11. Activity levels of sodium/potassium ATPase (NKA) from different concentrations of wastewater effluent (% WWE) for gills (A) and brains (B) of juvenile Chinook. Boxes represent the 25th percentile, median (middle) and 75th percentile, while lines extending from boxes represent the ranges excluding outliers. Dots represent outliers. Treatments that were significantly different from controls are marked with an asterisk. P-values represent the results from the Kruskal-Wallis tests.

Similarly, both lipid content and liver anomalies were affected by increased exposure to wastewater in the study:³⁴⁰

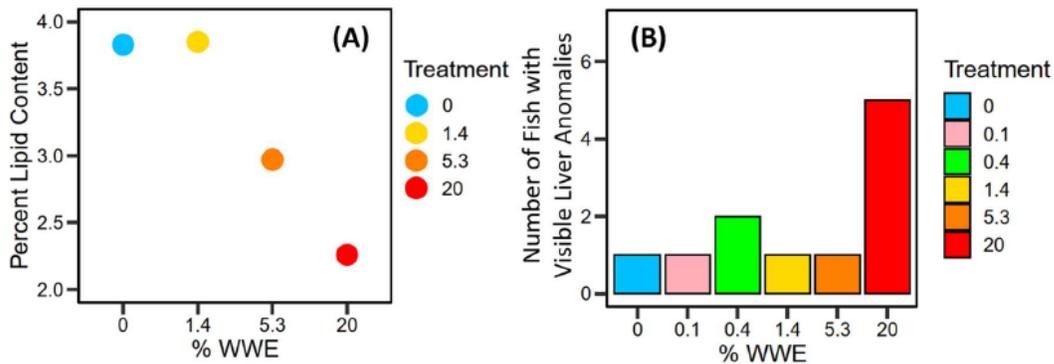


Figure 12. Lipid content and liver anomalies for fish exposed to wastewater effluent (WWE). (A) Percent lipid content of composited samples (n=7 fish) for treatments with tissue chemistry analysis. (B) Total number of fish in each treatment with a visible liver anomaly. Each fish with a visible liver anomaly was from a different replicate tank and there were a total of 7 tanks per treatment and 56 fish per treatment.

In summary, “[t]he present study is an example of how exposure to contaminants does not just alter one aspect of physiology but affects multiple pathways. For example, many agonists of endocrine receptors also affect metabolic pathways and behavior, highlighting that metabolism,

³⁴⁰ *Id.* at 56, fig. 12.

the endocrine system, stress, and brain function are linked.”³⁴¹ Noting that “[a]ll of the wastewater concentrations elicited changes in several important endogenous metabolites in juvenile Chinook,”³⁴² and integrating these multiple effects on salmon, the authors set out their conclusion:

Given that juvenile salmon are vulnerable to contaminant exposure, it is crucial to understand how anthropogenic pollution affects their physiology. Juvenile salmonid size while migrating through estuaries plays a pivotal role in salmonid survival (Burrows 1969; Tovey 1999; Beamish et al. 2004). Any decrease in growth or affected physiology that could decrease predator avoidance (reduced fitness, altered behavior, stress) could be fatal for a juvenile salmonid. Ocean type Chinook, used in this study, are more vulnerable to contaminated estuaries than stream type due to their increased use of this habitat (Quinn 2005). Contamination from multiple sources, including WWTPs, and the loss of critical rearing habitat exacerbate the threats to salmonid survival in Puget Sound.³⁴³

Demonstrating the far-field attributes of CECs, 10 analytes were detected in the estuarine water samples including five found at all sites: atenolol, cotinine, metformin, venlafaxine, and benzoylecgonine.³⁴⁴ The authors concluded that this finding “suggests that 1) effluent plume was not captured in the sampling, and 2) the 10 chemicals are present at a pseudo steady-state due to factors such as continual inputs, high persistence, and a well-mixed water mass.”³⁴⁵ The study also compared the laboratory and field results, finding that the observed field concentrations were the equivalent of the 0.4 percent dilution treatment and concluding: “We believe these results support our predictions of potential adverse effects to fish in the field based on our metabolomics and blood chemistry results for the 0.4% lab exposure treatment.”³⁴⁶

³⁴¹ *Id.* at 122.

³⁴² *Id.* at 120.

³⁴³ *Id.* at 123.

³⁴⁴ *Id.* at 81.

³⁴⁵ *Id.*

³⁴⁶ *Id.* at 83.

Yet to be published (after completion of peer review) is a 2023 study that has identified 57 high priority chemicals in Puget Sound treated sewage, “nearly half [of which] are antibiotics, about 18% are hormones, another 18% are pharmaceuticals, and 10% are perfluorinated substances.”³⁴⁷ Using multiple lines of evidence, this study identified CECs most likely to elicit a biological response, using “[m]onitoring data from water, wastewater, and tissue samples from 20 different campaigns combined with multiple biological response measures . . . compared with appropriate threshold values.”³⁴⁸ Out of 226 CECs detected, 57 were identified as high priority (“likely to cause a biological effect”) and 84 were denominated “Watch List” due to their “potential to cause biological effects.”³⁴⁹ Four biological response measures were used in this analysis: (1) marine predicted no effects concentrations based on experimental data and modeling; (2) hazard concentrations for five percent of species; (3) ecological hazard assessment screening values from the FWS’s Ecological Hazard Assessment; and *in vitro* high throughput screening.³⁵⁰ “More than one biological response measure was available for 150 compounds; 68 compounds were represented by two measures, 74 compounds were represented by three measures, and eight compounds were represented by four measures. Biological response ratios [“BRR”] were calculated for a total of 207 compounds.”³⁵¹ The analysis also demonstrated the importance of monitoring in fish and shellfish tissue, as well as sewage treatment effluent, as

³⁴⁷ John Ryan, KUOW, *Scientists try to keep up with chemical blizzard entering Puget Sound* (Feb. 2, 2023), available at <https://kuow.org/stories/scientists-try-to-keep-up-with-chemical-blizzard-entering-puget-sound>.

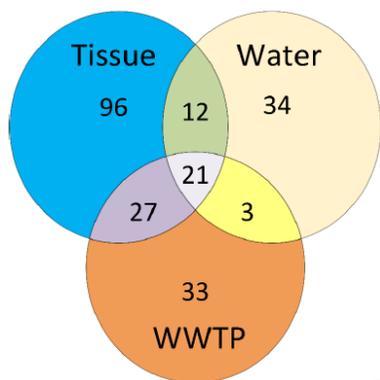
³⁴⁸ C. Andrew James, *et al.*, *The Screening and Prioritization of Contaminants of Emerging Concern in the Marine Environment Based on Multiple Biological Response Measures* (preprint article Feb. 19, 2023) at 2, available at https://papers.ssrn.com/sol3/papers.cfm?abstract_id=4363581.

³⁴⁹ *Id.*

³⁵⁰ *Id.* at 8–10.

³⁵¹ *Id.* at 19.

illustrated by this Venn diagram³⁵² and related table³⁵³ showing the number of individual compounds detected in each matrix:



Matrix	Biological Response Measure	Total Evaluated	Watch List (Cat 2)	High Priority (Cat 1)
Marine water	EAR	49	6	0
Marine water	TQ	68	9	1
Marine water	HQ _{HCS}	46	3	0
Marine water	HQ _{SV}	7	3	0
WWTP effluent	EAR	52	17	7
WWTP effluent	TQ	81	37	15
WWTP effluent	HQ _{HCS}	48	17	2
WWTP effluent	HQ _{SV}	9	4	5
Tissue (mussel)	EAR	26	7	4
Tissue (fish)	EAR	63	19	14

4. Marine Mammals and Other Aquatic and Aquatic-Dependent Species

Writing broadly in 2000, EPA noted that “[i]n wildlife, birds exposed to [bioaccumulative chemicals of concern] BCC have exhibited biochemical dysfunction and metabolic effects, behavioral/neurological disorders, and reproductive impairment.”³⁵⁴ Judging from EPA’s current website, the agency is not concerned about the effects of toxics on marine mammals and other aquatic and aquatic-dependent animals nationwide. It has, however, expressed concern about marine life in Puget Sound, for which it has awarded a “declining trend” based on its assessment that “the total number of marine species at risk in the Salish Sea has doubled from

³⁵² *Id.* at 19, fig. 1 (“Venn diagram showing the number of individual compounds detected in each matrix. The tissue numbers also account for detections in bile and liver samples which were not used to calculate BRRs.”).

³⁵³ *Id.* at 21 (“Summary of results of biological response ration (BRR) evaluation for marine water, WWTP effluent, and tissue samples. Marine water concentrations were either measured directly, or estimated with BCFs from mussel and fish tissue concentrations. The threshold values for each category for each BRR are included in Table 1.”).

³⁵⁴ Final Rule to Amend the Final Water Quality Guidance for the Great Lakes System To Prohibit Mixing Zones for Bioaccumulative Chemicals of Concern, 65 Fed. Reg. 67638, 67641 (Nov. 13, 2000) (hereinafter “Great Lakes Mixing Zones”).

2002 to 2015.”³⁵⁵ EPA noted that as of December 2015, in the Salish Sea, 126 marine species were at some risk of extinction, including 59 birds, 16 mammals, five invertebrates, and two reptiles.³⁵⁶ It noted too that of the 253 species of fish, 172 birds, 37 mammals, and “many more invertebrate species like crabs, mussels, shrimp, and worms” that use the Salish Sea, “[n]early 50% of these birds and 80% of these mammals depend on the ecosystem for habitat to feed, reproduce, and care for their offspring.”³⁵⁷ EPA confirms that the Sound’s Southern Resident killer whales are threatened by “heavy exposure to toxic pollutants.”³⁵⁸

The Puget Sound and Columbia River basin salmonids discussed above are prey for the endangered Southern Resident killer whale. In particular, these killer whales rely on the Chinook salmon because of their large size and high lipid content.³⁵⁹ In 2006, a study by the WDFW and NMFS assessed the source of elevated contaminant exposure in the Southern Resident as compared to other killer whale populations. The study concluded that “regional body burdens of contaminants in Pacific salmon, and Chinook salmon in particular, could contribute to the higher levels of contaminants in southern resident killer whales” because “[o]verall, concentrations of POPs were higher in coho and Chinook populations that have more coastal distributions than those measured in salmon species (e.g., chum, pink, sockeye) with more oceanic distributions.”³⁶⁰ In addition, “Chinook salmon that resided in Puget Sound in the

³⁵⁵ EPA, Salish Sea, *Marine Species at Risk*, available at <https://www.epa.gov/salish-sea/marine-species-risk>.

³⁵⁶ *Id.*

³⁵⁷ *Id.*

³⁵⁸ *Id.*

³⁵⁹ See e.g., Sandra O’Neill, et al., *Energy content of Pacific salmon as prey of northern and southern resident killer whales*, 25 *Endang Species Res* 265-281 (October 2017).

³⁶⁰ Sandra O’Neill, et al., *Regional patterns of persistent organic pollutants in five Pacific salmon species (*Oncorhynchus spp*) and their contributions to contaminant levels in northern and southern resident killer whales (*Orcinus orca*)*, 2006 Southern Resident Killer Whale Symposium, NMFS (April 3-5, 2006) at 3, 4.

winter rather than migrate to the Pacific Ocean (“residents”) had the highest concentrations of POPs[.]”³⁶¹ Other authors have observed with regard to PCBs, that

adult Chinook sampled in central Puget Sound exhibited mean concentrations of 86 µg/kg, which translated to 800 µg/fish.

* * *

Gockel and Mongillo (2013) noted that for killer whales to have blubber concentrations below a 17 mg/kg threshold, their prey would need to be less than 8 µg/kg tPCBs, which is lower than the values for offshore juvenile Chinook reported by O’Neill et al. (2015). A recent modeling effort evaluating the effects of PCBs on killer whale population growth predicts a decline for populations from various geographic locations with total PCB blubber concentrations ranging from 28-83 mg/kg lw (Desforges et al. 2018). All of the 14 SRKWs listed in Mongillo et al. (2012) exhibited concentrations of tPCBs in blubber close to or within this range considered by Desforges et al. (2018) to affect population growth of the world’s Orca whales. Total PCB concentrations higher than this range were predicted to have stronger effects on population growth.³⁶²

Of specific concern to NMFS are the additive properties of multiple toxic pollutants on endangered killer whales:

Health effects from exposure to PCBs, PBDEs, and DDTs should not be considered in isolation. Killer whales are exposed to a mixture of pollutants, some of which may interact synergistically and enhance toxicity, influencing the health of the Southern Residents. Although it is difficult to predict health effects from mixture interactions, it is important to predict the toxicity of such mixtures; disregarding the interactive effects may underestimate risk to an individual or to the population. Furthermore, we also stress the importance of establishing the impact on the health of killer whales of the transformed by-products, or metabolites, of the pollutants. The practice of examining only high doses of POPs may also underestimate the risk to the killer whales. Endocrine disruptors can produce non-linear dose–response effects and interact at lower doses than would occur with the isolated chemicals. Therefore, even low concentrations of persistent pollutants, when combined, have the potential to cause adverse effects in Southern Residents.³⁶³

NMFS focused on these persistent pollutants “because they are found at relatively high levels in

³⁶¹ *Id.* at 4.

³⁶² King County CECs, *supra* n. 329 at 126–127.

³⁶³ NMFS, *Exposure to a Mixture of Toxic Chemicals: Implications for the Health of Endangered Southern Resident Killer Whales* (Nov. 2016) at vii.

the whales” and they

have the ability to cause endocrine disruption, reproductive disruption or failure, immunotoxicity, neurotoxicity, neurobehavioral disruption, and cancer. The average concentration of blubber summed PCBs (Σ PCBs) in male Southern Resident killer whales sampled between 2004 and 2013 was $45,000 \pm 31,000$ ng/g lw (lipid weight), which exceeds a health effects threshold in harbor seals (*Phoca vitulina*). Average blubber Σ PBDEs in sampled Southern Residents were $4,800 \pm 3,500$ ng/g lw, with most individuals exceeding the levels associated with altered thyroid hormone levels in post-weaned and juvenile gray seals (*Halichoerus grypus*). Although there has been no report in the literature on a marine mammal health effect threshold for DDTs, Σ DDTs levels in the blubber of Southern Residents were high, and ranged from 1,200 to 210,000 ng/g lw.³⁶⁴

NMFS explained that in addition to the killer whales’ high body burden, other stressors increase the likelihood of a toxic response, including “nutritional stress from reduced Chinook salmon populations [that] may act synergistically with high POP levels in Southern Residents and result in deleterious health effects”³⁶⁵ and the timing of the exposure. When killer whale calves are exposed, toxics may compromise their immune system and increase disease susceptibility, “a large source of morbidity and mortality in marine mammals” as well as result in “alterations to the individual’s metabolism, impeded growth and development, delayed or premature physical or sexual maturity, reduced future fecundity, or reduced perinatal survival.”³⁶⁶ Finally, exposure during neurodevelopment can reduce learning, affecting a “killer whale’s capacity to successfully forage and interact with other pod members.”³⁶⁷

Other marine mammals in Puget Sound are affected by bioaccumulative chemicals, such as harbor seal pups that have been studied over many years because they are “highly contaminated, represent an integration of concentrations in a broad selection of prey in a region,

³⁶⁴ *Id.* at vii.

³⁶⁵ *Id.*

³⁶⁶ *Id.* at viii.

³⁶⁷ *Id.*

reflect health and physiological consequences from contaminant exposure and, with the utilization of non-emaciated live-captured pups, provide minimal inter-sample variability allowing sensitive detection of changes over space and time.”³⁶⁸ For example, a 2011 study found:

- harbor pups near urban areas were “more PCB- and PBDE-contaminated” than pups from less urban areas;
- “despite regulations imposed in the mid 1970s, PCBs remain the top ranked contaminant of concern in harbor seals”;
- “PBDE concentrations have been doubling every 3.5 years in the aquatic environment and are rapidly emerging as a concern to aquatic wildlife”;
- and “Vitamin A levels were lower in harbor seal pups from contaminated sites, and some gene expression endpoints (E α , hsp70, and PPAR γ) were associated with contaminant concentrations, suggesting that harbor seal health is affected by persistent contaminants in Puget Sound.”³⁶⁹

On the other side of the country, EPA has drawn similar conclusions in what it terms a “One Health Assessment” that the information gathered and analyzed by the Agency for Toxic Substances and Disease Registry (“ATSDR”) to evaluate human health impacts from members of the Penobscot Indian Tribe’s consuming toxic-contaminated fish and turtles in Maine, also affect wildlife:

Fish from the Penobscot River are also consumed by wildlife. The research team implemented a One Health Approach, which not only takes human risk into account, but also considers the interconnectedness with wildlife and environmental health. Several species depend on the Penobscot River for survival. Researchers calculated the impact of the contaminants on wildlife by concerting the fish fillet samples to whole fish concentrations and comparing them to wildlife values. The mercury, PCBs, and PFAS levels found in rainbow smelt, striped bass, and sea lamprey put animals who consume them at risk, including mink, otters, and eagles.³⁷⁰

³⁶⁸ Marie Noël, *et al.*, *Toxic Contaminants in Harbor Seal (Phoca vitulina) Pups from Puget Sound* (March 2011) (hereinafter “Puget Sound Seal Pup Contamination”).

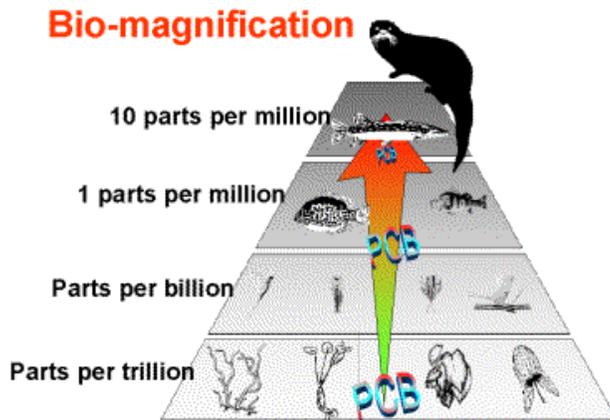
³⁶⁹ *Id.* at 51.

³⁷⁰ EPA, Related Topics: Science Matters, *One Health Assessment: Fish Returning to the Penobscot River* (Nov. 29, 2022), available at <https://www.epa.gov/sciencematters/one-health-assessment-fish-returning-penobscot-river>.

Mink and otter identified in Maine are well known across the country for their place in the food web, consuming a high level of aquatic life compared to their body weights. As the New York Department of Environmental Conservation describes river otter, they are:

at the top of the food chain, they have a greater chance of being exposed to elevated levels of environmental contaminants such as PCBs, DDT and its associated metabolites, and heavy metals such as cadmium and mercury. This means of exposure is referred to as bio-magnification. As contaminants accumulate in the organic materials and sediments on the bottom of a waterway, they become ingested by aquatic invertebrates such as snails, mussels, and insects. These are in turn consumed by fish, which may then be eaten by larger fish, all of which are consumed by river otter. This accumulating effect results in elevated levels of pollutants in river otter due to the ingestion of contaminated food items. At such high levels, some of these contaminants can have negative impacts on otter ranging from poor survivorship to sterility or infertility.³⁷¹

This process of biomagnification is illustrated as follows:³⁷²



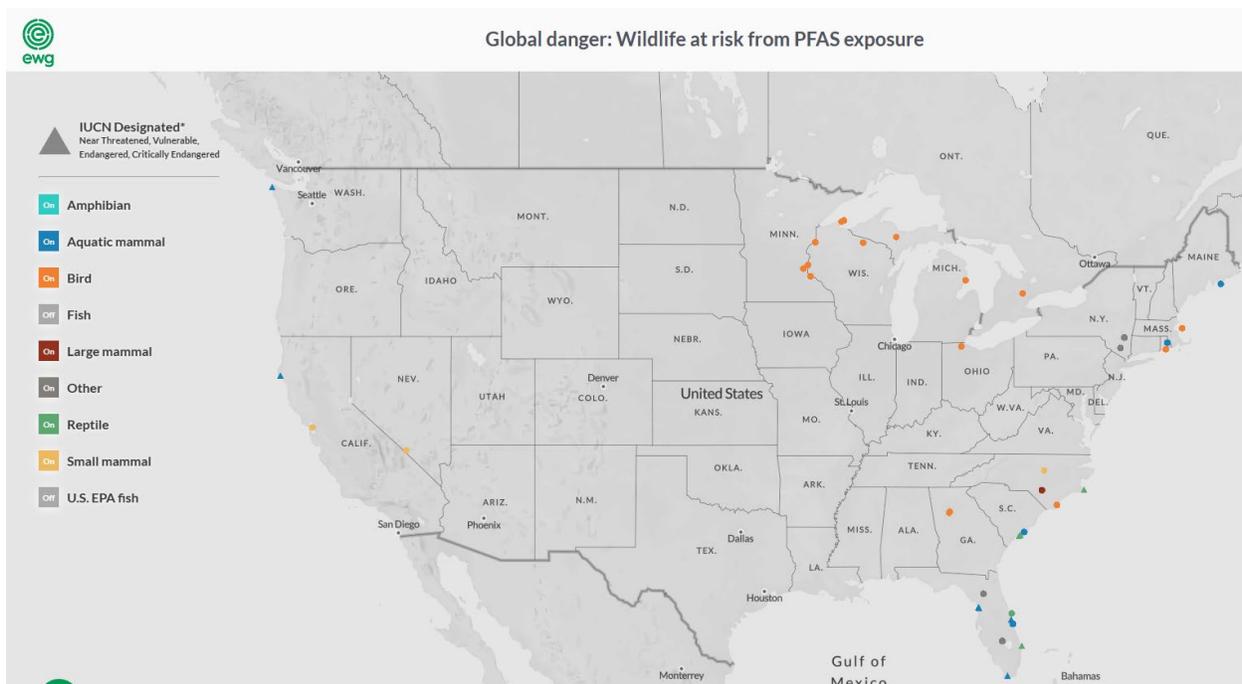
Similarly, levels of mercury, organochlorine pesticides, PCBs, PBDEs, and dioxins and furans have been assessed in river otters in New Jersey where levels of pesticides and PCBs were

³⁷¹ New York Department of Environmental Conservation, Animals, Plants, Aquatic Life, Mammals, *River Otter*, available at <https://www.dec.ny.gov/animals/9355.html>.

³⁷² *Id.*

found comparable to Oregon and Washington. The study concluded that while “the data suggest that contaminant concentrations are not high enough to adversely affect the overall otter population in New Jersey. . . . contaminant-related effects on the health or reproductive success of individual otters in some areas are possible.”³⁷³

A new effort has mapped the locations in the United States and the world where wildlife are at risk from the “forever chemical” PFAS, discussed in sub-sections V.B.3 and VII.B, *infra*. The results for wildlife, excluding fish, are shown in this map:³⁷⁴

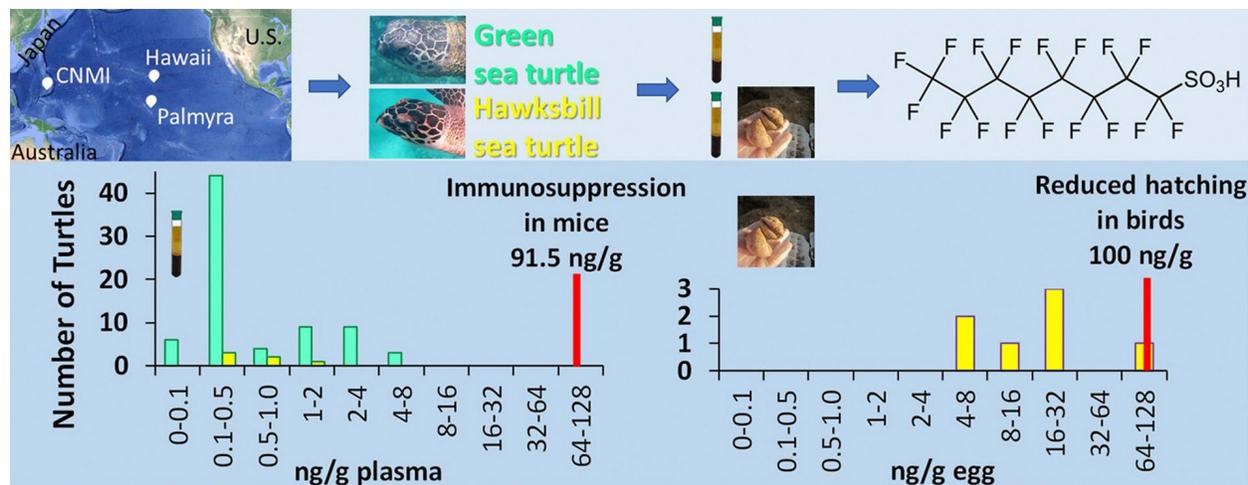


While concern for wildlife PFAS burdens has focused on implications for human consumption, studies demonstrate likely adverse consequences for wildlife itself. For example, a risk assessment was performed for PFAS concentrations in green (*Chelonia mydas*) and hawksbill

³⁷³ William Stansley *et al.*, *Mercury and halogenated organic contaminants in river otters (Lontra canadensis) in New Jersey, USA*, 10 *Environ Toxicol. Chem.* 2235–2242 (2010).

³⁷⁴ Environmental Working Group, *Global danger: Wildlife at risk from PFAS exposure*, available at https://www.ewg.org/interactive-maps/pfas_in_wildlife/map/ (fish data turned to “off”).

(*Eretmochelys imbricata*) turtles from the North Pacific including Hawaii that concluded the results were “similar to those documented along the Eastern U.S.,” and that “[t]wo contaminants (PFUnA and PFTriA) were related to reduced emergence success of hatchlings, which aligns with the risk assessment showing hawksbill egg PFOS concentrations are concerningly near concentrations causing developmental toxicity in birds,” as demonstrated by these graphs:³⁷⁵



PFAS substances in wildlife of San Francisco Bay, California, show significant declines in of PFOS in cormorant eggs and seal blood likely reflecting the 2002 U.S. phase-out of production, along with a shift to manufacture of shorter-chain PFAS compounds. However, “[o]f emerging concern are the stable concentrations of long-chain [perfluoroalkyl carboxylic acids] PFCAs in wildlife from certain locations in SF Bay, which suggest ongoing exposures, rather than declines,” likely the result of PFCS precursors (polyfluoroalkyl phosphate diesters) that “were

³⁷⁵ Cathryn Wood, *et al.*, *Sea turtles across the North Pacific are exposed to perfluoroalkyl substances*, 279 Environmental Pollution 116875 (June 15, 2011), available at <https://www.sciencedirect.com/science/article/abs/pii/S0269749121004577>.

observed at concentrations over an order of magnitude higher than PFCAs in sediment, highlighting their importance as a potential, on-going source of PFCAs to SF Bay wildlife.”³⁷⁶

This section of this Petition illustrates a strong basis to conclude that, despite the Clean Water Act, many of the nation’s waters are contaminated by toxic pollutants, both by chemicals on the Toxic Pollutants Lists as well as those that are not. This, along with the discussion in the next section below, pertaining to environmental injustice and health impacts to children, underscores the importance of updating the Toxic Pollutants Lists.

IV. FAILURE TO FULLY AND TIMELY CARRY OUT THE TECHNOLOGY- AND WATER QUALITY-BASED REQUIREMENTS OF THE CLEAN WATER ACT CAUSES ENVIRONMENTAL INJUSTICE AND HARM TO CHILDREN

EPA’s failure to update the grossly outdated Toxic Pollutant Lists to reflect current knowledge about the toxic contaminants that are harming designated uses in the nation’s waters not only diminishes the effectiveness of state and EPA water quality programs but results in EPA’s disregarding an integral part of its stated environmental justice mission: “to focus our attention on the environmental and public health challenges that face our nation’s minority, low-income, tribal, and indigenous populations.”³⁷⁷

The environmental justice movement began in the late 1960s, and gained traction in 1982 when protests over the siting of a toxic landfill in a predominantly African American community in Warren County, North Carolina drew national attention.³⁷⁸ In response, the U.S. General

³⁷⁶ Margaret D. Sedlak, *et al.*, *Per- and polyfluoroalkyl substances (PFASs) in San Francisco Bay wildlife: Temporal trends, exposure pathways, and notable presence of precursor compounds* 185 *Chemosphere* 1217 (Oct. 2017), available at <https://www.sciencedirect.com/science/article/abs/pii/S0045653517306331>.

³⁷⁷ EPA, *Office of Environmental Justice in Action*, https://www.epa.gov/sites/default/files/2017-09/documents/epa_office_of_environmental_justice_factsheet.pdf

³⁷⁸ EPA, Environmental Justice, *Environmental Justice Timeline*, available at <https://www.epa.gov/environmentaljustice/environmental-justice-timeline>.

Accounting Office (“GAO”) conducted a study that concluded “three out of four hazardous waste landfills examined were located in communities where African Americans made up at least twenty-six percent of the population, and whose family incomes were below the poverty level.”³⁷⁹

With environmental justice squarely in the public consciousness, President William Clinton issued Executive Order 12898 (“EO 12898”), directing federal agencies to develop environmental justice strategies to address disproportionately high and adverse human health or environmental effects of their programs on minority and low-income populations.³⁸⁰ EPA was thus charged with incorporating environmental justice—defined as the “fair treatment and meaningful involvement of all people regardless of race, color, national origin, or income, with respect to the development, implementation, and enforcement of environmental laws, regulations, and policies”—into its work.³⁸¹ In the years since EO 12898, EPA developed tools and guidance to achieve its stated goal: “to provide an environment where all people enjoy the same degree of protection from environmental and health hazards and equal access to the decision-making process to maintain a healthy environment in which to live, learn, and work.”³⁸²

According to an environmental justice advisory committee to EPA, “[c]onsumption and use of contaminated fish, aquatic plants, and wildlife is the primary route by which humans are exposed to many toxic contaminants.”³⁸³ The committee elaborated:

³⁷⁹ *Id.*

³⁸⁰ The President, *Executive Order 12898 of February 11, 1994, Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Communities*, available at https://www.epa.gov/sites/default/files/2015-02/documents/exec_order_12898.pdf.

³⁸¹ EPA, Environmental Justice, *Learn About Environmental Justice*, available at <https://www.epa.gov/environmentaljustice/learn-about-environmental-justice>.

³⁸² *Id.*

³⁸³ EPA, National Environmental Justice Advisory Council, *Fish Consumption and Environmental Justice, A Report developed from the National Environmental Justice Advisory Council Meeting of*

Consumption and use of contaminated fish, aquatic plants, and wildlife is an especially pressing concern for many communities of color, low-income communities, tribes, and other indigenous peoples, whose members may (1) consume fish, aquatic plants, and wildlife in greater quantities than does the general population; (2) consume and use different fish, aquatic plants, and wildlife than does the general population; (3) employ different practices in consuming and using fish, aquatic plants, and wildlife than does the general population; (4) consume and use fish, aquatic plants, and wildlife in cultural, traditional, religious, historical, economic, and legal contexts that differ from those of the general population.³⁸⁴

For this reason, the committee urged EPA to work expeditiously to reduce and clean up toxic pollution, “including but not limited to persistent bioaccumulative toxics” because “they *persist* in the environment for great lengths of time and because they *bioaccumulate* in the tissues of fish, aquatic plants, and wildlife, existing in greater quantities higher up the food chain.”³⁸⁵

The consumption of contaminated fish, aquatic plants, and wildlife, as well as contaminated drinking water, inequitably affects communities of color, low-income communities, tribes, and other indigenous people for multiple reasons, including the following:

- They “consume far greater quantities of fish than do members of the general population.”³⁸⁶
- They may eat “‘unusual’ species such as sea urchin, sea cucumbers or bottom-feeding fish.”³⁸⁷
- They may eat fat, head, skin, bones, eggs, internal organs, drippings, or cooking fluid, much of which has higher lipid content than consumed by the general population.³⁸⁸
- They may consume large amounts of fish, including from contaminated waters, to

December 3-6, 2001 (rev. Nov. 2002) (hereinafter “Fish Consumption Injustice”) at 13, available at https://www.epa.gov/sites/default/files/2015-02/documents/fish-consump-report_1102.pdf. See also e.g., Great Lakes Mixing Zones, 65 Fed. Reg. 67638, 67641 (Nov. 13, 2000) (“For humans, as is true for wildlife, the main route of exposure to [bioaccumulative chemicals of concern] BCCs is through the consumption of Great Lakes fish, which have “uptaken” and retained the pollutants from their surrounding environment and food. Potential adverse human health effects resulting from the consumption of contaminated fish include both the increased risk of cancer and the potential for systemic or noncancer risks such as kidney damage (U.S. EPA, 1997).”).

³⁸⁴ *Id.* at 14.

³⁸⁵ *Id.* at iii, v (emphasis original).

³⁸⁶ *Id.* at 26.

³⁸⁷ *Id.* at 35.

³⁸⁸ *Id.*; see also *id.* at 34–39 (examples provided).

subsist.

- The practices of consuming certain foods prepared in certain ways are for inalterable “cultural, traditional, religious, historical, and/or economic reasons.”³⁸⁹
- Background health and access to health care and nutrition is poorer than the general population.³⁹⁰

In Maine, the Penobscot Indian Nation—whose culture and traditions are “inextricably tied to the Penobscot River watershed”—contaminated waters are a barrier to tribal members’ engagement in cultural practices and threaten the tribe’s traditional lifestyle.³⁹¹ The federal government has slowly begun to evaluate the health impacts to tribal members from eating fish, shellfish, and in some instances animals, but taking little action but to caution tribal members not to consume contaminated foods. In 2021, the Agency for Toxic Substances and Disease Registry (“ATSDR”) finalized an evaluation of the risks of consuming anadromous fish by members of the Penobscot Indian Nation.³⁹² This evaluation considered dioxins, PCBs, mercury, PFAS/PFOS, and PBDEs, cautioning that the levels of these contaminants in anadromous fish species “could produce harmful effects.”³⁹³ For PFAS/PFOS, the ATSDR concluded only that “[s]tudies in humans and animals provide suggestive evidence that PFOS might contribute to cancer”³⁹⁴ and for PBDEs that tribal members “who eat any fish species at the highest intake rates for a year or more might be exposed to harmful levels of polybrominated diphenyl ethers (PBDEs).”³⁹⁵ Taking the toxic contaminants together, the agency cautioned the following:

³⁸⁹ *Id.* at 41.

³⁹⁰ *Id.* at 17.

³⁹¹ EPA, *The Penobscot River and Environmental Contaminants: Assessment of Tribal Exposure Through Sustenance Lifestyles* (Aug. 2015) at 1, available at https://cfpub.epa.gov/si/si_public_record_report.cfm?Lab=NHEERL&dirEntryId=309033 at 1.

³⁹² ATSDR, *Health Consultation Review of Anadromous Fish: Penobscot River, Penobscot Indian Nation, Indian Island, Maine* (May 19, 2021), available at https://www.atsdr.cdc.gov/hac/pha/PenobscotRiver/Penobscot_Indian_Nation_HC-508.pdf.

³⁹³ *See, e.g., id.* at 24.

³⁹⁴ *Id.* at 25.

³⁹⁵ *Id.* at 26.

- “Children should not eat any fish species at 1 ounce/day (or more)[.]”³⁹⁶
- Tribal “members should not eat any of the anadromous fish described in this health consultation because dioxin levels might cause harmful effects, including a significantly increased risk for liver cancer.”³⁹⁷
- “Children should not eat any striped bass because of PCBs and PBDEs. Adults should not eat striped bass daily or at 10 ounces per week because of PCBs and PBDEs.”³⁹⁸
- “Children should not eat any rainbow smelt, striped bass, or sea lamprey daily because of mercury levels” and “[c]hildren should not eat sea lamprey at 10 ounces per week because of mercury.”³⁹⁹

This was not the first evaluation of the risks of fish consumption to members of the Penobscot Nation. In 2006, following a request from the tribe to evaluate the health risks of exposure to contaminants from the Lincoln Pulp and Paper Mill at Lincoln, Maine, ATSDR calculated fish consumption limits based on dioxin and furans, PCBs, and methylmercury from resident fish species.⁴⁰⁰ In 2014, based on a multi-agency effort, the ATSDR once again calculated exposures to the same contaminants through resident fish, ducks, turtles, and plants, concluding that “members who ate fish and turtles were exposed to contaminants at levels of health concern” and recommending that “children younger than age 8 years, women who are breastfeeding, and women who are pregnant or who might become pregnant eat no Penobscot River fish.”⁴⁰¹ These earlier studies did not include PBDEs and PFAS/PFOS.

Tribes in the Pacific Northwest are another example of increased risks due to high fish consumption and poor water quality. Because Pacific Northwest tribal populations typically consume much more fish and shellfish than other people in the region, they are exposed to higher levels of toxic chemicals that bioaccumulate in aquatic life. In 1994, the Columbia River

³⁹⁶ *Id.*

³⁹⁷ *Id.* at viii.

³⁹⁸ *Id.*

³⁹⁹ *Id.*

⁴⁰⁰ *Id.* at ii.

⁴⁰¹ *Id.*

Intertribal Fish Commission (“CRITFC”) published a study on fish consumption rates in its four member tribes that concluded the “rates of tribal members’ consumption across gender, age groups, persons who live on- vs. off-reservation, fish consumers only, seasons, nursing mothers, fishers, and non-fishers range from 6 to 11 times higher than the national estimate used by USEPA.”⁴⁰² Subsequently, EPA analyzed fish tissue chemical concentrations for 132 chemicals from 24 sites located on the mainstem Columbia River and 15 related rivers and creeks.⁴⁰³ EPA summarized the risks posed by the toxics evaluated:

For adults in the general public with an average fish ingestion rate of about a meal³ per month (7.5 g/day), hazard indices were less than 1 and cancer risks were less than 1 in 10,000, except for a few of the more highly contaminated samples of mountain whitefish and white sturgeon. For adults in CRITFC’s member tribes, at the highest fish ingestion rate at about 48 meals [meal=8 ounces of fish] per month (389 g/day), hazard indices were greater than 1 for several species at some sites. Hazard indices (less than or equal to 8 at most sites) and cancer risks (7 in 10,000 to 2 in 1,000) were lowest for salmon, steelhead, eulachon and rainbow trout and highest (hazard indices greater than 100 and cancer risks up to 2 in 100 at some sites) for mountain whitefish and white sturgeon. For the general public, the hazard indices for children at the average fish ingestion rate were less for adults (0.9) at the average ingestion rate; the hazard indices for children at the high ingestion rate were 1.3 times greater than those for adults at the high ingestion rate. For CRITFC’s member tribes, the hazard indices for children at the average and high ingestion rates were 1.9 times greater than those for adults in CRITFC’s member tribes at the average and high ingestion rates, respectively.⁴⁰⁴

EPA concluded that “[a]dults in CRITFC’s member tribes who eat fish frequently (48 meals per month) over a period of 70 years may have cancer risks that are up to 50 times higher than those

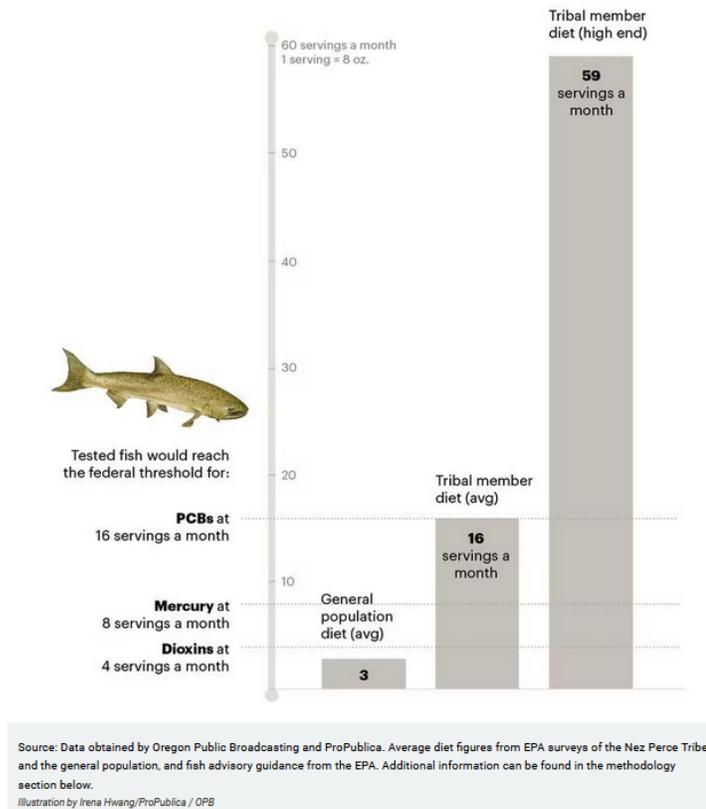
⁴⁰² Columbia River Intertribal Fish Commission, *A Fish Consumption Survey of the Umatilla, Nez Perce, Yakama, and Warm Springs Tribes of the Columbia River Basin* (Oct. 1994).

⁴⁰³ EPA, *Columbia River Basin Fish Contaminant Survey 1996-1998* (undated, 2002) (hereinafter “Columbia Fish Contamination Survey”), available at https://www.epa.gov/sites/default/files/documents/columbia_fish_contaminant_survey_1996-1998.pdf.

⁴⁰⁴ *Id.* at E-6–E-7.

in the general public who consume fish about once a month.”⁴⁰⁵ Similar risk calculations based on Columbia River fish tissue toxic residues were illustrated in a later news article that tested Columbia River salmon for contaminants:⁴⁰⁶

Graphic: Contaminants in Fish Put Tribal Members at Risk



In its study, while EPA suggested that tribal members could reduce fish consumption to reduce their health risks from chemical exposure, it limited its regulatory suggestions to a vague

⁴⁰⁵ EPA, *Columbia River Fish Contaminant Survey*, available at https://19january2017snapshot.epa.gov/columbiariver/columbia-river-fish-contaminant-survey_.html.

⁴⁰⁶ See Tony Schick, Oregon Public Broadcasting, and Maya Miller, ProPublica, *Unchecked pollution is contaminating the salmon that Pacific Northwest tribes eat* (Nov. 22, 2022) (hereinafter “Unchecked Pollution”), available at <https://www.opb.org/article/2022/11/22/pollution-contamination-threat-salmon-columbia-basin-pacific-northwest-tribes/>; see also Tony Schick and Maya Miller, *How we tested Columbia River salmon for contaminants* (Nov. 22, 2022), available at <https://www.opb.org/article/2022/11/22/how-opb-propublica-tested-columbia-river-salmon-for-contaminants/>.

conclusion that “[t]he results of this study confirm the need for regulatory agencies to continue to pursue rigorous controls on environmental pollutants and to remove those pollutants which have been dispersed into our ecosystems.”⁴⁰⁷ Suggesting to tribal members that they should limit fish consumption rather than taking appropriate regulatory action to safeguard fish supplies is itself an environmental injustice, especially for tribes for which fish harvesting and use is a longstanding and extremely important part of their culture. Although, under pressure from EPA, Oregon subsequently adopted new numeric human health criteria to reflect the CRITFC fish consumption survey,⁴⁰⁸ it has not taken any regulatory steps to reduce toxic pollution in the Columbia River basin, leaving tribal members in as much risk of adverse health consequences as they were prior to the criteria’s being updated.⁴⁰⁹

Other population groups in the Pacific Northwest have also been identified as consuming high levels of fish and shellfish, resulting in lack of protection for their health.⁴¹⁰ In addition to the CRITFC study, the Washington Department of Ecology found two additional tribal-specific

⁴⁰⁷ *Id.* at 11-229.

⁴⁰⁸ See Letter from Michael Bussell, EPA, to Neil Mullane, Oregon Department of Environmental Quality, Re: *EPA's Approval of New and Revised Human Health Water Quality Criteria for Toxics and Implementation Provisions in Oregon's Water Quality Standards Submitted on July 12 and 21, 2011* (Oct. 17, 2011), available at

<https://www.oregon.gov/deq/FilterDocs/shhEPAappLetter2011.pdf> (Oregon human health criteria now based on 175 grams/day fish consumption compared to previous 17.5 grams/day).

⁴⁰⁹ See *Unchecked Pollution*, *supra* n. 406 (“Jennifer Wigal, DEQ’s water quality administrator, said the standards were implemented not because of pollution but to ensure that tribal diets were represented. Wigal also said that when companies release harmful contaminants into the river, most are at such low concentrations that they are below the agency’s ability to detect them. Additionally, most of the contamination affecting fish, the DEQ said, comes not from those polluters but from runoff and erosion from industries like agriculture and logging. But the DEQ also has yet to curtail that source of pollution. Along the Willamette River, which flows through Oregon’s most populated areas and feeds into the Columbia, the EPA determined last year that the state needed to cut mercury pollution from these sources by at least 88% if it was going to meet its standards for protecting human health.”).

⁴¹⁰ See generally, Northwest Environmental Advocates, *Petition for Rulemaking Under the Clean Water Act Water Quality Criteria for Toxics in the State of Washington* (Oct. 28, 2013).

fish consumption surveys and one Asian and Pacific Islander survey technically defensible.⁴¹¹ It illustrated the results of the CRITFC study as follows:⁴¹²

Table 21. CRITFC Adult Fish Consumption Rates by Species Group and Source, Consumers Only

Population Tribal	Species Group	Harvest Source of Fish	Descriptive Statistics (g/day)				
			50 th Percentile	Mean	75 th Percentile	90 th Percentile	95 th Percentile
The 4 Tribes Affiliated With The Columbia River Inter-Tribal Fish Commission	All finfish	all	40.5	63.2	64.8	130.0	194.0
	Non-anadromous	all	20.9	32.6	33.4	67.0	99.9
	Anadromous	all	19.6	30.6	31.4	63.1	94.1
	All finfish	Columbia River Basin	35.6	55.6	57.0	114	171
	Non-anadromous	Columbia River Basin	18.4	28.6	29.4	58.9	87.9
	Anadromous	Columbia River Basin	17.3	27.0	27.7	55.5	82.8

See Polissar et al., 2012, Table E-1.

The other two tribal fish consumption surveys—of The Tulalip Tribes and Squaxin Island Tribe,⁴¹³ and of The Suquamish Tribe⁴¹⁴—also demonstrated high levels of fish and shellfish consumption:⁴¹⁵

⁴¹¹ See Final FCR Report, *supra* n. 199.

⁴¹² *Id.* at 48.

⁴¹³ See Kelly Toy et al., *A Fish Consumption Survey of the Tulalip and Squaxin Island Tribes of the Puget Sound Region* (Oct. 1996) available at <http://www.deq.state.or.us/wq/standards/docs/toxics/tulalipsquaxin1996.pdf>.

⁴¹⁴ See The Suquamish Tribe, *Fish Consumption Survey Of The Suquamish Indian Tribe Of The Port Madison Indian Reservation, Puget Sound Region* (Aug. 2000) available at https://www7.nau.edu/itep/main/iteps/ORCA/3877_ORCA.pdf.

⁴¹⁵ Final FCR Report, *supra* n. 199 at 55, 56, 61.

Table 23. Tulalip Tribal Adult Fish Consumption Rates by Species Group and Source

Population Tribal	Species Group	Harvest Source of Fish	Descriptive Statistics (g/day)				
			50 th Percentile	Mean	75 th Percentile	90 th Percentile	95 th Percentile
Tulalip	All Fish	All Sources	44.5	82.2	94.2	193	268
	Finfish	All Sources	22.3	44.1	49.1	110	204
	Shellfish	All Sources	15.4	42.6	40.1	113	141
	Non-anadromous	All Sources	20.1	45.9	52.4	118	151
	Anadromous	All Sources	16.8	38.1	43.3	92.1	191
	All	Puget Sound	29.9	59.5	75.0	139	237
	Finfish	Puget Sound	13.0	31.9	33.1	78.4	146
	Shellfish	Puget Sound	14.2	36.9	40.1	111	148
	Non-anadromous	Puget Sound	14.8	35.5	38.8	109	145
	Anadromous	Puget Sound	11.8	30.4	32.4	66.0	148

See Polissar et al., 2012, Table E-1.

Table 24. Squaxin Island Tribal Adult Fish Consumption Rates by Species Group and Source

Population Tribal	Species Group	Harvest Source of Fish	Descriptive Statistics (g/day)				
			50 th Percentile	Mean	75 th Percentile	90 th Percentile	95 th Percentile
Squaxin Island	All fish	All	44.5	83.7	94.4	206	280
	Finfish	All	31.4	65.5	82.3	150	208
	Shellfish	All	10.3	23.1	23.9	54.0	83.6
	Non-anadromous	All	15.2	28.7	32.3	70.5	95.9
	Anadromous	All	25.3	55.1	65.8	128	171
	All fish	Puget Sound	30.0	56.4	63.5	139	189
	Finfish	Puget Sound	21.6	45.0	56.5	103	143
	Shellfish	Puget Sound	6.4	14.3	14.8	33.5	51.9
	Non-anadromous	Puget Sound	6.5	12.3	13.9	30.3	41.2
	Anadromous	Puget Sound	20.2	44.1	52.6	103	137

See Polissar et al., 2012, Table E-1.

Table 26. Suquamish Tribal Adult Fish Consumption Rates by Species Group and Source

Population Tribal	Species Group	Harvest Source of Fish	Descriptive Statistics (g/day)				
			50 th Percentile	Mean	75 th Percentile	90 th Percentile	95 th Percentile
Suquamish Tribe	All	All Sources	132	214	284	489	797
	Shellfish	All Sources	64.7	134	145	363	615
	Non-anadromous*	All Sources	102	169	219	377	615
	Anadromous	All Sources	27.6	48.8	79.1	133	172
	All	Puget Sound	57.5	165	221	397	767
	Shellfish	Puget Sound	52.4	109	118	294	499
	Non-anadromous*	Puget Sound	49.1	126	116	380	674
	Anadromous	Puget Sound	21.8	38.6	62.5	105	136

See Polissar et al., 2012

*Based on an assumed n = 90 consumers.

Finally, Washington presented the results of a 1997 survey of Asian and Pacific Islanders in King County, Washington that demonstrated their high seafood consumption:⁴¹⁶

Table 30. API Adult Seafood Consumption Rates by Species Group and Source

Population API	Species Group	Source of Fish	Descriptive Statistics (g/day)		
			50 th Percentile	90 th Percentile	95 th Percentile
Asian-Pacific Islander (API)	Total seafood consumption	All sources	74.0	227	286
	All species	Harvested anywhere	6.5	25.9	58.8
	All species	Harvested from King County	5.7	22.2	48.4
	Non-anadromous species	Harvested anywhere	6.2	37.9	54.1
	Non-anadromous species	Harvested from King County	6.0	20.1	45.5

Sources: Adapted from Kissinger, 2005, Table 5. See also Polissar et al., 2012.

Table 31. API Seafood Consumption Rates Adjusted for Cooking Loss

Population API	Species Group	Source of Fish	Descriptive Statistics (g/day)		
			50 th Percentile	90 th Percentile	95 th Percentile
Asian-Pacific Islander (API)	Total seafood consumption	All sources	77.8	236	306
	All species	Harvested anywhere	6.9	49.1	76.3
	All species	Harvested from King County	5.8	25.5	57.1
	Non-anadromous species	Harvested anywhere	7.1	54.2	72.3
	Non-anadromous species	Harvested from King County	6.6	33.4	57.3

Source: Adapted from Kissinger, 2005, Table 8. See also Polissar et al., 2012.

More recently, a study on contaminant levels in seaweed was analyzed for the Salish Sea/Puget Sound to support “health-based consumption advisories . . . for consumers that include Tribes and First Nations, Asian and Pacific Islander community members, and recreational harvesters.”⁴¹⁷ Its results demonstrate that “concentrations of some chemical contaminants in Salish Sea seaweeds may pose a risk to human health when consumed at the rate specified by the

⁴¹⁶ *Id.* at 69. Data based on Ruth Sechena, et al., *Asian and Pacific Islander Seafood Consumption Study*, EPA 910/R-99-003 (May 27, 1999) available at https://depts.washington.edu/ceeh/downloads/API_Seafood_Study.pdf.

⁴¹⁷ Jennifer L. Hahn, et al., *Chemical contaminant levels in edible seaweeds of the Salish Sea and implications for their consumption*, PLoS ONE 17(9): e0269269 (Sept. 23, 2022) at 1, available at <https://doi.org/10.1371/journal.pone.0269269>.

[U.S. Food and Drug Administration] USFDA.”⁴¹⁸ A majority of sites exceeded U.S. screening levels for PCBs, two samples exceeded U.S. screening levels for benzo[a]pyrene, and three contaminants—cadmium, mercury, and lead—exceeded international limits at some sites.

In rural Alaska, fish is the most frequently consumed subsistence food. EPA reported the results of two tribal fish consumption surveys:

The first Alaska tribal fish consumption survey, an Assessment of Cook Inlet Tribes’ Subsistence Consumption, was conducted by Seldovia Village Tribe in 2012-2013. Modeled after the Columbia River Inter-Tribal Fish Commission survey, this study showed that for all fish and shellfish species consumed, the overall mean consumption rate (n=76) was 106.8 grams per day (g/d) and the 95th percentile consumption rate was 267.1 g/d.

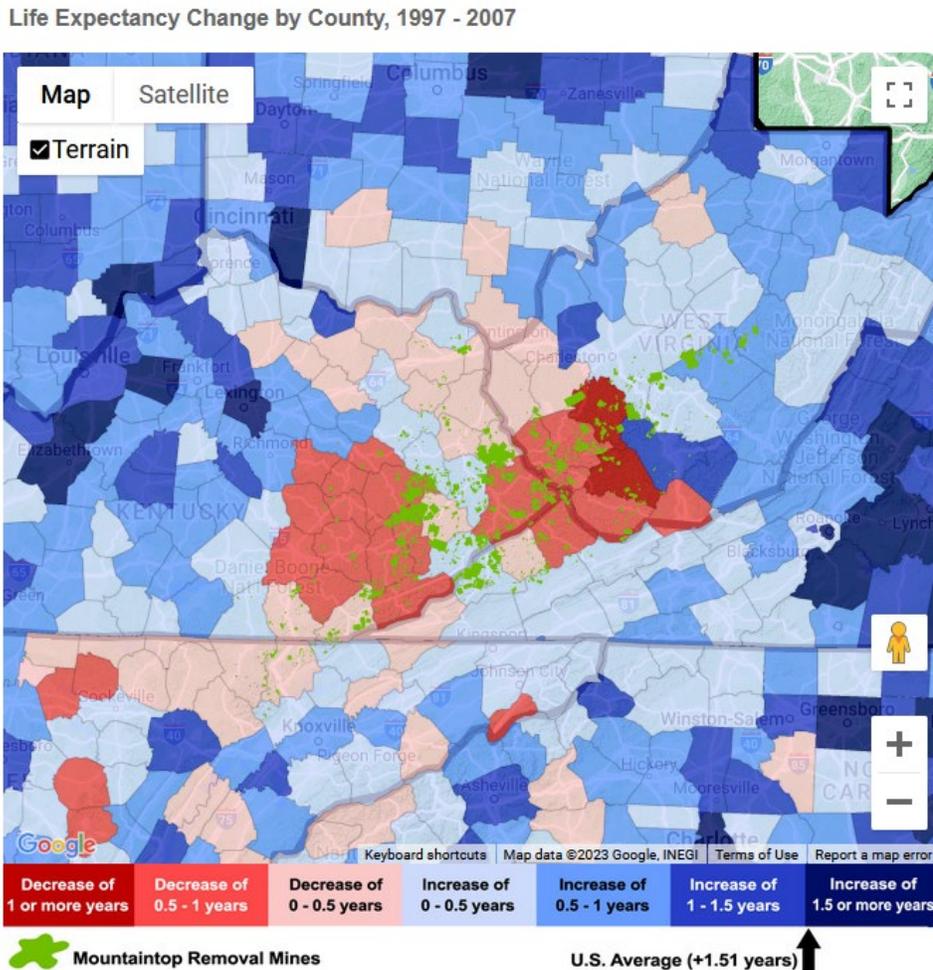
A second Alaska tribal fish consumption survey, an Assessment of Kodiak Island Tribes’ Seafood Consumption, was conducted by the Sun’aq Tribe of Kodiak in 2015-2016. Modeled after the Cook Inlet and Idaho Tribal surveys, this study showed that for all fish, shellfish, and marine mammal species consumed, the overall mean consumption rate (n=326) was 232.8 g/d and the 95th percentile consumption rate was 764.4 g/d.⁴¹⁹

Contaminated drinking water is also an injustice against communities, often low-income or lacking in political representation. Appalachia is a prime example, where the process of blowing up mountains to obtain ore, called “mountaintop removal mining,” has resulted in widespread contamination of drinking water wells. The removal of over 500 Appalachian mountains has resulted in massive human health consequences: “Twenty-one peer-reviewed scientific studies conducted from 2007-2012 . . . showed that . . . people living near the

⁴¹⁸ *Id.* at 22.

⁴¹⁹ EPA, *Fish and Shellfish Program Newsletter* (April 2018), available at <https://www.epa.gov/sites/default/files/2018-05/documents/fish-news-apr2018.pdf> at 19. Data based on Sun’aq Tribe of Kodiak, *Kodiak Tribes Seafood Consumption Assessment: Draft Final Report* (Dec. 2019), available at <https://sunaq.org/wp-content/uploads/2016/09/Kodiak-Tribes-Seafood-Consumption-Assessment-DRAFT-Final-Report-26Feb19-FINAL.pdf>; Seldovia Village Tribe, *Assessment of Cook Inlet Subsistence Consumption* (Sept. 30, 2013) available at <https://svt.org/wp-content/uploads/2022/04/Assessment-of-Cook-Inlet-Tribes-Subsistence-Consumption93013-FINAL.pdf>.

destruction are 50% more likely to die of cancer and 42% more likely to be born with birth defects compared with other people in Appalachia.”⁴²⁰ This change in life expectancy is illustrated by the following map (note green areas are mountaintop removal mines)⁴²¹ based on 21 studies identified by Coal River Mountain Watch:⁴²²



EPA has identified some of the costs of mountaintop mining in a 2011 report that curiously did not address the question of impacts to human health, but found extensive impacts

⁴²⁰ iLoveMountains.org, *The Human Cost of Coal, Mountaintop Removal's Effect on Health and the Economy*, available at <http://ilovemountains.org/the-human-cost>.

⁴²¹ *Id.*

⁴²² Coal River Mountain Watch, *Health Impacts*, available at <https://www.crmw.net/resources/health-impacts.php>.

to aquatic life:

[M]ountaintop mines and valley fills lead directly to five principal alterations of stream ecosystems:

1. springs and ephemeral, intermittent and perennial streams are permanently lost with the removal of the mountain and from burial under fill,
2. concentrations of major chemical ions are persistently elevated downstream,
3. degraded water quality reaches levels that are acutely lethal to organisms in standard aquatic toxicity tests,
4. selenium concentrations are elevated, reaching concentrations that have caused toxic effects in fish and birds, and
5. macroinvertebrate and fish communities are consistently degraded.⁴²³

Wikipedia has more information than EPA for the public to understand the human health effects of mountaintop mining and environmental justice concerns, including, for example the following:

Several studies have found that communities within the Appalachian region surrounding coal mining practices disproportionately experience negative health effects than communities with no coal mining. Such health disparities are largely attributed to the contamination of water and land associated with coal surface mining. MTR has increased salinity, metals, magnesium, and sulfates within Appalachian watersheds, threatening human health. Sixty-three percent of stream beds near coalfields within the Appalachia mountains have been identified as “impaired” due to high toxic chemical and metal contamination. In West Virginia, 14 counties are experiencing water that exceeds safe drinking water standards by seven times more than non-mining counties.⁴²⁴

Others have concluded that mountaintop removal mining is a form of environmental injustice

⁴²³ EPA, Risk Assessment, CADDIS, *The Effects Of Mountaintop Mines And Valley Fills On Aquatic Ecosystems Of The Central Appalachian Coalfields (2011 Final)*, available at <https://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=225743&CFID=63330774&CFTOKEN=63962894> (“The report did not review the impacts of MTM-VF on . . . human health.”); EPA, *The Effects Of Mountaintop Mines And Valley Fills On Aquatic Ecosystems Of The Central Appalachian Coalfields* (March, 2011), available at https://ordspub.epa.gov/ords/eims/eimscomm.getfile?p_download_id=501593.

⁴²⁴ Wikipedia, *Environmental justice and coal mining in Appalachia*, available at https://en.wikipedia.org/wiki/Environmental_justice_and_coal_mining_in_Appalachia (internal citations omitted).

based on evaluating poverty statistics and mortality and drawing the following conclusion:

Persons living in MTM areas experience persistently elevated poverty and mortality rates. Higher mortality is independently associated with both poverty and MTM, the latter effect suggestive of a possible environmental contribution from mining activities. Efforts to reduce longstanding health disparities in Appalachia must focus on those areas where disparities are concentrated: the Appalachian coalfields.⁴²⁵

In the 1990s, the USGS looked at both aquatic life and human health impacts, finding extensive water quality impacts from mountaintop mining, including:

Nickel, chromium, zinc, and certain toxic organic compounds were found in bed sediment in concentrations that could harm aquatic life. Elevated concentrations of cadmium, mercury, nickel, selenium, and zinc were measured in fish tissue at some sites.⁴²⁶

In the Appalachian Plateaus, iron and manganese concentrations exceeded USEPA drinking-water guidelines in at least 40 percent of the wells and in about 70 percent of wells near reclaimed surface coal mines. Elevated sulfate concentration and slightly acidic water were more common at wells within 1,000 feet of reclaimed mines than elsewhere.⁴²⁷

The USGS claimed that “[w]ater that exceeds these [iron and manganese] guidelines is unpleasant to drink . . . but it is not a health hazard.”⁴²⁸ Subsequent testing found manganese concentrations “‘way off the scale’ . . . ranging from nondetectable up to 4,063 ppb (the EPA recommends that manganese in drinking water not exceed 50 ppb).”⁴²⁹ Manganese is not on the Toxic Pollutants Lists.

⁴²⁵ Michale Hendryx, *Poverty and Mortality Disparities in Central Appalachia: Mountaintop Mining and Environmental Justice*, 4(3) *Journal of Health Disparities Research and Practice* 44-53 (Spring 2011), available at <https://digitalscholarship.unlv.edu/cgi/viewcontent.cgi?article=1005&context=jhdrp>.

⁴²⁶ USGS, *Water Quality in the Kanawha-New River Basin, West Virginia, Virginia, and North Carolina, 1996-98* (2000), available at <https://pubs.usgs.gov/circ/circ1204/pdf/circular1204.pdf> at 1.

⁴²⁷ *Id.* at 2.

⁴²⁸ *Id.*

⁴²⁹ David C. Holzman, *Mountaintop Removal Mining: Digging Into Community Health Concerns*, 199 *Environmental Health Perspectives* 11 (Nov. 2011), available at <https://web.archive.org/web/20170504161918/https://ehp.niehs.nih.gov/wp-content/uploads/119/11/ehp.119-a476.pdf> at 480.

USGS, however, is incorrect to disregard manganese as a health concern. To the contrary,

the massive production of manganese-containing compounds (metallurgic and chemical products, municipal wastewater discharges, sewage sludge, alloys, steel, iron, ceramics, fungicide products) has attracted the attention of scientists who investigated manganese as a potential emerging contaminant in the environment, and especially in the marine environment (CICAD 63, 2004). In humans, manganese excess is renowned for its role in neurotoxicity, associated with a characteristic syndrome called ‘manganese madness’ or ‘Parkinson-like’ diseases (Perl & Olanow, 2007). This neurodegenerative disorder is due to the accumulation of manganese inside intracellular compartments, such as the Golgi apparatus and mitochondria. In mammals, prenatal and postnatal exposure to manganese is associated with embryo-toxicity, fetal-toxicity, and decreased postnatal growth (Sanchez et al., 1993; Colomina et al., 1996).

* * *

In humans, the neurological damage induced by excessive manganese exposure has been well documented for over a century (Cooper, 1837; Mena et al., 1967; Normandin & Hazel, 2002; Takeda, 2003).⁴³⁰

A 2011 study evaluated the neurotoxicological effects of manganese from drinking water on the intellectual development of school-age children, finding that

[t]he median MnW in children’s home tap water was 34 µg/L (range, 1–2,700 µg/L). MnH increased with manganese intake from water consumption, but not with dietary manganese intake. Higher MnW and MnH were significantly associated with lower IQ scores. A 10-fold increase in MnW was associated with a decrease of 2.4 IQ points (95% confidence interval: –3.9 to –0.9; $p < 0.01$), adjusting for maternal intelligence, family income, and other potential confounders. There was a 6.2-point difference in IQ between children in the lowest and highest MnW quintiles. MnW was more strongly associated with Performance IQ than Verbal IQ.⁴³¹

The authors concluded: “The findings of this cross-sectional study suggest that exposure to manganese at levels common in groundwater is associated with intellectual impairment in

⁴³⁰ Annalisa Pinsino *et al.*, *Environmental Contamination*, Chapter 2 *Manganese: A New Emerging Contaminant in the Environment* (Feb. 2012) available at https://cdn.intechopen.com/pdfs/29309/InTech-Manganese_a_new_emerging_contaminant_in_the_environment.pdf at 1, 21.

⁴³¹ Maryse F. Bouchard, *et al.*, *Intellectual impairment in school-age children exposed to manganese from drinking water*, 119(1) *Environmental Health Perspectives* 138-43 (Jan. 2011), available at <https://pubmed.ncbi.nlm.nih.gov/20855239/>.

children.” Numerous other studies have concluded that “[e]xposure to manganese in water was associated with poorer neurobehavioral performances in children, even at low levels commonly encountered in North America.”⁴³²

Yet other studies have highlighted the environmental justice concerns of toxic metal levels in drinking water, including the non-priority pollutant manganese, because “[p]olicies tainted by environmental racism shape who has access to public water supplies, with Black People, Indigenous People, and People of Color (BIPOC) often excluded from municipal services. Thus, toxic metal exposure via private wells is an environmental justice (EJ) issue.”⁴³³ Twenty-four percent of the state’s population, or 2.4 million people, rely on private wells for drinking water.⁴³⁴ This study “developed four Toxic Metal Environmental Justice Indices (TM-EJIs) for inorganic arsenic (iAs), cadmium (Cd), lead (Pb), and manganese (Mn) to quantitatively identify areas of environmental injustice in NC” and found that “Mn had the greatest proportion (25.17%) of positive TM-EJIs, which are indicative of socioeconomically disadvantaged groups exposed to toxic metals. Positive TM-EJIs, particularly for Pb and Mn, were primarily located in eastern NC.”⁴³⁵ The study specifically cited to the fact that “exposure to Mn-contaminated drinking water can cause neurological effects, affecting memory, attention,

⁴³² Youssef Oulhote, *et al.*, *Neurobehavioral function in school-age children exposed to manganese in drinking water*, 122(12) *Environmental Health Perspectives* 1343-1350 (Dec. 2014), available at <https://pubmed.ncbi.nlm.nih.gov/25260096/>; see also PubMed, *Manganese exposure: cognitive, motor and behavioral effects on children: a review of recent findings*, available at <https://pubmed.ncbi.nlm.nih.gov/23486422/> (abstract concluding that “the adverse effects of manganese exposure on the developing brain is well demonstrated” and listing “similar articles.”).

⁴³³ Noemi Gavino-Lopez, *et al.*, *Developing Toxic Metal Environmental Justice Indices (TM-EJIs) for Arsenic, Cadmium, Lead, and Manganese Contamination in Private Drinking Wells in North Carolina*, 14(13) *Water (Basel)* 2088 (July 2022), available at <https://pubmed.ncbi.nlm.nih.gov/36452066/>.

⁴³⁴ *Id.* at 3.

⁴³⁵ *Id.* at 1.

and motor skills.”⁴³⁶

Not only are communities of color, low-income communities, tribes, and other indigenous peoples who “*depend* on healthy aquatic ecosystems and the fish, aquatic plants, and wildlife that these ecosystems support,” at much higher risk for adverse health consequences of consuming fish, shellfish, aquatic plants, and wildlife, but children are also at higher risk, particularly those in the most affected communities.⁴³⁷ This can be illustrated by EPA’s observation that “the hazard indices estimated for CRITFC’s member tribal children at the high ingestion rate were over 100 times those estimated for general public children at the average ingestion rate.”⁴³⁸ Similarly, when EPA prohibited mixing zones for some bioaccumulative pollutants in the Great Lakes System, part of its rationale was based on the vulnerability of children and fetuses:

As affirmed by commenters who support today’s rule, women who are pregnant and children, in particular, are at risk for being adversely affected by BCCs (U.S. EPA, 1997). BCCs can induce inheritable chromosomal changes in women that could result in birth defects in their infants, cross the human placenta contributing to exposure of the fetus through placental transfer, and accumulate in body tissues. Exposure to BCCs can result in decreased fertility, premature labor, spontaneous abortion, reproductive hormone disorders, increased stillbirths, lack of mammary function, reduced libido, and delayed estrus.

Children may be at greater risk than adults. Because BCCs can accumulate in human milk, women exposed to the pollutants who breastfeed could potentially pass the chemicals on to their infants. Risks to infants and children include central nervous system effects, mortality, low IQ scores, cataracts, congestive heart failure, skin disorders, cancers, immune system dysfunction and immunosuppression, skeletal disorders, neurological/behavioral effects, and

⁴³⁶ *Id.* at 2.

⁴³⁷ Fish Consumption Injustice, *supra* n. 383, at 42 (emphasis original) (taking as axiomatic that children are more susceptible to environmental contaminants); *see also id.* at 70 (mercury “associated with more subtle end points of neurotoxicity in children”); 73 (some studies show PCB causes adverse effects in children and developing fetuses); 79 (effects of lead on children are “particularly troubling”); 138 fn 440 (citing studies showing that tribal “children are uniquely susceptible to pollution and contaminants”)

⁴³⁸ Columbia Fish Contamination Survey, *supra* n. 403 at 6-92.

endocrinological disorders.⁴³⁹

As the ATSDR discusses, children, when compared with adults, are often especially susceptible to toxic exposures for multiple reasons including the following:

Childhood is a time of rapid growth and development. It is accompanied by

- changes in organ system functioning,
- metabolic capabilities,
- physical size, and
- behavior

that can dramatically modify the effects, the illness, or both caused by toxicant exposure.

Given the same amount of exposure to a toxicant, persons will vary in how susceptible they are to disease induced by the exposure. Among the factors affecting susceptibility are

- genes,
- sex,
- age,
- nutritional status,
- state of health (i.e., presence of other diseases), and
- biochemical differences such as chemical metabolism, speed of DNA repair, and regulation of net cell growth [Pitot and Dragan 1996].

* * *

For most agents, however, theory and empirical observations point to increased susceptibility to environmental hazards. This susceptibility begins in the preconception period and continues throughout

- fetal life,
- birth,
- infancy, and
- childhood.

Thus the U.S. Environmental Protection Agency (EPA) has suggested additional safety factors (e.g., 10-fold uncertainty factor, a 3.16-fold factor each for toxicokinetic and toxicodynamic variability) in regulating in utero and postnatal exposures to many environmental chemicals [Cresteil 1998; Renwick 1998;

⁴³⁹ Great Lakes Mixing Zones, 65 Fed. Reg. 67641 (Nov. 13, 2000).

Dourson et al. 2002].⁴⁴⁰

The ATSDR notes that there are three age-specific periods of susceptibility in children, when “children are exquisitely sensitive to any adverse effects of chemicals.”⁴⁴¹ However,

[e]ven within a given developmental stage, shorter exposure intervals may determine susceptibility for particular outcomes. Different organ systems develop at different rates. Broad windows of susceptibility and more specific periods of susceptibility (e.g., radiation effects on central nervous system development during the critical 8 to 15 weeks in utero) occur at each developmental stage [Faustman et al. 2000; ORISE 2010]. In most cases, however, the exact time is unknown when organ systems are susceptible to the actions of toxic chemicals.

In addition, the agency notes that of particular concern is the effect of “hormonally active agents.”⁴⁴² The ATSDR summarizes the vulnerability of children to toxic contaminant exposure: “The differing susceptibility of children to harm from environmental exposures results from their development—a dynamic process with many physiologic, metabolic, and behavioral aspects. Children are at increased risk because of their increased exposures and increased vulnerability.”⁴⁴³

These conclusions about the impacts of *in utero* and childhood exposure to chemicals are supported by the American Academy of Pediatrics (“AAP”) in its 2012 policy statement on pesticides in which it cites the following evidence:

Dosing experiments in animals clearly demonstrate the acute and chronic toxicity potential of multiple pesticides. Many pesticide chemicals are classified by the US EPA as carcinogens. The past decade has seen an expansion of the epidemiologic evidence base supporting adverse effects after acute and chronic

⁴⁴⁰ ATSDR, Principles of Pediatric Environmental Health, *Why Are Children Often Especially Susceptible to the Adverse Effects of Environmental Toxicants?* (hereinafter “Principles of Pediatric Environmental Health”) available at https://www.atsdr.cdc.gov/csem/pediatric-environmental-health/why_children.html; see also ATSDR, *ATSDR Case Studies in Environmental Medicine Principles of Pediatric Environmental Health* (original date Feb. 15, 2012), available at https://www.atsdr.cdc.gov/csem/ped_env_health/docs/ped_env_health.pdf.

⁴⁴¹ Principles of Pediatric Environmental Health, *supra* n. 440.

⁴⁴² *Id.*

⁴⁴³ *Id.*

pesticide exposure in children. This includes increasingly sophisticated studies addressing combined exposures and genetic susceptibility.

Chronic toxicity end points identified in epidemiologic studies include adverse birth outcomes including preterm birth, low birth weight, and congenital anomalies, pediatric cancers, neurobehavioral and cognitive deficits, and asthma. These are reviewed in the accompanying technical report. The evidence base is most robust for associations to pediatric cancer and adverse neurodevelopment. Multiple case-control studies and evidence reviews support a role for insecticides in risk of brain tumors and acute lymphocytic leukemia. Prospective contemporary birth cohort studies in the United States link early-life exposure to organophosphate insecticides with reductions in IQ and abnormal behaviors associated with attention-deficit/hyperactivity disorder and autism. The need to better understand the health implications of ongoing pesticide use practices on child health has benefited from these observational epidemiologic data.⁴⁴⁴

The AAP has published the results of studies on non-pesticide chemicals demonstrating its concern. For example, it published a study in 2018 on the association of *in utero* exposure to PBDEs, a common flame retardant added to consumer products, and the occurrence of infant hypospadias,⁴⁴⁵ “a birth defect in which the opening of the urethra is on the underside of the penis instead of at the tip.”⁴⁴⁶ PBDEs are not on the Toxic Pollutants Lists.

Other studies have looked at the rise of diagnoses of neurodevelopmental disabilities—including autism, attention-deficit hyperactivity disorder, dyslexia, and other cognitive

⁴⁴⁴ American Academy of Pediatrics, *Policy Statement: Pesticide Exposure in Children*, 130[6] *Pediatrics* e1757-1763 at e1759-e1760 (Dec. 2012) available at www.pediatrics.org/cgi/doi/10.1542/peds.2012-2757; see also American Academy of Pediatrics, *Technical Report: Pesticide Exposure in Children*, 130[6] *Pediatrics*, e1765–e1788 (Dec. 2012), available at <https://publications.aap.org/pediatrics/article/130/6/e1765/30343/Pesticide-Exposure-in-Children>.

⁴⁴⁵ American Academy of Pediatrics, Research Article: *Flame Retardants and Risk of Hypospadias*, 40[6] *Grand Rounds* 63 (2018), abstract available at <https://publications.aap.org/aapgrandrounds/article-abstract/40/6/63/88115/Flame-Retardants-and-Risk-of-Hypospadias?redirectedFrom=fulltext> (“Total PBDE levels were significantly higher in case parents than controls (51.4 pg/mg vs 35.8 pg/mg; P=.02). After adjusting for potential confounders, this association remained significant with a 48.2% (95% CI, 23.3%, 65.4%) higher PBDE level in case (vs control) parents. Case infant PBDE levels were significantly higher than case parent PBDE levels (P<.05) and, among the entire study population, maternal PBDE levels did not correlate with infant levels.”).

⁴⁴⁶ Mayo Clinic, Patient Care & Health Information, Diseases & Conditions, *Hypospadias*, available at <https://www.mayoclinic.org/diseases-conditions/hypospadias/symptoms-causes/syc-20355148>.

impairments—correlating them with studies that demonstrate industrial chemicals that injure the developing brain in children are among the known causes for this rise in prevalence.⁴⁴⁷

According to one evaluation, in 2006, a systematic review of literature identified five industrial chemicals as developmental neurotoxicants—lead, methylmercury, PCBs, arsenic, and toluene—and since 2006, epidemiological studies documented an additional six developmental neurotoxicants: manganese, fluoride, chlorpyrifos, DDT, tetrachloroethylene, and the PBDEs.⁴⁴⁸

Of this latter group, manganese, fluoride, chlorpyrifos, and PBDEs are not on the Toxic Pollutants Lists. The American Psychological Association has highlighted the connection between numerous chemicals and developing brains of children, citing studies that demonstrate the effects of endocrine disrupting chemicals on children’s learning and behavior.⁴⁴⁹ Endocrine disrupting chemicals (“EDC”)

have been shown to exert neurotoxic effects that are complex and lead to subtle impairments that are independent of, or indirectly related to, their effects on hormones. For instance, EDCs can disrupt the synthesis, transport, and release of many neurotransmitters, including dopamine, serotonin, norepinephrine, and glutamate, which play key roles in modulating behavior, cognition, learning, and memory. In addition, many neurons coexpress steroid hormone receptors during different stages of development, making them likely targets of EDCs. Therefore, EDCs impinging on steroid sensitive circuitry in the brain can exert effects on

⁴⁴⁷ See, e.g., National Scientific Council on the Developing Child, *Early Exposure to Toxic Substances Damages Brain Architecture, Working Paper No. 4* (Spring 2006), available at https://harvardcenter.wpenginpowered.com/wp-content/uploads/2006/05/Early_Exposure_Toxic_Substances_Brain_Architecture-.pdf; J. Grigg, *Environmental toxins; their impact on children’s health*, 89 *Archives of Disease in Childhood* 244-250 (2004), available at <https://adc.bmj.com/content/archdischild/89/3/244.full.pdf>.

⁴⁴⁸ Philippe Grandjean and Philip Landrigan, *Neurobehavioural effects of developmental toxicity*, 13 *Lancet Neurol* 330-338 (2014), available at <https://www.thelancet.com/action/showPdf?pii=S1474-4422%2813%2970278-3>. Note that this paper identifies a total of 214 substances known to be neurotoxicants to adults. *Id.* at 333, table 1; see also *id.* at Supplementary webappendix, available at [https://www.thelancet.com/cms/10.1016/S1474-4422\(13\)70278-3/attachment/57d395f7-ee37-492c-926a-343a58d302c6/mmcl.pdf](https://www.thelancet.com/cms/10.1016/S1474-4422(13)70278-3/attachment/57d395f7-ee37-492c-926a-343a58d302c6/mmcl.pdf) (lists of neurotoxicants).

⁴⁴⁹ American Psychological Association, *Chemical threats* 46[9] *Monitor on Psychology* 62 (Oct. 2015), available at <https://www.apa.org/monitor/2015/10/cover-chemical> (citing studies on the effects of agricultural chemicals, household chemicals, phthalates, bisphenol A, PBDEs).

cognition, learning, memory, and other nonreproductive behaviors, such as metabolism, as well as reproductive neuroendocrine systems.⁴⁵⁰

The information in this section of the Petition, together with the summaries of data on toxic releases in sub-section III.A.1 *supra*, demonstrates both that there is an inequitable distribution of toxic pollutants in the nation's waters and an inequitable human health effect from consumption of those toxics. These chemicals include many that are not on the Toxic Pollutants Lists. To give real world meaning to its environmental justice mission, EPA must incorporate currently unregulated pollutants into the CWA's regulatory programs, actions that can only be assured by adding these pollutants to the Toxic Pollutants Lists.

V. IMPLICATIONS OF EPA'S FAILURE TO UPDATE THE TOXIC POLLUTANTS LISTS ON NATIONAL EFFLUENT LIMITATIONS GUIDELINES AND PRETREATMENT STANDARDS

EPA's failure to update the Toxic Pollutants Lists has compromised the efficacy of both the technology- and water quality-based regulation of toxic pollutants in the nation's surface waters. In this section, we explain the need for the rules proposed by this petition to address failings in EPA's implementation of the CWA's technology-based approach. We begin in sub-section A with a description of how EPA's failure to update the toxic pollutant lists for 46 years has severely hampered the nation's ability to achieve the CWA's goal of eliminating toxic pollution through national effluent limitations guidelines that are key to implementing the technology-based approach to reducing toxic pollution. The manufacture of plastics is provided as an example. Sub-section B describes how this very same inaction has significantly

⁴⁵⁰ Thaddeus T. Schug, *et al.*, *Elucidating the Links Between Endocrine Disruptors and Neurodevelopment*, 156 *Endocrinology* 1941-1951 (June 1, 2015), available at <https://academic.oup.com/endo/article/156/6/1941/2422760>. See also, Bernard Weiss, *Endocrine Disruptors as a Threat to Neurological Function*, 305(1-2) *Journal of Neurological Science* 11-21 (June 15, 2011), available at <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3090512/pdf/nihms282912.pdf>.

undermined the regulation of industries that discharge toxic wastes to sewage collection system through the pretreatment program, thereby ignoring the statute’s prohibition on indirect discharges of pollutants that pass through sewage treatment facilities to the nation’s waters or that interfere with sewage treatment plant operation, resulting in ineffective sewage treatment and jeopardizing massive investments in that infrastructure. As just one example, this sub-section highlights the example of PFAS, a family of toxic pollutants well known to not be susceptible to removal through sewage treatment.

A. The CWA Goal of Eliminating Discharges Through Use of National Effluent Limitations Guidelines is Severely Hampered by EPA’s Failure to Maintain an Updated List of Toxic Pollutants

The Toxic Pollutant List has significant ramifications for pollution reductions required by the technology-based restrictions that apply to both direct and indirect dischargers of toxic pollutants under the CWA, as described in section II.B, *supra*. Each toxic pollutant on the list is subject to effluent limitations developed by EPA for categories or classes of dischargers,⁴⁵¹ limits that EPA is required to keep updated as treatment removal technology improves.⁴⁵² While EPA is required to develop effluent standards for all pollutants, historically it has focused heavily on toxic pollutants on the Priority Pollutant List, thus ignoring toxic pollutants not on the Toxic Pollutants Lists. To the extent EPA has relied on an assumption that treatment technologies for some pollutants will work for all pollutants, that has been proven incorrect by the experience with PFAS, as discussed in sub-section V.B.3, *infra*.

⁴⁵¹ CWA § 307(a)(2); *see also* CWA § 301(b)(2)(A) (the use of BAT “will result in reasonable further progress toward the national goal of eliminating the discharge of all pollutants” and “shall require the elimination of discharges of all pollutants if the Administrator finds . . . that such elimination is technologically and economically achievable[.]”).

⁴⁵² *Id.* *See also* CWA § 307(a)(3) (EPA is required to review all BAT-based effluent standards and prohibitions every three years).

Thus, for toxic pollutants on EPA’s Priority Pollutant List, pollution controls representing modern treatment technology are required for existing and new facilities, direct dischargers, and indirect so-called pretreating dischargers. Promulgated national ELGs that apply to priority pollutants include many industrial categories for sources of toxic pollutants such as manufacturing and processing oil and gas, plastics, medicines, ores and metals, foods, vegetative and fiber products, chemicals, glass, and large human activities such as hospitals and landfills.

1. EPA’s Failure to Update Toxic Pollutant Lists Undermines the Efficacy of National Effluent Limitation Guidelines

EPA has identified some toxic contaminants not on the Toxic Pollutant List that it considers to be sufficiently hazardous to aquatic life and human health that it has developed 304(a) recommended criteria for them *See* section VII.A and B *infra*. By sharp contrast, however, and despite mandatory statutory requirements for EPA to review and update ELGs frequently, it has not updated ELGs for most major industries for two decades and many for three, four, and approaching five decades⁴⁵³ and therefore has not established ELGs for those very same toxic pollutants. This lag in updating national ELGs and adopting standards for these toxic contaminants undermines the efficacy of the ELGs for these point source categories.

For example, nonylphenol, discussed at sub-section VII.A.11 *infra*, is used in rubber manufacturing, which has the oldest ELG at 47 years old.⁴⁵⁴ Some examples of the extreme age

⁴⁵³ Letter from Eric Schaeffer, Environmental Integrity Project *et al.* to EPA Administration Michael Regan, Re: *EPA’s Annual Review of Effluent Limitation Guidelines Under the Clean Water Act* (Sept. 22, 2021) (hereinafter “EIP Letter”) Attachment: *EPA’s Water Pollution Limits for Industries, Sorted from Oldest to Newest*.

⁴⁵⁴ *Id.* at 1.

of these ELGs were provided in a recent report on the successes and failures of the Clean Water Act.⁴⁵⁵

TABLE 4: AGE OF EPA WATER POLLUTION GUIDELINES FOR SELECT INDUSTRIES

Limits for Industrial Category (years)	Year of Promulgation	Year of Last Revision	Age of Pollution Limit
Rubber Manufacturing	1974	Never Revised	47
Asbestos Production	1974	1974	46
Seafood Processing	1974	1975	46
Dairy Processing	1974	1975	46
Soap Manufacturing	1974	1975	46
Tar & Asphalt	1975	Never Revised	46
Explosives Production	1976	Never Revised	45
Cement Manufacturing	1977	1977	44

Source: Federal Register. Effluent Limitation Guidelines (or ELG's) are technology-based standards that set discharge limits for individual industries, which EPA by law is supposed to review every five years and update to keep pace with improvements in technology. The examples above are only a portion of the 59 guidelines for industries.

Among those ELGs promulgated and/or last revised in the 1970s—40 to 50 years ago—are: rubber manufacturing; asbestos manufacturing; seafood processing; dairy products processing; ferroalloy manufacturing; soap and detergent manufacturing; ink formulating; paint formulating; tars and asphalt; canned and preserved fruits and vegetables processing; explosives manufacturing; gum and wood chemicals; hospitals; photographic; cement manufacturing; carbon black manufacturing; and mineral mining and processing. Those last promulgated or revised in the 1980s—30 to 40 years ago—include: timber products processing; textile mills; inorganic chemicals; electroplating; coil coating; electrical and electronic components; plastics molding and forming; petroleum refining; porcelain enameling; metal molding and casting; glass manufacturing; grain mills; phosphate manufacturing; sugar processing; copper forming; metal finishing; battery manufacturing; fertilizer manufacturing; hard rock mining; aluminum forming; and nonferrous metals forming and metal powders. ELGs last promulgated or revised in the

⁴⁵⁵ Environmental Integrity Project, *Clean Water Act at 50: Promises Half Kept at the Half-Century Mark* (March 17, 2022) (hereinafter “Clean Water Act at 50”) at 15 (Table 4).

1990s—20 to 30 years ago—include organic chemicals, plastics and synthetic fibers (1993 changes remain pending); leather tanning and finishing; pesticide chemicals; and landfills. Those last promulgated or revised in the 2000s—10 to 20 years ago—include transportation equipment cleaning; waste combustors; centralized waste treatment; coal mining; pharmaceutical manufacturing; metals products and machinery; meat and poultry products (pending since 2004); concentrated aquatic animal production; iron and steel manufacturing; pulp, paper, and paperboard; and concentrated animal feeding operations. Finally, those promulgated or revised in the 2010s include: airport deicing; construction and development; oil and gas extraction; dental offices; and steam electric power generating⁴⁵⁶ ELGs.⁴⁵⁷ These delays in maintaining an updated set of ELGs—and associated pretreatment standards—are not only concerning, but also indicates how out-of-step point source pollution controls often are with modern pollution control technology and the technology forcing objectives of the Clean Water Act.

One industrial sector alone—plastic production—is illustrative of the effects of EPA’s failure to update the toxics pollutants lists.⁴⁵⁸ Plastic production is responsible for generating numerous toxic pollutants during the production process, including through the use of additives to create the hundreds of different types of plastics that are currently manufactured.⁴⁵⁹ The industry is significant: over 1,200 facilities manufacture plastic and rubber products in the U.S.

⁴⁵⁶ Revisions have just been proposed for steam electric power generating ELGs. 88 Fed. Reg. 18440 (March 29, 2023).

⁴⁵⁷ See EIP Letter, *supra* n. 453.

⁴⁵⁸ Robert W. Adler and Carina E. Wells, *Plastics and the Limits of U.S. Environmental Law*, 47 Harvard Environmental Law Review 2–62 (2023) (hereinafter “Adler and Wells”). Petitioners extend their appreciation to the authors for use of an advance copy of this publication from which much of this discussion on plastics is derived.

⁴⁵⁹ See Anthony L. Andrady & Mike A. Neal, *Applications and Societal Benefits of Plastics*, 364 Phil. Trans. R. Soc. B (2009) (hereinafter “Andrady and Neal”), available at <https://royalsocietypublishing.org/doi/epdf/10.1098/rstb.2008.0304>.

In the year 2021 alone, these facilities produced 201.6 million pounds of waste reported through TRI, 39.3 million pounds of which was released or disposed of on- and off-site.⁴⁶⁰ And the industry is growing; worldwide, plastic production is expected to triple by 2050.⁴⁶¹ Since 2019, at least 42 new plastics facilities have opened, are under construction, or are in the permitting process in the United States.⁴⁶²

The additives used in the manufacture of plastic are a significant source of toxic chemicals. Nearly all plastic, for example, contains polymer stabilizers allowing them to be melted and molded without degrading the polymer.⁴⁶³ Other additives, such as phthalates, help to make the polymer more malleable.⁴⁶⁴ Because additive chemicals are not bonded to the polymer, they can leach out during product use.⁴⁶⁵ For example, “polycarbonate and polytetrafluoroethylene polymers pose significant toxic risks: the former leaches bisphenol chemicals and the latter can release per- and polyfluoroalkyl substances (or “PFAS”), both of which have adverse effects on human health.”⁴⁶⁶

Notwithstanding the toxic hazards posed by this industry, EPA last adopted ELGs for the

⁴⁶⁰ EPA, TRI, TRI Explorer, *Toxic Release Inventory Factsheet: Industry Sector: Plastics and Rubber*, 326 (2021), available at <https://enviro.epa.gov/triexplorer/industry.html?pYear=2021&pLoc=326&pParent=TRI&pDataSet=TRI> Q1. EPA includes plastic and rubber facilities in the same industry sector analysis; however, the top five establishments for total releases all manufacture plastic products.

⁴⁶¹ Roland Geyer, Jenna R. Jambeck & Kara Lavendar Law, *Production, Use, and Fate of All Plastics Ever Made*, 3 *Sci. Advances* (July 19, 2017) at 3, available at <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5517107/pdf/1700782.pdf>.

⁴⁶² Idna G. Castellón, *Cancer Alley and the Fight Against Environmental Racism*, 32 *Vill. Env't. L.J.* 15 (2021) at 6.

⁴⁶³ Andrady and Neal, *supra* n. 459.

⁴⁶⁴ *Id.*

⁴⁶⁵ John N. Hahladakis *et al.*, *An Overview of Chemical Additives Present in Plastics: Migration, Release, Fate and Environmental Impact During Their Use, Disposal and Recycling*, 344 *J. Hazardous Materials* 179 (2018), available at <https://www.sciencedirect.com/science/article/pii/S030438941730763X?via%3Dihub>.

⁴⁶⁶ Adler and Wells, *supra* n. 458 at 7.

plastics industry in 1987—35 years ago—pursuant to the consent decree in *Train*.⁴⁶⁷ Then, EPA described this category of Organic Chemicals, Plastics, and Synthetic Fibers (“OCPSF”) as manufacturing:

over 25,000 different organic chemicals, plastics, and synthetic fibers. However, less than half of these products are produced in excess of 1,000 pounds per year. The industry includes approximately 750 facilities whose principal or primary production activities are covered under the OCPSF SIC groups. There are approximately 200 other plants which are secondary producers of OCPSF products, i.e., OCPSF production is ancillary to their primary production activities. . . . Thus, the total number of plants to be regulated totally or in part by the OCPSF industry regulation is approximately 1,000.⁴⁶⁸

In describing the OCPSF industries 35 years ago, EPA pointed out that “an exceptionally wide variety of pollutants are found in the wastewaters of this industry” and “an unusually wide variety of toxic priority pollutants (both metals and organic compounds)” as well as “a large number of nonconventional pollutants.”⁴⁶⁹ Nonetheless, at the time EPA promulgated the ELGs for the OCPSF sector, it “focused its attention . . . on the conventional pollutants and on the 126 priority pollutants.”⁴⁷⁰ Moreover, the identification of the chemical constituents in the process wastewaters for this sector to be the subject of control began in 1977—45 years ago.⁴⁷¹ “EPA did not attempt to identify or quantify pollutants other than the priority toxic and conventional

⁴⁶⁷ Organic Chemicals and Plastics and Synthetic Fibers Category Effluent Limitations Guidelines, Pretreatment Standards, and New Source Performance Standards, 52 Fed. Reg. 42522 (Nov. 5, 1987) (hereinafter “OCPSF ELGs”) (codified at 40 C.F.R. Pt. 414).

⁴⁶⁸ *Id.* at 42525. EPA noted: “Secondary OCPSF plants may be part of other chemical producing industries such as the petroleum refining, inorganic chemicals, pharmaceuticals, and pesticides industries as well as chemical formulation industries such as the adhesives and sealants, the pain and ink, and the plastics molding and forming industries.” *Id.*

⁴⁶⁹ *Id.* at 42526.

⁴⁷⁰ *See generally* OCPSF ELGs, 52 Fed. Reg. 42522 (Nov. 5, 1987), *e.g., id.* at 42539 (limits based solely on priority pollutants); 52544 (“EPA has determined that in-plant biological treatment with an acclimated biomass is as effective as activated carbon absorption for removing priority pollutants such as polynuclear aromatic hydrocarbons, phthalate esters, acrylonitrile, phenol, and 2,3-dimethylephenol. EPA has thus selected this treatment for BAT control of these pollutants.”).

⁴⁷¹ *Id.* at 42544.

pollutants.”⁴⁷²

In promulgating the ELGs for the OCPSF sector, EPA first estimated the concentrations of priority pollutants from the sampling it conducted in the ELG development process.⁴⁷³ It concluded that the

current (1980) in-place treatment toxic pollutant annual loadings are estimated to be 1.6 million and 22.6 million pounds for direct and indirect dischargers respectively. The toxic pollutant estimated loadings for direct dischargers after compliance with BAT are 0.49 million pounds, and for indirect dischargers after compliance with PSES [Pretreatment Standards for Existing Sources] are 0.08 million pounds.⁴⁷⁴

EPA explained that it concluded “regulation beyond BPT is warranted” because “many OCPSF plants currently discharge significant amounts of toxics pollutants.”⁴⁷⁵ EPA also noted that “cyanide and toxic pollutant metals such as cadmium, chromium, and lead . . . are commonly found in cooling water additives” such that “[t]he presence of a portion of these metals and cyanide in the diluted effluent seems in many case to be caused by their presence in nonprocess cooling water.”⁴⁷⁶ EPA then evaluated treatment technologies and their costs.⁴⁷⁷ The end result of EPA’s evaluation was to issue BAT limits for 63 priority pollutants for facilities with end-of-pipe biological treatment for conventional pollutants (including 57 organic pollutants, five metals, and cyanide) and BAT limits for 59 priority pollutants (including 53 organic pollutants, five metals, and cyanide) for those facilities without end-of-pipe treatment.⁴⁷⁸ After evaluating the cost of establishing “concentration-based BAT effluent limitations based on the performance

⁴⁷² *Id.* (emphasis added).

⁴⁷³ *Id.* at 42528.

⁴⁷⁴ *Id.* at 42530.

⁴⁷⁵ *Id.*

⁴⁷⁶ *Id.*

⁴⁷⁷ *Id.* at 42550.

⁴⁷⁸ *Id.* at 42538.

of end-of-pipe treatment required to meet BPT limitations . . . plus in-plant control technologies which would remove priority pollutants from water stream from particularly processes prior to discharge to end-of-pipe treatment,” EPA established BAT based on end-of-pipe treatment for all plants other than a those whose annual OCPSF production is less than or equal to five million pounds.⁴⁷⁹ For small facilities, EPA set BAT as equal to BPT based on its evaluation of the cost impacts of requiring treatment at the BAT level.⁴⁸⁰ For future facilities, EPA chose BAT limits for 63 priority pollutants for those facilities with end-of-pipe biological treatment and 59 for those without.⁴⁸¹ EPA devoted less than one column in its Federal Register notice to contemplating whether the NSPS ELGs should be more stringent than BAT, merely concluding that it “has determined that NSPS will not cause a barrier to entry for any new source OCPSF plants.”⁴⁸² There was, however, no evaluation of whether new source OCPSF plants could afford to contain more toxic pollution than existing facilities.

In its promulgation of the final ELGs for OCPSF facilities for 63 priority pollutants,⁴⁸³ EPA did note this limitation on its narrow focus on priority pollutants, observing:

Readers should note that even though nonconventional pollutants and certain toxic pollutants are not directly limited by this regulation, they will nonetheless be indirectly controlled in many cases by the technologies used to comply with the promulgated limitations if they are present in treatable concentrations. While the degree of such indirect control will vary, in some cases unregulated pollutants will be substantially reduced by the operation of technologies installed to comply with limitations for related regulated pollutants.⁴⁸⁴

⁴⁷⁹ *Id.* at 42538–42539.

⁴⁸⁰ *Id.* at 42539.

⁴⁸¹ *Id.* at 42545.

⁴⁸² *Id.*

⁴⁸³ *Id.* at 42538.

⁴⁸⁴ *Id.* at 42544.

Regardless of whether EPA was correct in guessing 35 years ago that its ELGs could or would indirectly control unknown toxics, many of which were not in use at the time, there is no basis to believe that this observation remains true today, particularly given the massive change in the plastics industry and the identification of toxic contaminants well beyond the priority pollutants.

The ubiquitous and highly toxic PFAS pollutants are a key example. In fact, at the time, EPA specifically omitted regulation of 28 priority pollutants the basis for which there is no evidence EPA has ever revisited despite the passage of decades.⁴⁸⁵ Since then, the number of facilities, the increase in types of plastic products produced using different chemical additives than were evaluated in the 1987 ELG analysis, and the toxic loading from these facilities have all increased dramatically. As discussed above, in its ELG analysis EPA relied on a total facility number of 700, a number that stands at 1,200 today.

Moreover, today, OCPSF ELGs that are based on “discharges[’] not exceeding the quantity (mass) determined by multiplying the process wastewater flow subject to this subpart times the concentrations” set out in a table in the regulations—with the exception of chromium, copper, lead, nickel, zinc, and total cyanide⁴⁸⁶—cannot have the same effect on protecting the nation’s waters from the covered toxic pollutants as they did 35 years ago. Given the massive growth in the plastics industry in the ensuing years, the pollutant loading to the nation’s waters from the OCPSF sector can only have increased, a far cry from its being eliminated, as the CWA requires.

⁴⁸⁵ *Id.* at 42549–42550. *See also id.* at 42568 (bases include toxic pollutants purportedly “sufficiently controlled by existing technology”; detected only in a small number or unique sources, only in trace amounts unlikely to cause toxic effects, or sufficiently controlled by existing technology; and toxic pollutants that do not pass through or interfere with sewage treatment facilities).

⁴⁸⁶ These pollutants are covered by provisions in 40 C.F.R. § 414.91(b).

The effect of maintaining outdated Toxic Pollutants Lists is that EPA is severely undermining the technology-based approach to controlling toxic pollution in the nation’s waters. By updating the Toxic Pollutants Lists to reflect current use of chemicals and current understanding of their potential for adverse effects to human health and aquatic species, EPA will be required to carry out the CWA as Congress intended.

2. The Nationwide ELG Program Demonstrates the Need to Update the Toxic Pollutants Lists

In its effort to comply with CWA Section 304(m), EPA has issued a series of Effluent Guidelines Program Plans.⁴⁸⁷ EPA’s failure to update its Toxic Pollutants Lists underlies some of its compounding determinations not to update ELGs for a number of point source categories. For example, EPA determined in 2014 that its 2008 update to the ELGs for Concentrated Animal Feeding Operations (“CAFOs”) was sufficiently recent as to not require further consideration.⁴⁸⁸ But CAFOs are a major source of emerging contaminants of concern such as veterinary pharmaceuticals⁴⁸⁹ and the 2008 ELGs contain no reference to these pollutants and neither do EPA’s 2014 and 2023 ELG Plans in their consideration of CAFOs.

Because EPA has never added pollutants to the Toxic Pollutants Lists, generally

⁴⁸⁷ See, e.g., EPA, Effluent Guidelines, *Effluent Guidelines Plan*, available at <https://www.epa.gov/eg/effluent-guidelines-plan>.

⁴⁸⁸ *Id.* at 3-4-3-5, Table 3-1. EPA confirmed its position in EPA, *Effluent Guidelines Program Plan 15* (Jan. 2023) (hereinafter “2023 ELG Plan”), available at https://www.epa.gov/system/files/documents/2023-01/11143_ELG%20Plan%2015_508.pdf, at Appendix A—Response to Remand of ELG Plan 14 in *Food and Water Watch V.* EPA (No. 21-71084 9th Cir.) (EPA commits to a detailed study and notes its other ELG efforts, making no reference to CECs.)

⁴⁸⁹ See, e.g., JoAnn Burkholder, *et al.*, *Impacts of Waste From Concentrated Animal Feeding Operations on Water Quality*, 115(2) *Environmental Health Perspectives* 308 (Feb. 2007), available at <https://ehp.niehs.nih.gov/doi/epdf/10.1289/ehp.8839> (contaminants entering surface waters from CAFOs include veterinary antibiotics, hormones, and ammonia); see also Washington Department of Ecology, *Fact Sheet for the Draft Concentrated Animal Feeding Operation General Permits* (June 2022) (“Examples of CECs on CAFOS include pharmaceuticals, antimicrobials, disinfection by-products, and microplastics.”).

speaking, these non-listed pollutants are not evaluated by NPDES permit writers, monitoring is not required for them in NPDES permits, and effluent limits are not established. These gaps in pollution data constrict EPA’s review process in evaluating the need for new or revised ELGs. Even as independent scientists identified PFAS as a hazard⁴⁹⁰ and the State of Michigan subsequently found that electroplating and metal finishing industrial sectors were the most prevalent PFOS source categories in the state,⁴⁹¹ EPA was determining that “the Metal Finishing point source category as potentially discharging high concentrations of metals, particularly chromium, nickel, and zinc, to publically [sic] owned treatment works (POTWs). . . . EPA could not identify for further review any new pollutants of concern or wastewater discharges from industrial categories not currently regulated by ELGs.”⁴⁹² EPA did not mention PFAS as a concern with metal finishing.

In that same 2014 Plan, EPA identified:

[S]ix chemicals or classes of chemicals that are currently produced and have known or potential wastewater discharges: Benzidine dyes, Bisphenol A (BPA), Hexabromocyclododecane (HBCD), Nonylphenol and Nonylphenol Ethoxylates, Perfluorinated Chemicals (PFCs), and Phthalates. Another class of chemicals,

⁴⁹⁰ AECOM, *Evaluation of PFAS in Influent, Effluent, and Residuals of Wastewater Treatment Plants (WWTPs) in Michigan; Prepared in association with Michigan Department of Environment, Great Lakes, and Energy* (April 2021) (hereinafter “Michigan PFAS Report”) at 1 (“PFAS have been identified in WWTPs since the early 2000s during the 3M-sponsored Multi-City Study from Alabama, Tennessee, Georgia, and Florida. PFAS were also later identified in WWTPs from Minnesota, Iowa, California, Illinois, New York, Kentucky, Georgia, and Michigan (Boulanger, 2005; Higgins, 2005; Schultz, 2006; Sinclair, 2006; Loganathan, 2007; Sepulvado, 2011; Houtz, 2016).”); *see also id.* (“Effluents discharged from WWTPs and biosolids applied to the agricultural land for beneficial reuse have been identified as potential PFAS release pathways into the environment by the Interstate Technology and Regulatory Council (ITRC) (ITRC, 2017).”), (“Varying concentrations of perfluorooctane sulfonic acid (PFOS), perfluorooctanoic acid (PFOA), and other PFAS have been measured in surface waters in Michigan and biota worldwide in areas remote from known or suspected sources, including in Polar Regions where contamination could occur only through long-range environmental transport (Kannan, 2001; Giesy, 2001; Houde, 2011; Ye, 2008; Stahl, 2014; Custer, 2016; Williams, 2016).:).”)

⁴⁹¹ *Id.* at 21.

⁴⁹² 2014 ELG Plan, *supra* n. 40 at 4-4.

short-chain chlorinated paraffins (SCCPs) are no longer manufactured in the U.S., but they have been used in metal working and have the potential to be discharged in wastewater from this industry.

Additionally, two of the chemicals, Methylene Diphenyl Diisocyanate (MDI) and Toluene Diisocyanate (TDI), do not have significant wastewater discharges.

However, EPA identified that the hydrolysis byproducts of TDI and MDI, toluene diamine and methyl diphenyl diamine, may be present in industrial wastewater. One chemical category is being phased out of U.S. commerce; EPA does not intend to pursue further review for Penta, Octa, and Decabromodiphenyl Ethers (PBDEs).⁴⁹³

Nonylphenol remains unregulated by any EPA-promulgated ELG and is not mentioned in the 2023 ELG Plan, despite EPA's concerns about it as discussed in sub-sections VII.A.11 and V.A.3, *infra*. Of the other chemicals mentioned by EPA, benzidine and some phthalates are priority pollutants, the remainder are not. PFAS is notably barely mentioned in EPA's 2014 ELG Plan in which the agency complains that PFAS sources "contained limited documentation of actual measurements, and instead provided qualitative discussion,"⁴⁹⁴ and yet by 2023, the agency admitted that for the Plastics Molding and Forming Category, "EPA did not identify any PFAS discharge data in DMR or TRI because the category is not currently required to report discharges in NPDES permits or based on current TRI reporting criteria."⁴⁹⁵ Its failure to place PFAS on the Toxic Pollutants Lists ensured the CWA regulatory programs would not gather data on the chemical family. Instead, EPA turned to the Michigan and Wisconsin data on PFAS to obtain data not available from NPDES permits or TRI.⁴⁹⁶ Likewise, EPA's decision to not pursue the "unregulated pollutant" N,N-Dimethylformamide because it had insufficient data on

⁴⁹³ *Id.* at 4-4.

⁴⁹⁴ *Id.* at 3-7.

⁴⁹⁵ 2023 ELG Plan, *supra* n. 488 at 5-10

⁴⁹⁶ *Id.*

its use and that it appeared perhaps “only a small subset of facilities release this pollutant,”⁴⁹⁷ is all the more reason that it should appear on the Toxic Pollutants Lists to ensure that it will be regulated at a source-by-source level. Indeed, “EPA recommends that state and local permitting authorities consider applying water-quality-based effluent limits, as appropriate, to address any potential issues with phthalates or other pollutants in discharges from this category,”⁴⁹⁸ an option that is unlikely to occur so long as EPA fails to assign formal regulatory significance to the pollutant.

EPA determined that it would not update yet other ELGs because “it would be best to address the few facilities with significant dioxin discharges through permitting rather than through the development of national effluent guidelines”⁴⁹⁹ without explaining how it might be likely that these NPDES sources would be addressed “through permitting” given the extreme unlikelihood that a permit writer would develop WQBELs and a similar unlikelihood that a permit writer would develop TBELs based on best professional judgment. This is borne out by the fact that, these few facilities had not been subject to either WQBELs or TBELs to date to prevent the levels of “significant dioxin discharges.”⁵⁰⁰ Because “[f]or Pulp, Paper, and Paperboard, EPA determined that a majority of the estimated dioxin and dioxin-like compound releases reported to TRI were based on pollutant concentrations below the Method 1613B ML,” EPA concluded that “dioxin and dioxin-like compounds from pulp and paper facilities are not a hazard priority at this time.”⁵⁰¹ In offering this rationale, EPA omits the fact that internal

⁴⁹⁷ *Id.* at 5-11.

⁴⁹⁸ *Id.* at 5-11–5-12.

⁴⁹⁹ 2014 ELG Plan, *supra* n. 40 at 5-2 (internal citation omitted).

⁵⁰⁰ *Id.*

⁵⁰¹ *Id.* at 5-4.

monitoring can identify hazardous levels of toxic pollutants before they reach the outfall where they are discharged to waterbodies. For example, EPA noted in its Columbia River Basin Dioxin TMDL that for dioxin, “waste load allocations which result in total plant effluent concentration limits that are below the general analytical detection limit could be monitored for compliance by measuring concentrations in the combined bleach plant waste stream.”⁵⁰² This is precisely how NPDES permits implementing the TMDL’s wasteload allocations to pulp and paper facilities controlled a pollutant that is highly toxic at levels below detection.⁵⁰³

EPA’s similar discussion of its decision to not develop ELGs for some industries based on lack of economically achievable treatment, cites to the purported gap-fillers of TBELs based on best professional judgment and WQBELs based on water quality standards:

EPA is not suggesting that direct and indirect wastewater discharges associated with CBM extraction may not have negative environmental impacts and do not ever need to be controlled. On the contrary, EPA notes that in establishing NPDES permits, permitting authorities, in the absence of applicable ELGs, must establish technology-based effluent limits on a case-by-case basis using best professional judgment (BPJ), considering the same factors that EPA would consider in establishing an effluent guideline (40 CFR 125.3(c)(2)). Additional limitations based on water quality standards are also required to be included in the permits in certain circumstances to protect water quality should specific facilities’ discharges be found to cause, or have the reasonable potential to cause, violations of state water quality standards.⁵⁰⁴

But EPA’s reference to what permit writers “must” do pursuant to its own regulations for issuing NPDES permits ignores the fact that most of these permitting regulations are routinely ignored by permit writers from both states and EPA.

⁵⁰² EPA, *Total Maximum Daily Loading (TMDL) to Limit Discharges of 2,3,7,8-TCDD (Dioxin) to the Columbia River Basin* (Feb. 25, 1991) at C-2.

⁵⁰³ See e.g., Washington Department of Ecology, *National Pollutant Discharge Elimination System Waste Discharge Permit No. WA0003697 (Boise White Paper, L.L.C.)* (Feb. 23, 2018) at 8–9 (effluent limits for Bleach Plant Discharge).

⁵⁰⁴ *Id.* at 5-3–5-4.

Given EPA’s own findings that it is hampered by lack of data and other self-imposed constraints, merely including the impairment status of waterbodies⁵⁰⁵ in its ELG reviews, allegedly in response to environmental justice concerns, is certain to fail. Pollutants that are not on the Toxic Pollutants Lists are extremely unlikely to be the subject of monitoring and therefore are only identified by special studies. Even then, the results of these studies often do not result in waters being placed on a state’s CWA Section 303(d) list of impaired waters due to their refusal to consider sediment and tissue data, lack of screening values where there are no numeric criteria, and EPA’s failure to step in when states fail to meet the requirements of federal regulations as discussed in section VI *supra*. EPA has yet to address emerging pollutants, pharmaceuticals, and “unregulated pollutants” through any of its ELG plans, with the sole exception of PFAS and N,N-Dimethylformamide, demonstrating its general disinterest in using the technology-based approach of the CWA to address pollutants not on the outdated Toxic Pollutants Lists.⁵⁰⁶

3. Toxic Pollutants Acknowledged by EPA to Cause Extensive Contamination and Hazards in the Nation’s Waters are Not Subject to Technology-Based Limitations Because They Are Not on the Toxic Pollutant Lists

As with EPA’s defining PFAS as “nonconventional” rather than “toxic” pollutants, EPA’s choice to not update the Toxic Pollutants Lists fails to reflect the logic of other actions it has taken to address toxic pollutants about which it is concerned. For example, one toxic pollutant not covered by EPA’s ELGs due to its absence from the Toxic Pollutants Lists—despite EPA’s having determined that it posed a sufficiently great threat to aquatic life across the

⁵⁰⁵ 2023 ELG Plan, *supra* n. 488 at 5-14.

⁵⁰⁶ *Id.* at 5-11.

United States that it published recommended criteria for it—is nonylphenol.⁵⁰⁷ A driver of EPA’s concern in 2005 was the large—and increasing—production of nonylphenol:

Nonylphenol is produced in large quantities in the United States. Production was 147.2 million pounds (66.8 million kg) in 1980 (USITC 1981), 201.2 million pounds (91.3 million kg) in 1988 (USITC 1989), 230 million pounds (104 million kg) in 1998 (Harvilicz 1999), and demand is increasing about 2 percent annually.⁵⁰⁸

EPA notes some of the uses for nonylphenol in manufacturing that suggest it is used throughout many industries covered by now-outdated ELGs:

There is little direct use for nonylphenol except as a mixture with diisobutyl phthalate to color fuel oil for taxation purposes and with acylation to produce oxime as an agent to extract copper. Most nonylphenol is used as an intermediate in the production of other chemicals. Notably, nonionic surfactants of the nonylphenol ethoxylate type are produced through etherification of nonylphenol by condensation with ethylene oxide in the presence of a basic catalyst. The nonionic surfactants are used as oil soluble detergents and emulsifiers that can be sulfonated or phosphorylated to produce anionic detergents, lubricants, antistatic agents, high performance textile scouring agents, emulsifiers for agrichemicals, antioxidants for rubber manufacture, and lubricant oil additives (Reed 1978).⁵⁰⁹

According to EPA, compared to its recommended 304(a) acute criterion of 28 µg/L and chronic criterion of 6.6 µg/L,⁵¹⁰ levels of nonylphenol have been found in the aquatic environment at much higher levels:

- “4-nonylphenol at average concentrations ranging from 2 to 1,617 µg/L in eleven water samples associated with various industrial source”
- “in 25 percent of sites sampled in the Great Lakes at concentrations from 0.01 to 0.92 µg/L. They found nonylphenol in all sediment samples with concentrations ranging from 0.17 to 72 µg/g (dry weight)”
- “measured at 0.98 and 7.67 µg/L in the runoff as a result of aircraft deicer and antiicer fluid use”
- “found in approximately 30 percent of the water samples with concentrations ranging from about 0.20 to 0.64 µg/L. Approximately 71 percent of the sampling sites had

⁵⁰⁷ See 2005 Nonylphenol Criteria, *supra* n. 8.

⁵⁰⁸ *Id.* at 1.

⁵⁰⁹ *Id.*

⁵¹⁰ *Id.* at 34.

measurable concentrations of nonylphenol in the sediments at concentrations ranging from about 10 to 2,960 µg/kg”

- “reconnaissance of 95 organic wastewater contaminants in 139 U.S. streams conducted in 1999-2000 revealed that nonylphenol was one of the most commonly occurring contaminants and was measured at higher concentrations than most of the other contaminants”
- “concentrations in sediments below wastewater outfalls and found one site that had a sediment concentration of 54,400 µg/kg more than twenty years after the treatment plant ceased operation”
- “widely distributed in lower Great Lakes sediments and reached 37,000 µg/kg in sediments near sewage treatment plants”⁵¹¹

Despite the vast quantities of nonylphenol produced in the United States, the resulting accumulation in water column and sediments, and EPA’s concern about its toxic effects, this toxic pollutant is not addressed through EPA’s ELGs because it is not listed on the Toxic Pollutants Lists.⁵¹² The same is true of all other pollutants for which EPA has had sufficient concern to develop 304(a) recommended criteria but has not put on the Toxic Pollutants List, as described in section VII.A and B *infra*.

B. The CWA Goal of Eliminating Toxic Discharges Through Use of Pretreatment Standards is Hampered by EPA’s Failure to Maintain an Updated List of Toxic Pollutants

In 1986, more than one-third of all toxic pollutants entering the nation’s waters from publicly owned treatment works (“POTWs”) came from industrial discharges to public sewers.⁵¹³ The federal pretreatment program addresses some of those pollutants, namely those on the Toxic Pollutants Lists. For example, in 1991, EPA estimated that 190 to 204 million pounds of metals and 30 to 108 million pounds of organics were removed each year as a result of

⁵¹¹ *Id.* at 2–5.

⁵¹² See EPA, *Effluent Limitations Guidelines and Standards (ELG) Database* (hereinafter “ELG Database”), available at <https://owapps.epa.gov/elg/> (search on “nonylphenol” in Pollutant Search).

⁵¹³ EPA, *Introduction to the National Pretreatment Program*, EPA-833-B-98-002 (Feb. 1999) at iii.

pretreatment program requirements.⁵¹⁴ However, that same year, EPA estimated that approximately half of the mass of the most common toxics in publicly owned sewage treatment facilities' waste streams were released to surface waters, the rest contaminating sewage sludge and a small fraction volatilizing.⁵¹⁵

Publicly owned treatment works ("POTW") collect and treat wastewater from homes, commercial buildings, and industrial sources.⁵¹⁶ The POTW removes some harmful organisms and other contaminants from the sewage before it is discharged. POTWs are designed to treat domestic sewage but are not generally designed to remove specific toxic contaminants unless required to do so by water quality-based effluent limitations.⁵¹⁷ The CWA establishes secondary treatment as the ELG for POTWs.⁵¹⁸ Even so, many POTWs receive wastewater from industrial facilities that discharge into the collection system (along with commercial wastes, household toxics, and urban runoff in cases of combined sewer systems). Industrial facilities that discharge to POTW collection systems do not have NPDES permits as they would if they were direct dischargers to waters of the state. Instead, these indirect dischargers may or may not fall under the federal pretreatment program.

⁵¹⁴ *Id.* at 3.

⁵¹⁵ EPA, *Report to Congress National Pretreatment Program* (July 1991) at 6-6.

⁵¹⁶ Some industrial wastes are "hauled" waste, such as septage and wastes produced from hazardous waste clean-up.

⁵¹⁷ Generally, publicly owned sewage treatment plants are designed to treat domestic sewage only. Primary treatment is designed to remove large solids and smaller inorganic grit through methods such as screening and settling. Secondary treatment removes organic contaminants using microorganisms to consume biodegradable organics through such approaches as activated sludge, trickling filters, and rotating biological contactors. These facilities may also use tertiary treatment such as nitrification (to convert ammonia and nitrite to the less toxic nitrate), denitrification (to convert nitrate to molecular nitrogen), physical-chemical treatment (to remove dissolved metals and organics). Disinfection is used to kill any remaining human pathogens. The sewage sludge that is produced may be used as fertilizer, regulated under the biosolids program, or disposed of as waste. The CWA establishes secondary treatment as the ELG for publicly owned sewage treatment plants. *See* CWA § 301(b)(1)(B).

⁵¹⁸ *See* CWA § 301(b)(1)(B).

As designed by EPA, the federal pretreatment program consists of three basic components: (1) prohibited discharge standards; (2) categorical standards, and (3) local limits. EPA has established regulations that determine the respective responsibilities of government agencies, POTW authorities, and indirect discharging industries to implement federal pretreatment standards to control pollutants that may: (1) pass through or (2) interfere with POTW treatment processes, including interfering with the beneficial use of sewage sludge.⁵¹⁹ EPA regulations define and limit those toxic pollutants that “pass through,” and therefore must be regulated, as “a discharge which exits the POTW into waters of the United States in quantities or concentrations which, alone or in conjunction with a discharge or discharges from other sources, is a cause of a violation of any requirement of the POTW’s NPDES permit (including an increase in the magnitude or duration of a violation).”⁵²⁰ In contrast, however, the CWA defines “pass through” toxic pollutants that require pretreatment standards more broadly to include: (1) “those pollutants which are determined not to be susceptible to treatment by such treatment works or which would interfere with the operation of such treatment works”; and (2) “[any] pollutant [that] interferes with, passes through, or otherwise is incompatible with such works.”⁵²¹ The difference is that EPA has limited the requirement for pretreatment to only those pollutants that cause a violation of an NPDES permit, an unlikely scenario because most sewage treatment plants do not have effluent limitations on toxic pollutants in the first place.

EPA regulations that implement the statutory prohibition on “interference” are defined as that which—alone or in conjunction with other discharges—either (1) “inhibits or disrupts the

⁵¹⁹ 40 C.F.R. § 403.2(a), (b); 403.5(a).

⁵²⁰ 40 C.F.R. § 403.3(p).

⁵²¹ CWA § 307(b)(1).

POTW, its treatment processes or operations” and/or (2) “[t]herefore is a cause of a violation of any requirement of the POTW’s NPDES permit (including an increase in the magnitude or duration of a violation).”⁵²² It also includes eight categories of specific prohibitions including discharges that create a fire, explosion hazard, or corrosive structural damage; cause flow obstructions; contain heat that will inhibit biological activity; oils that will cause interference or pass through, or result in the presence of toxic gases; are composed of trucked or hauled pollutants except at designated locations; or any other discharges of “[a]ny pollutant, including oxygen demanding pollutants (BOD, etc.) released in a Discharge at a flow rate and/or pollutant concentration which will cause Interference with the POTW.”⁵²³ In addition to the likelihood that interference will result in additional pollution of the nation’s waters, it can impose additional costs on POTW operators, thus shifting costs from industrial dischargers to the public sector and jeopardizing public investments in wastewater infrastructure.

“Categorical standards” are national, uniform, technology-based standards that apply to indirect discharges for both existing and new sources. The goal of these categorical standards is to prevent the discharge of pollutants that could pass through, interfere with, or otherwise be incompatible with POTW operations. EPA has issued specific categorical standards for some industrial categories whereas it relies on general prohibitions and local limits for other categories. The categorical standards are intended to account for any pollutant removal that the POTW may accomplish. Dischargers are required to comply with categorical standards by a date certain, usually not more than three years after promulgation, while new source standards usually apply not longer than 90 days after a discharge commences. Categorical standards can

⁵²² 40 C.F.R. § 403.3(k) (omitting references to sewage sludge).

⁵²³ 40 C.F.R. § 403.5(b).

be concentration- or mass-based.⁵²⁴

As with ELGs, categorical standards only restrict certain pollutants in a given waste stream. Therefore, a source covered by categorical standards may have pollutants that are unregulated because they are not the subject of restrictions or because they are composed of the following: sanitary waste streams, demineralized backwash streams, boiler blowdown, noncontact cooling water, storm water, and any process waste streams based on the findings they contain none of the regulated pollutant or only trace amounts. According to EPA,

[t]he standards applicable to indirect dischargers (also called categorical pretreatment standards) are listed under each as pretreatment standards for existing sources (PSES) and pretreatment standards for new sources (PSNS). Not all ELGs contain PSES and PSNS; EPA implements PSES and PSNS for 35 (out of 58) industrial categories.⁵²⁵

The last category of pretreatment standards is local limits, which are specific discharge limits developed by POTWs in order to implement the federal regulations' general and specific discharge prohibitions on pass through and interference, and to address the specific needs of a POTW and its receiving waters.⁵²⁶ Federal regulations require local authorities to evaluate whether local limits are needed and to implement them if necessary.⁵²⁷ Although EPA states in guidance that local limits “should correct existing problems, prevent potential problems, protect the receiving waters, [and] improve sludge use options,”⁵²⁸ all of which suggests the potential

⁵²⁴ 40 C.F.R. § 403.6.

⁵²⁵ EPA, National Pollutant Discharge Elimination System (NPDES), *Pretreatment Standards and Requirements-Categorical Pretreatment Standards*, available at <https://www.epa.gov/npdes/pretreatment-standards-and-requirements-categorical-pretreatment-standards> (emphasis added).

⁵²⁶ 40 C.F.R. § 403.5(d).

⁵²⁷ 40 C.F.R. § 403.8(f)(4).

⁵²⁸ EPA, *Introduction to the National Pretreatment Program* (June 2011) at 3-8.

broad use of local limits to eliminate the discharge of toxic pollutants, in practice, the use of local limits is far more narrow. EPA's local limits guidance states that

[a]mong the factors a POTW should consider in developing local limits are the following: the POTW's efficiency in treating wastes; its history of compliance with its NPDES permit limits; the condition of the water body that receives its treated effluent; any water quality standards that are applicable to the water body receiving its effluent; the POTW's retention, use, and disposal of sewage sludge; and worker health and safety concerns.⁵²⁹

EPA also notes that while its categorical standards regulate “[p]rimarily Priority Pollutants listed under Clean Water Act Section 307 (toxic and non-conventional pollutants only),” local limits apply to “[a]ny pollutant that may cause pass through or interference.”⁵³⁰

The dependence of the pretreatment program on outdated ELGs that are themselves based on outdated Toxic Pollutants Lists undermines the pretreatment program's ability to interdict toxics before they are discharged to the nation's waters. In addition, the reliance on local limits to fill the gaps in the pretreatment program is misplaced. Few sewage treatment plants have limits on toxic pollutants for the reasons discussed in section VI of this petition, *infra*. And, as a practical matter, the toxic loading from indirect dischargers to sewage collection systems is significantly diluted by the domestic sewage, stormwater, and inflow and infiltration of groundwater such that concentrations of toxics in sewage influent or effluent may be low (including to the point of not being detected or quantified) even as toxic loading may be significant. This is particularly true for pollutants that concentrate in sediments and the food web.

⁵²⁹ *Id.* at 1-1.

⁵³⁰ *Id.* at 1-4, Table 1.1.

1. Pretreatment Local Limits are Invariably Limited to Priority Pollutants Making Technology-Based Pretreatment Standards Essential for Reducing Non-Priority Toxics

EPA’s observation that local limits apply to “any pollutants” is disingenuous given the extreme unlikelihood that toxic pollutants that are not on the Priority Pollutant List will even be identified in either an indirect discharger’s or POTW’s waste stream, let alone be identified as requiring water quality-based effluent limitations at the sewage treatment plant. This is in part due to the EPA application Form 2A NPDES for sewage treatment plants that allows but does not require an applicant to provide data on non-priority pollutants.⁵³¹ Similarly, Question No. A.10 on this form, a “Description of Receiving Waters,” does not require the applicant to provide any receiving water quality data or information that it may have gathered to the permitting agency. Moreover, the factors that EPA cites about whether a sewage treatment facility should consider developing local limits are seriously constrained by the ways in which NPDES WQBELs are developed, namely that they are based on inadequate data on receiving water and effluent quality and inadequate 303(d) lists of water quality impairments, are based on artificial dilution through the use of regulatory mixing zones, generally are not based on wasteload allocations from TMDLs, and generally fail to account for far-field effects such as sediment deposition and bioaccumulation in the food web, as explained in section VI *infra*. EPA’s suggestion that such local limits should be based on any “its history of compliance with its NPDES permit limits” is similarly flawed because there is little likelihood that there will be NPDES permit limits for non-priority toxics with which the sewage treatment facility has not

⁵³¹ See EPA, *Application Form 2A NPDES* at Part D, Expanded Effluent Testing Data (“Provide the indicated effluent testing information and any other information required by the permitting authority for each outfall through which effluent is discharged. . . . Indicate in the blank rows provided below any data you may have on pollutants not specifically listed in this form.”).

complied.

2. EPA Determinations of Pollutants Not Susceptible to Treatment by Sewage Treatment Facilities are Outdated and EPA Has Not Complied with the Statutory Requirement that it Update Pretreatment Standards

As described in sub-section II.B.2 *supra*, the CWA requires EPA “from time to time” to publish pretreatment standards “for those pollutants which are determined not to be susceptible to treatment” by publicly owned sewage treatment works.⁵³² In order to meet this requirement, EPA must therefore—from time to time—reevaluate whether its existing determinations of pollutants not susceptible to treatment remains either accurate or complete. It has not. Instead, EPA has relied heavily, and in some instances exclusively, on its Priority Pollutant List upon which to develop ELGs and therefore to develop pretreatment standards that are all based on ELGs, as discussed *supra* section V.A.1. As a consequence, EPA has not complied with the statute’s mandate that pretreatment standards “be established to prevent the discharge of any pollutant . . . which pollutant . . . passes through” publicly owned sewage treatment works.⁵³³ These requirements for a continued effort by EPA to identify and develop standards for toxic pollutants entering the nation’s waters through sewage treatment plants are re-emphasized by CWA Section 307(b)(2), which requires EPA “from time to time, as control technology, processes, operating methods, or other alternatives change, *revise such [pretreatment] standards[.]*”⁵³⁴

In order to evaluate pollutants that pass through sewage treatment plants, EPA must evaluate two questions. The first: Which toxic pollutants are substantially removed through

⁵³² CWA § 307(b)(1).

⁵³³ CWA § 307(b)(1).

⁵³⁴ CWA §307(b)(2) (emphasis added).

advanced nutrient removal technologies in sewage treatment plants? The second: Which toxic pollutants pass through sewage treatment plants regardless of the treatment technology employed such that only pretreatment can prevent them from entering the nation's waters? Because studies show both that many toxic pollutants are susceptible to removal with advanced sewage treatment for nutrient removal and that some toxic pollutants are not, EPA must answer both questions to ensure that an up-to-date federal pretreatment program serves the CWA's goals of eliminating toxic pollution in the nation's waters.

EPA knows that the technology-based requirements for sewage treatment plants—set by statute at “secondary treatment”—are not expected to and do not remove many toxic contaminants. Secondary treatment is a pollution abatement technology over a century old.⁵³⁵ It was also noted long ago—in the 1950s and 1960s—that secondary treatment did not reliably or predictably remove nitrogen or ammonia.⁵³⁶ Likewise, “[t]he problem of how to remove phosphorus in activated sludge processes was solved [in 1974 and] is now applied worldwide.”⁵³⁷ It is now well understood that secondary treatment is not adequate to ensure the removal of either nitrogen or phosphorus from sewage prior to discharge.⁵³⁸

⁵³⁵ See P.F. Cooper, *Historical Aspects of Wastewater Treatment, in Decentralized Sanitation and Reuse: Concepts, Systems and Implementation* (2001) at 27-28, available at <http://www.bvsde.paho.org/bvsacd/leeds/cooper.pdf>. The first use of activated sludge treatment systems to separate, aerate, and oxidize wastewater date from approximately 1913-1914, with the first full-fledged sewage treatment systems having come on-line in 1920.

⁵³⁶ *Id.* at 29.

⁵³⁷ *Id.* at 30.

⁵³⁸ See e.g., EPA, *Municipal Nutrient Removal Technologies Reference Document Volume 1—Technical Report ES-1* (Sept. 2008), available at <https://nepis.epa.gov/Exe/ZyPDF.cgi/P100GE8B.PDF?Dockey=P100GE8B.PDF>, at 1-1 (“Wastewater treatment plants (WWTPs) that use conventional biological treatment processes designed to meet secondary treatment effluent levels do not remove nitrogen or phosphorus to any substantial extent.”); EPA, *Primer for Municipal Wastewater Treatment Systems* (Sept. 2004), available at <https://www3.epa.gov/npdes/pubs/primer.pdf>, at 8 (“Conventional secondary biological treatment

In contrast, studies show that advanced secondary and tertiary treatment of sewage, in addition to the aeration and oxidation methods used by secondary treatment, are effective at removing nitrogen and phosphorus from wastewater.⁵³⁹ These treatments are, in turn, able to remove some toxic contaminants from influent. In an EPA study completed with the Washington Department of Ecology, nutrient removal technology was found to be efficacious in its ability to concurrently remove a wide array of toxic chemicals, concluding the results were “consistent with findings of published studies which reported that additional [wastewater treatment plant] nutrient removal provides better removal of [pharmaceuticals and personal care products] PPCPs than is achieved by secondary treatment technologies alone.”⁵⁴⁰ This study evaluated 72 pharmaceuticals and personal care products (“PPCP”), 27 hormones and steroids, and 73 semi-volatile organic compounds.⁵⁴¹ The summarized results showed that different nutrient removal technologies resulted in different levels of removal for the three categories of toxics.⁵⁴²

processes do not remove the phosphorus and nitrogen to any substantial extent—in fact, they may convert the organic forms of these substances into mineral form, making them more usable by plant life.”)

⁵³⁹ See e.g., EPA, *Case Studies on Implementing Low-Cost Modifications to Improve Nutrient Reduction at Wastewater Treatment Plants DRAFT – Version 1.0*, at 10 (August 2015), available at https://www.epa.gov/sites/production/files/2015-08/documents/case_studies_on_implementing_low-cost_modification_to_improve_potw_nutrient_reductioncombined_508_-_august.pdf.

⁵⁴⁰ Washington Department of Ecology/EPA, *Pharmaceuticals and Personal Care Products in Municipal Wastewater and their Removal by Nutrient Treatment Technologies (Jan. 2010)* (hereinafter “Removal of PPCPs”), at ix, 4 – 5 (reviewing existing studies that demonstrate removal of pharmaceuticals and personal care products); see also EPA, *Advanced Wastewater Treatment to Achieve Low Concentration of Phosphorus* (April 2007), available at <https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P1004JC4.TXT>.

⁵⁴¹ *Id.* at vii.

⁵⁴² Removal of PPCP, *supra* n. 540, at 43, table 26 (grouping treatment results that achieved at least an 80 percent reduction in the concentrations of pollutant categories).

Table 26. Categorical removal efficiencies in wastewater effluent by treatment type.

Category	PPCPs ¹⁶⁹⁴	Hormones/ Steroids	Semi-volatile Organics
High = >80% of analytes had at least 80% reduction in concentration	EBNR+F * EBNR+MF	EBNR+F * EBNR+MF * CA+F * AS+N * EBNR AD AS	EBNR * EBNR+F * CA+F AD
Moderate = 60-80% of analytes had at least 80% reduction in concentration	CA+F	--	AS+N AS
Low = <60% of the analytes had at least 80% reduction in concentration	EBNR AS+N AS AD	--	EBNR+MF

* = The treatment technologies that produced a 1-log reduction for at least 80% of the detected influent analytes.

Further evidence that EPA’s existing determinations of toxic pollutants not susceptible to removal by secondary treatment comes from its report citing a 2007 study that “performed a comprehensive analysis of the use of various membrane and activated carbon technologies on the removal of pharmaceuticals, endocrine-disrupting compounds, and personal care products.”⁵⁴³ EPA and Washington set out the results of this study as follows.⁵⁴⁴

⁵⁴³ *Id.* at 66, citing Snyder, S.A., *et al.*, *Role of Membranes and Activated Carbon in the Removal of Endocrine Disruptors and Pharmaceuticals*, 202 *Desalination* 156-181 (2006).

⁵⁴⁴ *Id.* at 76, Table A-6.

Table A-6. Comparison of wastewater treatment concentrations (ng/l) for a select set of pharmaceuticals.

(Modified from Snyder et al., 2006a; Drury et al., 2006; Ternes et al., 2002; Heberer et al., 2004.)

Chemical	Influent	Primary	Secondary	Micro-filtration	Ultra-filtration	PAC	EDR	MBR	RO	Double pass RO	Ozone
Acetaminophen	21,950	4095	<20	10	<10	53	3.4	<1.0	<1.0	<1.0	nd
Caffeine	58,550	6775	<20	6125	14	44	<10	<1.0	16	1.2	nd
Carbamazepine	299	138	110	271	147	35	18	205	<1.0	<1.0	<0.5
DEET	690	168	104	3365	103	18	112	37	3.4	<1.0	10
Erythromycin	479	9.4	336	507	357	17	<1.0	96	<1.0	<1.0	nd
Estradiol	<100	<1.0		<1.0		42	<1.0	<1.0	<1.0		nd
Estriol	226	67		<5.0		40	<5.0	<1.0	<5.0		nd
Ibuprofen	70,350	641	19	422		nd	5.4	4	<1.0	<1.0	nd
Meprobamate	520	92	693	341	715	19	71	236	<1.0	<1.0	97
Naproxen	21,000	599	<20	1205	17	2	<1.0	26	2	<1.0	<0.5
Oxybenzone	896	181	48	60	26	79	3.8	3.1	1.9	<1.0	nd
Sulfamethoxazole	234	103	90	805	56	23	<1.0	265	2	<1.0	3.2
TCEP	464	151	189	467	219	15	127	186	1.9	1.3	352
Triclosan	4,030	176	29	424	<10	71	<1.0	7.6	<1.0	<1.0	<1.0
Trimethoprim	699	144	186	409	158	72	<1.0	15	<1.0	<1.0	4.4

EDR - Electrodialysis reversal.

Additional studies support these findings. One, published by the Water Environment Research Foundation, built on the work done predominantly in Europe on minimum critical solids retention time to achieve good removal of endocrine disrupting compounds and pharmaceuticals, which were determined for 20 pharmaceuticals and personal care products to consistently remove greater than 80 percent of the compound.⁵⁴⁵ A Canadian study showed that the efficacy of removing the female sex hormones estradiol and estrone from treated sewage was greatly improved in sewage treatment plants that achieved nitrification.⁵⁴⁶ Likewise, tertiary treatment has been found to be the most effective method to remove estrogenic hormones from the discharge water.⁵⁴⁷ But, finally, there are many toxic compounds that cannot be addressed

⁵⁴⁵ Water Environment Research Foundation, *Fate of Pharmaceuticals and Personal Care Products Through Municipal Wastewater Treatment Processes* (2007) at iv.

⁵⁴⁶ See Servos et al., *Distribution of estrogens, 17 β -estradiol and estrone, in Canadian municipal wastewater treatment plants*, 336 *Science of the Total Environment*, 155 (Jan. 2005), available at <https://www.sciencedirect.com/science/article/pii/S0048969704004565>.

⁵⁴⁷ See Lucy Kirk et al., *Changes in estrogenic and androgenic activities at different stages of treatment in wastewater treatment works*, 21 *Environmental Toxicology and Chemistry* 972 (2002), available at <http://onlinelibrary.wiley.com/doi/10.1002/etc.5620210511/full>.

through sewage treatment: The USGS has concluded that the evidence demonstrates that “many such compounds survive wastewater treatment and biodegradation.”⁵⁴⁸

Despite EPA’s knowledge that use of nutrient removal technologies by sewage treatment plants can reduce toxics in discharges, including those not on the Priority Pollutant List, and that some pollutants are not susceptible to treatment by even tertiary treatment, EPA has not taken appropriate steps to address these issues. These steps should be to: (1) conduct a formal evaluation of all toxic pollutants—both those on and off the Toxic Pollutants Lists—for susceptibility to sewage treatment, both secondary and advanced; (2) address the class of toxic pollutants not susceptible to secondary treatment with new or revised pretreatment standards; (3) issue guidance for permit writers on how to write water quality-based effluent limitations for pollutants that are not susceptible to sewage treatment and are not primarily from sources subject to pretreatment standards (i.e., domestic sewage); and (4) address the class of toxic pollutants not susceptible to any form of treatment with new or revised pretreatment standards. By updating the Toxic Pollutants Lists, EPA will ensure that such updates are legally required.

3. PFAS is One Example of a Pollutant Not on the Toxic Pollutants Lists That is Not Susceptible to Treatment and Removal by Sewage Treatment Facilities

The family of PFAS chemicals is a well-known example of a pollutant that is not susceptible to treatment and removal by sewage treatment plants, regardless of nutrient removal technology. Moving well beyond the efforts of and requirements established by EPA for pretreatment, the Michigan Department of Environment, Great Lakes, and Energy (“EGLE”) has used its state pretreatment program to reduce PFAS in sewage treatment plant discharges.

⁵⁴⁸ Pharmaceuticals National Reconnaissance, *supra* n. 164 at 1210 (internal references omitted).

Michigan reported that “industrial discharges are expected to be the primary sources of PFAS” to sewage treatment plants.⁵⁴⁹ It cited the following as likely PFAS sources:⁵⁵⁰

- Electroplating & Metals Finishing Facilities
- Landfills
- Centralized Waste Management Facilities
- Airfields – Commercial, Private and Military
- Department of Defense (DoD) Facilities
- Fire Department Training Facilities
- Petroleum or Petrochemical Manufacturers and Storage Facilities
- Commercial Industrial Laundries
- Chemical Manufacturers
- Plastics Manufacturers
- Textile & Leather Facilities
- Paint Manufacturers
- Pulp & Paper Facilities

Because of the strong correlation of PFAS in treated sewage effluent with receipt of industrial discharges at sewage treatment facilities, EGLE focused its PFAS reduction program on pretreatment.⁵⁵¹ Unusually, given EPA’s failure to complete 304(a) recommended criteria for PFAS and to place PFAS on the Toxic Pollutants Lists, Michigan has adopted numeric human health and aquatic life criteria for the PFAS chemicals perfluorobutanesulfonate (“PFBS”), perfluorooctanoic acid (“PFOA”), and perfluorooctane sulfonate (“PFOS”).⁵⁵² When it identified a sewage treatment plant discharge in excess of the PFOS criterion, caused by a chrome plater, it “initiated the [industrial pretreatment program] IPP PFAS Initiative in February 2018 to reduce and/or eliminate PFOA and PFOS from industrial sources that may pass through WWTPs and enter lakes and streams, potentially causing fish consumption advisories or contaminating public drinking water supplies.”⁵⁵³ This initiative required all 95 of the

⁵⁴⁹ Michigan PFAS Report, *supra* n. 490.

⁵⁵⁰ *Id.* at 2.

⁵⁵¹ *Id.* at 3, 6.

⁵⁵² See Michigan EGLE, *Rule 57 Surface Water Quality Values* (Sept. 26, 2022), available at <https://www.michigan.gov/egle/-/media/Project/Websites/egle/Documents/Programs/WRD/SWAS/rule-57-values.xlsx?rev=91582d6bfa554b5aac0bf9d0667a75d&hash=8F8F76C57635BE8DD82D0C0F6D01EF8F>.

⁵⁵³ Michigan PFAS Report, *supra* n. 490 at 5.

Michigan’s sewage treatment plants with pretreatment programs to evaluate any pass-through of PFOA and PFAS, including to:

- Identify industrial users discharging to their system that were potential sources of PFOA and PFOS. Based on literature reviews and knowledge of Michigan, EGLE highlighted the following industrial categories as potential sources of PFOA and/or PFOS to WWTPs: metal finishers and electroplaters utilizing fume suppressants, tanneries, leather and fabric treaters, paper and packaging manufacturers, landfill leachate, centralized waste treaters, and sites where aqueous film-forming foam (AFFF) was used. WWTP staff was asked to evaluate these potential sources via surveys, records reviews, and industry staff interviews.
- Sample the effluent of those sources that were likely to have used PFOA and/or PFOS in the past or were currently using some type of PFAS-containing chemical in their processes.
- Sample the WWTP discharge (i.e., effluent) if sources were found to be discharging above a screening level, which EGLE recommended be set conservatively at the WQS for PFOA and PFOS.
- Require PFOA and PFOS reduction at confirmed sources through pollutant minimization plans, equipment/tank change out/cleanouts, product replacements, and treatment installation to remove PFOS before discharge (i.e., pretreatment).
- Recommend WWTPs develop technically-based local limits to determine PFOS and/or PFOA concentrations that can be discharged to the WWTP without passing through at levels exceeding WQS or interfering with the WWTP operation.
- Monitor the progress of industrial users reducing PFOA and PFOS.
- Submit reports and monitoring results as required by EGLE’s Water Resources Division (WRD).⁵⁵⁴

Those Michigan sewage treatment plants identified as having industrial sources of PFOA and PFAS were required to have NPDES permits with monitoring and source reduction programs. The program was a success: “For a subset of WWTPs, a total PFOS reduction between 88% to 99% was achieved through source reduction efforts[.]”⁵⁵⁵ The efficacy of using

⁵⁵⁴ *Id.* at 5.

⁵⁵⁵ *Id.* at 11.

granular activated carbon (“GAC”) to remove PFOS—considered to be the “regulatory driver” of the two PFAS pollutants⁵⁵⁶—is illustrated by the following results:⁵⁵⁷

Table 9. Substantial PFOS Reduction at WWTPs with Exceedances

Municipal WWTP	Recent PFOS, Effluent* (ng/L)	PFOS Reduction (highest to most recent)	Actions Taken to Reduce PFOS
Bronson WWTP	5	99%	Treatment (GAC) at source (1)
Howell WWTP	5	96%	Treatment (GAC/Resin) at source (1)
Ionia WWTP	<6	99%	Treatment (GAC) at source (1)
Kalamazoo WWTP	5	90%	Treatment (GAC) at source (2), change of water supply
KI Sawyer WWTP	9	96%	Eliminated leak of AFFF
Lapeer WWTP	8.2	99%	Treatment (GAC) at source (1)
Wixom WWTP	34	99%	Treatment (GAC) at source (1)

*Data received as of December 31, 2020

Michigan also found that sewage treatment plants without pretreatment programs “may still have industrial or commercial PFAS discharges that impact the WWTP.”⁵⁵⁸ In other words, the state determined that EPA’s pretreatment requirements were not sufficiently broad to capture important PFAS sources.

Michigan also evaluated categories of industrial dischargers, starting with 430 individual categorical industrial users (“CIU”) representing 18 pretreatment categories, from which 310 CIUs were sampled. Caution about using the results, particularly for other states, was expressed due to insufficient sample collection: “For example, category 419 (Petroleum Refining) had only one representative industry sampled multiple times, with the highest PFOA concentration of 710 ng/L and PFOS of 800 ng/L.”⁵⁵⁹ Nonetheless, the sampling demonstrated electroplating and metal finishing as the most prevalent PFOS source categories in the State of Michigan.⁵⁶⁰

⁵⁵⁶ *Id.* at 11.

⁵⁵⁷ *Id.* at 14.

⁵⁵⁸ *Id.* at 18.

⁵⁵⁹ *Id.* at 22.

⁵⁶⁰ *Id.* at 23.

Table 11. CIU PFAS Summary Results¹

Category Description	40 CFR Part	Total CIU	Number and (%) of CIU Sampled	PFOA Number and (%) of Detections	PFOA Minimum (Min) (ng/L)	PFOA Maximum (Max) (ng/L)	PFOS Number and (%) of Detections	PFOS Number and (%) of Sources (>WQS)	PFOS Minimum (Min) (ng/L)	PFOS Maximum (Max) (ng/L)
Textile Mills	410	1	1 (100%)	1 (100%)	7	114	1 (100%)	1 (100%)	2	36
Electroplating	413	46	44 (96%)	15 (34%)	1.6	19	29 (66%)	19 (66%)	0.4	50,000
Organic Chemicals, Plastics, and Synthetic Fibers	414	8	4 (50%)	2 (50%)	3	7	2 (50%)	0 (0%)	4	5
Soap and Detergent Manufacturing	417	6	1 (17%)	0 (0%)	---	---	0 (0%)	0 (0%)	---	---
Petroleum Refining	419	1	1 (100%)	1 (100%)	4	710	1 (100%)	1 (100%)	7	800
Iron and Steel Manufacturing	420	12	8 (67%)	3 (38%)	1.9	43	2 (25%)	0 (0%)	1.4	4
Steam Electric Power Generating	423	7	1 (14%)	0 (0%)	---	---	0 (0%)	0 (0%)	---	---
Leather Tanning and Finishing	425	1	1 (100%)	0 (0%)	---	---	1 (100%)	1 (100%)	10.0	14
Pulp, Paper, and Paperboard	430	4	4 (100%)	4 (100%)	13	110	4 (100%)	4 (100%)	2	190
Metal Finishing	433	281	212 (75%)	67 (32%)	0.3	740	71 (33%)	32 (15%)	0.7	240,000
Centralized Waste Treatment	437	17	14 (82%)	14 (100%)	0.5	3,000	13 (93%)	12 (86%)	1.1	53,000
Pharmaceutical Manufacturing	439	16	5 (31%)	0 (0%)	---	---	1 (20%)	0 (0%)	3	3
Transportation Equipment Cleaning	442	8	3 (38%)	3 (100%)	33	280	2 (67%)	1 (33%)	11	640
Paint Formulating	446	1	1 (100%)	1 (100%)	20	56	1 (100%)	1 (100%)	60	120
Plastics Molding and Forming	463	5	2 (40%)	1 (50%)	16	16	2 (100%)	1 (50%)	3	61
Aluminum Forming	467	10	5 (50%)	4 (80%)	1.5	5	5 (100%)	2 (40%)	1.7	5,200
Copper Forming	468	4	2 (50%)	0 (0%)	---	---	0 (0%)	0 (0%)	---	---
Electrical and Electronic Components	469	2	1 (50%)	1 (100%)	23	23	1 (100%)	0 (0%)	10	10
Total CIUs		430	310 (72%)							

¹Units are in nanograms per liter (ng/L) or parts per trillion (ppt)

In addition, Michigan sampled 256 Industrial Users (“IU”) and Significant Industrial Users (“SIU”) representing seven industry types. Michigan noted that where Category 414 Chemical Manufacturing and Category 430 Paper Manufacturing and Packaging were not designated as CIUs, nonetheless “concentrations were either similar or sometimes higher for the IU and SIU facilities than those categorized as CIUs,” concluding that “[t]his may indicate that the regulated processes that require an industrial facility to be listed as a CIU may not significantly affect the potential PFAS use. A facility could be a PFAS source under these two general industrial categories regardless of whether they are listed as an SIU, IU, or CIU.”⁵⁶¹

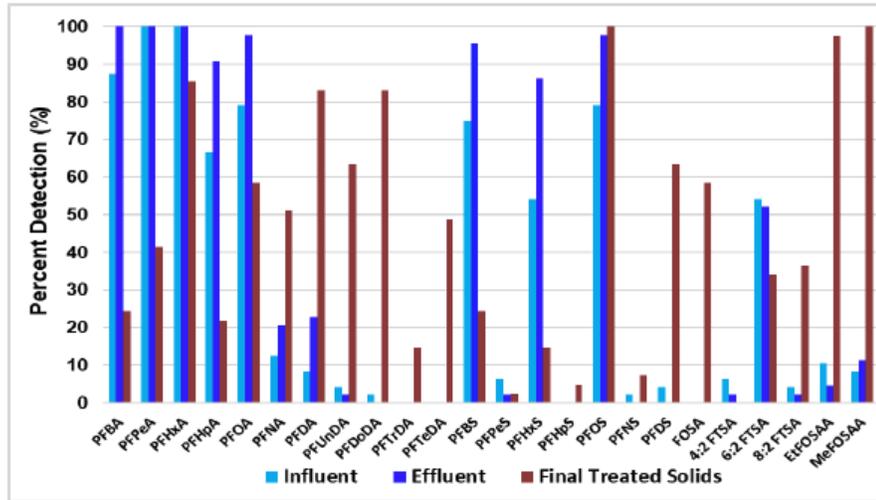
In addition to industrial source investigations, Michigan assessed 42 sewage treatment plants by sampling the influent, effluent, and associated residuals (e.g., sewage sludge).⁵⁶² PFAS was detected in all 134 aqueous samples and 69 out of 71 solids samples, as shown below.⁵⁶³

⁵⁶¹ *Id.* at 26.

⁵⁶² *Id.* at 33.

⁵⁶³ *Id.* at 33, 34.

Figure 23. Percent Detection of PFAS for 42 WWTPs Assessment



A total of 36 out of 42 effluent PFOA concentrations were higher than the influent, and a total of 19 out of 42 effluent PFOS concentrations were higher than the influent, “indicating the possible transformation of precursors and/or, at least in part, the recirculation of various treatment streams (e.g., waste activated sludge, centrate, filtrate) during WWTP operations.”⁵⁶⁴ Michigan reported that “[a]ll of the PFOA concentrations in both the influent and effluent samples were well below the PFOA [water quality standard] WQS of 420 ng/L. However, 15 influent and 14 effluent samples had PFOS concentrations above the PFOS WQS of 12 ng/L.”⁵⁶⁵

Michigan summarized its results as follows:

The significant and rapid drop in PFOS concentrations at WWTPs following source reduction indicates that the source reduction approach is highly effective. Treating PFOS at WWTPs is likely to be difficult and costly because sanitary sewage is a complex waste stream, larger flows would have to be treated, and treatment technologies are not yet sufficiently developed.⁵⁶⁶

* * *

⁵⁶⁴ *Id.* at 35. Other studies have observed the same increase in PFAS increases in effluent. See e.g., *id.* at 46, citing Timothy L. Coggan *et al.*, *An investigation into per- and polyfluoroalkyl substances (PFAS) in nineteen Australian wastewater treatment plants (WWTPs)*, Heliyon (Aug. 23, 2019).

⁵⁶⁵ *Id.* at 35.

⁵⁶⁶ *Id.* at 61.

At select WWTPs, additional aqueous and solid grab samples from various treatment processes were collected to further evaluate the fate of PFAS within the WWTPs The evaluation showed that wastewater treatment processes could not remove PFAS such as PFOA and PFOS, which passes through the WWTP, accumulates in the final treated solids, and is recirculated within the WWTP through various treatment streams.⁵⁶⁷

Put another way, pretreatment was found to be essential to reducing PFAS discharges to surface waters both directly through discharge of effluent and indirectly through the land application of sewage sludge.

Similar but much more limited efforts by the sewer utility Clean Water Services (“CWS”), in Oregon’s Tualatin River basin, also demonstrate how using local limits that are well beyond the requirements associated with an NPDES permit can reduce toxics in discharges, including PFAS, which is a non-priority pollutant. Between 2019 and 2022, CWS collected 229 samples of influent, effluent, biosolids, industrial outfalls and other strategic locations, such as both public and private maintenance holes.⁵⁶⁸ CWS confirmed the results of previous studies showing that influent levels of PFAS are widely variable and sewage treatment has not been shown efficacious for PFAS removal, and noting “poor removal through treatment processes and/or degradation of precursor compounds in PFAS,” the latter of which “has been shown to sometimes *increase* PFAS concentrations between influent and effluent.”⁵⁶⁹ The CWS results also confirmed that sewage treatment plants “with industrial discharges in their sewersheds tend to have higher concentrations of PFAS in their solids than those without[.]”⁵⁷⁰ The utility pointed to Michigan’s 2021 finding that by addressing industrial discharges of PFAS in only

⁵⁶⁷ *Id.* at 63–64.

⁵⁶⁸ See Scott Mansell, *et al.*, *Tracking Down and Addressing Sources of PFAS in Influent Flows Through Strategic Collection System Monitoring and Targeted Outreach* (WEFTEC 2022) (hereinafter “CWS PFAS Pretreatment”) at 19–20.

⁵⁶⁹ *Id.* at 23 (emphasis added).

⁵⁷⁰ *Id.* at 2.

seven of 35 sewage treatment plants studied, the state could “decrease the state-wide average PFOS biosolids concentration by an order of magnitude.”⁵⁷¹ The decrease was effected in Michigan by requiring industrial sources to “implement pretreatment such as granular activated carbon (GAC) which proved successful in reductions in the WRRF influent by 90-99%.”⁵⁷² CWS chose to use an outreach, rather than regulatory, approach to the largest PFAS contributors identified in its monitoring.⁵⁷³ The results—in influent, effluent, and biosolids—were measurable after the industrial source was identified and agreed to install controls, as shown below.⁵⁷⁴

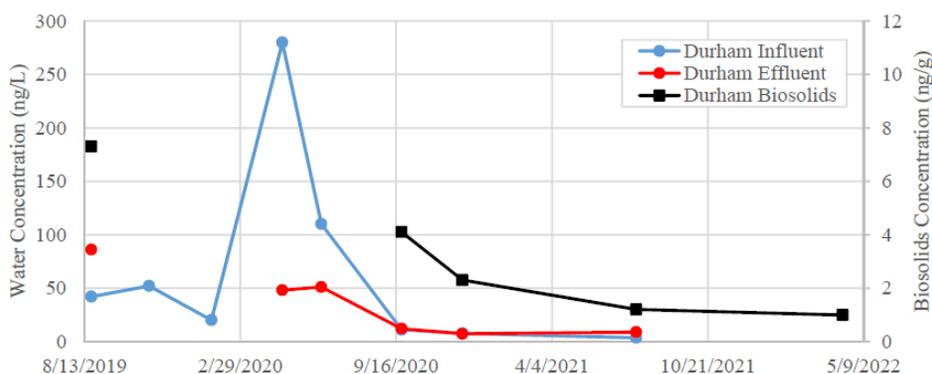


Figure 7: PFOA concentration in influent, effluent, and biosolids at the Durham WRRF

CWA concluded that PFAS reductions from the electronics manufacturer were highly successful as demonstrated in the following presentation:⁵⁷⁵

⁵⁷¹ *Id.*

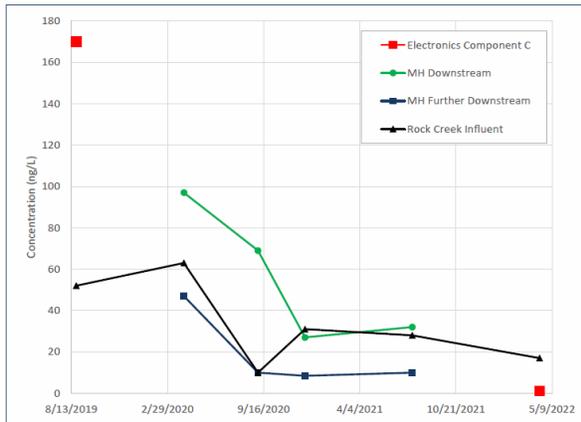
⁵⁷² *Id.*

⁵⁷³ *Id.* at 7.

⁵⁷⁴ *Id.* at 12.

⁵⁷⁵ Scott Mansell *et al.*, *Effectiveness of Targeted Monitoring and Outreach Programs to Reduce PFAS in Influent* (Sept. 7, 2022) (hereinafter “CWS PFAS Outreach”) found at page 40 of Clean Water Services, *Presentations on CEC combined*.

Pretreatment Successes



Downward trend never looked so good.

PFAS Compound	Landfill Leachate	EC-C (2019)	EC-C (2022)
Fraction of Influent Flow	0.3%	19%	17%
PFBS	38%	138%	3%
PFBA	27%	96%	1%
PFPeA	29%	89%	2%
6:2FTS	20%	3%	3%
PFHxA	34%	40%	1%
PFHpA	27%	65%	1%
PFOS	26%	33%	1%
PFOA	30%	27%	0%
PFNA	13%	65%	2%
PFDA	16%	29%	2%

CWS noted that it had a relatively small dataset from industrial outfalls (43 samples from 31 outfalls) but that, even so, it was able to conclude “PFAS concentrations in the landfill leachate are higher than the concentrations from any other industrial outfall by an order or magnitude or more for the most commonly detected PFAS in the influent[.]”⁵⁷⁶ It demonstrated the pattern of PFAS in landfill leachate:⁵⁷⁷

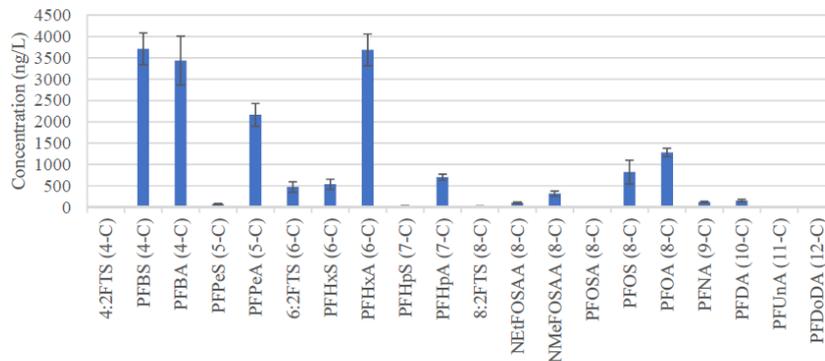


Figure 12: Average PFAS concentration from landfill leachate. Results below detection limits were estimated at half of the MDL. Error bars represent standard error. PFAS types with all non-detects were excluded from the figure for brevity.

⁵⁷⁶ CWS PFAS Pretreatment, *supra* n. 568 at 14.

⁵⁷⁷ *Id.*

And, excluding landfill leachate, patterns for other industrial sectors emerged:⁵⁷⁸

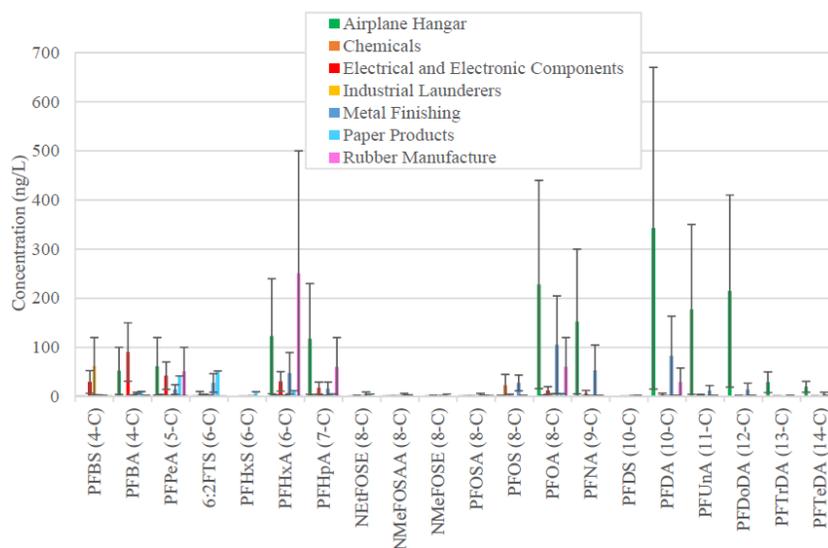


Figure 13: Average PFAS concentration from industrial outfalls by industrial sector excluding the landfill leachate. Results below detection limits were estimated at half of the MDL. Error bars represent standard error. PFAS types with all non-detects were excluded from the figure for brevity. The abrasive products sector excluded from figure because all samples were non-detects.

Based on its monitoring, CWS concluded that:

With the exception of the airplane hangar which historically hosted aeronautical fire-fighting training activities and some metal finishers that had some high concentrations of longer-chain compounds, the dominant forms of PFAS from the industrial sectors as a whole were similar to the dominant forms observed in the influent to the WRRFs. However, each one had its own ‘fingerprint’ with different sectors having the highest concentration for several of the types of PFAS. For example, PFBS was the only PFAS observed at measurable concentrations in the discharges from industrial launderers, while PFPeA and 6:2FTS were the only measurable types from the paper products sector. Rubber extruders, the airplane hangar, metal finishers, and the electronic and electrical component sectors had the highest concentration for most of the PFAS types (aside from the landfill leachate) in their discharges. The chemical industry had only very limited concentrations of PFOS in their discharge, though no specific PFAS manufacturing industries are located in the sewersheds to these WRRFs. While the PFAS concentrations in the discharges from the electronic and electrical component sector are not as high as some of the other industrial sectors, the PFAS types with the highest concentrations are similar to those observed in the influent to the Rock Creek WRRF and the landfill leachate. Some of these are

⁵⁷⁸ *Id.* at 15.

very large industries that account for a large portion of the industrial flow to their receiving WRRF, especially Rock Creek.⁵⁷⁹

CWS specifically concluded that both high source concentrations and high source flows (compared to overall influent) “are important factors when determining where to focus efforts on source control.”⁵⁸⁰ And, it found a high variability between individual industrial sources within the electrical/electronic component sector and within the metal finishing sector, as demonstrated by these graphs:⁵⁸¹

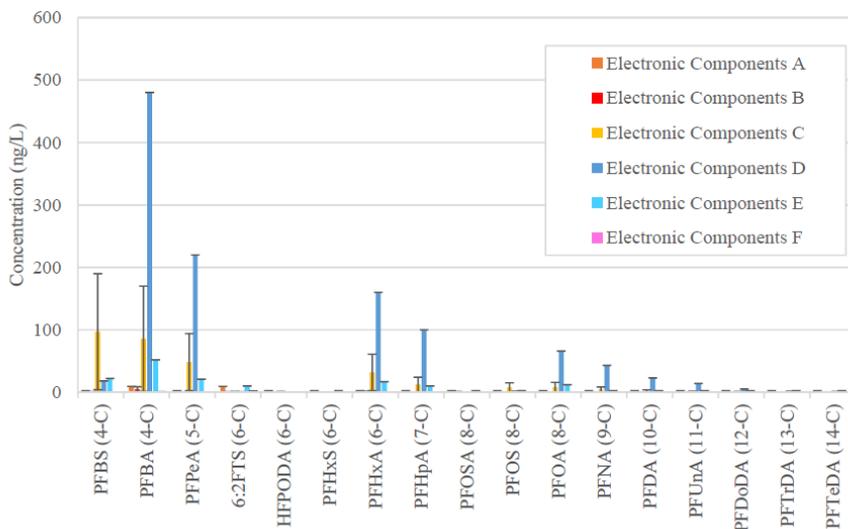


Figure 14: Average PFAS concentration in discharges from different industries within the electrical and electronic component sector. Results below detection limits were estimated at half of the MDL. Error bars represent standard error. PFAS types with all non-detects were excluded from the figure for brevity.

⁵⁷⁹ *Id.* at 15–16 (internal citations omitted).

⁵⁸⁰ *Id.* at 24.

⁵⁸¹ *Id.* at 16, 17.

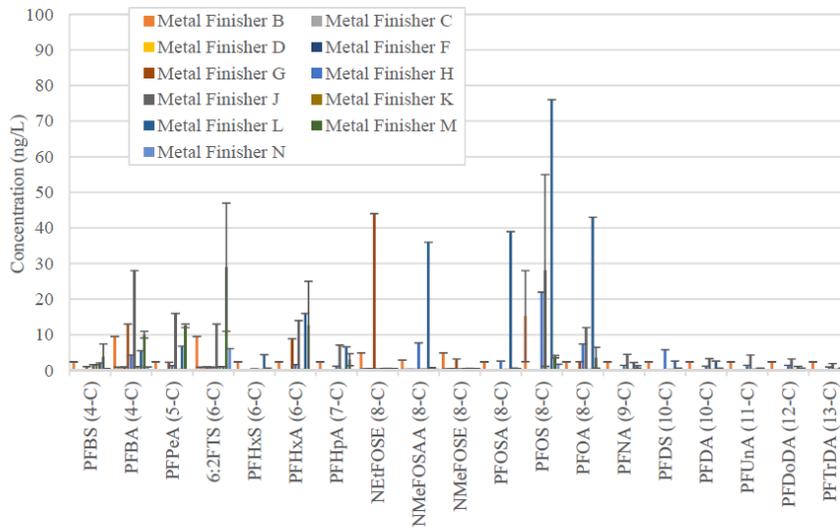


Figure 16: Average PFAS concentration in discharges from different industries within the metal finishing sector excluding Metal Finisher E and I. Results below detection limits were estimated at half of the MDL. Error bars represent standard error. PFAS types with all non-detects were excluded from the figure for brevity.

In response to the CWS outreach effort, a major unnamed electronic industrial source agreed to make “a significant effort to look deeper into their supply chain and collect samples from different parts of their industrial processes to locate the origins of the PFAS in their discharges.”⁵⁸² As this source began using “an advanced treatment system for reuse consisting of biological treatment . . . a membrane bioreactor, reverse osmosis, and brine concentration,” detectable PFAS concentrations decreased by a dramatic 86-98 percent.⁵⁸³

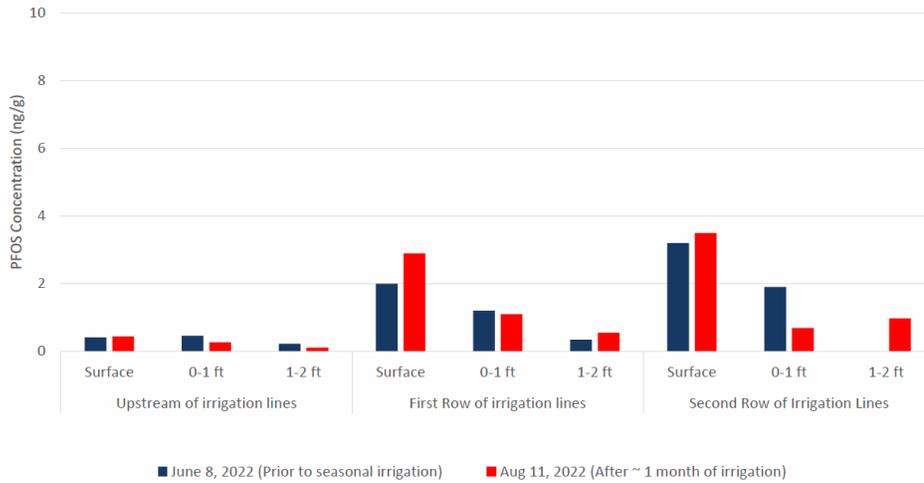
PFAS in sewage treatment plant effluent is also a concern for other media, namely contamination of soils and groundwater from PFAS in sewage sludge, known as “biosolids.” Given the known lack of efficacy of PFAS removal at sewage treatment plants and the evidence of biosolids contamination, pretreatment is the only means by which to keep PFAS out of the environment. While CWS is by no means the only source of information on PFAS-contaminated

⁵⁸² *Id.* at 21.

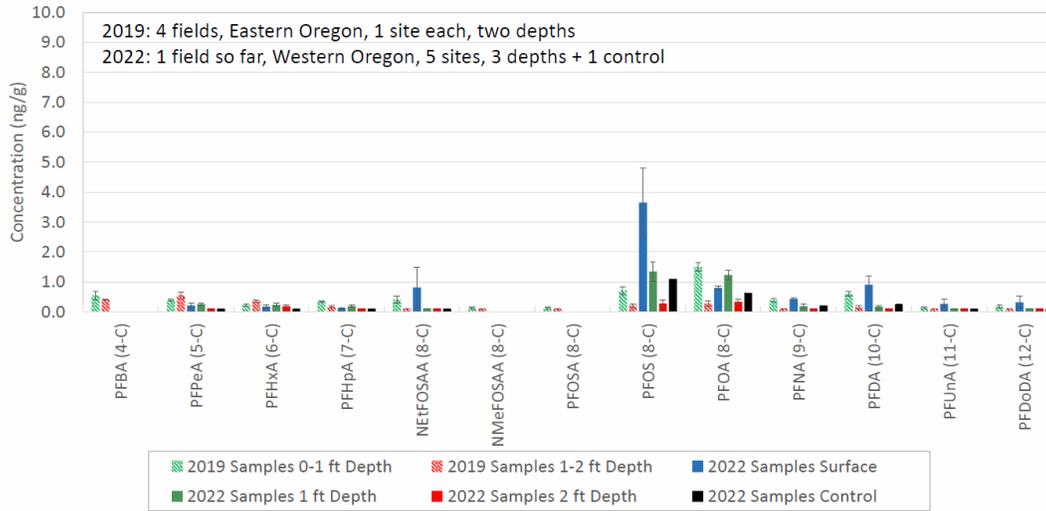
⁵⁸³ *Id.* at 21.

biosolids, it found that its sewage sludge distribution was causing PFAS contamination as demonstrated by the following two graphs.⁵⁸⁴

PFOS in Soils at Reuse Irrigation Site



PFAS in Biosolids-Amended Soils



CWS’s efforts to enhance its pretreatment program to address PFAS (and other toxic contaminants) demonstrates the power of the program to remove toxics from sewage discharges. Its actions were not, however, required by EPA because PFAS is not on the Toxic Pollutants

⁵⁸⁴ CWS PFAS Outreach *supra* n. 575 at 36, 37.

Lists and therefore not a part of the pretreatment program and not addressed in the utility's NPDES permit. Instead, this was a purely voluntary effort by a sewage discharger to use purely voluntary means of controlling indirect sources of PFAS. Michigan's program too, was not triggered by the CWA or EPA policies. While EPA has recently announced its intention to use EPA-issued NPDES permits to "to monitor for PFAS, include requirements to use best management practices like product substitution and good housekeeping practices, and establish practices to address PFAS-containing firefighting foams in storm water"⁵⁸⁵ and has recommended states to use monitoring and pretreatment to address PFAS in state-issued NPDES permits,⁵⁸⁶ EPA's track record of requiring either its own or states NPDES permits to meet federal legal requirements is poor.⁵⁸⁷ It has been particularly poor with regard to the pretreatment program.⁵⁸⁸ Perhaps more to the point, while it may be making progress with

⁵⁸⁵ EPA, Memorandum from Radhika Fox, Assistant Administrator, to EPA Water Division Directors, Re: *Addressing PFAS Discharges in EPA-Issued NPDES Permits and Expectations Where EPA is the Pretreatment Control Authority* (April 28, 2022), available at https://www.epa.gov/system/files/documents/2022-04/npdes_pfas-memo.pdf.

⁵⁸⁶ EPA, Memorandum from Radhika Fox, Assistant Administrator, to EPA Water Division Directors, Re: *Addressing PFAS Discharges in NPDES Permits and Through the Pretreatment Program and Monitoring Programs* (Dec. 5, 2022) (hereinafter "EPA PFAS Memo to States"), available at https://www.epa.gov/system/files/documents/2022-12/NPDES_PFAS_State%20Memo_December_2022.pdf.

⁵⁸⁷ See, e.g., Northwest Environmental Advocates, *Petition for Corrective Action or Withdrawal of Authorization from the State of Washington to Issue National Pollutant Discharge Elimination System Permits* (Feb. 13, 2017) (hereinafter "NWEA Washington NPDES Petition").

⁵⁸⁸ See, e.g., General Accounting Office ("GAO"), *WATER POLLUTION, Improved Monitoring and Enforcement Needed for Toxic Pollutants Entering Sewers* (April 1989) (finding: "Industrial users were in considerable noncompliance with discharge limits under the pretreatment program. . . . sampling inspections by major treatment plants disclosed that about 41 percent of their industrial users exceeded one or more applicable discharge limits during the 12-month period examined. Among the effects of such violations have been (1) the pass-through of untreated toxic pollutants to receiving waters, (2) interference with treatment plant operations or damage to plant facilities, and (3) exposure of treatment plant workers to health and safety problems. While EPA counts on treatment plants' enforcement programs to deter such violations, these plants have generally demonstrated a reluctance to take strong enforcement action when necessary." "Enforcement against noncomplying treatment plants by approval authorities has also been limited.") Follow up by the EPA was identified by the GAO as "business as usual" for ineffective audits and annual reports for sampling frequencies, sampling locations, and local

regard to identifying and controlling sources of PFAS, it waited for the horse to bolt before attempting to close the barn door on PFAS and it is ignoring all the other non-listed toxic chemicals that are causing similar kinds of harm. Placing toxic pollutants on the Toxic Pollutants Lists is a remedy for such continuing failures.

VI. THE WATER QUALITY-BASED APPROACH IN NPDES PERMITTING IS FAILING TO CONTROL TOXICS, UNDERSCORING THE IMPORTANCE OF IMPROVING BOTH THE TECHNOLOGY STANDARDS AND WATER QUALITY CRITERIA THAT ARE DRIVEN BY THE TOXIC POLLUTANTS LISTS

Updating the Toxic Pollutants Lists is of paramount importance to achieving the goals of the CWA because the lists are key drivers of the Act’s technology-based approach through mandatory effluent limits. The lists are thus amplified in importance if the water quality-based approach is not working well. It is not. The precision that EPA seeks to achieve when it establishes Section 304(a) recommended numeric criteria and acts on state water quality criteria is theoretically intended to result in a similarly precise level of protection of designated uses in the environment— aquatic species and people who consume water, fish, and shellfish—when those criteria are used in regulatory programs. Likewise, EPA requirements that a state identify the way in which it will interpret and apply its narrative criteria guarding against toxic effects have a similar goal.⁵⁸⁹

discharge limits. GAO, Water Pollution: Improved Monitoring and Enforcement Needed for Toxic Pollutants Entering Sewers, *Recommendations for Executive Action*, available at <https://www.gao.gov/products/rced-89-101>. Additionally, the GAO reported that “[t]he EPA response to prior recommendations indicates that deficiencies will not always be identified. In such cases, approval authorities will not amend POTW programs, as recommended” and that two recommendations pertaining to EPA evaluation of enforcement problems in pretreatment “will not take place until after all permits are renewed over the next 5 years.”).

⁵⁸⁹ 40 C.F.R. § 131.11(a)(2) (“Where a state adopts narrative criteria for toxic pollutants to protect designated uses, the State must provide information identifying the method by which the State intends to regulate point source discharges of toxic pollutants on water quality limited segments based on such narrative criteria.”).

But because the way in which EPA and states issue NPDES permits results in protection that could best be described as “watered down,” it is essential that EPA have a reliable technology-based approach with which to protect the nation’s waters. This watered-down protection manifests itself first in the relatively few permits that include water quality-based effluent limits (“WQBEL”) for toxics, despite the widespread contamination of the nation’s waters with toxic chemicals. And it manifests in weak WQBELs in the relatively rare instances they are included in an NPDES permit. This lack of WQBELs for toxics is not because the nation’s waters are free of toxics in toxic amounts. Nor is it because NPDES-permitted sources are not contributing to these toxic impairments. The multiple reasons for this outcome, which are discussed below, demonstrate the fundamental weakness of the water quality-based approach—despite its critical importance to protecting the nation’s waters—thereby emphasizing the importance of an up-to-date technology-based approach that, in turn, depends upon up-to-date Toxic Pollutants Lists.

Far from being up-to-date, EPA’s current program to carry out the technology-based approach is based instead on the seriously outdated ELGs and pretreatment standards, *see* section V *supra*, and the equally outdated Toxic Pollutants Lists. However, because Congress deemed the technology-based limit for municipal sewage treatment plants to be secondary treatment,⁵⁹⁰ use of WQBELs to control toxics and nutrients in treated sewage remains essential to protecting water quality because secondary treatment is not intended to, and largely does not, remove such pollutants. *See* sub-section V.B.2, *supra*. EPA’s failure to update the Toxic Pollutants Lists and to publish recommended 304(a) criteria for the newly-listed toxic pollutants seriously hampers

⁵⁹⁰ *See* CWA § 301(b)(1)(B).

the NPDES program's ability to control point sources of toxic contaminants through water quality-based limits that trigger the use of advanced treatment and, additionally, improved pretreatment of indirect industrial discharges. Thus, despite the failures and drawbacks of the water quality-based approach, it remains an essential part of the NPDES permitting program.

EPA and states might claim that the absence of numeric water quality criteria for the large number of new or newly discovered toxic pollutants can be addressed through the use of supplementary narrative criteria. As shown below, however, there are serious deficiencies in implementation of this approach, as well as ongoing problems with the use of numeric criteria in NPDES permits. The first and best solution to this problem—as discussed in the previous section—is to comprehensively update technology-based ELGs and permits, with a renewed focus on eliminating discharges of toxic pollutants entirely. However, to the extent that the water quality-based approach remains necessary as backup, or to control toxics until new ELGs are developed and implemented, updating the Toxic Pollutants Lists will strengthen that program by triggering development of new 304(a) recommended criteria and enforceable water quality standards for states. Thus, updating the Toxic Pollutant Lists is essential to improve both technology-based and water quality-based controls.

The limitations of the water quality-based approach as applied by EPA and the states when issuing NPDES permits start with the lack of information on both the effluent quality of the discharge and the receiving water quality, including limitations on the ways in which CWA Section 303(d) lists are prepared that constitute a formal determination that standards have been violated at or downstream of a discharge. To these problems are added the use of regulatory

mixing zones⁵⁹¹ to provide artificial dilution; a failure to evaluate the downstream effects of toxic pollutants with far-field impacts, including contamination of sediment and bioaccumulation; lack of wasteload allocations from TMDLs to address the cumulative effect of multiple sources of toxics; and a false assumption that whole effluent toxicity (“WET”) testing completely fills the gaps left by both (1) the program’s limited scope of chemical specific numeric criteria, and (2) the need to meet narrative criteria such as “no toxics in toxic amounts.” The discussion below illustrates how these failures work independently and together to result in a largely failed water quality-based approach to point source control of toxics, as the program is currently implemented.

To start, very few NPDES permits require permittees to gather information on the receiving stream into which the facility discharges, both at the point of discharge and downstream, both of which require protection.⁵⁹² As a result, permit writers often have insufficient data and information upon which to determine if a discharge is likely to cause or contribute to violations of water quality standards—prohibited by the CWA and EPA’s implementing regulations⁵⁹³—if the permit does not include a WQBEL. For example, for NPDES permits issued to municipal sewage dischargers to Puget Sound and its tributaries, multiple permit fact sheets include similar descriptions noting the presence of one or more toxic pollutants in facilities’ effluent where no receiving water quality data are available, the result of

⁵⁹¹ The phrase “regulatory mixing zone” is used in the sense EPA uses it, for example, in its Permit Writer’s Manual, *supra* n. 39, at 6-15 (“A regulatory mixing zone generally is expressed as a limited area or volume of water in any type of waterbody where initial dilution of a discharge takes place and within which the water quality standards allow certain water quality criteria to be exceeded.”). The CWA does not provide for mixing zones.

⁵⁹² Downstream protection is required. *See* 40 C.F.R. § 131.10(b); *see also* EPA, *Protection of Downstream Waters in Water Quality Standards: Frequently Asked Questions* (June 2014).

⁵⁹³ CWA § 301(b)(1)(C); 40 C.F.R. §§ 122.4(d), 122.44(d).

which is that permits are issued without QBELs for toxic pollutants.⁵⁹⁴ Here are just two of many such examples:

- Inner Budd Inlet, into which the LOTT facility discharges, is on the 303(d) list for the following toxic pollutants: “2-Methylnaphthalene, Acenaphthene, Acenaphthylene, Anthracene, Benz(a)anthracene, Benzo(a)pyrene, Benzo(b)fluorine, Benzo(b,k)fluoranthenes, Benzo(ghi)perylene, Benzo(k)fluorine, Bis(2-ethylhexyl) phthalate, Butylbenzyl phthalate, Chromium, Chrysene, Copper, Dibenz(a,h)anthracene, Dibenzofuran, Dissolved Oxygen, Fluoranthene, Fluorene, Indeno(1,2,3-cd)pyrene, Mercury, Naphthalene, PAHs, pH, Phenanthrene, Pyrene, Sediment Bioassay, Total PCBs, and Zinc.”⁵⁹⁵ The LOTT discharge was noted to have “toxics . . . in the discharge: ammonia, and heavy metals.” Despite the 303(d) listings that include heavy metals, the permit writer asserted that “[n]o valid ambient background data was available for heavy metals. A determination of reasonable potential using zero for background resulted in no reasonable potential.” In addition, the specific heavy metals in the LOTT discharge were not identified.
- The fact sheet for the Lakota Wastewater Treatment Plant permit states that “[t]he following toxic pollutants are present in the discharge: chlorine, ammonia, heavy metals (antimony, arsenic, cadmium, chromium, copper, lead, mercury, nickel, silver, and zinc), chloroform, toluene, 2,4-dichlorophenol, bis (2-ethylhexyl phthalate), butyl benzyl phthalate, and di-n-octyl phthalate. No valid ambient background data were available for chlorine and heavy metals (antimony, arsenic, cadmium, chromium, copper, lead, mercury, nickel, silver, and zinc), chloroform, toluene, 2,4-dichlorophenol, bis (2-ethylhexyl phthalate), butyl benzyl phthalate, and di-n-octyl phthalate. [Washington Department of] Ecology used zero for background.”⁵⁹⁶ The permittee was not required to gather the missing ambient background data.

In both of these cases, as with innumerable other permits in over 100 sewage treatment plants that discharge to Puget Sound and its tributaries, with few exceptions⁵⁹⁷ there are no QBELs for toxic contaminants nor did the permits include any requirement for monitoring the receiving

⁵⁹⁴ This discussion does not include chlorine and ammonia toxicity.

⁵⁹⁵ Washington Department of Ecology, *Fact Sheet for NPDES Permit WA0037061 Lott Alliance Budd Inlet Wastewater Treatment Plan Summary* (Sept. 1, 2005) (hereinafter “LOTT Fact Sheet”).

⁵⁹⁶ Washington Department of Ecology, *Fact Sheet for NPDES Permit No. WA0022624 Lakehaven Utility District Lakota Wastewater Treatment Plant* (Aug. 27, 2013) at 23.

⁵⁹⁷ There are six exceptions. Five of the six municipal permits with QBELs for toxic pollutants are for discharges to tributaries; LOTT is the only direct discharger to Puget Sound with a QBEL. One of these QBELs is for lead (Yelm) and the remaining five are for copper (LOTT, Buckley, Enumclaw, Orting, Mt. Vernon). Two of these six QBELs are for emergency outfalls only (Mt. Vernon, Yelm).

water despite the permitting agency’s having made an assumption of “zero for background” because there were no data or it chose to ignore the information it had.

This result can be explained in several ways, many of which are interwoven. First is the literal way in which permit writers interpret the CWA Section 303(d) list. Despite clear federal law that the need for effluent limits is not limited to situations where the discharge is of a pollutant to a waterbody that is impaired for that same pollutant,⁵⁹⁸ permit writers assume that a discharge will not cause or contribute to a violation of water quality standards if the receiving water at the precise location of the end-of-pipe is not on the 303(d) list. One example of this is the Washington Department of Ecology’s issuance of an NPDES permit to the Buckley Sewage Treatment Plant. This facility discharges to a waterbody segment of the White River for which the state has no data but which is listed as impaired for temperature both in the immediate upstream and downstream waterbody segments.⁵⁹⁹ Not only does Ecology not assume—in the absence of information—that the segment in the middle of two impaired segments is likely itself impaired for temperature but it has not required the permittee to gather data on the quality of the receiving water segment to confirm its illogical assumption.⁶⁰⁰ Likewise, the LOTT example provided above is similar in that while the receiving waterbody for the discharge is on the 303(d) list, the state lacks ambient water quality data and so dismisses the potential for the discharger to

⁵⁹⁸ See, e.g., *In re: City of Taunton Department of Public Works*, NPDES Appeal No. 15-08, slip op. at 38-39 (EAB May 3, 2016), aff’d. 895 F.3d 120 (1st Cir. 2018); at 38 (“NPDES regulations do not support the City’s contention that a permit authority must include effluent limits only for the pollutants discharged into receiving waters that are identified as impaired on the state’s 303(d) list.”).

⁵⁹⁹ Ecology, *Fact Sheet for City of Buckley Wastewater Treatment Plant National Pollutant Discharge Elimination System (NPDES) Permit WA 0023361* (2021) at 29-30 (no reference to temperature impairments); see also Washington Department of Ecology, *Water Quality Atlas Map* (assessment set to “temperature”).

⁶⁰⁰ Washington Department of Ecology, *National Pollutant Discharge Elimination System Waste Discharge Permit No. WA0023361* (no reference to a requirement to gather data on the receiving water segment).

contribute to the existing violations notwithstanding the fact that the same pollutants for which the waterbody is impaired are also present in the discharge.

Second is the fact that states' 303(d) lists are an inadequate basis upon which to conduct permit evaluations. For example, in Oregon, the state does not list any waters as impaired for toxics based on tissue residue, sediment data, or narrative criteria.⁶⁰¹ This means that the Oregon impaired waters list does not reflect toxic contamination that has bioaccumulated in the food web or accumulated in sediment, the two matrices where contamination by persistent toxics is most likely to be found in the aquatic environment and most likely to be both detectable and quantifiable. In contrast, although Washington lists impaired waters on the basis of tissue residue and is the only state in the country with sediment quality criteria, its 303(d) listings can be and often are exceedingly narrow. For example, despite the endangered Southern Resident killer whales' being known as among the most contaminated marine mammals in the world,⁶⁰² of the entirety of Puget Sound where they live, Washington has identified only a single grid cell of 0.836 square kilometers⁶⁰³ as impaired due to toxic contamination in killer whales, and that

⁶⁰¹ See e.g., Oregon Department of Environmental Quality, Assessment Methodology Updates, *Future Integrated Report cycle updates*, available at <https://www.oregon.gov/deq/wq/Pages/Integrated-Report-Improvements.aspx> (“Short-term updates” to include “Oregon’s methodology for listing segments of waterbodies as “impaired” for toxics does not currently include a method for assessing toxics data in tissue other than mercury.” In addition, “DEQ will evaluate the use of guidance values, or concentrations for toxic compounds that do not have WQ criteria that DEQ may use in application of Oregon’s Toxic Substances Narrative (340-041-0033(2)).”).

⁶⁰² EPA, Salish Sea, Southern Resident Killer Whales, Why Is It Happening?, Current Threats to Killer Whale Recovery, *Pollution and Contaminants*, available at: <https://www.epa.gov/salish-sea/southern-resident-killer-whales#about> (“Individuals have been found to carry some of the highest PCB concentrations reported in animals, with levels in blubber exceeding those known to affect the health of other marine mammals. Other contaminant levels, such as the levels of DDT and PBDEs, are also found in high levels, especially in juvenile killer whales.”).

⁶⁰³ This is roughly 2,460 feet by 3,660 feet. See Ecology, *Water Quality Program Policy, Assessment of Water Quality for the Clean Water Act Section 303(d) and 305(b) Integrated Report* (July 2012) at 5.

impairment is limited to dioxins.⁶⁰⁴ Two additional listings for the same grid cell are the only listings that reflect contamination of Puget Sound harbor seals, for total furans and PCBs, despite the seals' widespread presence in the Sound and years of data collection on their contamination by toxics.⁶⁰⁵

Third, underlying this state and EPA resistance to properly identifying waters impaired by toxics on the state 303(d) lists is the lack of monitoring data. A recent report discussed the lack of state-funded monitoring of waterbodies:

The true extent of the nation's water pollution is unknown because few states monitor all their waterways. Due to limited funding and budget cuts, many state environmental agencies do not have the staff to test all their waters within mandated time periods – usually between six and 10 years, depending on state rules. For example, Missouri and Arkansas assessed only five percent of their river and stream miles in their most recent period. Across the U.S., 73 percent of rivers and stream miles were not assessed during the most recent cycle, and the same is true for 49 percent of lake acres and 24 percent of bay areas.⁶⁰⁶

This finding is not new. In 2000, the GAO testified that:

States' 303(d) lists may not accurately reflect the extent of pollution problems in the nation's waters because many waters have not been assessed. In our survey, only six states responded that they have a majority of the data needed to fully assess all their waters. This response is consistent with the relatively low percentage of waters that states reported assessing for the National Water Quality Inventory. In 1996, for example, states assessed 19 percent of the nation's rivers and streams and 40 percent of the lakes and reservoirs.⁶⁰⁷

GAO noted that

⁶⁰⁴ Washington Department of Ecology, Listing ID No. 36166.

⁶⁰⁵ Washington Department of Ecology, Listing ID Nos. 36167, 36168; *see* Puget Sound Seal Pup Contamination *supra*, n. 368.

⁶⁰⁶ *The Clean Water Act at 50*, *supra* n. 455 at 7.

⁶⁰⁷ GAO, *Water Quality: Identification and Remediation of Polluted Waters Impeded by Data Gaps*, Testimony Before the Subcommittee on Fisheries, Wildlife, and Water, Committee on Environment and Public Works, United States Senate by Statement of Peter F. Guerrero, Director, Environmental Protection Issues, Resources, Community, and Economic Development Division (March 23, 2000) at 4, *available at* <https://www.gao.gov/assets/t-rccd-00-131.pdf>.

studies that have more thoroughly monitored water quality conditions—either through monitoring previously untested waters or conducting different types of monitoring tests—have identified additional pollution problems. For example, a 1993 EPA-funded study of toxins in lakes showed widespread elevated levels of mercury in Maine lakes, despite Maine officials’ assumption that these waters were likely meeting standards because they are in areas with little or no human activity. . . . [A] study conducted by Ohio’s environmental protection agency found that using additional types of monitoring tests identified a significant number of pollution problems in waters that had been shown by other monitoring efforts to be meeting standards.⁶⁰⁸

Finally, GAO reported that 45 states reported that “lack of resources was a key limitation to making more progress on improving water quality.”⁶⁰⁹ In the absence of state and federal resources for data collection, NPDES permittees should be required to gather data to justify permits without effluent limits for toxics but such permit provisions are the rare exception.

Fourth, the corollary to the permit writer’s generally not having adequate information on toxics in the receiving water is that he or she generally does not have sufficient information on effluent quality either. More often than not, the data available on effluent quality are a single toxic pollutant scan—limited to priority pollutants—that does not provide sufficient information about the levels of pollutants in the effluent from which a permit writer can derive a WQBEL. Unless a permit specifically requires the collection of sufficient additional data, the same problem will arise in the development of the subsequent permit, and the permit after that, *ad infinitum*. Permits rarely require the collection of additional toxics data because the permit writer makes a finding that there is no “reasonable potential” for the source to cause or contribute to violations of water quality standards, notwithstanding that lack of information led to the finding in the first instance. Additionally, in general, any requirement that

⁶⁰⁸ *Id.* at 5.

⁶⁰⁹ *Id.* at 10.

a permittee obtain additional information on its effluent quality is limited to those contaminants on the Priority Pollutant List.

Every so often, the lack of effluent quality information triggers a permit response, generally to gather data prior to the next issuance of a permit. For example, despite Washington’s having been evaluating the effect of nitrogen on Puget Sound since at least the early 1990s—a period of three decades—only in December 2021, when the state issued a nitrogen general permit to marine dischargers of treated sewage, did it require the collection of consistent data on effluent nitrogen levels from all such sources.⁶¹⁰ The situation for toxics is even worse because, despite innumerable studies and papers on toxic pollutants in Puget Sound, many of which are cited in this petition, most permits do not require much in the way of effluent monitoring and we are not aware of any that require monitoring of constituents not on the Toxic Pollutants Lists. For example, EPA itself has just now proposed to include a requirement for effluent monitoring of 40 PFAS chemicals in its second draft NPDES permit for the Lummi Tribal Sewer and Water District’s Sandy Point Wastewater Treatment Plant first proposed in 2021.⁶¹¹

Fifth, where there are data on toxics, permit writers almost uniformly use regulatory mixing zones to artificially dilute the effluent prior to evaluating whether it is causing or contributing to violations of water quality standards. This is true, even when a discharge is to an impaired water where, by definition, no assimilative capacity remains. For example, a permit issued in Montana found no dilution was available for copper and zinc but

⁶¹⁰ Washington Department of Ecology, *Puget Sound Nutrient General Permit* (Dec. 1, 2021).

⁶¹¹ EPA, *Authorization to Discharge Under the National Pollutant Discharge Elimination System for Permit No. WA0025658 Lummi Sandy Point Wastewater Treatment Plant* (undated draft, April 2023) (hereinafter “Sandy Point draft permit”), at I.B.1, 9 (table 2: PFAS Chemicals to be Analyzed).

because the city of “Helena cannot currently meet the average monthly limits for copper and zinc, previous limits will be retained and a mixing zone study will be required to consider the potential for a standard mixing zone[.]”⁶¹² In that same permit, the permit authority identified ammonia as an impairment in the receiving water but granted a mixing zone nonetheless.⁶¹³ Permits issued by the Oregon Department of Environmental Quality for temperature, a parameter that in Oregon waters is more often than not impaired in receiving waters, routinely allow additional temperature. Some permit writers even allow for a facility to dilute its effluent through use of a regulatory mixing zone to achieve the mass load limits established in wasteload allocations from Total Maximum Daily Loads (“TMDL”), which utterly defeats the purpose of the TMDL.⁶¹⁴ A 15-year old evaluation of mixing zones for municipal and industrial dischargers in the Puget Sound region found that most permits were granted the maximum sized mixing zones and that mixing zones were used to avoid WQBELs for multiple dischargers of bis(2-ethylhexyl)-phthalate, arsenic, copper, lead, and zinc.⁶¹⁵ (This is despite Washington State case law that “a mixing zone is an exception to the water quality standards that should only be granted in limited instances” and because of their ability to “bioaccumulate and biomagnify, a mixing

⁶¹² Montana Department of Environmental Quality, Montana Pollutant Discharge Elimination System (MPDES) *Fact Sheet, City of Helena, NPDES Permit No. MT0022641*, at 8 (table 8), 14.

⁶¹³ *See id.* at 6, 8.

⁶¹⁴ *See e.g.*, Oregon Department of Environmental Quality, *National Pollutant Discharge Elimination System Permit Renewal Fact Sheet Oregon Department of Fish and Wildlife - Marion Forks Fish Hatchery Final* (Dec. 20, 2022) at 28–29 (wasteload allocation for Minto outfalls given mixing zone). Even in the context of a prohibition on allowing mixing zones for bioaccumulating toxics in the Great Lakes System, EPA noted that “Great Lakes States are not absolutely foreclosed from authorizing a mixing zone for BCCs in the context of a TMDL.” *Great Lakes Mixing Zones*, 65 Fed. Reg. 67638, 67645 (Nov. 13, 2000).

⁶¹⁵ People for Puget Sound, *Toxic Chemicals in Puget Sound: The Impact of Mixing Zones on Permitted Discharges* (June 2, 2008) at 48.

zone for PCBs should rarely, if ever, be granted.”⁶¹⁶) Agencies’ use of regulatory mixing zones to increase the allowable discharge of toxic pollutants renders the effort to precisely establish numeric water quality criteria a farce.

While it should be axiomatic that a discharge of a pollutant into a waterbody impaired by that same pollutant is “causing and contributing” to a violation of water quality standards, EPA has declined to take a position on this: “EPA does not have a general policy on the availability of mixing zones in impaired waters at this time and generally defers to States on this issue.”⁶¹⁷ It also, confoundingly, stated: “When background levels of the pollutant for which a mixing zone is sought already exceed the applicable criterion in the receiving water, there may be no available dilution, despite the availability of a mixing zone.”⁶¹⁸

In contrast, EPA prohibits, with some exceptions, mixing zones for some bioaccumulative chemicals of concern (“BCC”) in the Great Lakes System.⁶¹⁹ As EPA explained in its final action to reinstate the prohibition, its effect would be “that NPDES permit limitations for BCCs discharged to the Great Lakes System must be set no higher than water quality criteria.”⁶²⁰ EPA noted that by 2000, the States of Illinois, Indiana, Minnesota,

⁶¹⁶ Washington State Pollution Control Hearings Board, *Puget Soundkeeper Alliance v. Ecology and Seattle Iron & Metals Corp*, PCHB No. 13-137c Findings of Fact, Conclusions of Law, and Order (July 23, 2015).

⁶¹⁷ Great Lakes Mixing Zones, 65 Fed. Reg. 67638-67651, 67645 (Nov. 13, 2000). *See also* Permit Writers’ Manual *supra* n. 39 at 6-21 (allowing states to adopt whatever policies they choose: “Recall as well that for some pollutants (e.g., pathogens in waters designated for primary contact recreation, bioaccumulative pollutants), the water quality standards or implementing procedures might not authorize any dilution allowance, even where the effluent and receiving water mix rapidly and completely.”).

⁶¹⁸ *Id.*, noting that “Exceptions might be where there are no currently available data for calculating background values as provided in [procedure] 3.B.9 or where anticipated loading reductions would lower background levels (see 3.C.3.b.iii) and “free up” assimilative capacity for use in calculating WLAs.”

⁶¹⁹ *Id.*; 40 C.F.R. § Part 132, Appendix F, Procedure 3.C (Mixing Zones for Bioaccumulative Chemicals of Concern (BCCs)).

⁶²⁰ *Id.* at 67639. For the Great Lakes System, EPA defined a BCC as “in essence, as any chemical that (1) accumulates in aquatic organisms by a human health bioaccumulation factor (BAF) greater than 1000

Michigan, and Wisconsin had adopted such provisions.⁶²¹ The agency cited its rationale:

A large number of scientists, policy makers, and other stakeholders in the Great Lakes and Canada agree on the need to virtually eliminate BCCs from the Great Lakes Basin and to reduce the size of BCC mixing zones to the maximum extent possible. This is because BCCs, due to their persistent and bioaccumulative nature, are incompatible with mixing zones. By definition, BCCs are chemicals that do not degrade over time. These chemicals accumulate in organisms living in the water and become more concentrated as they move up the food chain—from biota to fish and wildlife to humans. Because the effects of these chemicals are not mitigated by dilution, using a mixing zone to “dilute” BCC discharges is not appropriate. Commenters pointed out that dilution and dispersion are inadequate substitutes for removing and treating the BCCs before they are discharged to the Great Lakes’ waters. EPA agrees with these commenters because it is the mass of BCCs that poses a problem, not just the concentration. Because dioxins, mercury, polychlorinated biphenyls (PCBs) and other BCCs degrade over long periods of time or do not degrade at all, their buildup in pockets of sediments creates “hot spots” in the environment in which bioaccumulation of toxics in fish and other aquatic organisms can occur at levels that significantly exceed safe levels for consumption by wildlife and humans.

* * *

Because the food web that accumulates BCCs can be concentrated in tributaries, bays, and other areas where natural sinks exist—and where fish species are more diverse and productive—the elimination of mixing zones will reduce the probability of adverse effects on these organisms and those that consume them. Fewer pollutants entering the waters will reduce the detrimental effects already discovered in various fish species and wildlife.⁶²²

EPA concluded that “[p]rohibiting mixing zones for BCCs in the Great Lakes System can reduce the natural sink masses below point source discharges by a factor of 10 to 100 in some circumstances.”⁶²³ And it noted:

Had the Guidance’s framework been in place 30 years ago when the effects of PCBs from point source discharges began to emerge, States could have moved quickly to control these pollutants, avoiding millions of dollars in cleanup costs, human health impacts, and other environmental damage. . . . With low concentrations of new chemicals being introduced into the environment every

(after considering various specified factors), and (2) has the potential upon entering surface waters to cause adverse effects, either by itself or in the form of its toxic transformation product, as a result of that accumulation. See 40 CFR 132.2.” *Id.*

⁶²¹ *Id.* at 67640.

⁶²² *Id.* at 67640–67641.

⁶²³ *Id.* at 67642

year, it would be prudent to try to avoid future cleanup costs now.⁶²⁴

Sixth, in using regulatory mixing zones and making reasonable potential determinations, permit writers falsely assume that toxic contaminants do not have a downstream effect, for example to contaminate sediment in depositional areas or to contaminate the food web through bioaccumulation. This, of course, makes no logical sense because the concentration of a toxic pollutant at the point of discharge is irrelevant to its contribution of loading to a waterbody. But it is common. For example, the fact sheets for most Washington Department of Ecology permits include the following boilerplate assertion:

Pollutants in an effluent may affect the aquatic environment near the point of discharge (near field) or at a considerable distance from the point of discharge (far field). Toxic pollutants, for example, are near-field pollutants--their adverse effects diminish rapidly with mixing in the receiving water.⁶²⁵

This flawed assertion that toxic pollutants do not have far-field effects becomes the basis for an evaluation of toxics that is limited to the end-of-pipe impacts. If, for example, the waterbody segment into which a facility discharges is not on the 303(d) list, even if the water is listed as impaired downstream for a toxic pollutant, the permit writer will assume that the permitted discharge is not causing or contributing to violations of water quality standards.

Contrary to Washington's assertion, however, not all toxic pollutants are near-field pollutants whose "adverse effects diminish rapidly." The very definition of a persistent toxic pollutant is that its effects do not diminish rapidly.⁶²⁶ As EPA notes in its 1985 guidance on how

⁶²⁴ *Id.* (emphasis added).

⁶²⁵ See, e.g., LOTT Fact Sheet, *supra* n. 595 at 13.

⁶²⁶ See, e.g., EPA, Pacific Southwest, *Persistent, Bioaccumulative and Toxic Chemicals (PBTs)*, available at <https://archive.epa.gov/region9/waste/archive/web/html/pbts.html> ("PBT pollutants pose risks because they are toxic even in small quantities, persist in ecosystems, bioaccumulate in food chains, and can travel great distances (via equipment or products, food, or the environment). The threats are widely recognized, and the environmental legacy of PBTs is clear as well[.]").

to issue NPDES permits to sources of toxic pollutants, “[f]ate modeling of the toxicant to estimate its behavior after discharge can be an important step in establishing water quality-based permit limits.”⁶²⁷ Specifically, after discussing the use of regulatory mixing zones, EPA notes that “[o]ne disadvantage of the chemical-specific approach is that the bioavailability of the toxicant after discharge is not measured. However, persistence can be modeled and the persistence of specific toxicants can be accounted for in making impact predictions and setting controls.”⁶²⁸ EPA’s guidance provides minimal advice on how a permitted discharge of heavy metals or organic chemicals can be monitored for fate and transport modeling.⁶²⁹ But there is no evidence that most, if indeed any, permit writers model the persistence, fate, and transport of pollutants after they have completed a mixing zone evaluation based on pollutant concentrations and concluded that a discharge of a toxic pollutant has no reasonable potential to cause or contribute to violations of water quality standards. While EPA states that “any chemical that has high potential for persistence and bioaccumulation *should* be a matter of concern until it can be demonstrated that there are no adverse environmental and human health effects resulting from discharge,”⁶³⁰ it has not elaborated on how permit writers *must* evaluate this “matter of concern” and include appropriate effluent limits in permits to ensure there is no adverse impact.

The gap between “should” and “must” is huge. This is demonstrated by a study, in which the GAO focused on permitting of nonindustrial toxics discharged in treated sewage.⁶³¹ Citing an Office of Technology Assessment estimate that “household wastewater accounts for 15

⁶²⁷ EPA, *Technical Support Document for Water Quality-based Toxics Control* (Sept. 1985) (hereinafter “EPA Toxics TSD”) at 1.

⁶²⁸ *Id.* at 5.

⁶²⁹ *See id.* at 21.

⁶³⁰ *Id.* at 22

⁶³¹ GAO, *Nonindustrial Wastewater Pollution Can Be Better Managed* (Dec. 1991), available at <https://www.gao.gov/assets/rced-92-40.pdf>.

percent of regulated toxic pollutants entering treatment plants,” the estimate did not take into account pollutants not on the Toxic Pollutants Lists.⁶³² Focused on regulated metals, it cited

a Seattle, Washington, treatment plant estimates that up to 26 percent of the arsenic entering the plant originates from household laundry detergents, dishwashing detergents, and bleach. A Palo Alto, California, treatment plant determined that up to 81 percent of the silver entering the plant comes from nonindustrial sources. Other studies point to car washes, dry cleaners, and a wide variety of household products as contributors of lead, mercury, phosphorus, oil, and benzene to treatment plants.⁶³³

GAO pointed to the voluntary efforts by the Palo Alto plant to impose “limits on silver discharges from commercial photo processors, hospitals, and dental offices” and cited “many states in the Great Lakes and Chesapeake Bay areas [that] have banned the use of phosphates.”⁶³⁴ But it pointed to EPA’s having constrained itself “primarily to providing information and guidance to states, localities, and the public,” and indicted EPA for its failure to take steps to address the scope and seriousness of this problem that are “warranted now.”⁶³⁵ EPA’s response was a milquetoast “should it determine additional nonindustrial source controls are necessary, it will develop appropriate and cost-effective measures.”⁶³⁶ It has not so determined, and even on PFAS, the subject of grave EPA concern, its new guidance on NPDES permitting does not include the serious actions of “revising discharge permits, regulatory initiatives, or product bans” that GAO urged upon the agency⁶³⁷ but, rather, is limited to EPA’s “recommendations”⁶³⁸ that states consider various actions. Its assertion that NPDES permits “must include water quality-

⁶³² *Id.* at 3.

⁶³³ *Id.* at 3–4.

⁶³⁴ *Id.* at 4.

⁶³⁵ *Id.*

⁶³⁶ GAO, *Water Pollution: Nonindustrial Wastewater Pollution Can Be Better Managed, Recommendations for Executive Action*, available at <https://www.gao.gov/products/rced-92-40>.

⁶³⁷ *Id.*

⁶³⁸ EPA PFAS Memo to States, *supra* n. 586.

based effluent limits (WQBELs) as derived from state water quality standards, in addition to TBELs developed on a BPJ basis, if necessary to achieve water quality standards, including state narrative criteria for water quality,”⁶³⁹ is certainly a true statement of the rule of law but it is a rule that by permit writers is far “more honored in the breach than the observance.”⁶⁴⁰

Moreover, in its 1985 guidance, EPA specifically cautioned against the use of regulatory mixing zones for bioaccumulative toxics by advising that “a smaller mixing zone or no zone at all can be required in site-specific cases. Bioaccumulative pollutants in particular may exert unexpected impacts if a mixing zone is allowed.”⁶⁴¹ As EPA explained further, a water quality-based limit “for nutrients or bioaccumulative pollutants could be expressed as the required average effluent quality because the total loading of these pollutants is of concern.”⁶⁴² The use of a mixing zone analysis, however, is entirely focused on concentrations of pollutants, not pollutant loading. In 1998, EPA added in non-regulatory commentary that:

The impacts of bioaccumulative compounds may extend beyond the boundaries of a given mixing zone with resulting impairment of a water body’s designated uses, particularly where stationary species (e.g. shellfish) are present, where uncertainties exist regarding the assimilative capacity of a water body or where bioaccumulation in the food chain is known to be a problem. Sediment contamination has also become a major concern in both flowing and non-flowing water bodies. Concerns about sediment contamination require additional attention since typical mixing zone evaluations focus only on water column toxicity. The effects of persistent and bioaccumulative pollutants may not be detected for some distance from the point of discharge, well outside the mixing zone, or possibly not in the water column at all.⁶⁴³

⁶³⁹ *Id.* at 3–4.

⁶⁴⁰ William Shakespeare, *Hamlet* (1602), Act 1, Scene 4.

⁶⁴¹ EPA Toxics TSD, *supra* n. 627 at 32 (emphasis added).

⁶⁴² *Id.* at 50.

⁶⁴³ Standards ANPRM, *supra* n. 131 at 36791.

Despite these concerns about toxics that simply mirror factual reality, nothing in EPA’s permit writer’s manual describes, let alone requires, permitting consistent with them.

In its 1985 Toxics TSD guidance, EPA further states that ambient criteria for metals, organometallic compounds, and organic compounds “will adequately control those compounds because the criteria are based on bioaccumulation where appropriate.”⁶⁴⁴ But the state adoption of criteria that consider bioaccumulation effects and EPA’s subsequent approval of them does not factor in the additional dilution of regulatory mixing zones and permit writers’ failure to consider far-field effects, which render the criteria nearly irrelevant. EPA itself did not dismiss the importance of bioaccumulation of toxics, finding that it “is a potentially significant route of exposure to human populations” and “may also cause severe impacts on aquatic organisms and other wildlife.”⁶⁴⁵ Instead, rather unhelpfully for a technical guidance, EPA proposed that permit writers should pay “[p]articular attention . . . to bioaccumulative pollutants.”⁶⁴⁶

EPA has not updated its now 38-year old technical guidance on how to issue NPDES permits for toxics. Instead, in its newer permitting guidance, issued in 2010, EPA continues to cite to the Toxics TSD.⁶⁴⁷ In this newer permitting guidance, EPA notes the ability for permit writers to establish “internal outfalls” pursuant to 40 C.F.R. § 122.45(h) that “might be necessary to ensure proper treatment of persistent, bioaccumulative, and toxic pollutants that are discharged in concentrations below analytic detection levels at the final effluent outfall or other pollutants that may be diluted by flows (e.g., cooling water) not containing the pollutant.”⁶⁴⁸ There is,

⁶⁴⁴ EPA Toxics TSD, *supra* n. 627 at 22.

⁶⁴⁵ *Id.* at 26.

⁶⁴⁶ *Id.* at 28.

⁶⁴⁷ *See, e.g.*, Permit Writers’ Manual *supra* n. 39 at 6-17.

⁶⁴⁸ *Id.* at 5-39.

however, no meaningful distinction between not allowing for dilution within a facility and not allowing a regulatory mixing zone at the point of discharge for the same purpose, other than to assure detection and quantification. Bizarrely, despite the passage of 25 years following the publication of the EPA Toxics TSD, EPA’s 2010 permitting guidance makes little reference to permitting bioaccumulative toxic pollutants other than the possible need for permittees to conduct studies and the need to “assess many complex human health effects, including carcinogenicity, teratogenicity, mutagenicity, bioaccumulation, and synergistic propensities” if a state has sought a variance from water quality standards.⁶⁴⁹ Apparently EPA does not consider these issues relevant if a state has not sought a variance. EPA also notes that “[i]mpacts to wildlife should also be considered in evaluating the discharge of toxicants,”⁶⁵⁰ while providing absolutely no information on how a permit writer is supposed to accomplish this task.⁶⁵¹

Returning to Washington, which is the only state with EPA-approved sediment quality criteria, the result of the state’s narrow view that toxics are only near-field pollutants, cited above, results in permitting decisions such as that for Bremerton, the effluent for which contains mercury.⁶⁵² The outfall for Bremerton is located in a grid cell of Puget Sound for which there are no data on sediment quality.⁶⁵³ This cell is literally surrounded, however, by 303(d) listings for mercury in sediment⁶⁵⁴ and to the north by a cell that is not listed as impaired for sediment

⁶⁴⁹ *Id.* at 5-41.

⁶⁵⁰ EPA Toxics TSD, *supra* n. 627 at 30.

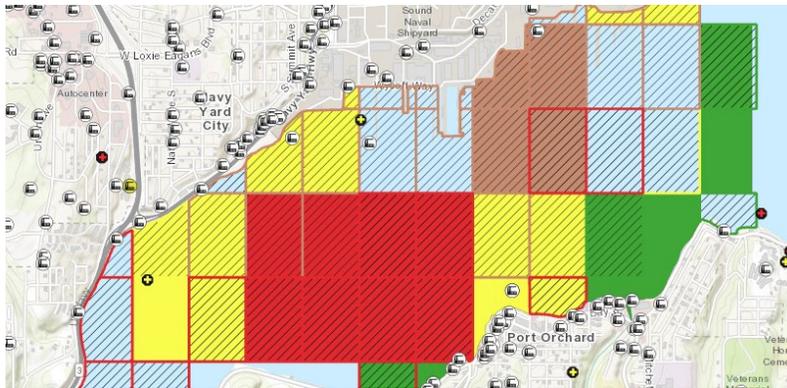
⁶⁵¹ In addition to EPA’s Toxics TSD’s not including any information on how to issue NPDES permits to protect wildlife, neither does the more recent EPA NPDES Permit Writers’ Manual (Sept. 2010) (no reference to how to ensure permits protect wildlife).

⁶⁵² Washington Department of Ecology, *Fact Sheet for NPDES Permit WA0029289 Bremerton Wastewater Treatment Plant* (June 21, 2013) (hereinafter “Bremerton Fact Sheet”) at 36.

⁶⁵³ Washington Department of Ecology, *Bremerton and 303(d) listings for sediment mercury* (Nov. 20, 2022).

⁶⁵⁴ *Id.*

quality but for which data show sediment bioassays exceeded the criterion.⁶⁵⁵ Similarly, the outfall is located in a cell that is not 303(d) listed for mercury in tissue because although the tissue samples taken exceeded the criteria, the data were deemed insufficient for a 303(d) listing.⁶⁵⁶ Multiple cells to the east of the outfall, however, are 303(d) listed for mercury in tissue.⁶⁵⁷



In issuing this facility its 2013 permit, the state found that it has the potential to cause a violation of sediment quality standards⁶⁵⁸ but included no effluent limitations to address this potential, instead only including a permit condition that Bremerton demonstrate that either “[t]he point of discharge is not an area of deposition” or that “[t]oxics do not accumulate in the sediments even through the point of discharge is a depositional area.”⁶⁵⁹ It is unclear why, given that the data for the 303(d) assessment findings showing that toxics are accumulating in sediments, why Washington had not previously required Bremerton to assess its contribution to the 303(d) listings. It is unclear why Washington persists in stating that toxics are near-field

⁶⁵⁵ Washington Department of Ecology, *Assessment Listing ID No. 508185*.

⁶⁵⁶ Washington Department of Ecology, *Listing ID No. 88702*.

⁶⁵⁷ Washington Department of Ecology, *Bremerton and assessment for sediment and tissue mercury* (Nov. 30, 2022).

⁶⁵⁸ Bremerton Fact Sheet, *supra* n. 652 at 36.

⁶⁵⁹ *Id.* at 36.

pollutants when the presence of extensive areas of sediment and tissue contamination that are not in the immediate vicinity of outfalls are a demonstration that toxics accumulate and move, a concept widely known as “fate and transport.” It is unclear why this 2013 permit did not require data collection and include a reopener provision to establish WQBELs for toxic pollutants. And, finally, it is unclear why the only provision pertaining to sediment quality is limited to “the point of discharge.”

In *Puget Soundkeeper Alliance v. Ecology and Seattle Iron & Metals Corp.*, the Washington State Pollution Control Hearings Board (“PCHB”) discussed this very type of permitting failure and found it contrary to law. At the time of Seattle Iron & Metals 2013 permit, “water column data on background levels for PCBs in the Duwamish River were lacking . . . [and] [a]s a result, Ecology was unable to determine whether the [Lower Duwamish River] had available assimilative capacity for additional PCBs. At the time the 2013 Permit was being drafted, the stretch of river in question was not listed on the state’s 303(d) list for PCBs.”⁶⁶⁰ However, subsequent data “shows that PCB levels in the Green River above the Duwamish River exceed applicable human health criteria. [State employee] Mr. Shervey acknowledged that this more recent data suggests the [Lower Duwamish River] lacks additional assimilative capacity for PCBs, and that it would not probably be appropriate to grant a mixing zone in the future.”⁶⁶¹ The use of the mixing zone in the 2013 permit “raised the calculated limits for copper, lead, mercury, silver, zinc, and PCBs by a factor of 5.3 in the acute zone and 30.2 in the chronic zone.”⁶⁶² The PCHB held: “Given their persistence and ability to bioaccumulate and

⁶⁶⁰ *Puget Soundkeeper Alliance v. Ecology and Seattle Iron & Metals Corp.*, PCHB No. 13-137, Findings of Fact, Conclusions of Law, Order (July 23, 2015) at ¶ 33.

⁶⁶¹ *Id.* at ¶ 19.

⁶⁶² *Id.*

biomagnify, a mixing zone for PCBs should rarely, if ever, be granted,” noting that “[t]he granting of a mixing zone to [the source] for PCBs is counterproductive” to the effort to clean up contaminated sediments in the river.

Despite this legal holding, Washington permit writers have not stopped addressing impacts of bioaccumulative and persistent toxic pollutants by conducting a mixing zone evaluation to determine if a source has a reasonable potential to violate water quality standards.⁶⁶³ Nor has it stopped the state’s permit writers from focusing solely on the assessed water quality of the 303(d) segment into which a source discharges. In fact, recent permits show that the state agency simply ignores public comments about sources’ contributions to downstream toxic impairments.⁶⁶⁴ EPA has not weighed in to encourage the state to stop using mixing zones for such toxic pollutants.

Seventh, permit writers often assume that whole-effluent toxicity (“WET”) testing using bioassays fills all gaps left by numeric criteria to ensure compliance with narrative criteria that prohibit toxic effects to designated uses. While WET testing is useful because it

⁶⁶³ See, e.g., Washington Department of Ecology, *Fact Sheet for NPDES Permit WA0029181 West Point Wastewater Treatment Plant and Combined Sewer Overflows* (April 5, 2023) at 73 (receiving water not deemed impaired for PCBs and mercury because while “regions of Elliott Bay and Central and South Puget Sound as impaired (Category 5) for . . . PCBs and mercury in fish tissue. . . the list also shows the water segments in the immediate vicinities of the West Point, Alki, and Carkeek outfalls . . . does not include listings in any categories for PCB and mercury.”), 82-83 (“Chronic dilution factors are typically assessed using the maximum 4-day average flow, except for pesticides and PCBs that use 24-hour flow rates.”), 131-132 (reasonable potential calculations using mixing zones for toxics).

⁶⁶⁴ See, e.g., Letter from Nina Bell, NWEA, to Carey Cholski, Washington Department of Ecology, Re: *Draft NPDES Permit for Sumner Wastewater Treatment Plant, Permit No. WA0023353* (Dec. 28, 2020) at 23–26 (discussing the need to issue a permit that addresses downstream impairments for conventional and toxic pollutants due to 303(d) listings and scientists’ conclusion that the Puyallup river is highly contaminated with metals, PPCPs, PCBs, PBDEs and likely causing adverse effects to Chinook salmon). Ecology’s response to these comments was two-fold: (1) to ignore all comments on pollutants other than nutrients; and (2) to assert that “Ecology has assessed the reasonable potential for the discharge to violate water quality standards in the near field[.]” Washington Department of Ecology, *Fact Sheet for City of Sumner Wastewater Treatment Plan National Pollution Discharge Elimination System (NPDES) Permit WA0023353* (Feb. 4, 2021) at 68–69.

evaluates to a limited extent the “aggregate toxicity of all constituents in a complex effluent,” it leaves additional gaps and is supposed to supplement but not replace adequate pollutant-specific WQBELs. As EPA itself pointed out in its 1985 Toxics TSD guidance:

The principal disadvantages of the whole-effluent approach are:

- Effluent toxicity treatability data are lacking, and treatment engineers and permit writers are unfamiliar with the procedures.
- Where there are chemical/physical conditions (pH changes, photolysis, etc.) present that act on toxicants in such a way as to “release” toxicity downstream, such toxicity may not be measured in the effluent.
- Properties of specific chemicals in complex effluents (such as bioaccumulation, carcinogenicity) are not assessed.⁶⁶⁵

Yet, incorporating the potential for a toxic pollutant to bioaccumulate is a key issue in establishing pollution controls for persistent toxic pollutants, rendering WET testing alone inadequate to ensure protection of designated uses and compliance with narrative criteria guarding against toxic effects. Even the existing WET testing, endorsed by EPA, is not based on a consistent quantitative measure. In the 1998 Advanced Notice of Proposed Rulemaking (“ANPRM”), EPA considered requiring states to “specify implementation of toxicity criteria and test methods as a required means to implement the narrative water quality criteria” because “[s]uch a quantification serves, in conjunction with numeric criteria for individual pollutants and biological criteria, to establish an integrated and fully protective basis for assessment and control of pollutants.”⁶⁶⁶ It did not.

WET tests were endorsed in a limited fashion by the National Marine Fisheries Service and U.S. Fish & Wildlife Service (“USFWS”) in their biological opinions for EPA’s proposed approval of Idaho numeric toxic criteria. NMFS found that “[a]ddressing mixture toxicity

⁶⁶⁵ EPA Toxics TSD, *supra* n. 627 at 2 (emphasis added).

⁶⁶⁶ Standards ANPRM, *supra* n. 131 at 36768.

through the use of WET testing and instream bioassessment are practical and reasonable approaches for addressing the expected increased toxicity of a given concentration of a chemical in the presence of other chemicals.”⁶⁶⁷ But it also noted that “the assessment triggers on WET tests may not be sensitive enough to protect listed salmonids with reasonable certainty, and biomonitoring has not always been well defined.”⁶⁶⁸

In reality, few permit writers engage in any evaluation of compliance with narrative criteria other than WET testing. WET testing is not sufficient to address the numerous elements required by some narrative criteria. For example, the Oregon narrative toxics criterion requires: “Toxic substances may not be introduced above natural background levels in waters of the state in amounts, concentrations, or combinations that may be harmful, may chemically change to harmful forms in the environment, or may accumulate in sediments or bioaccumulate in aquatic life or wildlife to levels that adversely affect public health, safety, or welfare or aquatic life, wildlife or other designated beneficial uses.”⁶⁶⁹ WET testing does not address protection of sediment, bioaccumulation in the food web, and harm to wildlife.

Eighth, one key method of assuring that multiple toxic pollution sources are all adequately regulated is the development of Total Maximum Daily Loads (“TMDLs”). But in many states, very few TMDLs have been completed for toxic contaminants and of those completed, very few include wasteload allocations to NPDES-permitted sources.⁶⁷⁰ For

⁶⁶⁷ NMFS Idaho BiOp, *supra* n. 260 at 92. See also USFWS, *Biological Opinion for the Idaho Water Quality Standards for Numeric Water Quality Criteria for Toxic Pollutants* (June 25, 2015) at 273 (example of reasonable and prudent alternative to address jeopardy opinion includes WET testing for copper).

⁶⁶⁸ *Id.*

⁶⁶⁹ OAR 340-041-0033(1).

⁶⁷⁰ EPA’s ATTAINS database does not allow the public to search for all TMDLs completed except by one parameter at a time, one state at a time, and state submissions are not sufficiently consistent to allow for comparisons (e.g., Texas documents are non-sortable pdfs whereas New Jersey’s 303(d) list is a

example, in Washington State, TMDLs for toxics may appear to have been a considerable body of work, albeit one that ceased after 2007.⁶⁷¹ A closer look shows that these TMDLs have not, however, had a meaningful effect on Washington's permitting program as there are only a total of 20 NPDES sources covered by wasteload allocations for toxics in over 30 years of TMDL development, six of which were driven by the requirements of the one-time provisions of CWA Section 304(i).⁶⁷² (Petitioners do not know how many of these wasteload allocations have been used in subsequently issued NPDES permits as WQBELs.) In contrast, 1,213 segment-parameters for toxics are on the current EPA-approved list of impaired waters in Washington.⁶⁷³

Similarly, in Oregon, few TMDLs have been completed for toxics and even fewer include wasteload allocations for NPDES-permitted sources.⁶⁷⁴ Only 13 NPDES sources are named in the TMDLs developed over a similar period of three decades and of those, only three

spreadsheet). See EPA, *How's My Waterway?*, available at <https://mywaterway.epa.gov/>. For this reason, examples are given here rather than national data.

⁶⁷¹ TMDLs for toxics in Washington waters include: Grays Harbor Dioxin TMDL (1992) (2 NPDES); Inner Bellingham Bay Contaminated Sediments TMDL (2001) (1 NPDES); Lake Chelan Watershed DDT and PCB TMDL (2006) (0 NPDES); Lower Okanogan River Watershed Toxics TMDL (2004) (4 NPDES); Lower Similkameen River Arsenic TMDL (2004) (1 NPDES); Lower Snohomish River Dioxin TMDL (1992) (1 NPDES); Lower Yakima DDT TMDL (1998) (0 NPDES); Mill Creek Chlorine TMDL (1997) (1 NPDES); Mission Creek Watersheds Toxics TMDL (2007) (0 NPDES); Palouse River Watershed Toxics TMDL (2007) (3 NPDES); Spokane River Metals TMDL (1999) (0 NPDES); Stillaguamish River Watershed Multiparameter TMDL (2005) (0 NPDES); Upper Yakima Turbidity, TSS, and OCP TMDL (2002) (1 NPDES); Walla Walla River Watershed Toxics TMDL (2006) (1 NPDES); Wildcat Creek Multiparameter TMDL (1993) (1 NPDES); and EPA's Columbia River Basin Dioxin TMDL (1991) (4 NPDES).

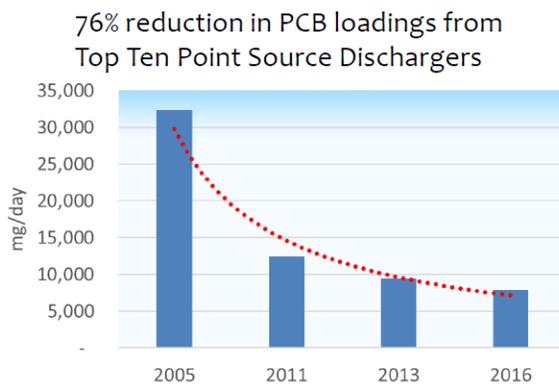
⁶⁷² The dioxin TMDLs for Grays Harbor and the Columbia River were developed to satisfy CWA Section 304(i).

⁶⁷³ Washington Assessment Database, *supra* n. 272 (search Current, Category 5 and 4a, all toxic parameters).

⁶⁷⁴ TMDLs for toxics in Oregon include: Columbia Slough DDT/DDE, Dieldrin, Dioxin, PCBs TMDL (1998) (0 NPDES, stormwater only); Lower Willamette (Johnson Creek) DDT, Dieldrin TMDL (2008) (0 NPDES, stormwater only); Molalla-Pudding Subbasin Iron, DDT, Dieldrin TMDL (2008) (10 NPDES, all "current conditions" for iron, DDT, and dieldrin); Snake River DDT, Dieldrin, Mercury TMDL (2004) (0 NPDES); Willamette Mercury TMDL (2006/2019/2021) (mercury minimization measures required or use of current conditions); and EPA's Columbia Basin dioxin TMDL (1991) (3 NPDES).

(3) require pollutant reductions, the three subject to EPA’s Columbia River basin TMDL for dioxin completed to satisfy CWA Section 304(l). In contrast to this pace of TMDL production for toxics in Oregon waters, 483 segment-parameters for toxics are on the current EPA-approved list of impaired waters for Oregon. It must be noted in this regard that Oregon does not place waters on its 303(d) list for toxic tissue residue or sediment contamination.

TMDLs can be effective in reducing toxic discharges as demonstrated by some of the results of Delaware River Basin Commission TMDLs for PCBs, as illustrated by this graph focused on a 76 percent reduction of PCB loadings from 10 primary sources of PCBs:⁶⁷⁵



Even where no TMDL has been established, the discharge of a pollutant into a waterbody that is impaired by that pollutant or a related pollutant is the very definition of a source’s “causing or contributing to a violation of water quality standards.” For this reason, the First Circuit Court of Appeals has explained that the absence of a TMDL, or other similar extensive analysis, is no defense for a permit writer’s failure to find reasonable potential and to establish a water quality-based effluent limit:

⁶⁷⁵ Delaware River Basin Commission, *Lessons Learned from other watersheds: Delaware River Basin Contaminants of Emerging Concern Surveys & PCB TMDL* (May 23, 2019) (hereinafter “Delaware Lessons Learned”) at 23, available at https://www.nj.gov/drbc/library/documents/macgillivray_ChesapeakeSTACworkshop_may2019.pdf.

TMDLs take time and resources to develop and have proven to be difficult to get just right; thus, under EPA regulations, permitting authorities must adopt interim measures to bring water bodies into compliance with water quality standards. *Id.* § 1313(e)(3); 40 C.F.R. § 122.44(d); *see also, e.g.*, 43 Fed. Reg. 60,662, 60,665 (Dec. 28, 1978) (“EPA recognizes that State development of TMDL’s and wasteload allocations for all water quality limited segments will be a lengthy process. Water quality standards will continue to be enforced during this process. Development of TMDL’s . . . is not a necessary prerequisite to adoption or enforcement of water quality standards . . .”).

* * *

[N]either the CWA nor EPA regulations permit the EPA to delay issuance of a new permit indefinitely until better science can be developed, even where there is some uncertainty in the existing data. . . . The Act’s goal of “eliminat[ing]” the discharge of pollutants by 1985 underscores the importance of making progress on the available data. 33 U.S.C. § 1251(a)(1).⁶⁷⁶

Similarly, the Environmental Appeals Board has held the same:

Where TMDLs have not been established, water quality-based effluent limitations in NPDES permits must nonetheless comply with applicable water quality standards. In discussing the relationship between NPDES permitting and TMDLs, EPA has explained that the applicable NPDES rules require the permitting authority to establish necessary effluent limits, even if 303(d) listing determinations and subsequent TMDLs lag behind. 54 Fed. Reg. 23,868, 23,878, 23,879 (June 2, 1989); *see also In re Upper Blackstone Water Pollution Abatement Dist.*, 14 E.A.D. 577, 604-05 (EAB 2010) (expressly rejecting the idea that the permitting authority cannot proceed to determine permit effluent limits where a TMDL has yet to be established), *aff’d*. 690 F.3d 9 (1st Cir. 2012), *cert. denied*, 133 S. Ct. 2382 (2013).⁶⁷⁷

And, in the absence of TMDLs, other EPA regulations require a permit writer to conduct some form of cumulative effects analysis as part of determining the applicable WQBEL:

When determining whether a discharge causes, has the reasonable potential to cause, or contributes to an in-stream excursion above a narrative or numeric criteria within a State water quality standard, the permitting authority shall use

⁶⁷⁶ *Upper Blackstone Water Pollution Abatement Dist. v. U.S. EPA*, 690 F.3d 14, n. 8 (1st Cir. 2012), *cert. denied*, 133 S. Ct. 2382 (2013).

⁶⁷⁷ *In re: City of Taunton Department of Public Works*, NPDES Appeal No. 15-08, slip op. at 11 (EAB May 3, 2016), *aff’d*. 895 F.3d 120 (1st Cir. 2018); *see also id.* at 40-41 (citing, *inter alia*, National Pollutant Discharge Elimination System; Surface Water Toxics Control Program, 54 Fed. Reg. 23,868, 23,879 (June 2, 1989) (clarifying in the preamble to 40 C.F.R. § 122.44 that subsection (d)(1)(vii) “do[es] not allow the permitting authority to delay developing and issuing a permit if a wasteload allocation has not already been developed and approved”).

procedures which account for existing controls on point and nonpoint sources of pollution, the variability of the pollutant or pollutant parameter in the effluent, the sensitivity of the species to toxicity testing (when evaluating whole effluent toxicity), and where appropriate, the dilution of the effluent in the receiving water.⁶⁷⁸

In all the NPDES permits that Petitioners have ever reviewed, it has never seen any attempt to address this federal requirement. To the contrary, not only have some states explicitly made TMDLs a prerequisite to a WQBEL—Washington State is one example⁶⁷⁹—but EPA has actually endorsed such an illegal approach saying: “In Washington, if a facility is not causing water quality impairment, the discharge is allowed until a TMDL is developed.”⁶⁸⁰

Finally, in its 1985 Toxics TSD permitting guidance, EPA repeatedly drew attention to the limitations of its Toxic Pollutants Lists, particularly for the absence of bioaccumulative pollutants. It noted:

The problem in addressing effluent discharges involves organic compounds that are not among the priority pollutants for which there are criteria.⁶⁸¹

* * *

It is important to note that the priority pollutant list is not sufficient for chemical-specific analysis because it is so limited in scope. There are many more hazardous toxicants discharged than are listed.⁶⁸²

* * *

⁶⁷⁸ 40 C.F.R. § 122.44(d)(1)(ii).

⁶⁷⁹ See NWEA Washington NPDES Petition, *supra* n. 587 at 19–21, 48–50, 52–56. EPA’s response to this issue was to note that “Ecology’s Manual is merely a guidance document, and permit writers are not required to follow the principles set forth in this guidance document. Moreover, as explained in this response, Ecology is currently establishing narrative WQBELs for nitrogen even though a TMDL has not been developed.” EPA, *Decision Document, Final Response to Petition to Withdraw Washington’s National Pollutant Discharge Elimination System (NPDES) Permit Program* (Nov. 29, 2021) at 6, n. 9.

⁶⁸⁰ Memorandum from Deborah G. Nagle, Acting Director, EPA Water Permits Division, Office of Wastewater Management, to Michael Bussell, Director, Office of Water and Watersheds, Region 10 Re: *2009 Regional National Pollutant Elimination System (NPDES) Program Review for Region 10* (Jan. 13, 2011), available at <https://fortress.wa.gov/ecy/publications/publications/0501006.pdf>. (EPA also cited favorably an example of the state’s having postponed a WQBEL for the Yakima Sewage Treatment plan because its use of a technology-based effluent limits “will prohibit the facility from further impairment of the Yakima River.”).

⁶⁸¹ EPA Toxics TSD, *supra* n. 627 at 22 (emphasis added).

⁶⁸² *Id.* at 26 (emphasis added).

It should be recognized from the outset that it is usually impossible to identify all the compounds contained in a wastewater. Typically, only a small fraction of the total organic carbon (TOC) can be accounted for as specific chemicals. Therefore, a substantial effort should be put into identifying constituents through means other than chemical analysis. The best way to accomplish this is through a detailed process evaluation.

A process evaluation is a study in which components in the wastewater are determined from an analysis of feedstocks, manufacturing processes, products, byproducts, and pollution control in place. The result is a list of compounds or classes of compounds with a high probability of being present in the wastewater. Chemical analysis should also be conducted for not only the priority pollutants but also nonpriority pollutant peaks.⁶⁸³

Accordingly, this EPA guidance identifies “three approaches to identifying non-priority bioaccumulative pollutants in effluents.”⁶⁸⁴ First, “[a]quatic biota can be collected upstream and downstream of an effluent outfall and analyzed for specific compounds or any compound present in downstream biota that is not present in upstream biota.” EPA notes this approach is expensive but it is “the only way to assess bioaccumulation.”⁶⁸⁵ The second approach is to look at what is being manufactured by the facility.⁶⁸⁶ Last, EPA notes that high pressure liquid chromatography analysis can be used when “effluent is too complex to list all compounds,” followed by an evaluation of the toxicological properties of the identified classes or individual chemicals.⁶⁸⁷ EPA lists numerous sources of information, including the Chemical Information System, a database of over 250,000 substances.⁶⁸⁸ Petitioners have never seen any permit writer’s evaluation that has looked at pollutants beyond those on the Toxic Pollutants Lists.⁶⁸⁹

⁶⁸³ *Id.* at 28 (emphasis added).

⁶⁸⁴ *Id.* at 28.

⁶⁸⁵ *Id.* at 28 (emphasis added).

⁶⁸⁶ *Id.* at 28.

⁶⁸⁷ *Id.* at 29.

⁶⁸⁸ *Id.*

⁶⁸⁹ There are examples of dischargers conducting their evaluations of discharges of toxic pollutants not on the Toxic Pollutants Lists of their own accord.

Point sources subject to NPDES permits are, of course, not the only sources of toxic pollution that could be addressed by a robust TMDL program. There are two significant hurdles to overcome, however. The first is the lack of TMDLs for toxic contaminants, as discussed above. The second is a weakness in the TMDL program regarding clarity in and implementation of load allocations to nonpoint sources. This significant weakness was identified by the U.S. Government Accountability Office (“GAO”) in 2014.⁶⁹⁰ In a sample of 25 TMDLs reviewed,

17 of 25 long-established TMDLs they reviewed did not show that addressing identified stressors would help attain water quality standards; 12 contained vague or no information on actions that need to be taken, or by whom, for implementation; and 15 did not contain features to help ensure that TMDLs are revised if need be. GAO’s review showed that EPA’s existing regulations do not explicitly require TMDLs to include these key features, and without such features in TMDLs—or in addition to TMDLs—impaired water bodies are unlikely to attain standards.⁶⁹¹

For nonpoint sources, the results were particularly damning:

In response to GAO’s survey, state officials reported that long-established TMDLs generally do not exhibit factors most helpful for attaining water quality standards, particularly for nonpoint source pollution (e.g., farms and storm water runoff). The officials reported that landowner participation and adequate funding—factors they viewed as among the most helpful in implementing TMDLs—were not present in the implementation activities of at least two-thirds of long-established TMDLs, particularly those of nonpoint source TMDLs.⁶⁹²

Even GAO did not recognize the extent of the problem regarding TMDLs’ failure to guide needed nonpoint source pollution controls. It cited an example of a TMDL with more rather than less detail on the nonpoint source problem:

For example, the TMDL for the South Santiam River in Oregon describes and locates soil conditions, vegetation, and human uses affecting the river and its

⁶⁹⁰ GAO, *Clean Water Act: Changes Needed if Key EPA Program is to Help Fulfill the Nation’s Water Quality Goals* (Dec. 2013) (hereinafter “Changes Needed to TMDL Program”), available at <https://www.gao.gov/assets/gao-14-80.pdf>.

⁶⁹¹ *Id.* at Highlights.

⁶⁹² *Id.*

tributaries. It also describes in detail specific steps to address elevated temperature in the river, including restoring stream channels, native vegetation, and natural streamflow, and it aligns such steps with the specific conditions and areas within the TMDL's geographic boundary. Other state TMDLs simply direct that runoff from all nonpoint sources is to fall below the overall load allocated among them.⁶⁹³

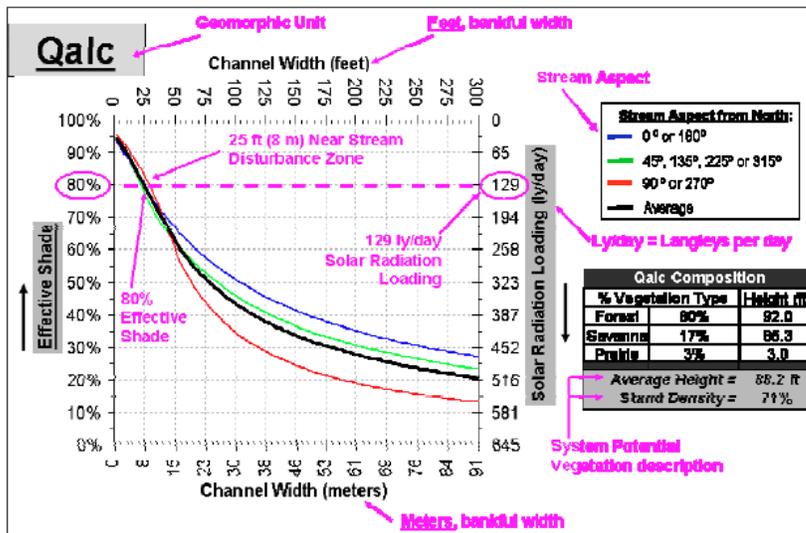
But what GAO missed in this description of the South Santiam TMDLs is that the TMDL does not include any "information on actions that need to be taken, or by whom" even as it discusses the problems leading to the water quality impairment. There is a world of difference between a TMDL that *describes* a need for "native vegetation" and one that states *how much* native streamside vegetation is required to meet the load allocation. The raging argument over whether Oregon's and Washington's logging practices are sufficient to meet water quality standards, for example, is not *whether* riparian vegetation protects streams from warming but *precisely how wide* that riparian buffer needs to be.⁶⁹⁴ The South Santiam temperature TMDLs cited by the GAO do not establish the BMPs needed for riparian vegetation but, rather, describe a "surrogate measure . . . [that] is percent effective shade expressed as a shade curve."⁶⁹⁵ As shown below,

⁶⁹³ *Id.* at 24.

⁶⁹⁴ See, e.g., EPA, National Oceanic and Atmospheric Administration ("NOAA"), *NOAA/EPA Finding that Oregon has Not Submitted a Fully Approvable Coastal Nonpoint Program* (Jan. 30, 2015) at 1 ("the State has not adopted additional management measures applicable to forestry that are necessary to achieve and maintain applicable water quality standards under Clean Water Act section 303 and to protect designated uses. NOAA and EPA first identified and notified the State of the need to implement the additional measures in 1998."), 4 ("Based on the results of a number of studies including those summarized below, NOAA and EPA previously determined and continue to find that additional management measures (beyond those in FPA rules and the voluntary program) for forestry riparian protection around medium-sized and small fish-bearing streams and non-fish-bearing streams are necessary to attain and maintain water quality standards and to protect designated uses."), 6 ("As early as 1999, the IMST study found that the FPA rule requirements related to riparian buffers and large woody debris needed to be improved."). At a result of the federal agencies' disapproval of Oregon's Coastal Nonpoint Pollution Control Program under the Coastal Zone Act Reauthorization Amendments ("CZARA"), EPA and NOAA have withheld over \$8 million in federal grant funds.

⁶⁹⁵ Oregon Department of Environmental Quality, *Willamette Basin TMDL, Chapter 9: South Santiam Subbasin* (Sept. 2006) at 9-33.

the TMDL explains how a landowner can identify what part of a shade curve applies to his or her land but nowhere in the TMDL (or its implementation plan) does it show how a shade curve answers the key question: how wide does the riparian buffer need to be to meet the load allocation?⁶⁹⁶



Example: A tributary to the Willamette River on the west bank near Independence with a stream aspect from north of 0° or 180° (blue line) and a channel width of 25 feet: using the blue line to determine the loading capacity from the x-axis identify the 25 feet (8 m) mark and read the y-axis, the solar radiation loading would be 129 Langley's/day with 80% effective shade when system potential vegetation is applied to the left and right bank of the stream reach. System potential vegetation identifies the riparian average height, 88.2 feet (26.9 m), and stand density (tree canopy density), 71 %, that would be established in the riparian area. If it is difficult to determine the streams aspect from north, the average stream aspect from north, black line, can be used to determine the solar radiation loading and effective shade.

As a result, not a single TMDL completed in Oregon or Washington over three decades has resulted in changes in logging practices despite findings that such logging practices are not meeting water quality standards.⁶⁹⁷ Not a single TMDL has made clear what riparian buffers or other BMPs are required for agricultural lands to meet load allocations.

⁶⁹⁶ *Id.* at 9-36.

⁶⁹⁷ See, e.g., Letter from Laura Watson, Director, Washington Department of Ecology, to Forest Practices Board Members (Dec. 3, 2021) at 1 (noting that 22 years have passed waiting for improvements in logging practices, including “rules related to non-fish bearing perennial streams (Type Np riparian buffers) in Western Washington” during which time TMDLs pertaining to commercial logging on private lands have been suspending in an agreement termed the “Clean Water Assurances”), 3 (noting that the State “Auditor’s Office concluded that the program is not ‘operating as intended’ and that, without needed changes, the “program would continue to languish.”), 3 (citing 2009 findings that “the prescriptions associated with Type Np rules have the greatest potential risk of violating the water quality standards.”).

GAO correctly points out that some states have formal TMDL implementation plans that could, in theory, contain such information⁶⁹⁸ but in general these plans do not add any of the information just described as lacking from the TMDLs themselves. For example, although logging and farming are the greatest source of Oregon’s ubiquitous temperature impairments, the 2006 implementation plan for the entire Willamette River basin, including the South Santiam subbasin says this about the TMDL’s implications for changed practices, which is nothing at all:

Forest operators conducting operations in accordance with the Forest Practices Act (FPA) are considered to be in compliance with water quality standards.⁶⁹⁹

* * *

The Environmental Quality Commission, Board of Forestry, [Oregon Department of Environmental Quality] ODEQ, and [Oregon Department of Forestry] ODF have agreed that these pollution control measures will be relied upon to result in achievement of state water quality standards.⁷⁰⁰

This same effect of no change to logging and farming practices applies equally to Oregon’s Willamette River basin mercury TMDLs, which found those two sources drive the mercury impairments (mobilizing atmospheric deposition of mercury).⁷⁰¹ In EPA’s reasonable assurances that nonpoint sources will be controlled sufficiently to allow for wasteload allocations

⁶⁹⁸ Changes Needed to TMDL Program, *supra* n. 690 at 26.

⁶⁹⁹ Oregon Department of Environmental Quality, *Willamette Basin TMDL Chapter 14: Water Quality Management Plan* (Sept. 2006) at 14-12.

⁷⁰⁰ *Id.* at 14-19.

⁷⁰¹ EPA issued a TMDL for mercury in 2021, largely relying upon the state’s earlier 2019 TMDL that it had disapproved. See EPA, Impaired Waters and TMDLs, *Willamette Basin Mercury TMDL*, available at <https://www.epa.gov/tmdl/willamette-basin-mercury-tmdl>. In its TMDL, EPA described the outcome: “ODEQ’s 2019 TMDL assigned an 88% load reduction to mercury in sediment erosion from agriculture, forestry, developed land outside of urban DMAs or MS4s, and ‘other’ nonpoint source load categories such as water impoundments and water conveyance entities. Greater reductions are needed in subbasins that did not achieve ODEQ’s 2019 TMDL target. EPA conducted an analysis where the needed reduction from this category was incrementally increased above 88% to the point where, in combination with other allocation adjustments, the TMDL target would be met in each subbasin. These needed changes in sediment erosion varied (89 – 97%) by subbasin due to land use and loading differences between subbasins, and due to the magnitude of departure in meeting the TMDL target in ODEQ’s 2019 TMDL. EPA, *Total Maximum Daily Load (TMDL) for Mercury in the Willamette Basin, Oregon* (Feb., 4, 2021) at 8; see also *id.* at 14 (Table 3. Percent Reductions for Land Managers in the Willamette River Basin and Subbasins).

to point sources, EPA merely cites the Water Quality Management Plan prepared for the 2006 temperature TMDLs, cited above, that in turn cites the existing logging and farming practices.⁷⁰²

The GAO made the following recommendation pertaining to TMDLs and nonpoint source controls:

To enhance the likelihood that TMDLs support the nation's waters' attainment of water quality standards and to strengthen water quality management, the Administrator of EPA should develop and issue new regulations requiring that TMDLs include additional elements--and consider requiring the elements that are now optional--specifically, elements reflecting key features identified by NRC as necessary for attaining water quality standards, such as comprehensive identification of impairment and plans to monitor water bodies to verify that water quality is improving.⁷⁰³

GAO recently pointed out that this priority recommendation remains “open” because EPA continues to refuse to use its only means to address nonpoint source pollution that does not involve withholding funding to state agencies:

Since June 2020, EPA officials stated that they believe this action has been implemented. They said the agency has taken several actions that change the focus of the total maximum daily loads (TMDL) program to focus efforts on implementing TMDLs. First, EPA developed a TMDL Vision document to focus on integrating and implementing different efforts to restore and protect the nation's aquatic resources. Second, EPA held regional meetings to discuss different TMDL topics such as monitoring, implementation, and reasonable assurance. Included in these discussions were good practices and examples. EPA officials stated that these actions have changed the focus of the program in the place of regulations. We agree that these actions are helpful and can take the agency and states in the direction of improving the TMDL program. However, the actions do not carry the force of regulations and we believe that the problems of nonpoint source pollution require stronger action such as regulations to be resolved. In July 2020, EPA officials told us they did not believe the agency could issue the recommended regulations under the agency's current authority. The officials also stated that EPA had no plans to develop TMDL regulations to address our recommendation. As of March 2023, EPA officials told us that the

⁷⁰² *Id.* at 15.

⁷⁰³ GAO, Reports & Testimonies, Clean Water Act: Changes Needed If Key EPA Program Is to Help Fulfill the Nation's Water Quality Goals, *Recommendations for Executive Action*, available at <https://www.gao.gov/products/gao-14-80>.

agency had not changed its position. We continue to believe that EPA has the authority to issue the regulations we recommended, so long as it follows all applicable procedural and substantive requirements. We also believe that the problems of nonpoint source pollution, which is a major contributor to pollution in our nation's waters, require stronger actions such as issuing new regulations. To fully implement our recommendation, EPA would have to develop TMDL regulations that include additional elements—such as comprehensive identification of impairment and plans to monitor water bodies to verify that water quality is improving—to ensure that TMDLs help water bodies attain water quality standards.⁷⁰⁴

Pollution controls applied to nonpoint sources are almost uniformly described as “best management practices” (“BMPs”). Defined in the context of NPDES permits,

BMPs typically involve requirements like operating procedures, treatment requirements, practices to control runoff, spillage or leaks, sludge or waste disposal, or drainage from raw material storage; they can also be structural requirements including tarpaulins, retention ponds, or devices such as berms to channel water away from pollutant sources, and treatment facilities. *See NRDC v. Sw. Marine, Inc.*, 236 F.3d 985, 991 n. 1 (9th Cir.2000). Examples of BMPs that have been accepted as substitutes for effluent limits include: nutrient management plans for concentrated animal feeding operations, *see Waterkeeper All.*, 399 F.3d at 497, 502, filtration of stormwater runoff from ditches before it enters rivers and streams (by timber companies), and constructing roads with surfacing that minimizes sediment in runoff (by timber companies), *see Decker v. Nw. Env'tl. Def. Ctr.*, — U.S. —, 133 S.Ct. 1326, 1338, 185 L.Ed.2d 447 (2013).

Natural Resources Defense Council v. U.S. E.P.A., 808 F.3d 579 (2d Cir. 2015). BMPs for nonpoint sources are similar and often include methods of protecting the immediate riparian area next to a waterbody with a vegetated riparian buffer, methods of protecting the riparian buffer from degradation, keeping applications of toxic materials such as pesticides away from waterbodies and from entering groundwater, and preventing various types of polluted runoff and erosion by limiting plowing and other activities that release soil to water.

TMDLs, however, rarely describe the load allocations made to nonpoint sources in terms

⁷⁰⁴ *Id.*

of practical, easy to understand and easy to evaluate, BMPs. Instead, load allocations are simply amounts of a pollutant that can enter a waterbody from nonpoint sources. In some instances, those numeric load allocations are translated into “surrogate measures,” but even these are largely unusable because they do not describe the actions needed as BMPs but, rather as obscure and unmeasurable metrics.

The water quality-based approach has, in theory, tremendous potential to fill the gaps left by the technology-based approach in protecting the nation’s designated uses from toxic pollution. In practice, however, the strengths of the water quality-based approach are routinely jettisoned by agency policies and permit writers. Even if they were not, Congress never intended the water quality-based approach to carry the entire burden of reaching CWA goals.

VII. TOXIC POLLUTANTS NOT APPROPRIATELY REGULATED BECAUSE OF OUTDATED TOXIC POLLUTANTS LISTS

Just as the ELGs for priority pollutants are supposed to be the front-line protection of the nation’s waters from toxic pollution, as discussed in section V *supra*, water quality standards are intended to ensure additional pollution controls are implemented where they are required to protect water quality. (See section VI for a discussion of just how imperfectly this water quality-based regulation is working.) However, while OCPSF facilities are subject only to 35-year-old ELGs as described above, they are likewise not subject to updated water quality standards that reflect the latest scientific understandings of how chemicals affect human health and species or, for many chemicals, any criteria at all. For example, polybrominated diphenyl ethers (“PBDEs”) are chemicals used to produce plastics for which EPA has not published recommended criteria, as discussed *infra* at sub-section VII.E.3; PBDEs are not on the Toxic Pollutants Lists.

Another example of toxic pollutants used in the OCPSF sector with outdated criteria is phthalates, chemicals known as plasticizers that make plastics more durable.⁷⁰⁵ EPA adopted its 304(a) recommended criteria for phthalate esters in October 1980—42 years ago.⁷⁰⁶ The 110-page criteria document includes extensive analysis of the literature available—as of the late 1970s—on aquatic and mammalian toxicity⁷⁰⁷ and human health effects.⁷⁰⁸ EPA has not reviewed or updated the recommended criteria for phthalates since 1980,⁷⁰⁹ despite considerable new information about human exposure,⁷¹⁰ human health effects,⁷¹¹ and aquatic environment exposure and toxicity of phthalates.⁷¹² Therefore, to the extent EPA is relying on the water quality-based approach to regulate toxics from point sources to fill the gap left by its outdated ELGs, it is clear that this approach, too, is based on the science of over a half century ago.

The combination of outdated and nonexistent water quality criteria for toxics and significant barriers to their use in restricting the discharge of toxic pollution, discussed in section VI *supra*, heightens the importance of ELGs and pretreatment standards to protect the nation’s waters. EPA’s action to develop ELGs and pretreatment standards for toxic pollutants, however,

⁷⁰⁵ See Center for Disease Control and Prevention, National Biomonitoring Program, *Phthalates Factsheet*, available at https://www.cdc.gov/biomonitoring/Phthalates_FactSheet.html (hereinafter “CDC Phthalates Factsheet”).

⁷⁰⁶ EPA, *Ambient Water Quality Criteria for Phthalate Esters* (1980), available at <https://www.epa.gov/sites/default/files/2019-03/documents/ambient-wqc-phthalateesters-1980.pdf>.

⁷⁰⁷ See *id.* at B-1 *et seq.*

⁷⁰⁸ See *id.* at C-1 *et seq.*

⁷⁰⁹ See EPA, *Quality Criteria for Water 1986* (May 1986), at *Water Quality Criteria Summary* (table depicting dates of adoption) (hereinafter “1986 Recommended Criteria”).

⁷¹⁰ See CDC Phthalates Factsheet, *supra* n. 710.

⁷¹¹ See, e.g., Sailas Benjamin *et al.*, *Phthalates Impact Human Health: Epidemiological Evidences and Plausible Mechanism of Action*, 340 *J. Hazardous Materials* 360 (2017).

⁷¹² See, e.g., Ying Zhang *et al.*, *Hazards of Phthalates (PAEs) Exposure: A Review of Aquatic Animal Toxicology Studies*, 771 *Science of the Total Env’t.* 145418 (2021).

is triggered by pollutants being placed on the Toxic Pollutant List, demonstrating the importance of granting this petition.

There are multiple categories of toxic pollutants where EPA's actions and other data and information demonstrate that the Toxic Pollutant List must be updated pursuant to CWA Section 307(a)(1). In this section, we look at some of these different categories. Sub-section A reviews toxic pollutants that EPA has already determined pose a sufficiently significant threat to public health and the environment that it has developed Section 304(a) recommended criteria for them, notwithstanding its choice to not add them to the Toxic Pollutant List. Likewise, in sub-section B we examine EPA's ongoing development of recommended criteria for toxic pollutants for which EPA has made this same determination and is in the process of developing 304(a) criteria: PFAS and PFOA. Sub-section C discusses toxic pollutants that EPA considers a sufficient threat to public health and the environment that it requires data collection on them in the Toxics Release Inventory program but has likewise not placed them on the Toxic Pollutant List. Similarly, sub-section D identifies toxic pollutants identified by EPA as requiring source control to prevent the ongoing contamination of Superfund sites but which EPA has not designed as priority pollutants. In sub-section E, the petition discusses the issue of so-called "contaminants of emerging concern," decades after they first "emerged" as a toxic threat and for which EPA has taken no regulatory actions. This includes the broad category as well as some individual pollutants. Sub-section F provides information on pesticides that are not on the Toxic Pollutant List but that have long been identified as posing "jeopardy" to ESA-listed threatened and endangered species.

EPA is required by CWA Section 307(a)(1) to "take into account the toxicity" of toxic pollutants and "its persistence, degradability, the usual or potential presence of the affected

organisms in any waters, the importance of the affected organisms and the nature and extent of the effect of the toxic pollutant on such organisms” in revising the Toxic Pollutants List. The pollutants and chemical families discussed in each of these sub-sections below are known in the scientific literature and EPA’s own analysis to be persistent, often ubiquitous, and to have adverse effects on a range of species. It is EPA’s responsibility to systematically evaluate all relevant monitoring data and scientific information to determine which new pollutants to list. This Petition only includes examples that suggest the large number of pollutants that must be listed or further evaluated for listing on the Toxics Pollutants Lists. It is not, however, intended to be exhaustive. Rather, it illustrates the kind of analysis EPA should undertake on an ongoing basis.

A. Pollutants for Which EPA Has Developed Section 304(a) Recommended Water Quality Criteria but Which are Not Subject to the Requirements of CWA Section 303(c)(2)(B)

Notwithstanding EPA’s failure to update the Toxic Pollutant List, the agency has developed numerous 304(a) recommended criteria for toxic pollutants it has deemed to pose a hazard to aquatic life. For this reason and the reasons discussed below, each of these toxic pollutants should be added to the Toxic Pollutant List. These include, along with the dates of adoption or revision of recommended aquatic life criteria the following pollutants that are not on the Toxics Pollutants Lists:

- Aluminum (2018)
- Ammonia⁷¹³ (2013 freshwater; 1989 saltwater)
- Carbaryl (2012)
- Chloride (dissolved) (1988)
- Chlorine (total residual) (1986)

⁷¹³ CWA § 301(g) names the following pollutants as “nonconventional”: ammonia, chlorine, color, iron, and total phenols. With the exception of color, these pollutants act with a “toxic” effect as defined in Section 502(13).

- Chloropyrifos (1986)
- Demeton (1985)
- Diazinon (2005)
- Guthion (1986)
- Iron (1986)
- Malathion (1986)
- Methoxychlor (1986)
- Mirex (1986)
- Nonylphenol (2005)
- Parathion (1995)
- Sulfide-Hydrogen Sulfide (1986)
- Tributyltin (2004)

1. Aluminum

EPA first published 304(a) recommended criteria for aluminum in 1988 to “protect aquatic life from harmful effects of aluminum toxicity.”⁷¹⁴ In 2018, due to EPA’s obligation under a legal settlement to update the State of Oregon’s aluminum criteria,⁷¹⁵ EPA updated its 304(a) criteria to reflect the latest scientific knowledge, including using studies from 1988 to 2017 that more accurately reflect the toxicity of aluminum to aquatic life.⁷¹⁶ These updated recommendations now take into account the effects of pH, total hardness, and dissolved organic carbon on aluminum toxicity to aquatic life.⁷¹⁷ The EPA obligation stemmed from a finding by the National Marine Fisheries Service (“NMFS”) determination that aluminum at levels allowed by the Oregon criteria, that reflected EPA 304(a) criteria, posted “jeopardy” to the continuing existence of certain salmonids in Oregon waters listed as threatened or endangered under the Endangered Species Act (“ESA”).⁷¹⁸

⁷¹⁴ 2018 Aluminum Recommended Criteria, *supra* n. 286, at xi-xii.

⁷¹⁵ *See Northwest Environmental Advocates v. U.S. EPA*, 3:15-cv-00663-BR (D. Or. 2015).

⁷¹⁶ 2018 Aluminum Recommended Criteria, *supra* n. 286 at xi.

⁷¹⁷ *Id.* at xii.

⁷¹⁸ NMFS, *Jeopardy and Destruction or Adverse Modification of Critical Habitat Endangered Species Act Biological Opinion for Environmental Protection Agency’s Proposed Approval of Certain Oregon*

According to EPA, “[e]levated levels of aluminum can affect some species ability to regulate ions, like salts, and inhibit respiratory functions, like breathing.”⁷¹⁹ Aluminum accumulates on gill surfaces of fish and damages epithelial cells, causing loss of plasma ions and reduced ion uptake and gas exchange.⁷²⁰ This interrupts gill function, often causes necrosis, and ultimately causes fish to die.⁷²¹ In invertebrates, aluminum is believed to disrupt certain ion concentrations, resulting in sodium loss, which can lead to changes in respiratory efficiency in sensitive species.⁷²² Aluminum is also thought to interfere with salt regulation, causing reduction in whole body sodium and chloride concentration, ultimately resulting in the death of aquatic species.⁷²³ According to NMFS, the toxic effects of aluminum on salmon can be summarized as follows (referring to the Oregon adopted and EPA recommended criteria):

The available evidence for indicates that listed species exposed to waters equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (high-intensity), reduced growth (high-intensity), impairment of essential behaviors related to successful rearing and migration (moderately-high-intensity), cellular trauma (moderate intensity), and physiological trauma (moderately-high-intensity).⁷²⁴

Aluminum can enter water from human activities such as drinking⁷²⁵ and wastewater treatment, mining, and other industrial processes.⁷²⁶ Other anthropogenic sources include

Administrative Rules Related to Revised Water Quality Criteria for Toxic Pollutants (Aug. 14, 2012) (hereinafter “NMFS Oregon Toxics BiOp”).

⁷¹⁹ Aquatic Life Ambient Water Quality Criteria for Aluminum in Freshwater, 83 Fed. Reg. 65663, 65,664 (Dec. 21, 2018); 2018 Aluminum Recommended Criteria, *supra* n. 286 at 11.

⁷²⁰ *Id.*

⁷²¹ *Id.*

⁷²² *Id.*

⁷²³ *Id.*

⁷²⁴ NMFS Oregon Toxics BiOp, *supra* n. 718 at 249.

⁷²⁵ Fluoride increases the toxicity of aluminum. *See* 2018 Aluminum Recommended Criteria, *supra* n. 286 at 14.

⁷²⁶ *Id.* at 2.

mining tailings, fossil fuel, air emissions, and agricultural fertilizers.⁷²⁷ In the marine environment, the mixing of aluminum with the high salinity and pH of the ocean waters increases the toxicity of aluminum to aquatic species.⁷²⁸ Data collected by the USGS National Water Quality Assessment program between 1992 and 2003 revealed a 90th percentile concentration of dissolved aluminum, illuminating the high concentrations of aluminum that exist in the nation’s waters.⁷²⁹ Aluminum becomes particularly toxic when combined with acid rain, where researchers observe aluminum is responsible for the demise of biotic communities because it becomes more soluble and more toxic to aquatic biota at acidic pH levels.⁷³⁰

2. Ammonia

EPA’s 304(a) recommended criteria for ammonia was developed in 2013 (freshwater) and 1989 (salt water).⁷³¹ As with aluminum, ammonia was the subject of a NMFS jeopardy call pursuant to the ESA with regard to EPA’s approval of Oregon’s updated toxic criteria.⁷³² EPA updated the criteria to protect aquatic life from toxic effects of ammonia in freshwater through 2013 criteria that supersede EPA’s 1999 recommendations.⁷³³ Ammonia is a significant pollutant in the nation’s waters; EPA’s most recent report on the Toxics Release Inventory (“TRI”) shows that ammonia accounts for 21 percent of total releases by chemicals in 2020 that are not nitrate compounds discharged directly into waterbodies.⁷³⁴

⁷²⁷ *Id.* at 3.

⁷²⁸ *Id.*

⁷²⁹ *Id.* at 4.

⁷³⁰ *Id.* at 10.

⁷³¹ EPA, *Aquatic Life Ambient Water Quality Criteria for Ammonia - Freshwater (2013)* (Aug. 2013) (hereinafter “Ammonia Fact Sheet”), available at https://www.epa.gov/sites/default/files/2015-08/documents/fact_sheet_aquatic-life-ambient-water-quality-criteria-for-ammonia-freshwater-2013.pdf.

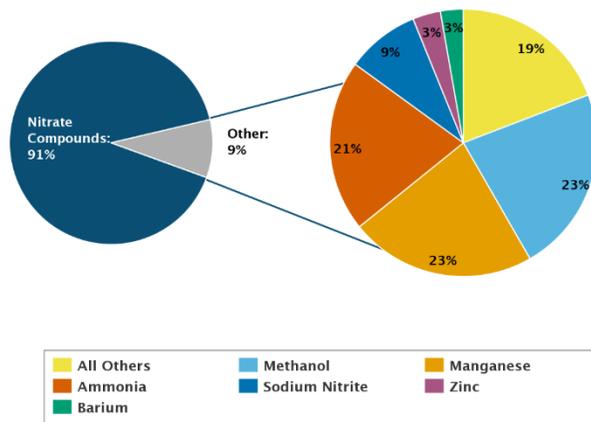
⁷³² NMFS Oregon Toxics BiOp, *supra* n. 718.

⁷³³ See Ammonia Criteria website, *supra* n. 285.

⁷³⁴ EPA, TRI National Analysis, Water Releases by Chemical & Industry,

Water Releases by Chemical, 2020

193.61 million pounds



Ammonia is widely considered one of the most detrimental pollutants in the aquatic environment because it is extremely toxic and ubiquitous in surface water.⁷³⁵ The chemical is commonly used in production of commercial fertilizer, which accounts for approximately 90 percent of ammonia produced, as well as in the chemical, pharmaceutical, mining, metal finishing, and petroleum industries.⁷³⁶ Ammonia occurs naturally in organic waste decomposition, atmospheric gas exchange, animal and human waste products, and nitrogen fixation and is therefore discharged from sewage treatment plants and animal facilities as well as indirect sources like nitrogen fixation, air deposition, and agricultural runoff.⁷³⁷ Despite efforts to keep ammonia concentrations from being unacceptably high,⁷³⁸ in 2010, industrial releases of ammonia to ten large aquatic ecosystems (e.g., Chesapeake Bay, Puget Sound, Great Lakes)

Water Releases By Chemical, available at <https://www.epa.gov/trinationalanalysis/water-releases-chemical-industry>.

⁷³⁵ EPA, *Aquatic Life Ambient Water Quality Criteria for Ammonia – Freshwater 2013* (April 2013) (hereinafter “2013 Ammonia Recommended Criteria”) available at <https://www.epa.gov/sites/default/files/2015-08/documents/aquatic-life-ambient-water-quality-criteria-for-ammonia-freshwater-2013.pdf>.

⁷³⁶ *Id.* at 5.

⁷³⁷ *Id.* at 5.

⁷³⁸ *Id.* at 6.

were reported to total approximately 1.3 million pounds,⁷³⁹ and roughly 3.71 million pounds of ammonia was documented as discharged from reporting industries into the surface waters of the United States in 2020.⁷⁴⁰

Once ammonia has reached a water body, it is challenging for organisms to excrete, leading to a toxic build up in their internal tissues and blood.⁷⁴¹ A variety of environmental factors can contribute or detract from the toxicity of ammonia to organisms, including pH and temperature.⁷⁴² EPA's 2013 ammonia criteria recommendations consider freshwater toxicity information for ammonia, including toxicity studies for sensitive unionid mussels and gill-breathing snails and are more stringent than previous criteria to reflect the updated knowledge of ammonia's toxicity to these species.⁷⁴³ Unionid mussels are found in almost every state in the country, and approximately 25 percent of 300 freshwater unionid mussel taxa in the United States are federally-listed as endangered or threatened species.⁷⁴⁴

According to NMFS, effects on salmonids from ammonia can be summarized as follows (pertaining to Oregon ammonia criteria in place in 2012):

The available evidence for indicates that listed species exposed to water equal to the acute or chronic criteria concentrations will suffer acute and chronic toxic effects including mortality (high-intensity), reduced growth (high-intensity), impairment of essential behaviors related to successful rearing and migration (moderately-high-intensity), cellular trauma (high-intensity), physiological trauma (high-intensity), impairment of biochemical processes (high-intensity), and sublethal effects—ACR-NOEC analysis—(moderately-high-intensity to high-intensity).⁷⁴⁵

⁷³⁹ *Id.*

⁷⁴⁰ *Id.*

⁷⁴¹ Ammonia Fact Sheet, *supra* n. 731 at 1.

⁷⁴² *Id.*

⁷⁴³ *Id.*

⁷⁴⁴ 2013 Ammonia Recommended Criteria, *supra* n. 735 at 22.

⁷⁴⁵ NMFS Oregon Toxics BiOp, *supra* n. 718 at 239.

3. Carbaryl, Chlorpyrifos, Diazinon, and Malathion

Of the non-priority pollutants for which EPA has recommended 304(a) criteria, four are currently-used pesticides for which NMFS has completed ESA consultation with EPA on its pesticide registrations under the Federal Insecticide, Fungicide, and Rodenticide Act (“FIFRA”): carbaryl, chlorpyrifos, diazinon, and malathion. In 2017, NMFS, in collaboration with EPA, issued a biological opinion evaluating the effects of the pesticides chlorpyrifos, diazinon, and malathion on federally-listed species and designated critical habitats, concluding that the three pesticides are likely to jeopardize 38 ESA-listed species—including several listed anadromous fish and Southern Resident killer whales—and adversely modify 37 designated critical habitats.⁷⁴⁶ As a result, EPA determined that current application methods of chlorpyrifos, diazinon, and malathion are expected to produce aquatic concentrations of the three pesticides that are likely to cause harm to 77 federally-listed species and 50 designated critical habitats.⁷⁴⁷ In June 2022, NMFS issued a revised biological opinion in which it concluded that the effects of EPA’s approval of the registration of these pesticides would not jeopardize ESA-listed species because the approval action included “additional conservation measures”⁷⁴⁸ by EPA described as

⁷⁴⁶ NMFS, *Biological Opinion on the Environmental Protection Agency’s Registration of Pesticides containing Chlorpyrifos, Diazinon, and Malathion* (Dec. 29, 2017), available at https://repository.library.noaa.gov/view/noaa/16997/noaa_16997_DS1.pdf?download-document-submit=Download.

⁷⁴⁷ EPA, *Chlorpyrifos Executive Summary for ESA Assessment* (undated), available at <https://www3.epa.gov/pesticides/nas/final/chlorpyrifos/executivesummary.docx>; EPA, *Executive Summary for Diazinon ESA Assessment* (undated), available at: <https://www3.epa.gov/pesticides/nas/final/diazinon/executive-summary-diazinon.docx>; EPA, *Executive Summary for Malathion ESA Assessment* (undated), available at: <https://www3.epa.gov/pesticides/nas/final/malathion/executive-summary.docx>.

⁷⁴⁸ NMFS, *Revised Conference and Biological Opinion on the Environmental Protection Agency’s Registration Review of Pesticide Products containing Chlorpyrifos, Malathion, and Diazinon* (June 30, 2022) (hereinafter “2022 Chlorpyrifos BiOp”) at 1192, available at <https://www.fisheries.noaa.gov/resource/document/biological-opinion-chlorpyrifos-diazinon-and-malathion>.

follows:

The primary mechanisms for addressing these elements include removal of label authorization for some high risk uses (e.g. aerial applications of diazinon and wide area applications of chlorpyrifos for ants; option 1a) and implementation of mitigation to reach targets for drift and runoff reductions outlined in Point System (draft Opinion, option 1c). The measures will be incorporated through a combination of general label changes and through enforceable geographically specific requirements specified on EPA Endangered Species Protection Program Bulletins.⁷⁴⁹

Additional mitigation measures are also required.⁷⁵⁰ Notwithstanding these measures, NMFS determined that the risk to numerous threatened and endangered species from these pesticides is “high”⁷⁵¹ and that in the absence of mitigating actions, they pose jeopardy to these species.⁷⁵²

EPA has published 304(a) recommended criteria for these three pesticides. The criteria for chlorpyrifos⁷⁵³ and malathion⁷⁵⁴ both date to 1986 and the criteria for diazinon⁷⁵⁵ to 2005—all pre-dating the NMFS biological opinion’s finding jeopardy at levels deemed acceptable to EPA. This strongly suggests that EPA’s 304(a) criteria are outdated and insufficiently protective of aquatic life.

In 2009, NMFS concluded that pesticide products containing carbaryl are likely to jeopardize many populations of ESA-listed Pacific salmonids and destroy or adversely modify

⁷⁴⁹ *Id.* at 3.

⁷⁵⁰ *See id.* at 3, 1194–1202.

⁷⁵¹ *See id.* at 693–696 (table 263), 698–701 (table 264), 702–705 (table 265).

⁷⁵² *See id.* at 810–812 (table 272), 900–903 (Table 275), 992–995 (table 278).

⁷⁵³ 1986 Recommended Criteria, *supra* n. 709 (neither chlorpyrifos nor its CAS number (2921882) are found in this document but numeric criteria are listed in EPA’s National Recommended Aquatic Life Criteria table, *available at* <https://www.epa.gov/wqc/national-recommended-water-quality-criteria-aquatic-life-criteria-table>, with a citation to the 1986 Recommended Criteria.)

⁷⁵⁴ EPA, *Aquatic Life Ambient Water Quality Criteria Diazinon FINAL* (Dec. 2005), *available at* <https://www.epa.gov/sites/default/files/2019-03/documents/ambient-wqc-diazinon-final.pdf>.

⁷⁵⁵ 1986 Recommended Criteria, *supra* n. 709.

their designated critical habitat.⁷⁵⁶ EPA published 304(a) aquatic life criteria for carbaryl in 2012, noting that “[c]arbaryl is the second most frequently found insecticide in water, with detections in approximately 50% of urban streams.”⁷⁵⁷ The reason is obvious:

Carbaryl is registered in the United States for use in controlling more than 160 insect pests on over 115 agricultural and non-crop use applications, including home and garden uses (U.S. EPA 2007a; U.S. EPA 2010a). Major uses include insect control on lawns, home gardens, citrus, fruit, forage and field crops, forests, nuts, ornamentals, rangeland, turf, shade trees, poultry and pets (U.S. EPA 2010a). Agricultural crops with the greatest annual use of carbaryl include apples, pecans, grapes, alfalfa, oranges, and corn. Carbaryl was the third most commonly used conventional pesticide used in homes and gardens in 2005 and 2007 with a range of 4 to 6 million pounds of active ingredient used annually (U.S. EPA 2011).⁷⁵⁸

As described by EPA, carbaryl causes inhibition of the enzyme acetylcholinesterase (“AChE”) at synaptic junctions in the nervous system. AChE breaks down the neurotransmitter acetylcholine but inhibition of AChE results in the accumulation of acetylcholine in the nerve synapses leading to continual firing of nerve pulses throughout the nervous system. This buildup results in uncontrolled movement, paralysis, convulsions, tetany, and possible death as respiratory, circulatory and other vital body systems fail.⁷⁵⁹ The NMFS evaluation concluded:

[C]arbaryl will impair swimming of salmonids, kill salmonid prey, and in certain circumstances kill salmonids when exposed for sufficient durations. The effect concentrations shown in the figure [below] do not account for the potential enhanced toxicity of carbaryl to salmonids or their prey items in aquatic habitats where other AChE inhibitors are present. We also note that pH is a major factor in carbaryl’s persistence in aquatic habitats. At pHs above 8, carbaryl breaks down fairly rapidly (half-life of 24 h) while at pHs less than 8 carbaryl is much more resistant to hydrolysis (half-life of 1- 30 d for pH of 7.9 – 5.7). The pH of

⁷⁵⁶ NMFS, *National Marine Fisheries Service Endangered Species Act Section 7 Consultation Biological Opinion Environmental Protection Agency Registration of Pesticides Containing Carbaryl, Carbofuran, and Methomyl* (April 20, 2009) (hereinafter “2009 Carbaryl BiOp”), available at <https://media.fisheries.noaa.gov/dam-migration/63806531carbamate.pdf>.

⁷⁵⁷ EPA, *Aquatic Life Ambient Water Quality Criteria for Carbaryl – 2012* (April 2012) available at <https://downloads.regulations.gov/EPA-HQ-OW-2011-0787-0006/content.pdf> at vi.

⁷⁵⁸ *Id.*

⁷⁵⁹ *Id.* at 3.

natural surface waters commonly ranges from 7 to 9, thus pH is an important consideration when evaluating toxicity of carbaryl.⁷⁶⁰

NMFS concluded that “[p]rey survival appears to be the most sensitive endpoint,” as demonstrated in the figure below, although “effect concentrations shown in the figure do not account for the potential enhanced toxicity of carbaryl to salmonids or their prey items in aquatic habitats where other AChE inhibitors are present.”⁷⁶¹

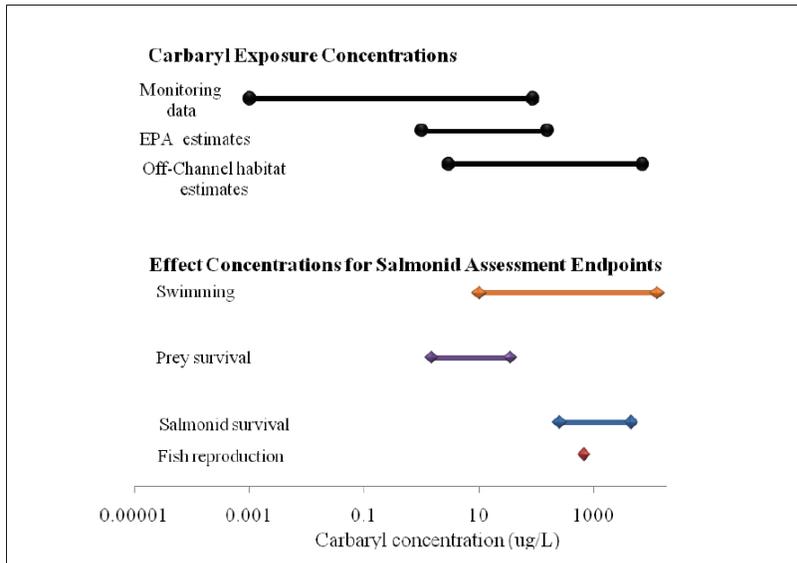


Figure 39. Carbaryl exposure concentrations and salmonid assessment endpoints' effect concentrations in µg/L

Although EPA has issued 304(a) criteria for these four highly toxic, current-use pesticides, none of them is on the Toxic Pollutants Lists and three of them pre-date modern analysis of their toxic effects to aquatic species, particularly threatened and endangered species.

4. Chloride

EPA published 304(a) criteria for chloride in 1988 after increased salinization of

⁷⁶⁰ 2009 NMFS Carbaryl BiOp, *supra* n. 756 at 365.

⁷⁶¹ *Id.*

freshwaters had been measured since around 1900.⁷⁶² Chloride levels continue to rise. For example, the U.S. Geological Survey (“USGS”) found in 2009 that “[c]hloride levels above the recommended federal criteria set to protect aquatic life were found in more than 40 percent of urban streams tested.”⁷⁶³ Chloride derives from salt, urban and agricultural runoff, discharges from municipal wastewater plants, industrial plants, and drilling of oil and gas wells.⁷⁶⁴

In the over 35 years since EPA published its chloride recommendations, scientists have continued to assess its contribution to water quality and species health. There is strong evidence that EPA’s recommended criteria are not protective. For example, researchers in Colorado found that effects on macroinvertebrate communities were observed at concentrations considerably lower than those reported from traditional laboratory toxicity tests, and below the EPA criterion value for chloride of 230 mg/L.⁷⁶⁵ The researchers concluded that “MgCl₂ road deicer has the potential to impair montane stream benthic communities at relatively low ionic concentrations, and regulatory agencies should manage for and establish regionally appropriate application rates for this stressor.”⁷⁶⁶ The USGS states that the effects of salinization “may be vastly different among species and among different life stages.”⁷⁶⁷ For example, chloride tolerance levels for

⁷⁶² EPA, *Ambient Water Quality Criteria for Chloride – 1988* (Feb. 1988) (hereinafter “1988 Chloride Recommended Criteria”) available at <https://www.epa.gov/sites/default/files/2018-08/documents/chloride-aquatic-life-criteria-1988.pdf>.

⁷⁶³ Science Daily, *Chloride Found At Levels That Can Harm Aquatic Life In Urban Streams Of Northern US* (Sept. 17, 2009), available at <https://www.sciencedaily.com/releases/2009/09/090916123513.htm>.

⁷⁶⁴ 1988 Chloride Recommended Criteria, *supra* n. 762 at 1.

⁷⁶⁵ Christopher J. Kotalik *et al.*, *Effects of magnesium chloride road deicer on montane stream benthic communities*, *Hydrobiologia* DOI 10.1007/s10750-017-3212-5 (April 26, 2017), available at https://www.researchgate.net/publication/316490087_Effects_of_magnesium_chloride_road_deicer_on_montane_stream_benthic_communities/link/5ace39a50f7e9b1896580c1e/download

⁷⁶⁶ *Id.*

⁷⁶⁷ USGS, Eastern Ecological Science Center, *Assessing the effects of chloride exposure on aquatic organisms* (March 29, 2019), available at <https://www.usgs.gov/centers/eesc/science/assessing-effects-chloride-exposure-aquatic-organisms>. See also Patricia L. Gillis, *Assessing the toxicity of sodium*

brook trout, *salvelinus fontinalis*, are as low as 3.1 mg/L.⁷⁶⁸

In addition, researchers have concluded that water hardness plays a significant role in the toxicity of chloride to those species. A team using nine freshwater species proposed that the recommended criteria be based on water hardness, ranging from 64 mg/L chloride at 10 mg/L hardness to 388 mg/L chloride at 160 mg/L hardness (as CaCO₃). As they summarized, “current water quality guidelines for chloride may be overly conservative in water with moderate-to-high hardness, and may not be sufficiently protective under soft-water conditions.”⁷⁶⁹

5. Chlorine

EPA’s 304(a) criteria for chlorine date to 1986.⁷⁷⁰ Chlorine is commonly used in ponds and reservoirs to control algae growth, in sewage treatment, to adjust the taste and odor of tap waters, and to treat wood pulp in producing paper products.⁷⁷¹ Toxicity to aquatic species depends on how much chlorine remains after its initial dissipation, and how much free chlorine and chloramines are created.⁷⁷² Additionally, use of chlorine causes chlorination of many organic compounds that are closely allied to compounds in wastewater effluents, resulting in creation of products entirely different from the original material and facilitates a chlorine

chloride to the glochidia of freshwater mussels: Implications for salinization of surface waters 159 Environmental Pollution, 1702-1708 (June 2011), available at <https://www.sciencedirect.com/science/article/abs/pii/S0269749111001060>.

⁷⁶⁸ Michael Meador *et al.*, *Quantifying tolerance indicator values for common stream fish species of the United States*, 7 Ecological Indicators 329, 334 (2007) table 2, available at <https://www.sciencedirect.com/science/article/abs/pii/S1470160X06000227?via%3Dihub#!>.

⁷⁶⁹ James Elphick, *et al.*, *Chronic Toxicity of Chloride to Freshwater Species: Effects of Hardness and Implications for Water Quality Guidelines* 30 Environmental Toxicology and Chemistry 239-246 (2011), available at <https://setac.onlinelibrary.wiley.com/doi/10.1002/etc.365>.

⁷⁷⁰ 1986 Recommended Criteria, *supra* n. 709.

⁷⁷¹ William Brungs, *Effects of Residual Chlorine on Aquatic Life*, 45(10) Journal (Water Pollution Control Federation) 2180-2193 (Oct. 1973) available at <https://www.jstor.org/stable/pdf/25038016.pdf> at 2.

⁷⁷² *Id.* at 3.

reaction that is toxic to aquatic species.⁷⁷³ The effect of chlorine on aquatic life is also dependent on another toxic pollutant—ammonia—that can affect the ratio of free chlorine and chloramines in water, as well as pH, temperature, and the period of time in which the reaction of chlorine and other substances takes place.⁷⁷⁴

Since 1986, new studies have looked at the effect of chlorine on aquatic life, primarily byproducts of chlorination and additive effects of chlorine and other pollutants. For example, elevated temperatures can make sublethal chlorine concentrations lethal to Great Lakes fishes:

When chlorination is used at temperatures near the thermal maxima, but not sufficiently high to exclude fish, high mortality rates can be expected. Most of the fish that lose equilibrium during exposure do not survive. Fish exposed to sublethal levels of chlorine become lethargic and often gulp air and frequently suffer increased predation pressures from birds and other fish. Additionally, hematological and biochemical disturbances, and potentially irreversible gill damage, may impair the lifetime fitness of fish exposed to chlorine.⁷⁷⁵

Chlorination of treated sewage, which contains high concentrations of organic carbon and ammonia, requires sufficiently high chlorine dosage to effect its disinfection purpose. While a 1992 study concluded that “[o]rganochlorinated by-products represent a minor part of the added compound,” the authors urged “further investigations to precisely evaluate their potential hazard to marine life because of their persistence and mutagenic character.”⁷⁷⁶ The authors demonstrated that “[f]or chlorine produced oxidants first signs of deleterious effects appear at concentrations as low as $0.1 \text{ mg} \cdot \text{l}^{-1}$, about two orders of magnitude lower than the actual

⁷⁷³ *Id.*

⁷⁷⁴ *Id.*

⁷⁷⁵ S.J. Cooke and J.F. Schreer, *Additive Effects of Chlorinated Biocides and Water Temperature on Fish in Thermal Effluents with Emphasis on the Great Lakes*, 9(2) *Reviews in Fisheries Science* 69-113 (June 24, 2010), available at <https://www.tandfonline.com/doi/abs/10.1080/20016491101717>.

⁷⁷⁶ A. Abarnou and L. Miossec, *Chlorinated waters discharged to the marine environment chemistry and environmental impact. An overview*, 126 *Science of the Total Environment* 173–197 (Sept. 1992), available at <https://www.sciencedirect.com/science/article/abs/pii/004896979290490J>.

concentrations in chlorinated effluents.”⁷⁷⁷ Similarly, a 2013 study looked at the formation of trihalomethanes (“THMs”) from chlorination of treated sewage, concluding that “THMs formation may be the direct reason for the increase of toxicity in the secondary effluent when chlorine was used as disinfectant.”⁷⁷⁸

6. Demeton

Demeton, commonly called Systox, is a systemic organophosphate insecticide.⁷⁷⁹ EPA published its 304(a) criteria for protection of aquatic life from demeton in 1986.⁷⁸⁰ EPA noted high toxicity values in carp, goldfish, flathead minnow, channel catfish, guppy, rainbow trout, and bluegill.⁷⁸¹ EPA also noted acute toxicity to invertebrates, with smaller crustaceans showing particular susceptibility.⁷⁸² Overall, EPA found that demeton is highly toxic to fish, and very highly toxic to freshwater invertebrates.⁷⁸³

According to the EPA Pesticide Fact Sheet, issued on February 27, 1985, demeton application sites at the time included vegetable crops, orchards, and ornamentals, including greenhouse.⁷⁸⁴ EPA determined that demeton is readily absorbed by plants, though according to the agency no data was available at the time to assess the environmental effects of demeton, nor its potential for contamination of groundwater.⁷⁸⁵

Due to the dangers it poses to both aquatic life and human health, all registrations of

⁷⁷⁷ *Id.*

⁷⁷⁸ Maoni N. Wu, *et al.*, *Characteristics of THMFP increase in secondary effluent and its potential toxicity*, 261 *Journal of Hazardous Materials* 325–331 (Oct. 15, 2013), *available at* <https://www.sciencedirect.com/science/article/abs/pii/S0304389413005013>.

⁷⁷⁹ EPA, *Pesticide Fact Sheet: Demeton* (Feb. 27, 1985) (hereinafter “Demeton Fact Sheet”).

⁷⁸⁰ 1986 Recommended Criteria, *supra* n. 709 at 115.

⁷⁸¹ *Id.*

⁷⁸² *Id.* at 116.

⁷⁸³ Demeton Fact Sheet, *supra* n. 779 at 2.

⁷⁸⁴ *Id.* at 1.

⁷⁸⁵ *Id.* at 2.

demeton have been cancelled.⁷⁸⁶ While the USGS found no use of demeton since 2005, scientists have pointed out that even non-persistent organic phosphate insecticides, such as demeton, do not disappear when released into the environment.⁷⁸⁷ In discussing the release of demeton into the environment, research microbiologist and professor of environmental toxicology Wendall W. Kilgore noted that in the process, “several other chemicals are formed – one is 100 times more toxic and another is 1,000 times more toxic than demeton itself.”⁷⁸⁸ There is no indication that EPA has evaluated these other chemicals.

7. Guthion

Guthion, commonly referred to as Azinphos-methyl or AZM, is an organophosphate insecticide⁷⁸⁹ for which EPA published 304(a) aquatic life criteria in 1986.⁷⁹⁰ The agency noted decreased spawning in flathead minnows, as well as fish mortality caused by repetitive exposure.⁷⁹¹ Additionally, EPA noted that after being exposed to guthion, it took several weeks for the enzyme acetylcholinesterase, which is essential to nerve function, to return to normal, despite exposure being discontinued.⁷⁹²

⁷⁸⁶ See EPA, *Search by Product*, available at

https://ordspub.epa.gov/ords/pesticides/f?p=113:5:::RP:PPLS_STATUS:9 (search on Systox).

⁷⁸⁷ USGS, Pesticide National Synthesis Project, *Estimated Annual Agricultural Pesticide Use* (hereinafter “USGS Pesticide Map”), available at

https://water.usgs.gov/nawqa/pnsp/usage/maps/show_map.php?year=2005&map=DEMETON&hilo=L;

Harrison C. Dunning, *Pests, Poisons, and the Living Law: The Control of Pesticides in California's*

Imperial Valley, 2 Ecology L. Q. 633, 679 (1972), [https://heinonline-](https://heinonline-org.library.lcproxy.org/HOL/Page?handle=hein.journals/eclawq2&id=651&collection=journals&index=)

[org.library.lcproxy.org/HOL/Page?handle=hein.journals/eclawq2&id=651&collection=journals&index=.](https://heinonline-org.library.lcproxy.org/HOL/Page?handle=hein.journals/eclawq2&id=651&collection=journals&index=)

⁷⁸⁸ *Id.* at 679.

⁷⁸⁹ EPA, *Pesticide Fact Sheet: Azinphos-methyl (Guthion)* (Sept. 30, 1986) (hereinafter “Guthion Fact Sheet”).

⁷⁹⁰ 1986 Recommended Criteria, *supra* n. 709 at 141.

⁷⁹¹ *Id.* at 141-42.

⁷⁹² *Id.* at 142.

According to EPA, guthion application sites included fruit and field crops, vegetables, tobacco, ornamentals, and forest trees.⁷⁹³ Guthion has a high potential to reach surface water after these applications through both spray drift and runoff.⁷⁹⁴ Due in large part to its toxicity to humans, guthion is no longer registered for use.⁷⁹⁵ According to the USGS, guthion has not been used since 2015 but was heavily used across the country prior to that date.⁷⁹⁶ Notably, available data in 1986 indicated that guthion exhibited low soil mobility and poor solubility in water, meaning it is more likely to stick to soil in the environment and does not easily dissolve in water.⁷⁹⁷

8. Iron

EPA's iron criteria were last updated in 1986.⁷⁹⁸ Iron is, by weight, the fourth most plentiful element comprising earth's crust,⁷⁹⁹ resulting in the release of iron to waterbodies where soil is disturbed, such as coal and hard rock mining,⁸⁰⁰ logging, and farming.⁸⁰¹

⁷⁹³ Guthion Fact Sheet, *supra* n. 789 at 1.

⁷⁹⁴ Science Direct, *Azinphos-Methyl*, available at <https://www.sciencedirect.com/topics/agricultural-and-biological-sciences/azinphos-methyl>.

⁷⁹⁵ Azinphos-methyl; Product Cancellation Order and Amendments to Terminate Uses, 73 Fed. Reg. 9328 (Feb. 20, 2008) (as amended. Azinphos-methyl; Product Cancellation Order and Amendments to Terminate Uses; Correction, 73 Fed. Reg. 16006 (March 28, 2008)).

⁷⁹⁶ USGS Pesticide Map, *supra* n. 787, available at https://water.usgs.gov/nawqa/pnsp/usage/maps/show_map.php?year=2015&map=AZINPHOSMETHYL&hilo=L.

⁷⁹⁷ Guthion Fact Sheet, *supra* n. 789 at 3; National Library of Medicine, PubChem, compound Summary, *Azinphos-methyl*, available at <https://pubchem.ncbi.nlm.nih.gov/compound/Azinphos-methyl>.

⁷⁹⁸ 1986 Recommended Criteria, *supra* n. 709.

⁷⁹⁹ *Id.* at 161.

⁸⁰⁰ Pete Cadmus, *et al.*, *Chronic Toxicity of Ferric Iron for North American Aquatic Organisms: Derivation of a Chronic Water Quality Criterion Using Single Species and Mesocosm Data* (hereinafter "Chronic Toxicity of Iron") 74 *Archives of Environmental Contamination and Toxicology* 605–615 (Jan. 22, 2018) available at <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5893738/> at 1.

⁸⁰¹ See Kari-Matti Vuori, *Direct and Indirect Effects of Iron on River Ecosystems*, 32 *An.. Zool. Fennici* 317-329 (Nov. 1995) (hereinafter "Effects of Iron") at 325–326, available at https://www.researchgate.net/publication/241686376_Direct_and_Indirect_effects_of_iron_on_river_eco_systems.

According to EPA, ferrous (or bivalent) and ferric (or trivalent) irons can be harmful to aquatic environments, particularly at high concentrations.⁸⁰² The ferrous form of iron persists in waters that do not contain dissolved oxygen, while the ferric form of iron is insoluble and fails to dissolve in water.⁸⁰³ Iron can cause physical stress to organisms; affect the “fate, bioavailability and toxic effects of trace metals and organic toxicants”; have “deleterious effects on aquatic invertebrates” along with non-toxic effects on the availability of nutrients and light.⁸⁰⁴ Although EPA has not updated its analysis of iron since 1986, studies done as far back as the 1990s have shown that iron has toxic effects on aquatic species.⁸⁰⁵ When accumulated, iron can interfere with aquatic species’ ability to properly feed and develop.⁸⁰⁶ Recent studies show that EPA’s analysis of the appropriate iron criterion is based on too small a sampling and underestimates the toxicity of iron to aquatic life, recommending that “modernization of water quality criteria [for iron] should include data generated from mesocosm experiments and other lines of evidence” in order to “reduce[] the risk of calculating an underprotective [final chronic value] FCV for total Fe.”⁸⁰⁷ British Columbia adopted criteria for iron in 2008, demonstrating its continuing concern about its effects on aquatic life.⁸⁰⁸

9. Mirex

⁸⁰² 1986 Recommended Criteria, *supra* n. 709 at 161.

⁸⁰³ *Id.*

⁸⁰⁴ Effects of Iron, *supra* n. 801 at 321–325.

⁸⁰⁵ *See, e.g., id.*

⁸⁰⁶ Chronic Toxicity of Iron, *supra* n. 800 at 7.

⁸⁰⁷ *Id.* at 1, 610–613.

⁸⁰⁸ *See* Ministry of the Environment, Province of British Columbia, *Ambient Aquatic Life Guidelines for Iron Overview Report* (March 2008), available at <https://www2.gov.bc.ca/assets/gov/environment/air-land-water/water/waterquality/water-quality-guidelines/approved-wqgs/iron-or.pdf>.

While not produced in the United States since 1977, mirex was used to control fire ants, and as flame retardant for plastics, rubber, paint, paper, and electronics.⁸⁰⁹ EPA has not updated the water quality criteria for mirex since 1986.⁸¹⁰ Mirex has been noted in at least 9 of 1,867 hazardous waste sites that have been proposed for inclusion on EPA's National Priorities List.⁸¹¹ Most mirex releases were to surface waters of the United States, and high levels continue to be reported in especially high numbers in the Great Lakes region, but have also been more dramatically detected in groundwater in New Jersey and South Carolina.⁸¹²

In a study done on crayfish in the 1980s, mortality rates were extraordinarily high after exposure to mirex, indicating an extreme toxicity of mirex in crayfish.⁸¹³ In other species, mirex caused gill and kidney breakdown in goldfish, and stunted growth in bluegills.⁸¹⁴ Male and female reproductive systems appear to be harmed by mirex, and prenatal and postnatal development may be inhibited also.⁸¹⁵ EPA identified mirex as a known cancer-causing toxin to animals.⁸¹⁶ It has also been shown to negatively affect the endocrine system.⁸¹⁷

In laboratory and field tests, mirex accumulated through the food chain and in estuaries after traveling from treated lands to untreated areas and estuary animals.⁸¹⁸ Accumulation of

⁸⁰⁹ 1986 Recommended Criteria, *supra* n. 709 at 186; *see also* EPA, Mirex, Nease Chemical Superfund Site, Salem, Ohio (Nov. 1992) (hereinafter "Nease Superfund Site"), available at <https://semspub.epa.gov/work/05/238521.pdf> at 1.

⁸¹⁰ 1986 Recommended Criteria, *supra* n. 709 at 186.

⁸¹¹ ATSDR, *Mirex and Chlordecone, Chapter 5. Potential for Human Exposure* (hereinafter "ATSDR Mirex") available at <https://www.atsdr.cdc.gov/ToxProfiles/tp66-c5.pdf> at 147.

⁸¹² *Id.* at 2-3.

⁸¹³ 1986 Recommended Criteria, *supra* n. 709 at 186 (ingestion of one mirex particle killed crayfish).

⁸¹⁴ *Id.*

⁸¹⁵ ATSDR Mirex, *supra* n. 811 at 15.

⁸¹⁶ Nease Superfund Site, *supra* n. 809 at 3.

⁸¹⁷ ATSDR Mirex, *supra* n. 811 at 15.

⁸¹⁸ 1986 Recommended Criteria, *supra* n. 709 at 186, 189.

mirex was particularly high in predators due to its ability to bioaccumulate and biomagnify.⁸¹⁹ For example, Mirex found in high levels in fish, game, and waterfowl have triggered fish consumption advisories in New York, Pennsylvania, and Ohio.⁸²⁰ Individuals exposed to mirex in the environment have higher levels of the chemical and women in those areas have passed detectable levels of mirex to their infants.⁸²¹ EPA noted that, “considering the extreme toxicity and potential for bioaccumulation, every effort should be made to keep mirex bait particles out of water containing aquatic organisms.”⁸²²

Mirex appears to have longevity in the environment due to its tendency to attach to soil and sediment, and is detected in food, animals, aquatic organisms, water, sediment, soil, and air.⁸²³ Due to its ability to move easily through sediments in water, mirex is most commonly still found in water bodies.⁸²⁴

10. Methoxychlor

EPA recommended criteria for methoxychlor was last updated in 1986.⁸²⁵ Methoxychlor is very persistent when released into soil or water, often attaching to soil, and leaching into

⁸¹⁹ *Id.* at 148, 186.

⁸²⁰ *Id.* at 149.

⁸²¹ *Id.* at 150

⁸²² *Id.* at 190.

⁸²³ Centers for Disease Control and Prevention, National Biomonitoring Program, Biomonitoring Summary, *Organichlorine Pesticides Overview, Mirex*, available at https://www.cdc.gov/biomonitoring/Mirex_BiomonitoringSummary.html.

⁸²⁴ Nease Superfund Site, *supra* n. 809 at 1.

⁸²⁵ 1986 Recommended Criteria, *supra* n. 709 at 182.

groundwater.⁸²⁶ Banned by EPA in 2000, methoxychlor remains in the environment.⁸²⁷

At relatively low levels, methoxychlor may cause reduced hatchability of fathead minnow embryos, and at higher levels, may prevent spawning.⁸²⁸ Yellow perch shows reduced growth when exposed to moderate levels of methoxychlor for several months.⁸²⁹ Presence of methoxychlor may also increase transgenerational diseases.⁸³⁰ Some bioaccumulation has occurred in aquatic species,⁸³¹ with bioaccumulation more common in shellfish than fish.⁸³²

Methoxychlor has been found in human adipose tissues, umbilical cord blood, and human breast milk.⁸³³ Increased quantities of the insecticide have been noted in studies on elephant seals.⁸³⁴ The U.S. Fish and Wildlife Service “has determined that the ‘use of methoxychlor as a mosquito larvicide may jeopardize the continued existence of certain endangered species.’”⁸³⁵

11. Nonylphenol

Nonylphenol was considered a sufficient threat to water quality that EPA developed recommended criteria for some compounds of the pollutant in 2005. Presumably among its reasons was that, according to the agency, “nonylphenol is produced in large quantities in the

⁸²⁶ Persistent Organic Pollutants Review Committee, *Methoxychlor Draft Risk Profile* (Feb. 2020) (hereinafter “Methoxychlor Risk Profile”) available at [⁸²⁷ EPA, *Methoxychlor*, available at <https://www.epa.gov/sites/default/files/2016-09/documents/methoxychlor.pdf> at 1.](https://echa.europa.eu/documents/10162/b65a738e-b50f-64e5-cbb0-f2711d49c25e#:~:text=After%201992%2C%20production%20of%20methoxychlor,(US%20EPA%2C%202004) at 2.</p></div><div data-bbox=)

⁸²⁸ 1986 Recommended Criteria, *supra* n. 709 at 182.

⁸²⁹ *Id.*

⁸³⁰ Methoxychlor Risk Profile, *supra* n. 826 at 2.

⁸³¹ 1986 Recommended Criteria, *supra* n. 709 at 183.

⁸³² EPA, *Consumer Factsheet on: METHOXYCHLOR*, available at <https://nepis.epa.gov/Exe/ZyPDF.cgi/P1013QEV.PDF?Dockey=P1013QEV.PDF> at 2.

⁸³³ Methoxychlor Risk Profile, *supra* n. 826 at 2.

⁸³⁴ *Id.*

⁸³⁵ Beyond Pesticides, Chemical Watch Factsheet, *Methoxychlor*, available at <https://www.beyondpesticides.org/assets/media/documents/pesticides/factsheets/Methoxychlor.pdf>

United States.”⁸³⁶ It is discharged to waters from sewage treatment plants, airports, and industries.⁸³⁷ “A reconnaissance of 95 organic wastewater contaminants in 139 U.S. streams conducted in 1999-2000 revealed that nonylphenol was one of the most commonly occurring contaminants and was measured at higher concentrations than most of the other contaminants[.]”⁸³⁸

In 2010, pursuant to TSCA, EPA reiterated its concern about nonylphenols:

Nonylphenol (NP) and nonylphenol ethoxylates (NPEs) are produced in large volumes, with uses that lead to widespread release to the aquatic environment. NP is persistent in the aquatic environment, moderately bioaccumulative, and extremely toxic to aquatic organisms.

* * *

NP and certain oligomeric NPEs are highly toxic to aquatic organisms, are moderately bioaccumulative in mollusks, are persistent in the aquatic environment, and accumulate in soils and sediments (EPA, 2005).⁸³⁹

While EPA’s recommended criteria do not directly address nonylphenol’s estrogenic effects on aquatic organisms, the agency reports they are significant and have been addressed in the criteria.⁸⁴⁰ It also asserts that to the extent that it is incorrect, it has “activities underway” to

⁸³⁶ 2005 Nonylphenol Criteria, *supra* n. 8 at 1.

⁸³⁷ *Id.* at 2.

⁸³⁸ *Id.* at 3.

⁸³⁹ EPA, *Nonylphenol (NP) and Nonylphenol Ethoxylates (NPEs) Action Plan* (Aug. 18, 2010) at 1.

⁸⁴⁰ EPA claims that it has taken estrogenic effects into account: “Whole organism endpoints such as reproductive and growth effects are used to derive aquatic life ambient water quality criteria for nonylphenol. To the extent that such endpoints reflect the integration of molecular, biochemical and tissue-level effects at the whole organism level, the nonylphenol criteria address the estrogenicity of nonylphenol. For example, while vitellogenin is a commonly used biomarker indicative of exposure to estrogenic compounds, measurement of this molecular/biochemical endpoint alone does not necessarily indicate adverse effect on population relevant endpoints such as survival, growth and reproduction. However, several studies have demonstrated that vitellogenin induction can be accompanied by decreased fecundity (egg production) of breeding pairs of fathead minnows exposed chronically to estrogenic compounds (Ankley et al.). The chronic toxicity studies used in deriving the nonylphenol criteria (Table 6) included assessment of effects on growth and reproduction endpoints in aquatic organisms. Hence, to the extent that these endpoints are the result of effects on the endocrine system (although this was not definitively demonstrated in any of the tests by use of a concomittant measure of an estrogen-receptor specific endpoint), the estrogenic effects of nonylphenol have been considered in deriving the aquatic life ambient water quality criteria for nonylphenol.” *Id.* at 7–8.

do so.⁸⁴¹ Vitellogenin is a protein produced in the liver of female oviparous species and deposited in the ovaries as the primary material for yolk in the ova. Male fish normally produce very little vitellogenin. EPA cites a number of studies that demonstrate significant increases in vitellogenin production in rainbow trout and other fish exposed to nonylphenol as well as a study that found “concentrations of > 0.3 to 0.4 µg/L did appear to reduce fecundity.”⁸⁴² Effects on egg hatchability and the development of ovo-testis tissue was also reported.⁸⁴³ Finally, EPA concluded that “the ability of nonylphenol to induce estrogenic effects has seldom been reported at concentrations below the freshwater Final Chronic Value of 6.5965 µg/L.”⁸⁴⁴ However, the criteria document cites the multi-generational effects of nonylphenol on fish found by Schwaiger *et al.* (2002):

The present findings indicate that [nonylphenol], in an environmentally relevant concentration range, acts as a weak estrogen in directly exposed adult male rainbow trout as indicated by elevated plasma vitellogenin levels. Reproduction success was reduced as indicated by decreased hatching rates. Hormonal imbalances detected in the offspring of exposed fish indicate a transgenerational effect mediated by the endocrine system.⁸⁴⁵

⁸⁴¹ *See id.* at 7–8 (“EPA has activities underway to develop scientific methods for considering endocrine effects, such as the estrogenicity of nonylphenol, in Agency risk assessments. Under the Federal Food, Drug and Cosmetic Act (FFDCA), as amended by the Food Quality Protection Act (FQPA), EPA is required to develop a screening program to determine whether certain substances (including all pesticide active and other ingredients) “may have an effect in humans that is similar to an effect produced by a naturally-occurring estrogen, or other such endocrine effects as the Administrator may designate”. Following the recommendations of its Endocrine Disruptor Screening and Testing Advisory Committee (EDSTAC), EPA determined that there was scientific basis for including, as part of the program, the androgen and thyroid hormone systems, in addition to the estrogen hormone system. EPA also adopted EDSTAC’s recommendation that the Program include evaluations of potential effects in aquatic life and wildlife. When the appropriate screening and or testing protocols being considered under the Agency’s Endocrine Disruptor Screening Program have been developed, nonylphenol may be subjected to additional screening and or testing to better characterize effects related to endocrine systems.”).

⁸⁴² *Id.* at 26-27.

⁸⁴³ *Id.* at 28-29.

⁸⁴⁴ *Id.* at 29.

⁸⁴⁵ J. Schwaiger, *et al.*, *How estrogenic is nonylphenol? A transgenerational study using rainbow trout (Oncorhynchus mykiss) as a test organism*, *Aquat Toxicol.* (2002) 59(3-4):177-89, available at: <https://pubmed.ncbi.nlm.nih.gov/12127735/>.

A 2022 study of toxic contaminants in Puget Sound observed that EPA’s “chronic water quality criterion (WQC) for nonylphenol in marine systems is 1.7 µg/L (USEPA 2005b), which was the mean value of the observed effluent concentration in this study,” concluding that “[n]onylphenols should be considered in any risk assessment of WWTP effluent.”⁸⁴⁶ In Washington State, they are not because the state does not have numeric criteria for nonylphenol. Writing about how these compounds may affect marine mammals, the authors stated that “we do know that low water concentrations can result in high tissue concentrations because these compounds are very hydrophobic” and “[g]iven the extremely low effect concentrations for estrogen hormones (sub ng/L); concentrations of nonylphenols in the low ppb range should be considered for additional analysis to determine potential effects on wildlife.”⁸⁴⁷ This study was followed shortly by the 2023 paper identifying 4-nonylphenol as predominating northeast Pacific killer whales’ toxic burden and identifying it as having the highest transfer rates of toxics from mothers to fetuses, as high as 95 percent.⁸⁴⁸

12. Parathion

Parathion, also referred to as ethyl parathion, is an organophosphate pesticide.⁸⁴⁹ EPA established CWA section 304(a) criteria for parathion in 1986 for aquatic life.⁸⁵⁰ EPA noted that brown bullheads exposed to high concentrations of parathion exhibited tremors, convulsions, deformities, and hemorrhages.⁸⁵¹ Additionally, reproductive impairment and deformities were observed in flathead minnows.⁸⁵² At the time, EPA acknowledged that limited information was

⁸⁴⁶ King County CECs, *supra* n. 329, at 123.

⁸⁴⁷ *Id.* at 129.

⁸⁴⁸ CECs and Killer Whales, *supra* n. 6.

⁸⁴⁹ 1986 Recommended Criteria, *supra* n. 709 at 226.

⁸⁵⁰ *Id.*

⁸⁵¹ *Id.*

⁸⁵² *Id.*

available concerning the persistence of parathion in water, though two scientists cited by EPA suggested that the breakdown of parathion could result in compounds more toxic than parathion itself.⁸⁵³

According to EPA, parathion is among the most highly toxic chemicals that has been registered with the agency.⁸⁵⁴ It is classified as a possible human carcinogen, and is extremely toxic to humans by all routes of exposure.⁸⁵⁵ Parathion is formulated as a liquid and applied only using aerial equipment on various food crops.⁸⁵⁶ EPA published a product cancellation order for parathion in 2010, stating existing stocks of parathion products could be used until depleted or until December 2013 without causing unreasonable risk to human health and the environment.⁸⁵⁷ Despite this, the USGS estimates that the last use of parathion was in 2016.⁸⁵⁸

13. Tributyltin

Tributyltin was partially banned for use by Congress in 1988 and banned for use as an antifouling paint by EPA in 2005 because it is extremely toxic to marine life, causing shell deformation, reproductive aberrations, endocrine disruption, and bio-accumulation in predator species including marine mammals.⁸⁵⁹ In developing recommended criteria for tributyltin, EPA

⁸⁵³ *Id.* at 229.

⁸⁵⁴ EPA R.E.D. Facts, *Ethyl Parathion, Pesticide Registration* (Sept. 2000) available at https://www3.epa.gov/pesticides/chem_search/reg_actions/reregistration/fs_PC-057501_1-Sep-00.pdf.

⁸⁵⁵ *Id.*

⁸⁵⁶ *Id.*

⁸⁵⁷ Methyl Parathion; Cancellation Order for Certain Pesticide Registrations, 75 Fed. Reg. 41482 (July 16, 2010).

⁸⁵⁸ USGS, National Water-Quality Assessment, *Parathion*, available at https://water.usgs.gov/nawqa/pnsp/usage/maps/show_map.php?year=2005&map=PARATHION&hilo=L&disp=Parathion.

⁸⁵⁹ See Sea Grant Law Center, *Restrictions on the Use of Marine Antifouling Paints Containing Tributyltin and Copper* (Oct. 2004, updated Aug. 2005), available at <http://nsglc.olemiss.edu/Advisory/Antifouling.pdf> at 3 (describes EPA as having negotiated the voluntary cancellation of pesticide registrations with coating vendors. See *id.*, n. 5. The Organotin Antifouling

stated that “problem in the aquatic environment because it is extremely toxic to non-target organisms, is linked to imposex and immuno-suppression in snails and bivalves, and can be persistent.”⁸⁶⁰ EPA also noted that tributyltin’s “principal use . . . is as a stabilizer in the manufacturing of plastic products, for example, as an anti-yellowing agent in clear plastics and as a catalyst in poly(vinyl chloride) products (Piver 1973). Another and less extensive use of organotins is as a biocide (fungicide, bactericide, insecticide) and as a preservative for wood, textiles, paper, leather and electrical equipment.”⁸⁶¹ See sub-section VI.E.5 *infra* for further discussion on the hazards of TBT and its family of organotins.

B. Toxic Pollutants for Which EPA is Currently Developing Section 304(a) Recommended Criteria but Are Not on the Toxic Pollutants Lists: PFAS and PFOA

In addition to the toxic pollutants not on the Toxic Pollutants Lists for which EPA has already developed recommended criteria, there are two for which EPA has recently proposed Section 304(a) criteria: perfluorooctane sulfonate (“PFOS”)⁸⁶² and perfluorooctanoic acid (“PFOA”),⁸⁶³ both of which belong to the per- and poly-fluoroalkyl substances (“PFAS”) group of chemicals. PFAS are widely used human-made chemicals that—because EPA has not regulated them—are now ubiquitous in the environment, including in drinking water and fish.⁸⁶⁴

Paint Control Act also directed EPA to issue final water quality standards for organotin compounds by March 30, 1989. *Id.*)

⁸⁶⁰ EPA, *Ambient Aquatic Life Water Quality Criteria for Tributyltin (TBT) – Final* (Dec. 2003), available at <https://www.epa.gov/sites/default/files/2019-02/documents/ambient-wqc-tributyltin-final.pdf> at 3.

⁸⁶¹ *Id.* at 1.

⁸⁶² EPA, Water Quality Criteria, *Aquatic Life Criteria - Perfluorooctane Sulfonate (PFOS)*, available at <https://www.epa.gov/wqc/aquatic-life-criteria-perfluorooctane-sulfonate-pfos>.

⁸⁶³ EPA, Water Quality Criteria, *Aquatic Life Criteria - Perfluorooctanoic Acid*, available at <https://www.epa.gov/wqc/aquatic-life-criteria-perfluorooctanoic-acid-pfoa>.

⁸⁶⁴ EPA, *Our Current Understanding of PFAS*, <https://www.epa.gov/pfas/our-current-understanding-human-health-and-environmental-risks-pfas> (hereinafter “PFAS Current Understanding”).

PFAS have historically been used to make a wide range of customer and industrial products.⁸⁶⁵ PFAS manufacturing and processing facilities, manufacturing facilities, airports, and military installations are just a few of the sources of PFAS to water, soil, and air.⁸⁶⁶ There are thousands of PFAS, with PFOA and PFOS being two of the most widely used and studied.⁸⁶⁷ PFAS share the common feature of being very slow to break down and persisting in people, animals, and the environment for long periods of time.⁸⁶⁸ PFAS are resistant to biodegradation, direct photolysis, atmospheric photooxidation, and hydrolysis.⁸⁶⁹

As of June 2022, 2,858 locations in 50 states and two territories were known to be contaminated with PFAS chemicals.⁸⁷⁰ A mapping tool shows known locations of PFAS in public water systems (blue dots), on or near military bases (purple dots), and other known sites such as airports, industrial plants and dumps, and firefighter training sites (red dots).⁸⁷¹ These sources result in both point and nonpoint PFAS pollution in the nation's waters.

⁸⁶⁵ ATSDR, *What are PFAS?* <https://www.atsdr.cdc.gov/pfas/health-effects/overview.html>.

⁸⁶⁶ EPA, *Research on Per- and Polyfluoroalkyl Substances (PFAS)*, available at <https://www.epa.gov/chemical-research/research-and-polyfluoroalkyl-substances-pfas>.

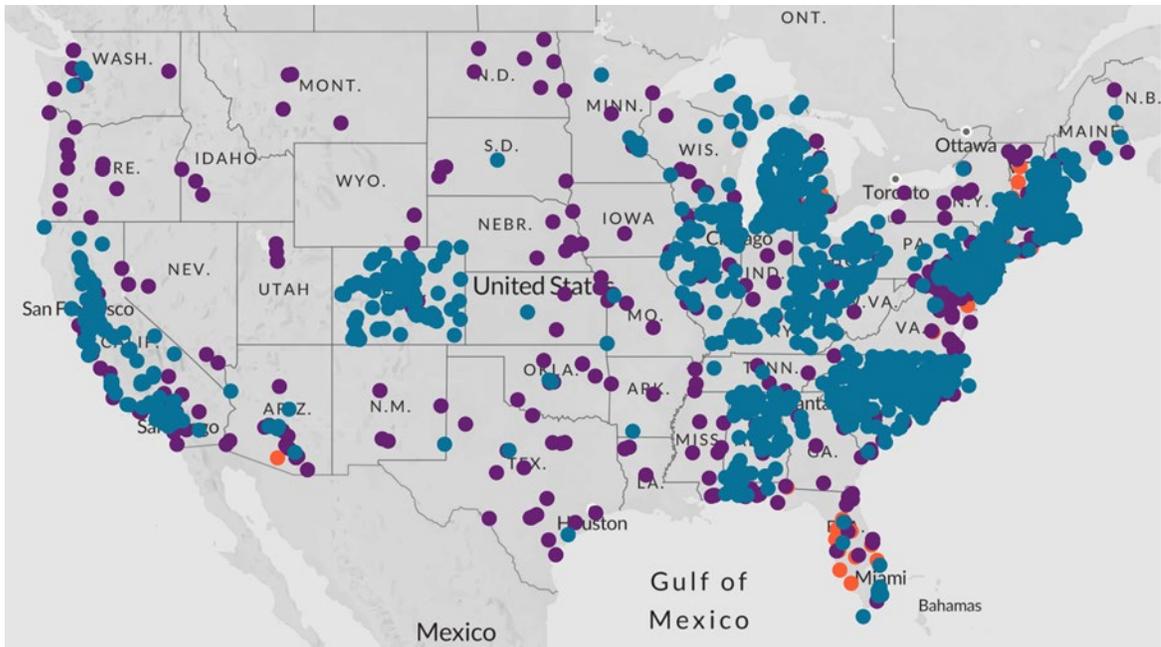
⁸⁶⁷ PFAS Current Understanding, *supra* n. 864.

⁸⁶⁸ EPA, *PFAS Explained*, available at <https://www.epa.gov/pfas/pfas-explained>.

⁸⁶⁹ ATSDR, *ToxGuide for Perfluoroalkyls* (March 2020), <https://www.atsdr.cdc.gov/toxguides/toxguide-200.pdf> at 2 (hereinafter "ATSDR PFAS ToxGuide").

⁸⁷⁰ Environmental Working Group/ Northeastern University Social Science Environmental Health Research Institute, *PFAS Contamination in the U.S. (June 8, 2022)*, available at https://www.ewg.org/interactive-maps/pfas_contamination/.

⁸⁷¹ Environmental Working Group, *Mapping the PFAS Contamination Crisis* (April 23, 2020), available at <https://www.ewg.org/news-insights/news/mapping-pfas-contamination-crisis>.



In fact, PFAS are ubiquitous in the nation’s surface waters. In a recent reconnaissance of 114 waterways across the country, at least one type of PFAS was found in 83 percent of the waters as shown below.⁸⁷²

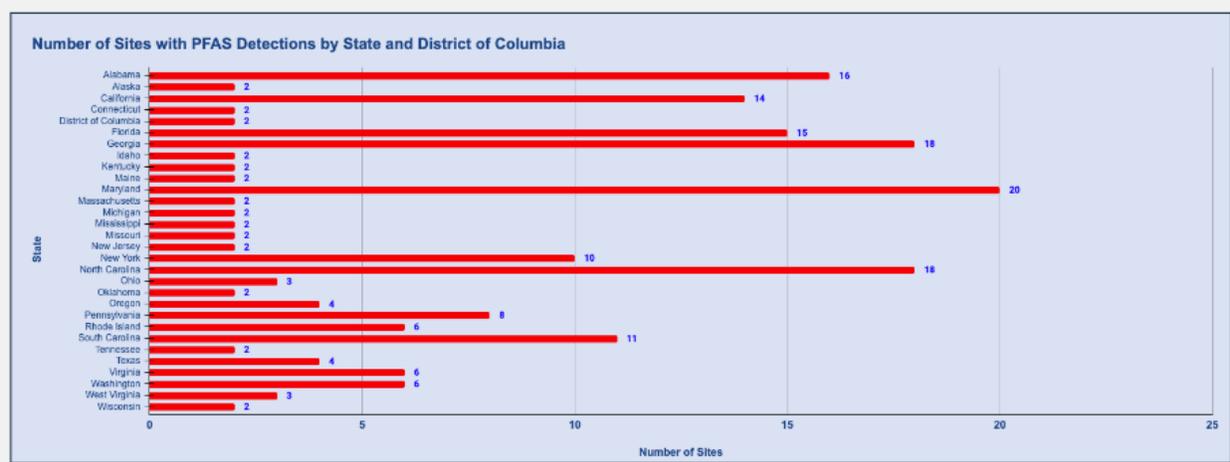


FIGURE 3
Number of Water Sample Sites With PFAS Detections by States and D.C.

⁸⁷² Waterkeeper Alliance, *Invisible Unbreakable Unnatural: PFAS Contamination of U.S. Surface Waters* (Oct. 2022) at 21, available at <https://waterkeeper.org/wp-content/uploads/2022/10/Waterkeeper-Alliance-PFAS-Report-FINAL-10.14.22.pdf>.

Several waterbodies—creeks connected to the Potomac River in Maryland, the Lower Susquehanna River in Pennsylvania, and the Niagara River in New York—had levels of PFAS contamination thousands to hundreds of thousands times higher than what experts say is safe for drinking water.⁸⁷³ (An estimated 65 percent of Americans source their drinking water from surface waters similar to those sampled.⁸⁷⁴) While PFOS and PFOA, the subjects of EPA’s proposed recommended criteria, were the most widely detected, other PFAS detections were also high, as illustrated by the following graph:⁸⁷⁵

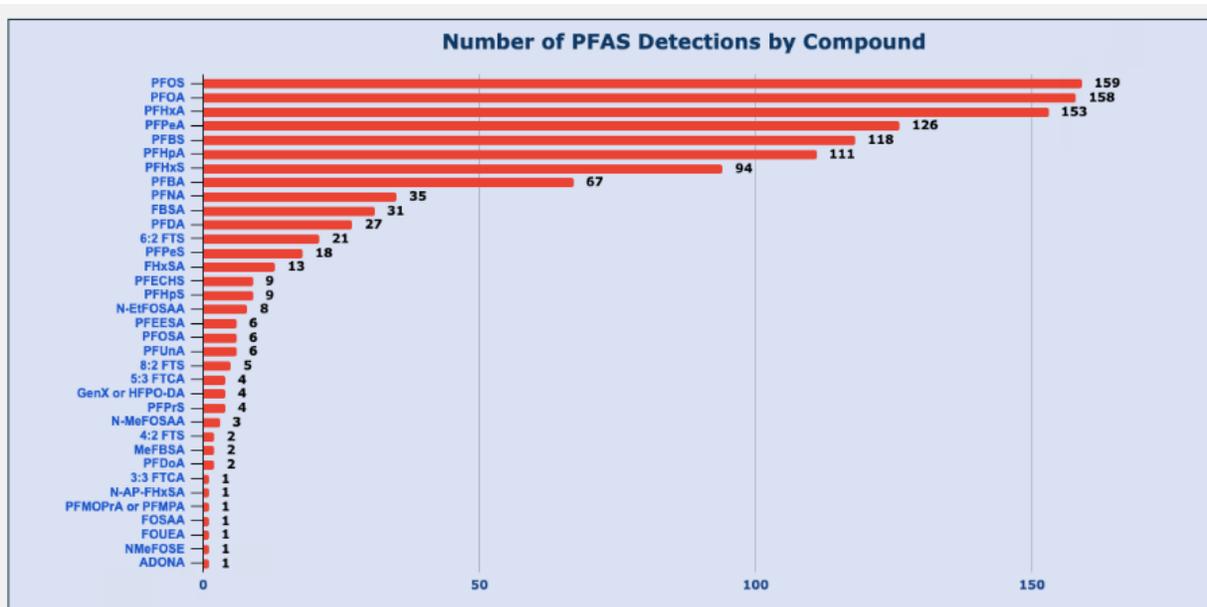


FIGURE 4
Total Number of PFAS Detections by Compound

Likewise, recent testing in the Great Lakes region revealed elevated levels of PFAS in fish tissue, raising human health concerns. In January 2021, the Wisconsin Department of Natural Resources and the Wisconsin Department of Health Services recommended a PFAS-

⁸⁷³ *Id.* at 24.

⁸⁷⁴ *Id.*

⁸⁷⁵ *Id.* at 31.

based fish consumption advisory for Lake Superior based on sampling results that found elevated levels of PFOS in rainbow smelt.⁸⁷⁶ The Michigan Department of Health and Human Services subsequently issued similar guidance recommending that individuals limit Lake Superior smelt consumption to one serving per month.⁸⁷⁷ And, in March 2021, the Minnesota Department of Health included new rainbow smelt consumption guidance based on high levels of PFOS.⁸⁷⁸

Since 1999, the Center for Disease Control (“CDC”) has measured at least 12 PFAS in the blood serum of participants 12 years and older in the National Health and Nutrition Examination Survey.⁸⁷⁹ The CDC found four PFAs—PFOA, PFOS, perfluorohexane sulfonic acid (“PFHxS”), and perfluorononanoic acid (“PFNA”)—in the serum of nearly all people tested, indicating widespread exposure to these PFAS in the U.S. population.⁸⁸⁰ This exposure to PFAS is linked to a myriad of harmful human health effects, including: decreased fertility and/or increased high blood pressure in pregnant women; increased risk of cancers, including prostate, kidney, and testicular cancers; reduced ability of the body’s immune system to fight infections, including reduced vaccine response; interference with the body’s natural hormones; and increased cholesterol levels and/or risk of obesity.⁸⁸¹

Children are especially vulnerable to PFAS exposure, which can also cause developmental effects or delays, including low birth weight, accelerated puberty, bone

⁸⁷⁶ EPA, *Fish and Shellfish Program Newsletter* (May 2021) (hereinafter “Fish and Shellfish Newsletter”), available at <https://www.epa.gov/sites/default/files/2021-06/documents/fish-news-may2021.pdf> at 1.

⁸⁷⁷ *Id.* at 3.

⁸⁷⁸ *Id.* at 4.

⁸⁷⁹ CDC, *Per- and Polyfluorinated Substances (PFAS) Factsheet*, available at https://www.cdc.gov/biomonitoring/PFAS_FactSheet.html.

⁸⁸⁰ *Id.*

⁸⁸¹ PFAS Current Understanding, *supra* n. 864.

variations, or behavioral changes.⁸⁸² Additionally, because children are still developing, they may be more sensitive to the harmful effects of PFAS and are likely exposed more often than adults.⁸⁸³ Infants can be exposed both through contaminated breast milk and formula made with contaminated water.⁸⁸⁴ In addition, recent science suggests maternal transfer of PFAS *in utero*.⁸⁸⁵ The smaller body size and mechanism of movement of young children also puts them at greater risk of exposure than adults. Children drink more water, eat more food, and “breathe more air per pound of body weight than adults.”⁸⁸⁶ Additionally, young children crawl on floors and often put things in their mouths, which leads to a “higher risk of exposure to PFAS in carpets, household dust, toys, and cleaning products” to which dietary additions present greater risks.⁸⁸⁷

PFAS also bioaccumulate in wildlife.⁸⁸⁸ In Michigan, deer, muskrats, and tree swallows were all found with high PFAS levels and testing of other species demonstrated “PFOS contamination across a range of aquatic taxa, including mollusks, arthropods, and amphibians.”⁸⁸⁹ In December of 2020 researchers documented negative health effects of PFOS on the Eastern oyster. Results indicated that cellular damage occurred even during short periods

⁸⁸² *Id.*

⁸⁸³ *Id.*

⁸⁸⁴ *Id.*

⁸⁸⁵ *Id.*

⁸⁸⁶ *Id.*

⁸⁸⁷ *Id.*

⁸⁸⁸ Memorandum from Gary Klase and Abiy Mussa, Michigan Department of Health and Human Services, Division of Environmental Health, to Deb MacKenzie-Taylor, Toxicology & Response Section Manager, Re: *Public Health Advisory for Wildlife from Clark’s Marsh* (Dec. 8, 2019), available at <https://www.michigan.gov/-/media/Project/Websites/pfasresponse/Folder1/Folder1/Memo-2019-12-08-Public-Health-Advisory-Clarks-Marsh.pdf?rev=59aa5ab597264b02b6b0e53f8e0ac11e>.

⁸⁸⁹ *Id.* at 4.

of exposure.⁸⁹⁰ Numerous states have taken a range of regulatory and non-regulatory actions to address PFAS.⁸⁹¹

EPA’s response to the hazards of PFAS has been marked by decades of inaction that could largely be characterized as ‘closing the barn door after the horse has bolted.’ In 1998, EPA was informed about the tendency of PFOS to build up in blood and was offered studies that showed liver damage from PFAS exposure.⁸⁹² In 2006, EPA’s Science Advisory Board found PFOA to be a “likely human carcinogen.”⁸⁹³ In 2016, EPA set a non-enforceable health advisory level at 70 parts per trillion for PFOA and PFOS in drinking water—far above levels that independent researchers said was safe.⁸⁹⁴ In 2019, EPA issued a PFAS Action Plan and promptly missed a self-imposed deadline to issue a plan to set enforceable legal limits for PFOA and PFOS in drinking water.⁸⁹⁵

Finally, in October 2021, EPA announced its PFAS Roadmap that details timelines for the agency to take a myriad of regulatory and non-regulatory actions including to set drinking water standards, recommended aquatic life and human health criteria,⁸⁹⁶ wastewater treatment guidelines, health assessments and hazardous substance designations for several PFAS chemicals.⁸⁹⁷ As EPA states on its website: “Under the Biden-Harris Administration, EPA has restored scientific integrity and accelerated the pace of research and actions needed to tackle the

⁸⁹⁰ EPA, Fish and Shellfish Program Newsletter *supra* n. 876 at 7.

⁸⁹¹ See Environmental Council of the States, *Processes & Considerations for Setting State PFAS Standards* (Feb. 2020, updated March 2022).

⁸⁹² Scott Faber, Environmental Working Group, *For 20-Plus Years, EPA has Failed to Regulate ‘Forever Chemicals’* (Jan. 9, 2020), available at <https://www.ewg.org/research/20-plus-years-epa-has-failed-regulate-forever-chemicals>.

⁸⁹³ *Id.*

⁸⁹⁴ *Id.*

⁸⁹⁵ *Id.*

⁸⁹⁶ EPA PFAS Roadmap, *supra* n. 2 at 15.

⁸⁹⁷ *Id.*

PFAS crisis and protect American communities.”⁸⁹⁸ This flurry of EPA actions to address PFAS include—all since January 2021:

- Inactive PFAS Significant New Use Rule;
- Industrial Wastewater and Permitting PFAS;⁸⁹⁹
- Proposed Rule to Enhance Reporting of PFAS Data to the Toxics Release Inventory;
- Proposed Hazardous Substance Designation for PFOA and PFO;
- New Drinking Water Health Advisories and \$1 Billion in Bipartisan Infrastructure Law Funding;
- National PFAS Testing Strategy Test Order;
- Adding Five PFAS to Contaminated Site Cleanup Tables;
- Draft Aquatic Life Criteria for PFOA and PFOS;
- Addressing PFAS in National Pollutant Discharge Elimination System (NPDES) Permitting;
- Draft Adsorbable Organic Fluorine Method;
- Expanding PFAS Monitoring in Drinking Water;
- Science Advisory Board Review of Draft PFOA/PFOS Scientific Documents;
- Initiation of Two Rulemaking Efforts Under RCRA;
- Final Human Health Toxicity Assessment for GenX Chemicals;
- National PFAS Testing Strategy; PFBA Toxicity Assessment Released for Public Comment; Released Preliminary Toxics Release Inventory Data on PFAS;
- Rule Development for designating PFOA/PFOS as CERCLA Hazardous Substances;
- Expanding Data Collection Efforts on PFAS;

⁸⁹⁸ EPA, PFOA, PFOS and Other PFAS, *EPA Actions to Address PFAS*, available at <https://www.epa.gov/pfas/epa-actions-address-pfas>. Note one example of addressing PFAS in NPDES permits is illustrated by EPA Region 10’s proposed issuance of a permit including 40 PFAS chemicals. See Sandy Point draft permit, *supra* n. 611, Section I.B.9 (Table 2) (proposing to require monitoring of perfluoroalkyl carboxylic acids, perfluoroalkyl sulfonic acids (acid form), fluorotelomer sulfonic acids, perfluorooctane sulfonamides, perfluorooctane sulfonamidoacetic acids, perfluorooctane sulfonamide ethanols, per- and polyfluoroether carboxylic acids, ether sulfonic acids, and fluorotelomer carboxylic acids).

⁸⁹⁹ EPA recently announced an interest in updating the OCPSF ELGs but only for the limited purpose of addressing “discharges from [OCPSF] manufacturers of per- and polyfluoroalkyl substances (PFAS).” *Advance Notice of Proposed Rulemaking, Clean Water Act Effluent Limitations Guidelines and Standards for the Organic Chemicals, Plastics and Synthetic Fibers Point Source Category*, 86 Fed. Reg. 14560 (Mar. 17, 2021). Because PFAS compounds have not been designated by EPA as priority pollutants, EPA asserts that it is seeking to regulate them as a “nonconventional pollutant, as they are not defined as a toxic or conventional pollutant in the CWA or the Code of Federal Regulations (CFR).” *Id.* at 14562. EPA is bending over backwards to not regulate PFAS as a toxic pollutant, and more specifically to not add PFAS to the Toxic Pollutants Lists, because the PFAS family of chemicals clearly meets the statutory definition of a “toxic pollutant” at CWA Section 502(13).

- EPA Council on PFAS;
- Updated Toxicity Assessment for PFBS;
- Robust Review Process for new PFAS;
- Establishing a National Primary Drinking Water Standard for PFOA/PFO;
- Planning to Conduct Expanded Nationwide Monitoring for PFAS in Drinking Water⁹⁰⁰

Yet, despite EPA’s impressive attention to regulation of PFAS, its PFAS Roadmap fails to include inclusion of PFAS on the Toxic Pollutants Lists. Doing so is a necessary step to ensure that all of the work accomplished by EPA in the Biden/Harris administration is not allowed to languish should, for example, a new administration choose to change the course of federal regulation of PFAS.

C. Persistent Bioaccumulative Toxics Pollutants Covered by the Toxics Release Inventory Program That are Not on the Toxic Pollutant or Priority Pollutant Lists

Section 313 of the 1986 Emergency Planning and Community Right-to-Know Act (“EPCRA”) includes the EPA compilation of the Toxics Release Inventory (“TRI”) wherein covered manufacturing sectors are required to report their annual releases to the environment of identified toxic chemicals.⁹⁰¹ Chemicals covered by TRI (“TRI List”) are those that cause one or more of the following adverse impacts: cancer or other chronic human health effects, significant adverse acute human health effects, and significant adverse environmental effects.⁹⁰² The current TRI List contains 787 individual chemicals and 33 chemical categories.⁹⁰³ Six of the

⁹⁰⁰ *See id.*

⁹⁰¹ 42 U.S.C. § 11001 *et seq.* (1986).

⁹⁰² EPA, Toxics Release Inventory (TRI) Program, *What is the Toxics Release Inventory?*, available at <https://www.epa.gov/toxics-release-inventory-tri-program/tri-listed-chemicals>.

⁹⁰³ *Id.* *See also*, EPA, *Table II. EPCRA Section 313 Chemical List For Reporting Year 2021 (including Toxic Chemical Categories)*, available at https://www.epa.gov/system/files/documents/2022-03/ry-2021-tri-chemical-list-03-07-2022_0.pdf; EPA, *Changes To The TRI List Of Toxic Chemicals* (Jan. 24, 2022), available at <https://www.epa.gov/system/files/documents/2023-01/TRI%20Chemical%20List%20Changes%2001%2024%202022.pdf>.

chemical categories include several compounds such as the category of dioxin and dioxin-like compounds, which includes 17 individual compounds.⁹⁰⁴ Consequently, the TRI List contains around a total of 890 chemicals and compounds that may pose a threat to human health and the environment. Of those, there are hundreds that are not on the Toxic Pollutants Lists despite EPA's having determined they pose a threat to human health and the environment.

In addition, EPA has classified 16 chemicals and 5 chemical categories on the TRI List "of special concern" and created reporting thresholds that are lower than other TRI chemicals.⁹⁰⁵ Of these, the following individual and chemical categories are not on the Toxic Pollutants Lists: isodrin, methoxychlor, octachlorostyrene, pendimethalin, pentaclorobenzene, tetrabromobisphenol A, trifluran, and compounds of hexabromocyclododecane ("HBCD"), lead, mercury, polycyclic aromatic compounds ("PAC"), "dioxin-like compounds," and some PCBs.⁹⁰⁶ As the following examples demonstrate, these TRI chemicals of "special concern" particularly warrant placement on the Toxic Pollutants Lists for the same reason they are included in the TRI program:

- Isodrin is toxic to humans, a suspected carcinogen, and because it mainly affects the central nervous system, exposure can lead to convulsions.⁹⁰⁷ Additionally, reproductive

⁹⁰⁴ The diisocyanates category includes 20 compounds; the dioxin and dioxin-like category includes 17 compounds; the hexabromocyclododecane category includes 2 compounds; the nonylphenol category includes 6 compounds; the nonylphenol ethoxylates category includes 13 compounds; and the polycyclic aromatic compound category includes 25 compounds.

⁹⁰⁵ EPA, TRI National Analysis, *Chemicals of Special Concern*, available at <https://www.epa.gov/trinationalanalysis/chemicals-special-concern>.

⁹⁰⁶ EPA, Toxics Release Inventory (TRI) Program, *Persistent Bioaccumulative Toxic (PBT) Chemicals Covered by the TRI Program*, available at <https://www.epa.gov/toxics-release-inventory-tri-program/persistent-bioaccumulative-toxic-pbt-chemicals-covered-tri>.

⁹⁰⁷ National Library of Medicine, PubChem, *Isodrin (insecticide)*, available at <https://pubchem.ncbi.nlm.nih.gov/compound/12310946>.

and liver effects have been reported.⁹⁰⁸ Isodrin is an environmental hazard, as it is very toxic to aquatic life and has long-lasting effects.⁹⁰⁹

- Pentachlorobenzene is a persistent organic pollutant.⁹¹⁰ Pentachlorobenzene is an environmental hazard, as it is very toxic to aquatic life and has long-lasting effects.⁹¹¹ In 2021, 142 pounds of this chemical were reported to have been directly discharged to the Tule Lake watershed, located along the California/Oregon border and including the Tule Lake National Wildlife Refuge.⁹¹²
- Tetrabromobisphenol A is a probable carcinogen and very toxic to aquatic life. There was one direct discharge reported for tetrabromobisphenol A, and three indirect discharges reported in 2021.⁹¹³ The top receiving watershed for direct TRI pounds per year was Beaverdam Creek-Ohio River, with 10 pounds per year reported.⁹¹⁴

EPA has also designated the category of “dioxin and dioxin-like compounds” as TRI chemicals of “special concern.”⁹¹⁵ Dioxins are persistent organic pollutants that are highly toxic to humans and cause cancer, reproductive and developmental problems, damage to the immune system, and endocrine disruption.⁹¹⁶ “Dioxin-like compounds” refers to a group of toxic

⁹⁰⁸ *Id.*

⁹⁰⁹ *Id.*

⁹¹⁰ PubChem, *Pentachlorobenzene*, available at <https://pubchem.ncbi.nlm.nih.gov/compound/11855>.

⁹¹¹ *Id.*

⁹¹² EPA, ECHO Enforcement and Compliance History Online, *Water Pollution Search Results* (search set to pentachlorobenzene in 2021), search engine available at <https://echo.epa.gov/trends/loading-tool/water-pollution-search>.

⁹¹³ *Id.* (search set to Tetrabromobisphenol A in 2021).

⁹¹⁴ *Id.*

⁹¹⁵ Dioxin and Dioxin-like Compounds; Toxic Equivalency Information; Community Right-To-Know Toxic Chemical Release Reporting, Fed. Reg., 26544 (May 10, 2007) (16 dioxin and furan compounds in addition to 2,3,7,8-TCDD are required to be summed for TRI reporting purposes).

⁹¹⁶ EPA, *Learn about Dioxin*, available at <https://www.epa.gov/dioxin/learn-about-dioxin> (hereinafter “Learn About Dioxin”); see also Sally S. White and Linda S. Birnbaum, *An Overview of the Effects of Dioxins and Dioxin-like Compounds on Vertebrates, as Documented in Human and Ecological Epidemiology*, 27(4) *J. Environ Sci Health C Environ Carcinog Ecotoxicol Rev.* 197-211 (Oct. 2009), available at <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2788749/pdf/nihms150859.pdf>.

chemicals with a similar chemical structure, similar physical-chemical properties, and invoke similar toxic responses; all are persistent and bioaccumulative.⁹¹⁷

In 2002, EPA issued recommended human health criteria for the chemical compound 2,3,7,8-TCDD. Its website states that these criteria “should be used in conjunction with the recommended toxicity equivalence factors for dioxin and dioxin-like compounds (USEPA, 2010) to account for the additive effects of other dioxin-like compounds,”⁹¹⁸ a citation to recommended toxicity equivalence factors for dioxin-like compounds.⁹¹⁹ However, because the recommendation is only on the agency’s website and not in the published recommended criteria, it may not be treated by the states or EPA as recommended criteria. Moreover, “dioxin-like compounds” are not found on either the Toxic Pollutant List or the Priority Pollutant List, relegating those compounds to a lesser legal status than the listed chemicals: the individual compound 2,3,7,8-TCDD and the category of PCBs.

Similarly, polybrominated biphenyls (“PBBs”) are a class of TRI-listed chemical that is structurally similar to the class of polybrominated diphenyl ethers (“PBDEs”).⁹²⁰ PBBs and PBDEs are brominated flame-retardant chemicals found in a variety of products, including furniture, electrical and electronic equipment, textiles, and other household products.⁹²¹ While EPA notes their similarity—they both “may act as endocrine disruptors in humans and other

⁹¹⁷ Learn About Dioxin, *supra* n. 916.

⁹¹⁸ EPA, *National Recommended Water Quality Criteria - Human Health Criteria Table*, available at <https://www.epa.gov/wqc/national-recommended-water-quality-criteria-human-health-criteria-table>.

⁹¹⁹ EPA, *Recommended Toxicity Equivalence Factors (TEFs) for Human Health Risk Assessments of 2,3,7,8-Tetrachlorodibenzo-p-dioxin and Dioxin-Like Compounds* (Dec. 2010), available at <https://www.epa.gov/sites/default/files/2013-09/documents/tefs-for-dioxin-epa-00-r-10-005-final.pdf>

⁹²⁰ EPA, *Emerging Contaminants – Polybrominated Diphenyl Ethers (PBDE) and Polybrominated Biphenyls (PBB)* (April 2008).

⁹²¹ *Id.*

animals” and some individual homologs are classified as possible human carcinogens⁹²²—EPA has banned PBBs but included them in the TRI List while it has not included PBDEs in the TRI while observing that they are in “widespread use in the US” and “there is growing concern about their persistence in the environment and their tendency to bioaccumulate in the food chain.”⁹²³ Neither is included on the Toxic Pollutants Lists.

Looking at toxic chemicals discharged directly or indirectly to surface waters from sources in the “Plastics Materials, Synthetic Resins, and Nonvulcanizable Elastomers” group of dischargers (SIC Code 2821), that are on the TRI list but not on the Toxic Pollutants Lists, demonstrates how outdated the Toxic Pollutants Lists are.⁹²⁴ Of the 23 pages of TRI pollutants not on the Toxic Pollutants Lists, over 115 individual pollutants and chemical families were discharged by this group of manufacturing sources.⁹²⁵

D. Toxic Pollutants Identified as in Need of Source Control Pursuant to the CWA in CERCLA Actions to Remedy Contaminated Sediments

The Comprehensive Environmental Response, Compensation, and Liability Act (“CERCLA”), commonly known as “Superfund,” was enacted by Congress in 1980.⁹²⁶ CERCLA provides broad federal authority to respond to releases or threatened releases of hazardous substances that may endanger public health or the environment from closed abandoned hazardous waste sites. The law authorizes both short-term removals and long-term

⁹²² *Id.*

⁹²³ *Id.* See EPA, *List of Lists* (Dec. 2022), available at https://www.epa.gov/system/files/documents/2022-12/List_of_Lists_Compiled_December%202022.pdf at 14 (PBBs included in TRI).

⁹²⁴ See *2021 Data from the Toxics Release Inventory*, derived from EPA, ECHO Water Pollution Search, <https://echo.epa.gov/trends/loading-tool/water-pollution-search> (showing all TRI list pollutants that are not also on the Toxics Pollutants Lists and highlighting chemicals that were reported as discharged from SIC Code 2821 in 2021).

⁹²⁵ *Id.*

⁹²⁶ 42 U.S.C. § 9601 *et seq.*

remedial actions associated with releases or threats of releases of hazardous substances. These actions can be conducted only at sites listed on EPA's National Priorities List ("NPL").

In addition to the primary clean-up focus at Superfund sites, regulatory agencies must also ensure that the sites are not re-contaminated with toxic chemicals from any source: "If a site includes a source that could result in significant recontamination, source control measures will likely be necessary as part of that response action."⁹²⁷ This "source control" is defined in EPA regulations as: "the construction or installation and start-up of those actions necessary to prevent the continued release of hazardous substances or pollutants or contaminants (primarily from a source on top of or within the ground, or in buildings or other structures) in the environment."⁹²⁸ By implementing source controls, regulatory agencies can assure the goal of environmental protection as well as safeguarding the investment of tax dollars spent on cleaning up Superfund sites. EPA has explained its importance:

Identifying and controlling contaminant sources typically is critical to the effectiveness of any Superfund sediment cleanup. Source control generally is defined for the purposes of this guidance as those efforts are taken to eliminate or reduce, to the extent practicable, the release of contaminants from direct and indirect continuing sources to the water body under investigation. At some sediment sites, the original sources of the contamination have already been controlled, but subsequent sources such as contaminated floodplain soils, storm water discharges, and seeps of ground water or non-aqueous phase liquids (NAPLs) may continue to introduce contamination to a site. At sites with significant sediment mobility, areas of higher contaminant concentration may act as continuing sources for less-contaminated areas.⁹²⁹

⁹²⁷ EPA, Memorandum from Marianne Lamon Horinko to Superfund National Managers, RCRA Senior Policy Advisors, *Principles for Managing Contaminated Sediment Risks at Hazardous Waste Sites* (Feb. 12, 2002) (hereinafter "Sediment Risk Management") at 2.

⁹²⁸ 40 C.F.R. § 300.5.

⁹²⁹ EPA, *Contaminated Sediment Remediation Guidance for Hazardous Waste Sites* (2005) at 2-20, available at <https://semspub.epa.gov/work/HQ/174471.pdf>.

EPA cites the role of the CWA to support source control for Superfund sites, for example, where “[s]ome sources, especially those outside the boundaries of the Superfund or RCRA site, may best be handled under another authority, such as the CWA or a state program.”⁹³⁰ Specifically, EPA notes that permitting of toxic discharges is an important consideration for source control:

Source control may include application of regulatory mechanisms and remedial technologies to be implemented according to [applicable or relevant and appropriate requirements] ARARs, including the application of technology-based and water quality-based National Pollutant Discharge Elimination System (NPDES) permitting to achieve and maintain sediment cleanup levels.”⁹³¹

In addition, because EPA notes that there is “a need to balance the desire for watershed-wide solutions with practical considerations affecting a subset of responsible parties” and that “[a] critical question often is whether an action in one part of the watershed is likely to result in significant and lasting risk reduction, given the probable timetable for other actions in the watershed,” source control actions may include “[p]ollutant load reductions of point and nonpoint sources based on a TMDL.”⁹³²

Both NPDES permitting and TMDLs depend on water quality standards for which reason EPA identifies standards as important but, in addition noting that “water quality standards . . .

⁹³⁰ *Id.* See also *id.* at 3-9, 3-11, 3-12 (potential federal ARARs based on the Clean Water Act).

⁹³¹ *Id.* at 2-20.

⁹³² *Id.* at 2-20 – 2-21. See also *id.* at 3-8 (“EPA-established TMDLs are not promulgated as rules, are not enforceable, and, therefore, are not ARARs. TMDLs established by states, territories or authorized Indian tribes may or may not be promulgated as rules. Therefore, TMDLs established by states, territories, or authorized Indian tribes, should be evaluated on a regulation-specific and site-specific basis. Even if a TMDL is not an ARAR, it may aid in setting protective cleanup levels and may be appropriately a TBC. Project managers should work closely with regional EPA Water program and state personnel to coordinate matters relating to TMDLs. The project manager should remember that even when a TMDL or wasteload allocation is not enforceable, the water quality standards on which they are based may be ARARs. TMDLs can also be useful in helping project managers evaluate the impacts of continuing sources, contaminant transport, and fate and effects. Similarly, Superfund’s RI/FS may provide useful information and analysis to the federal and state water programs charged with developing TMDLs.”).

may be ARARs.”⁹³³ Called out as particularly important are sediment quality criteria and “biological standards”⁹³⁴ as well as standards that “may be potential ARARs for surface water when water is discharged from dewatering or treatment areas or as effluent from confined disposal facilities.”⁹³⁵ In short, water quality standards are the basis for source control efforts, including the development of best management practices for non-NPDES sources to prevent continued toxic releases.⁹³⁶

EPA urges Superfund and other hazardous waste site managers to “[a]s early in the process as possible, . . . try to identify all direct and indirect continuing sources of significant contamination to the sediments under investigation” namely the entire range of water pollution sources, including:

discharges from industries or sewage treatment plants, spills, precipitation runoff, erosion of contaminated soil from stream banks or adjacent land, contaminated groundwater and non-aqueous phase liquid contributions, discharges from storm water and combined sewer outfalls, upstream contributions, and air deposition.⁹³⁷

Toxic pollutants found in contaminated sediments at CERCLA sites can be released into water by mining, dredging, breached impoundments, incised channels, channel modification, eroding and/or collapsing stream banks, impervious surfaces, lack of connectivity with a floodplain, impoundments, scoured streambeds upstream, stored soil or waste, and many other methods.⁹³⁸ Each activity mobilizes contaminated sediment and allows it to reach or move

⁹³³ *Id.* at 3-8.

⁹³⁴ *Id.* at 2-17.

⁹³⁵ *Id.* at 3-8.

⁹³⁶ *Id.* at 2-21.

⁹³⁷ Sediment Risk Management, *supra* n. 927 at 2.

⁹³⁸ EPA, CADDIS Volume 2, *Sediments*, available at <https://www.epa.gov/caddis-vol2/sediments>.

within waterbodies. Finer sediment is particularly able to move easily through water to contaminate the food web.⁹³⁹

The following tables demonstrate that there are many persistent contaminants of concern (“COC”) at Superfund sites that are or may not be on the Toxic Pollutants Lists.

Tier 1 CERCLA National Priority Sites ⁹⁴⁰	CERCLA Persistent Contaminants of Concern Not on the Toxic Pollutant List
Centredale Manor Restoration Project	<ul style="list-style-type: none"> • dioxins/furans TEQ • aluminum • barium • vanadium
Pine Street Canal	<ul style="list-style-type: none"> • metals
Bridgeport Rental & Oil Services	<ul style="list-style-type: none"> • total petroleum hydrocarbons
Lipari Landfill	<ul style="list-style-type: none"> • 4-methyl-2-pentanone • total xylenes
Reynolds Metals Co.	<ul style="list-style-type: none"> • TDBFs
Roebing Steel Co.	<ul style="list-style-type: none"> • metals
Copper Basin Mining District Site	<ul style="list-style-type: none"> • iron
Kerr-Mcgee (Kress Creek/West Branch of Dupage River)	<ul style="list-style-type: none"> • radium 226 • radium 228
Iron Mountain Mine	<ul style="list-style-type: none"> • iron
Commencement Bay, Near Shore/Tide Flats	<ul style="list-style-type: none"> • dioxins/furans • 4-methyl-phenol
Harbor Island (Lead)	<ul style="list-style-type: none"> • tributyltin
Ketchikan Pulp Company	<ul style="list-style-type: none"> • 4-methyl-phenol • ammonia • sulfide
McCormick & Baxter Creosoting Co. (Portland)	<ul style="list-style-type: none"> • dioxins/furans

⁹³⁹ *Id.*

⁹⁴⁰ This chart is based on information contained in EPA’s Tier 1 Sediment Site List spreadsheet, *available at* <https://www.epa.gov/superfund/large-sediment-sites-tiers-1-2>. Given EPA’s use of broad and/or vague descriptions of relevant COCs on the spreadsheet and/or inconsistencies in how EPA referred to various COCs, this list may not be complete and/or overinclusive. For example, dioxin(s), furan(s), PCB(s), and PAHs may or may not be covered on the Toxic Pollutants Lists. The spreadsheet filename suggests the spreadsheet was last updated in July 2015.

Tier 2 CERCLA Active Sites ⁹⁴¹	CERCLA Persistent Contaminants of Concern Not on the Toxic Pollutant List
Diamond Alkali – Lower Passaic River & Newark Bay Newark, New Jersey	<ul style="list-style-type: none"> • dioxins • pesticides • metals • petroleum hydrocarbons
Operable Unit (OU) 4: Interim Cleanup in the upper 9 miles of the Lower Passaic River Study Area	<ul style="list-style-type: none"> • dioxins • petroleum hydrocarbons • pesticides • metals • 2,4,5-trichlorophenol • 2,4-dichloropophenoxyacetic acid • 2,4,5-trichlorophenoxyacetic acid
Tittabawassee River, Saginaw River and Bay Midland and Saginaw Counties, Michigan	<ul style="list-style-type: none"> • dioxins/furans
Upper Columbia River, Washington State	<ul style="list-style-type: none"> • iron • manganese • uranium • pesticides • dioxins/furans

For source control activities to address Superfund (or other hazardous waste) sites to be effective, actions taken pursuant to the CWA must be based on protective water quality standards and/or national effluent limitation guidelines. Neither approach can be effective where toxic contaminants are not on the Toxic Pollutants Lists.

E. Contaminants of Emerging Concern—A Group of Unregulated Toxic Pollutants for Which EPA Largely Has No Plan to Regulate

“Contaminants of emerging concern” (“CECs”) include a wide range of pollutants for which EPA has no plans to regulate with the exception of PFAS and PFOS as described in subsection VII.B, *supra*. This subsection of the petition discusses CECs as a general group, the

⁹⁴¹ This chart is based on EPA memoranda for the included sites, *available at* <https://www.epa.gov/superfund/large-sediment-sites-tiers-1-2>. Given EPA’s use of broad and/or vague descriptions of relevant COCs on the spreadsheet and/or inconsistencies in how EPA referred to various COCs, this list may not be complete or may be overinclusive.

subset of CECs known as “pharmaceuticals and personal care products” (“PPCPs”), and a few examples of individual or chemical family pollutants that are considered to be CECs and the reasons why they need to be regulated.

1. “Contaminants of Emerging Concern”

With the exception of the PFAS described in sub-section VII.B *supra*, EPA has made no progress in regulating CECs. Defined to include many different kinds of chemicals and substances—including industrial chemicals, pharmaceuticals and personal care products, medical and agricultural products, and microplastics—the concern about the adverse effects of these contaminants has long since “emerged” and is now well known. EPA has described these CECs to include the following:

- Persistent organic pollutants (POPs) such as polybrominated diphenyl ethers (PBDEs; used in flame retardants, furniture foam, plastics, etc.) and other global organic contaminants such as perfluorinated organic acids;
- Pharmaceuticals and personal care products (PPCPs), including a wide suite of human prescribed drugs (e.g., antidepressants, blood pressure), over-the-counter medications (e.g., ibuprofen), bactericides (e.g., triclosan), sunscreens, synthetic musks;
- Veterinary medicines such as antimicrobials, antibiotics, anti-fungals, growth promoters and hormones;
- Endocrine-disrupting chemicals (EDCs), including synthetic estrogens (e.g., 17 α -ethynylestradiol, which also is a PCPP) and androgens (e.g., trenbolone, a veterinary drug), naturally occurring estrogens (e.g., 17 β -estradiol, testosterone), as well as many others (e.g., organochlorine pesticides, alkylphenols) capable of modulating normal hormonal functions and steroidal synthesis in aquatic organisms;
- Nanomaterials such as carbon nanotubes or nano-scale particulate titanium dioxide, of which little is known about either their environmental fate or effects.⁹⁴²

⁹⁴² EPA CEC White Paper, *supra* n. 146 at 2. Note that the concept of endocrine disruption was introduced in 1991. See Wingspread Conference, *Statement from the Work Session on Chemically-Induced Alterations in Sexual Development: The Wildlife/Human Connection Consensus Statement* (July 1991), available at http://endocrinedisruption.org/assets/media/documents/wingspread_consensus_statement.pdf.

Writing in 2008—14 years ago—EPA stated that “[w]idespread uses, some indication of chemical persistence, effects found in natural systems, and public concerns have made clear the need for EPA to develop criteria that can be used to help assess and manage potential risk of some CECs in the aquatic environment.”⁹⁴³ And yet, a decade and a half later, EPA has managed to only just this last year issue draft recommended criteria for two related CECs pursuant to the CWA: PFAS and PFOS.⁹⁴⁴

⁹⁴³ *Id.* at 2.

⁹⁴⁴ EPA has developed final Endocrine Disruptor Screening Program Test Guidelines that “are generally intended to meet testing requirements under TSCA, FIFRA and FFDCA to determine if a chemical substance may pose a risk to human health or the environment due to the disruption of the endocrine system.” See EPA, Test Guidelines for Pesticides and Toxic Substances, *Series 890 - Endocrine Disruptor Screening Program Test Guidelines*, available at <https://www.epa.gov/test-guidelines-pesticides-and-toxic-substances/series-890-endocrine-disruptor-screening-program>. See also, EPA, *Availability of New Approach Methodologies (NAMs) in the Endocrine Disruptor Screening Program (EDSP)* (Dec. 13, 2022); EPA, *Endocrine Disruptor Screening Program for the 21st Century: (EDSP21 Work Plan) The Incorporation of In Silico Models and In Vitro High Throughput Assays in the Endocrine Disruptor Screening Program (EDSP) for Prioritization and Screening Summary Overview* (Sept. 30, 2011), available at https://www.epa.gov/sites/default/files/2015-07/documents/edsp21_work_plan_summary_overview_final.pdf. It has also evaluated the science on nonmonotonic dose responses—where a response may also be greater at lower doses. See EPA, Safer Chemicals Research, *Nonmonotonic Dose Responses as They Apply to Estrogen, Androgen, and Thyroid Pathways and EPA Testing and Assessment Procedures*, available at <https://www.epa.gov/chemical-research/nonmonotonic-dose-responses-they-apply-estrogen-androgen-and-thyroid-pathways-and>; EPA, Safer Chemicals Research, *Endocrine Disruption Research: Testing for Potential Low-Dose Effects*, available at <https://www.epa.gov/chemical-research/endocrine-disruption-research-testing-potential-low-dose-effects>. See also EPA, *ORD Science Integration: EPA’s Nonmonotonic Dose Response Curve (NMDRC) Workplan* (undated), available at <https://www.epa.gov/sites/default/files/2014-03/documents/nmdrc-06212012.pdf>. The agency has been conducting a screening program to identify chemicals with the potential to interact with the endocrine system and to conduct more in-depth testing of those identified. See EPA, Safer Chemicals Research, *Research on Endocrine Disruptors*, available at <https://www.epa.gov/chemical-research/research-endocrine-disruptors>. This includes research specifically focused on water quality, namely chemicals from animal feeding operations, from sewage sludge, and from treated sewage. See EPA, Safer Chemical Research, *Research on Detecting Endocrine Disrupting Chemicals from Animal Feeding Operations*, available at <https://www.epa.gov/chemical-research/research-detecting-endocrine-disrupting-chemicals-animal-feeding-operations>; EPA, Safer Chemicals Research, *Research on Evaluating Concentrations of Endocrine Disrupting Chemicals in Biosolids (Sewage Sludge)*, available at <https://www.epa.gov/chemical-research/research-evaluating-concentrations-endocrine-disrupting-chemicals-biosolids>; and EPA, Safer Chemicals Research, *Assessing Endocrine Disrupting Chemicals In Landfills, Solid Waste Sites and Wastewater*, available at <https://www.epa.gov/chemical-research/assessing-endocrine-disrupting-chemicals-landfills-solid-waste-sites-and>.

EPA itself described the hazards associated with the unregulated discharge of these toxic pollutants:

Some CECs are similar to conventional toxic pollutants in that they are associated with industrial releases, whereas many others are used by the general public every day in homes, on farms, by businesses and industry (Daughton and Ternes 1999). PPCPs acting as [endocrine-disrupting chemicals] EDCs can be released directly to the environment after passing through wastewater treatment processes, which are typically not designed to remove these pollutants from the effluent (Halling-Sorensen et al. 1998). Sludge from secondary treatment processes are land-applied as biosolids, supplying CECs which may leach or run off into nearby bodies of water. Pharmaceuticals used in animal feeding operations may be released to the environment in animal wastes via direct discharge of aquaculture products (i.e., antibiotics), the excretion of substances in animal urine and feces of livestock animals, and the washoff of topical treatments from livestock animals (Boxall et al. 2003).

EDCs discharged at WWTPs are one group of CECs with potentially widespread environmental effects (Folmar et al. 1996; Folmar et al. 2001; Jobling et al. 1998; Woodling et al. 2006). Although particular concern has been expressed about the anthropogenic EDCs, there are also natural estrogens (estradiol and its metabolites estriol and estrone) entering the aquatic environment through wastewater discharge and excretion from domestic animals. Furthermore, little is known about the environmental occurrence, fate and, transport for any of these compounds after they enter aquatic ecosystems. Many of the man-made compounds have been in use for a long time, and there is concern about pharmacologically active ingredients and personal care products that are designed to stimulate a physiological response in humans, plants, and animals (Daughton and Ternes 1999).⁹⁴⁵

As EPA points out, the “[f]requent detection of compounds by itself does not constitute a need for [aquatic life criteria].”⁹⁴⁶ But the agency concludes that endocrine-disrupting CECs have “received the most attention because field studies from around the world have demonstrated that very low concentrations of some of these compounds can significantly impact natural populations of aquatic vertebrates,” going on to explain:

⁹⁴⁵ EPA CEC White Paper, *supra* n. 146 at 2-3.

⁹⁴⁶ *Id.* at 3.

For example, observational field studies (Jobling et al. 1998) have shown a high occurrence of intersex (the presence of both male and female characteristics) in wild populations of a fish known as roach (*Rutilus rutilus*) in rivers in the United Kingdom that are downstream from WWTPs. Similar results have recently been reported for white sucker (*Catostomus commersoni*) in northern Colorado, U.S.A (Woodling et al. 2006). In a multiyear study by Kidd et al. (2007), the authors showed that environmentally relevant concentrations of ethynylestradiol, EE2, caused reproductive failure and near collapse of a natural fathead minnow population in an experimental lake, and also had deleterious effects on the reproductive biology of the pearl dace. These direct effects resulting in loss of forage fish have led to cascading effects on the lake trout population due to lack of prey (Kidd, personal communication). Researchers from the U.S. Geological Survey (USGS) have observed intersex and testis-ova (the presence of eggs in the testis) in bass species collected from the Potomac River and its tributaries in West Virginia, Maryland, and Washington DC, and also quantified EDCs in their blood (Blazer et al. 2007; Chambers and Leiker 2006). The occurrence of intersex fish in the Potomac River, as well as documented occurrence of this and related effects in other waters of the US and internationally, prompted Congressional hearings that were held in October 2006 to inquire about the “State of the Science on EDCs in the Environment,” as well as EPA activities associated with EDCs.⁹⁴⁷

Similarly, in 2012, the National Marine Fisheries Service (“NMFS”) expressed concern about CECs and their effect on fish and marine mammals:

Recent studies suggest that certain pharmaceuticals and personal care products (PPCPs) may also accumulate in killer whales. Synthetic musks and antibacterial chemicals (e.g. Triclosan) have been detected in dolphins and porpoises in coastal waters off Japan and the southeastern United States and in harbor seals off the California Coast (Fair *et al.* 2009, Kannan *et al.* 2005, Nakata 2005, Nakata *et al.* 2007). A wider range of PPCPs, including anti-depressants, cholesterol lowering drugs, antihistamines, and drugs affecting blood pressure and cholesterol levels have been detected in tissues of fish from urban areas and sites near wastewater treatment plants (Brooks *et al.* 2005, Ramirez *et al.* 2009), suggesting possible contamination of prey. As yet we have no data on concentrations of PPCPs in either killer whales or their prey species, but they could be a concern because of their widespread occurrence, potential for biomagnification, and biological activity.⁹⁴⁸

⁹⁴⁷ *Id.*

⁹⁴⁸ NMFS Oregon Toxics BiOp, *supra* n. 718 at 82.

NMFS specifically called out the following CECs as persistent pollutants that may pose a risk to resident killer whales: flame retardants, especially PBBs and PBDEs; PFOS; DBT Dibutyltin; polychlorinated paraffins (“PCPs”); polychlorinated naphthalenes (“PCNs”); alkyl-phenol ethoxylates (“APEs”); TBT; and polychlorinated terphenyls (“PCTs”)—all of which it indicated are persistent and bioaccumulate.⁹⁴⁹ The abstract of a recently published paper contains additional insight into CECs in killer whales:

Killer whales (*Orcinus orca*) have been deemed one of the most contaminated cetacean species in the world. . . . Here, we quantify CECs [alkylphenols (APs), triclosan, methyl triclosan, and per- and polyfluoroalkyl substances (PFAS)] and new POPs [hexabromocyclododecane (HBCCD), PFOS, PFOA, and PFHxS] in skeletal muscle and liver samples of these sentinel species and investigate *in utero* transfer of these contaminants. Samples were collected from necropsied individuals from 2006 to 2018. . . . AP and PFAS contaminants were the most prevalent compounds; 4-nonylphenol (4NP) was the predominant AP (median 40.84 ng/g ww), and interestingly, 7:3-fluorotelomer carboxylic acid (7:3 FTCA) was the primary PFAS (median 66.35 ng/g ww). Maternal transfer ratios indicated 4NP as the most transferred contaminant from the dam to the fetus, with maternal transfer rates as high as 95.1%. Although too few killer whales have been screened for CECs and new POPs to infer the magnitude of contamination impact, these results raise concerns regarding pathological implications and potential impacts on fetal development and production of a viable neonate. This study outlines CEC and new POP concentrations in killer whales of the NEP and provides scientifically derived evidence to support and inform regulation to mitigate pollutant sources and contamination of Southern Resident killer whale critical habitat and other marine ecosystems.⁹⁵⁰

In the Great Lakes, the U.S. Fish and Wildlife Service has led efforts to characterize CECs in freshwater. Noting that “limited studies using current environmentally relevant concentrations of chemicals [show] . . . risks to fish and wildlife are evident [creating] . . . an increasing urgency to address data gaps that are vital to resource management decisions,” FWS

⁹⁴⁹ *Id.* at 83–84, Table 2.4.4.1.4.

⁹⁵⁰ CECs and Killer Whales, *supra* n. 6.

issued a report on CECs in water, sediment, and fish tissue.⁹⁵¹ The report noted that “[m]ost appearances and increases in chemical concentrations in sediments occurred at sites immediately downstream from wastewater treatment plants and at sites with predominantly developed land use,” as illustrated by the following bar chart:⁹⁵²

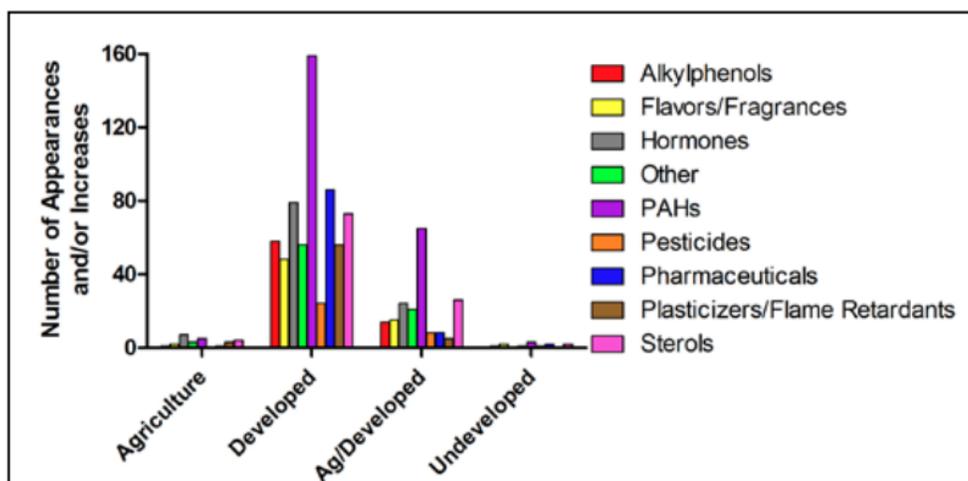


Figure 18. Number of appearances and increases in sediment by chemical class and land use grouping.

In sediment, 22 percent of CECs were detected while in water 11 percent of CECs were detected. CECs “more frequently detected in sediment compared to water were alkylphenols, favors/fragrances, hormones, PAHs, and sterols[.]”⁹⁵³ Perfluorodecanoic acid (“PFDA”), PFOS, and perfluoroundecanoic acid were detected at a 100 percent detection rate in the livers of benthic and pelagic species.⁹⁵⁴ The FWS pointed to the failure to regulate the discharge from point sources by its choice of sampling locations, as demonstrated by this bar chart:⁹⁵⁵

⁹⁵¹ FWS, *Contaminants of Emerging Concern in the Great Lakes Basin: A Report on Sediment, Water, and Fish Tissue Chemistry Collected in 2010-2012* (undated) at 1, available at <https://www.fws.gov/media/contaminants-emerging-concern-great-lakes-basin-report-sediment-water-and-fish-tissue>.

⁹⁵² *Id.* at 1, 25.

⁹⁵³ *Id.* at 18.

⁹⁵⁴ *Id.* at 27.

⁹⁵⁵ *Id.* at 25.

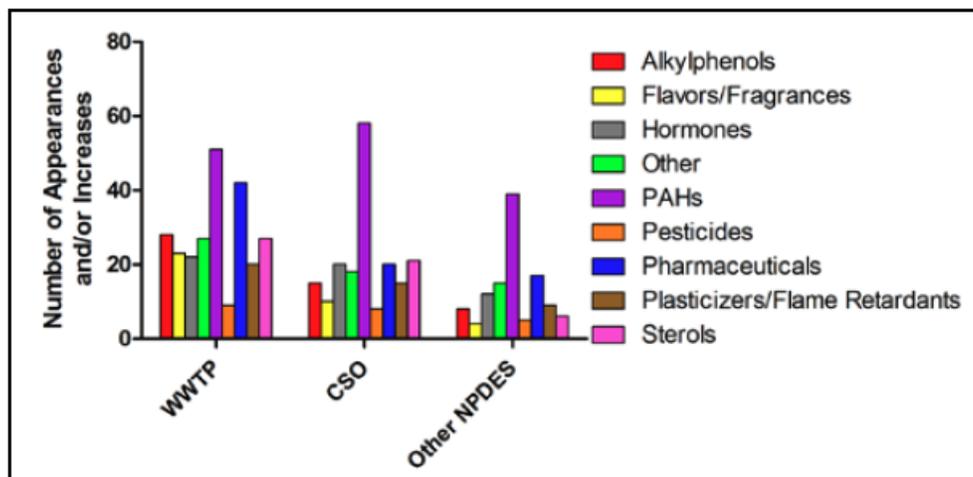


Figure 17. Number of appearances and increases in sediment by chemical class and point source type.

A later study on Great Lakes lake sturgeon found that “44 different PPCPs were identified in serum and gamete samples across sites, with 22 PPCPs identified in at least 25% of serum samples and three PPCPs identified in 25% of gamete samples” with “[m]any of the PPCPs and PBDEs identified in this study . . . associated with population level effects at environmentally-relevant concentrations based on previous research.”⁹⁵⁶ In particular, the authors cited:

[T]he effects of PPCP exposures to fish, particularly for antidepressants. Selective serotonin reuptake inhibitors (SSRIs) (e.g., sertraline) have been shown to influence fish behavior in both short and longer-term toxicity tests, which could have implications for survival, reproduction, and populations overall (Mennigen et al., 2011; Weinberger and Klaper, 2014; Ansai et al., 2016; Ziegler et al., 2020). Exposure to SSRIs can impact predator avoidance and reproductive behaviors (Valenti et al., 2012; Weinberger and Klaper, 2014; Pelli and Connaughton, 2015). Chronic exposure to fluoxetine has also been shown to lead to altered sexual development in fish, however exposure concentrations were

⁹⁵⁶ Jo A. Banda, et al., *Characterization of pharmaceuticals, personal care products, and polybrominated diphenyl ethers in lake sturgeon serum and gametes*, 266 *Environmental Pollution* 115051 (2020) at 7, available at <https://reader.elsevier.com/reader/sd/pii/S0269749120312471?token=1C8FC32C1E3AC63293CB5C232F46C19594ACBA8CBE137D1C17063AE1441865EF8EE591C00C2072AC56C92F4E915DD768&originRegion=us-east-1&originCreation=20230306180643>.

much higher than observed environmental concentrations (Henry and Black, 2008). Furthermore, exposure of fish to SSRIs during early life stages can lead to altered development, growth, and behavioral effects (Pelli and Connaughton, 2015; Huang et al., 2019) in addition to behavioral changes that last beyond the early life stages of exposure into adulthood (Kellner et al., 2018). SSRIs were not the only antidepressants identified in lake sturgeon tissues as amitriptyline (a tricyclic antidepressant) or its metabolite 10-hydroxy amitriptyline were detected in serum samples across sites and in eggs from the St. Lawrence River. Similar to the behavioral effects elicited by SSRIs, acute and chronic exposure to low concentrations of amitriptyline caused zebrafish to spend more time near the top of the tank, in addition to chronic exposure causing elevated whole-brain norepinephrine and dopamine concentrations (Demin et al., 2017; Meshalkina et al., 2018). If similar effects are seen in lake sturgeon this could make them more vulnerable to predation or collisions with boaters, affect their ability to adequately feed, or possibly even alter their opportunity for successful reproduction, all potentially leading to population level effects.⁹⁵⁷

They cited the “limited research on the effects of long term-exposure to antibiotics on fish, however available studies suggest that antibiotics can also influence growth, development, reproduction, and behavior in fish” and concluded that “the prevalence of PBDEs across the Great Lakes basin and the potential for PBDEs to disrupt thyroid production and consequently the imprinting process in young sturgeon should be given careful attention by managers.”⁹⁵⁸

Great Lakes tributaries have also been studied. Data collected in 2010–2013, representing 41 percent of the inflow to the Great Lakes, found that on average, samples from urban watersheds had nearly four times the number of detected compounds and four times the total sample concentration.⁹⁵⁹ Findings were summarized as follows:

Water quality benchmarks for individual OWCs were exceeded at 20 sites, and at 7 sites benchmarks were exceeded by a factor of 10 or more. The compounds with the most frequent water quality benchmark exceedances were the PAHs benzo[a]pyrene, pyrene, fluoranthene, and anthracene, the detergent metabolite 4-

⁹⁵⁷ *Id.* at 7–8.

⁹⁵⁸ *Id.* at 8, 10 (citations omitted).

⁹⁵⁹ Austin K. Baldwin, *et al.*, *Organic contaminants in Great Lakes tributaries: Prevalence and potential aquatic toxicity*, 554-555 *Science of the Total Environment* 42–52 (2016), available at <https://www.sciencedirect.com/science/article/pii/S0048969716303485>.

nonylphenol, and the herbicide atrazine. Computed estradiol equivalency quotients (EEQs) using only nonsteroidal endocrine-active compounds indicated medium to high risk of estrogenic effects (intersex or vitellogenin induction) at 10 sites. EEQs at 3 sites were comparable to values reported in effluent.⁹⁶⁰

Similarly, a subsequent study on six Great Lakes tributaries examined over 2,250 resident and caged sunfish (*Lepomis ssp.*) for morphological and physiological endpoints and related them to CEC occurrence, which were both ubiquitous across the study and highest in their presence and concentrations in water and sediment in effluent dominated rivers and downstream of sewage treatment plants.⁹⁶¹ The authors concluded:

Canonical Redundancy Analysis revealed consistent patterns of biological consequences of CEC exposure across all six tributaries. Increasing plasma glucose concentrations, likely as a result of pollutant-induced metabolic stress, were associated with increased relative liver size and greater prominence of hepatocyte vacuoles. These indicators of pollutant exposure were inversely correlated with indicators of reproductive potential including smaller gonad size and less mature gametes. The current study highlights the need for greater integration of chemical and biological studies and suggests that CECs in the Laurentian Great Lakes Basin may adversely affect the reproductive potential of exposed fish populations.⁹⁶²

The ecosystem risk of the PPCP subset of CECs in the Great Lakes has been found to be of medium or high ecological risk; despite dilution, “the concentrations found in this study, and their corresponding risk quotient, indicate a significant threat by PPCPs to the health of the Great

⁹⁶⁰ *Id.* at 42–43.

⁹⁶¹ Linnea M. Thomas, *et al.*, *Contaminants of emerging concern in tributaries to the Laurentian Great Lakes: II. Biological consequences of exposure*, PLoS ONE 12(9) e0184725 (Sept. 27, 2017) available at <https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0184725>; see also Sarah M. Elliott, *et al.*, *Contaminants of emerging concern in tributaries to the Laurentian Great Lakes: I. Patterns of occurrence*, PLoS ONE 12(9): e0182868 (Sept. 27, 2017) available at <https://doi.org/10.1371/journal.pone.0182868>.

⁹⁶² *Id.* at 1/36.

Lakes, particularly near shore organisms.”⁹⁶³ Thirty-two PPCPs were detected in the water of Lake Michigan and 30 in sediments:

PPCPs were frequently detected in the water and sediments at the ng L⁻¹ level, including sites 3.2 km from shore in Lake Michigan at concentrations that are estimated to cause environmental concern. At the concentrations detected, medium or high risk was associated with twenty-four compounds in the final effluent, and fourteen were found to be of medium or high risk in Lake Michigan. The most frequently detected PPCPs were metformin, caffeine, sulfamethoxazole, and triclosan. Given the widespread detection of PPCPs, these pollutants are not ephemeral and pose an environmental risk to the sixth largest lake in the world. Therefore, high dilution is not adequate to mitigate the risk from this cocktail of PPCPs and the potential ecological risk for large lake systems is much higher than previously understood.⁹⁶⁴

Some of these results are illustrated by the following bar chart:⁹⁶⁵

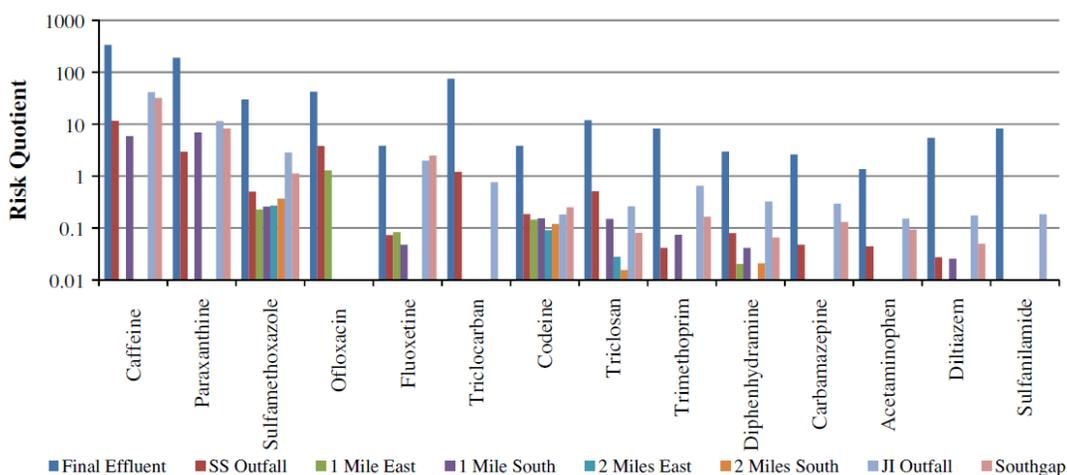


Fig. 3. Risk quotient for 14 PPCPs in wastewater effluent and in Lake Michigan (RQ > 1 is high risk, RQ from 0.1 to 1 is medium risk, and RQ < 0.1 is low risk).

Efforts to develop assessment tools for CECs in the Great Lakes have been led by FWS because despite the fact that “[m]any CECs are ubiquitous in surface waters. . . . [and that] [a]nalytical methods have been developed for detecting hundreds of individual CECs in ambient

⁹⁶³ Benjamin D. Blair, *et al.*, *Pharmaceuticals and personal care products found in the Great Lakes above concentrations of environmental concern*, 93 *Chemosphere* 2116–2123 (2013), available at <https://www.sciencedirect.com/science/article/pii/S0045653513010412>.

⁹⁶⁴ *Id.* at 2122.

⁹⁶⁵ *Id.* at 2121.

waters . . . few official agency guidelines or benchmarks exist for CECs in surface waters in the United States (U.S.) and few CECs are regulated as environmental contaminants.”⁹⁶⁶ The paper presented screening values for 14 substances: 4-Androstene-3, 17-dione; Bisphenol A; Carbamazepine; Citalopram; N,N-diethyl-meta-toluamide (DEET); Diphenhydramine; Estrone; Hexahydrohexamethylcyclopentabenzopyran (HHCB); Ibuprofen; Lidocaine; β -Sitosterol; Tris(2-butoxyethyl) phosphate (TBEP); Triclosan; and Venlafaxine.⁹⁶⁷

EPA plans to evaluate CECs in the Columbia River Basin. In a 2014 paper, outlining a research and monitoring strategy focusing on CECs, EPA summarized the basis of its concern:

A variety of CECs have been detected in the Columbia River Basin. Some examples are:

- PAHs, PCBs, and PBDEs were found throughout the lower River and in river water, sediment, and juvenile Chinook salmon. These contaminants are moving from river water and sediment into salmon prey and then into salmon tissue (LCREP 2007).
- In surficial bed sediments sampled from the lower Columbia River main stem and several tributaries, 49 different CECs were detected, with endocrine-disrupting compounds (contaminants that block or mimic hormones in the body and cause harm to fish and wildlife) detected at 22 of 23 sites sampled (Nilsen and others, 2013).
- A myriad of pharmaceuticals and personal care products were detected in the effluent from numerous WWTPs discharging to the Columbia River (Morace 2012).⁹⁶⁸

⁹⁶⁶ FWS, *Ecological Hazard Assessment of Contaminants of Emerging Concern in the U.S. Great Lakes Basin, Part B – Supplemental Document: Technical Resources for Ecological Hazard Assessments of Contaminants of Emerging Concern in Freshwater Fish* (Sept. 2019) at 1, available at <https://www.fws.gov/media/ecological-hazard-assessment-contaminants-emerging-concern-us-great-lakes-basin-part-b>.

⁹⁶⁷ See *id.* at 73.

⁹⁶⁸ EPA, *Columbia River Toxics Reduction Working Group: Strategy For Measuring, Documenting And Reducing Chemicals of Emerging Concern* (July 2014) (hereinafter “Columbia CEC Strategy”), available at <https://www.epa.gov/sites/default/files/2014-07/documents/columbia-river-cec-strategy-july2014.pdf>, at 2.

In addition to PBDEs, discussed in sub-section VII.E.3 *infra*, EPA noted that “[t]he presence of estrogen-like compounds in the waters of the Basin is evidenced by vitellogenin⁹⁶⁹ induction in juvenile salmonids (LCREP 2007). . . . Estrogen-like compounds in effluent from Portland’s WWTP (Morace 2012) are concentrated enough that the resulting estrogenicity in the Columbia River could be the equivalent of 1 ng/L estrogen—a concentration that could cause endocrine disruption in some aquatic species.”⁹⁷⁰ EPA noted that while pharmaceuticals are highly diluted and thus less often detected in mainstem Willamette and Columbia Rivers,

[i]n contrast, many more personal-care-product ingredients were detected in surficial-bed-sediment samples collected throughout the Lower Columbia River Basin, where endocrine-disrupting compounds were detected at 22 of 23 sites (Nilsen et al. 2007; Nilsen et al. 2014). Similarly, several personal-care-product chemicals were found in WWTP effluent (Morace 2012), which raises the question of which compounds may be partitioning into the biosolids during the treatment process and at what levels.”⁹⁷¹

Finally, EPA cited Washington’s having found perfluorinated compounds (“PFC”) at “(38 to 910 ng/g) in osprey eggs collected from the Lower Columbia River (Furl and Meredith, 2010).”⁹⁷² EPA summarized the concerns about CECs in the basin as “the contaminant and bioindicator results support the hypothesis that contaminants in the environment both correlate to bioaccumulation and cause genetic and reproductive impacts within the food web.”⁹⁷³ And,

⁹⁶⁹ Vitellogenin, an egg yolk protein normally produced in adult female fish, is an indicator of exogenous estrogen exposure when found in juvenile or male fish. *Id.* at 7.

⁹⁷⁰ *Id.* at 7. This trigger value used in this paper may very well have been too high. See Justin M. Conley, et al., *Occurrence and In Vitro Bioactivity of Estrogen, Androgen, and Glucocorticoid Compounds in a Nationwide Screen of United States Stream Waters*, 51 *Environ. Sci. Technol.* 4781-4791 (2017) at 4787 (citing 1 ng/L as a trigger value and reporting trigger values considerably lower and noting “the influence that in vitro-to-in vivo extrapolation of chemical potency has on the interpretation and use of in vitro bioassays for environmental monitoring.”). See also Justin M. Conley, et al., *Comparison of in vitro estrogenic activity and estrogen concentrations in source and treated waters from 25 U.S. drinking water treatment plants*, 579 *Science of the Total Environment* 1610-16-17 (2017).

⁹⁷¹ Columbia CEC Strategy, *supra* n. 968 at 7.

⁹⁷² *Id.*

⁹⁷³ *Id.* at 9.

despite finding low levels of pharmaceuticals in mainstem Columbia basin ambient waters, EPA concluded that they are of concern:

Pharmaceuticals, by intent, are biologically active, therefore, although their exact effects on wildlife are not yet fully documented, their presence in the environment would be expected to have ecological effects (Williams 2005). Pharmaceuticals and other CECs delivered through WWTP effluent can be considered to have “pseudo-persistence” because of the continual input of these compounds (Smtal 2008). The effects of continuous low-level exposure to these CECs, particularly during sensitive life stages, as well as effects of long-term exposure to these complex mixtures are further unknowns (Daughton and Ternes 1999; Han et al. 2010).⁹⁷⁴

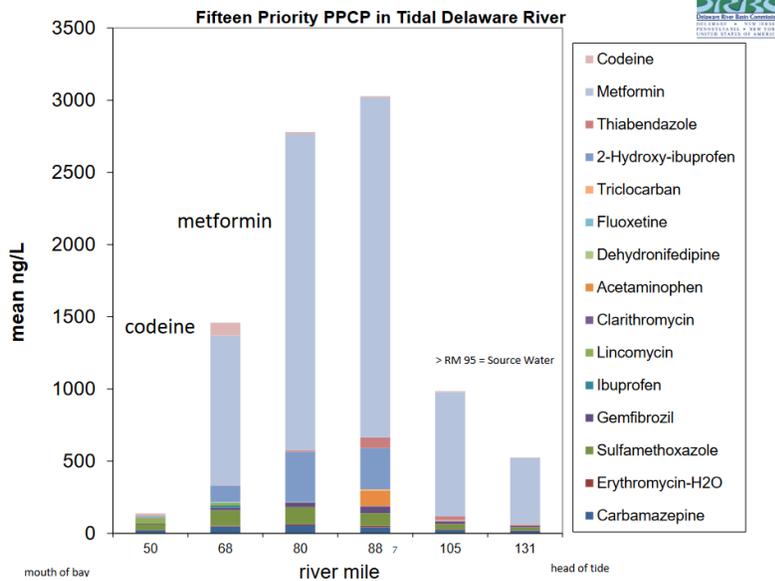
On the East coast, the Delaware River Basin Commission (“DRBC”)—covering parts of Delaware, New Jersey, Pennsylvania, and New York—has investigated CECs because they “persist in the environment and have been detected in people and other living organisms. Many of these compounds are currently unregulated and not routinely monitored.”⁹⁷⁵ The DRBC engaged in special studies and continues to perform “periodic monitoring for CECs in surface water, sediment, and fish tissue.”⁹⁷⁶ Samples from 2004, 2007, and 2009 looking at the PPCP subset of CECs demonstrated geographic variability as shown by the following graph:⁹⁷⁷

⁹⁷⁴ *Id.* at 9.

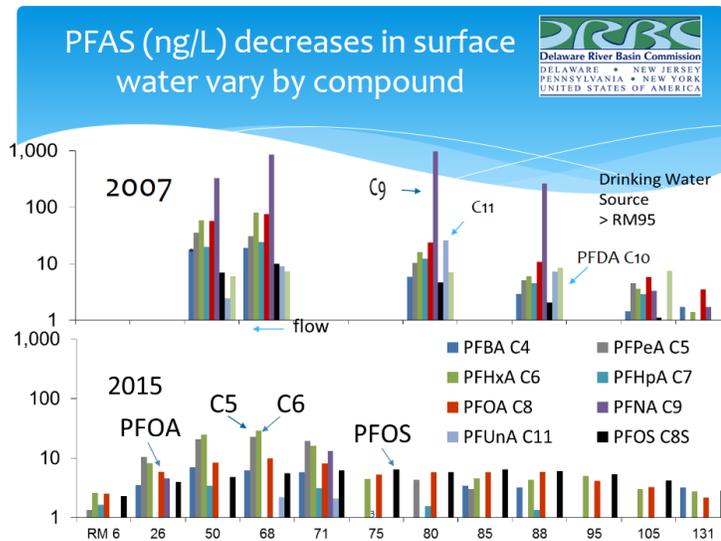
⁹⁷⁵ Delaware River Basin Commission, Water Quality, *Contaminants of Emerging Concern*, available at <https://www.nj.gov/drbc/programs/quality/cecs.html>.

⁹⁷⁶ *Id.*

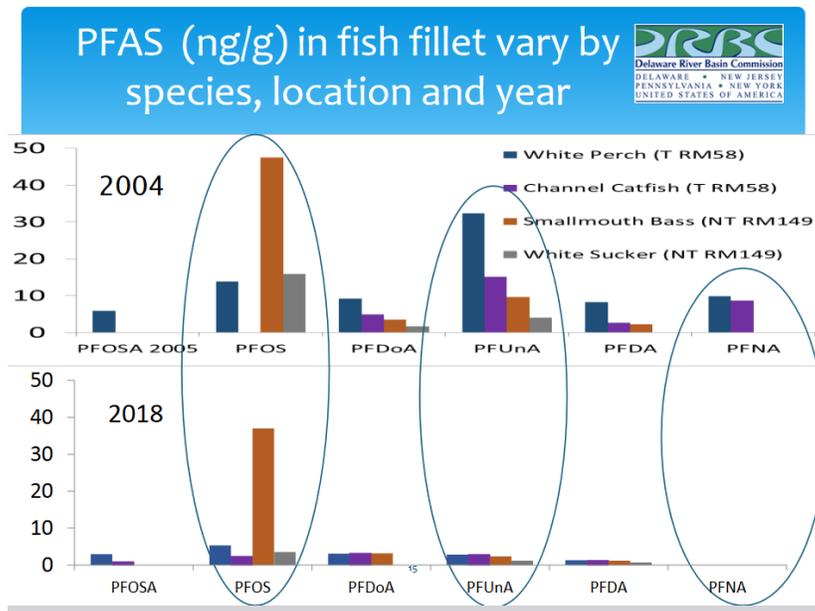
⁹⁷⁷ Delaware Lessons Learned, *supra* n. 675 at 7.



Similarly, monitoring for PFAS demonstrated geographic and species variability: ⁹⁷⁸



⁹⁷⁸ *Id.* at 13, 15.



A 2014–2014 DRBC study of PPCPs in Pennsylvania tributaries of the Delaware River identified “compounds such as ibuprofen, triclocarban and dehydronifedipine” as concerning when comparing environmental concentrations with predicted results, and expressing particular concern for exposure to pregnant women and children from drinking water and fish.⁹⁷⁹ These findings are illustrated by the following chart:⁹⁸⁰

⁹⁷⁹ Djordje Vilimanovic *et al.*, *Occurrence and aquatic toxicity of contaminants of emerging concern (CECs) in tributaries of an urbanized section of the Delaware River Watershed*, 7(4) AIMS Environmental Science 302-319 (July 9, 2020) at 314, 315, available at <https://www.aimspress.com/article/10.3934/environsci.2020019>.

⁹⁸⁰ *Id.* at 309; see also *id.* at 308 (“RR < 0.1 indicates minimal risk and the 1.0 > RR ≥ 0.1 indicates moderate concern”). “PNEC” stands for predicted no effect concentration.

Table 2. PPCP in the Pennsylvania tributaries of the Delaware River listed in order of relative risk.

Analyte	Number of analysis	Detection Frequency (%)	Concentration Range (ng/L)	Median concentration, (ng/L)	Maximum Concentration (ng/L)	PNEC, (ng/L)	RR
Triclocarban	60	100	6.3–265	36.7	265	190 ^a	1.395
Diphenhydramine	60	88	0.6–101	8.0	101	100 ^b	1.010
Carbamazepine	60	97	2.3–243	80.6	243	500 ^c	0.486
Erythromycin	60	32	0.2–58	7.5	58	300 ^d	0.193
Ibuprofen	60	97	3.7–152	19.8	152	1000 ^b	0.152
Clarithromycin	60	92	0.1–16	4.6	16	120 ^d	0.133
Sulfamethoxazole	20	90	1.0–24	5.8	24	200 ^b	0.120
Ranitidine	58	91	0.1–106	7.6	106	6200 ^e	0.017
Gemfibrozil	60	95	0.2–93	7.4	93	7800 ^f	0.012
Metformin	50	100	933–9258	3563	9258	1030000 ^b	0.009
Guanylyurea	39	85	2.9–620	45.7	620	160000 ^g	0.004
Thiabendazole	60	97	0.1–191	6.1	191	74000 ^h	0.003
Diltiazem	60	75	0.4–8.2	1.8	8.2	8200 ⁱ	0.001
Trimethoprim	60	88	0.1–63	7.9	63	120700 ⁱ	0.001
Dehydronifedipine	60	90	0.3–8.6	2.4	8.6	2899000 ^h	0.001<

Notes: ^a[27]; ^b[28]; ^c[29]; ^d[30]; ^e[31]; ^f[32]; ^g[33]; ^h[34]; ⁱ[35].

The DRBC concluded the following:

- Natural and synthetic hormones “have been ranked in the top chemicals in U.S. surface waters for potential ecological effects warranting further study in the Delaware River Basin.”
- The predominant PFAS is perfluorononanoic acid (PFNA) in surface water and perfluoroundecanoic acid (PFUDA) in fish tissue.
- While nonylphenol concentrations did not exceed EPA’s recommended criteria, “because of widespread occurrence in the environment and the evolving knowledge of ecotoxicity, NP and [nonylphenol ethoxylates] NPEOs should continue to be characterized as a contaminant of emerging concern in Delaware River Basin studies.”
- Because of the low levels of PBDEs found in water, future monitoring of PBDE in the Delaware River Basin should focus on bioaccumulation in fish tissue and other biota.⁹⁸¹

And the DRBC noted there was limited usefulness of the EPA recommended criteria for nonylphenol:

In interpreting the concentrations of NP in the environment with regard to the criteria, it should be noted that since studies in the literature that measured estrogenic effects by NP did not meet data quality for deriving criteria, they were not included in the calculation of the USEPA criteria for NP. However, chronic toxicity data used in the derivation of the criteria did include growth and

⁹⁸¹ Delaware River Basin Commission, *Contaminants of Emerging Concern in the Tidal Delaware River, Pilot Monitoring Survey 2007-2009* (July 2012, rev. Aug. 2013) at 44, available at <https://www.nj.gov/drbc/library/documents/contaminants-of-emerging-concernAug2013rev.pdf>.

reproduction endpoints. Therefore, to the extent that these chronic toxicity endpoints include the effect of endocrine disruption, the estrogenicity of NP is included in the derivation of the criteria. In short, upon development of standardized tests for estrogenicity, the criteria will certainly be revised.⁹⁸²

The report observed, as well, that published studies indicate that surface waters “containing between 1 to 10 µg/l [of nonylphenol] are at some risk and surface water at >10 µg/l are at a significant risk of environmental harm” and that “the lipophilic NP can bioaccumulate.”⁹⁸³

EPA and other agencies’ findings that very low concentrations of some of endocrine-disrupting compounds can significantly impact natural populations of aquatic vertebrates and human health addresses some of the primary considerations set out in Section 307(a)(1), namely their toxicity, potential for biomagnification, ubiquity in the environment, and their effect on aquatic species, including prey species. For these CECs to be included in the regulatory programs of the CWA, they must be placed on the Toxic Pollutants Lists.

2. Pharmaceuticals and Personal Care Products (PPCPs)

One subset of CECs is pharmaceuticals and personal care products (“PPCPs”). As EPA has observed, “[r]esearchers monitoring the environment find PPCPs nearly everywhere domestic wastewater is discharged. . . . The human health effects resulting from daily exposure to low concentrations of PPCPs are unclear, but there are some documented impacts to wildlife from PPCPs in the environment.”⁹⁸⁴ EPA has defined PPCPs as follows:

PPCPs include drugs made for humans and animals; they include prescription and over-the counter drugs. They also include diagnostic agents such as x-ray contrast media, nutraceuticals (bioactive chemicals in nutritional supplements), and excipients (inert ingredients such as pill coatings) (Motzer, 2006). The PPCP

⁹⁸² *Id.* at 42.

⁹⁸³ *Id.*, citing Vazquez-Duhalt, *et al.*, *Nonylphenol, an integrated vision of a pollutant*, 4(1) *Appl. Ecol. and Environ. Res.* 1-25 (2005).

⁹⁸⁴ Removal of PPCPs, *supra* n. 540, Appendix A. Pharmaceuticals and Personal Care Products in the Environment: A Literature Review at 55.

definition also includes illicit drugs, personal care products (chemicals in consumer products), and veterinary medicines (Daughton and Ternes, 1999).

Personal care products are items that individuals use every day to take care of themselves. They include a wide variety of products: shampoo, deodorant, toothpaste, lotions, make-up, aftershave lotions, hair dyes, anti-dandruff shampoos, teeth whiteners, sunless tanning products, colognes, and fragrances. There are over 10,500 different chemicals used in personal care products. Only 11% of these chemicals have been tested for human health safety in the United States.⁹⁸⁵

The fate and transport of an individual PPCP depends on its chemical structure.⁹⁸⁶ The primary sources are residences, hospitals and other medical facilities, manufacturers, illicit drug labs, livestock, pesticide use, aquaculture, pets, reclaimed water, and on-site septic systems, making sewage treatment plants significant “secondary sources.”⁹⁸⁷

In 2010, EPA reported on the nationwide extent of PPCPs in surface water:

From 1999 to 2000, the USGS (Kolpin et al., 2002; Barnes et al., 2002) conducted the first national assessment of pharmaceuticals in U.S. streams. This study sampled 139 streams in 30 states. This study also captured a variety of hydrogeologic, climatic, and land-use settings. Ninety-five PPCPs were analyzed, and 82 (86%) were detected in the aquatic environment. All of the 95 chemicals tested are used extensively by the general public. Eighty percent of the sites had at least 1 PPCP detected, and 75% of the sites had multiple PPCPs detected. Concentrations were low, generally in the $\mu\text{g}/\text{l}$ range. Standards have been established for only 14 of the compounds, and rarely were any of these standards exceeded. The lack of standards is due to the limited information about potential human and aquatic health effects.

Certain types of organic chemicals were detected more frequently than others. Steroids, non-prescription drugs, and insect repellent were the three groups most frequently detected during this study. Detergent metabolites, plasticizers, steroids, and non-prescription drugs were found at the highest concentrations.

The organic chemicals chosen for monitoring in this study were aimed at pharmaceuticals, personal care products, biogenic hormones, and other household chemicals released directly into the environment after wastewater treatment processes. The high level of occurrence indicates that many compounds are not

⁹⁸⁵ *Id.* at 56.

⁹⁸⁶ *Id.* at 57.

⁹⁸⁷ *Id.* at 58, fig. A-1 Environmental Fate of PPCPs.

sufficiently removed by the wastewater treatment processes. The presence of PPCPs in sediments is an area which was determined to need more attention.

The most frequently detected compounds are shown in Figure A-2. These include coprostanol, cholesterol, N,N-diethyltoluamide, caffeine, triclosan, tri(2-chloroethyl)phosphate, and 4-nonylphenol. Thirty-three of the 95 compounds monitored are endocrine-disrupting compounds, and all 33 were detected during the study.⁹⁸⁸

The most frequently detected compounds found in surface water in this reconnaissance study are shown in the graph below.⁹⁸⁹

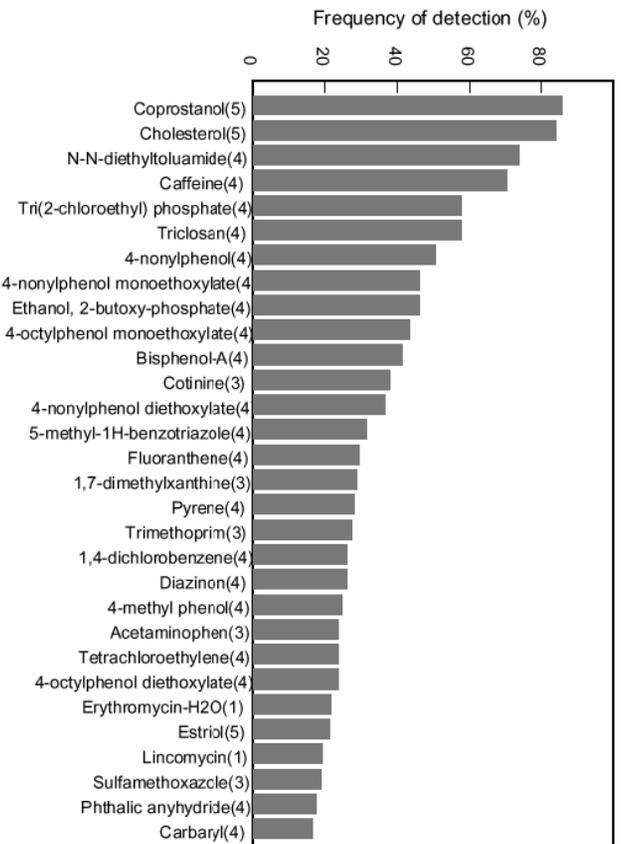


Figure A-2. Most frequently detected compounds in the National Reconnaissance Study of PPCPs in surface water conducted by the USGS (Kolpin et al., 2002; Barnes et al., 2002).

Subsequent to the U.S. Geological Survey's national assessment, researchers sampled for PPCPs upstream and downstream of sewage treatment plants in northern Arkansas, demonstrating that the majority of PPCPs come from such facilities.⁹⁹⁰

⁹⁸⁸ *Id.* at 59.

⁹⁸⁹ *Id.* at 60.

⁹⁹⁰ *Id.* at 61, Fig. A-3, citing Galloway, J.M., et al., *Occurrence of Pharmaceuticals and Other Organic Wastewater Constituents in Selected Streams in Northern Arkansas* (2004), USGS Virginia. Scientific Investigations Report 2005-5140.

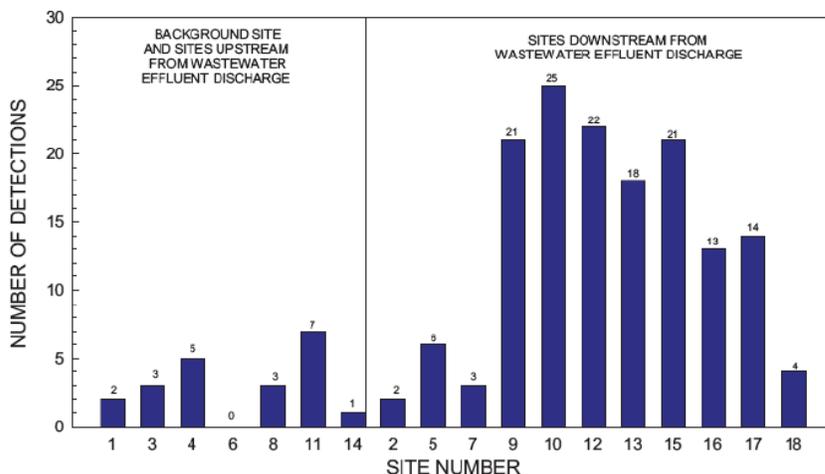


Figure A-3. Summary of pharmaceuticals and other organic wastewater constituents detected from selected sites in northern Arkansas, March and April 2004 (Galloway et al., 2005).

The most frequently detected PPCPs in this study were the following: caffeine, phenol, para-cresol, and acetyl hexamethyl tetrahydronaphthalene. A 2002 study of PPCPs in the Las Vegas Wash, Nevada found that carbamazepine, dehydronifedipine, acetaminophen, cimetidine, codeine, and diltiazem were detected in 83 percent of samples.⁹⁹¹ A 2005 study provided a detection frequency of PPCPs near 10 sewage treatment plants across the nation as follows: cotinine (92.5 %), cholesterol (90 %), carbamazepine (82.5 %), tonalide (AHTN) (80 %), tri(2-chloroethyl) phosphate (TCEP) (75 %), codeine (72.5 %), ethyl citrate (72.5 %), sitosterol (72.5 %), sulfamethoxazole (72.5 %), caffeine (70 %), ethanol, 2-butoxy-phosphate (70 %), N-N-diethyltoluamide (DEET) (70 %), tributylphosphate (70 %), benzophenone (67.5 %), diltiazem (67.5 %), 4-nonylphenol diethoxylate (62.5 %), 4-nonylphenol monoethoxylate (62.5 %), triclosan (62.5 %), coprostanol (60 %), trimethoprim (60 %), (dehydronifedipine (57.5 %), galaxolide (HHCB) (57.5 %), diphenhydramine (55 %), acetaminophen (50 %), diazinon (47.5 %), 5-methyl-1 H-benzotriazole (45 %), phenol (40 %), triphenyl phosphate (37.5 %), 1,7-

⁹⁹¹ *Id.* at 61.

Dimethylxanthine (35 %), 4-octylphenol diethoxylate (32.5 %), bisphenol A (30 %), 1,4-dichlorobenzene (27.5 %).⁹⁹² Additional PPCPs recommended for evaluation by additional studies include: 2,6-dimethylnaphthalene; 3,4-dichlorophenyl isocyanate; 4-methyl phenol; 4-nonylphenol; 4-octylphenol monoethoxylate; carbaryl; cimetidine; erythromycin; estriol; fluoranthene; fluoxetine; gemfibrozil; isophorone; lincomycin; miconazole; pentachlorophenol; phthalic anhydride; pyrene; ranitidine; salbutamol (albuterol); tetrachloroethylene; thiabendazole; tri(dichloroisopropyl)phosphate; warfarin.⁹⁹³

EPA and Washington reported several wildlife studies that have demonstrated the toxic effects of PPCPs including diclofenac in vultures (acute kidney failure) and dicofol in alligators (altered hormone concentrations and exhibited modified reproductive anatomy and function).⁹⁹⁴ Sexual disruption and reproductive problems have been widely reported in fish. For example, EPA reported a 2003 study on white sucker fish in Boulder Creek, Colorado caught both upstream and downstream of a sewage treatment plant that

analyzed effluent quality and found a number of endocrine-disrupting compounds including alkylphenols, bisphenol A, and reproductive steroids. They noted a number of effects in the downstream fish that were not present upstream. The male to female ratio upstream was roughly equal, but downstream of the WWTP, the ratio was 90% female and 10% male. The remaining downstream males all showed significant signs of abnormal reproductive organs. Additionally, the downstream female population also exhibited reproductive abnormalities. Intersex fish and elevated vitellogenin in juvenile fish were only detected downstream of the WWTP outfall.⁹⁹⁵

⁹⁹² *Id.* at 81, Table A-10, citing Glassmeyer, S.T., *et al.*, *Transport of Chemical and Microbial Compounds from Known Wastewater Discharges: Potential for Use as Indicators of Human Fecal Contamination*, 39 Environmental Science and Technology No. 14, 5157-5169 (2005)

⁹⁹³ *Id.* at 83–84, Table A-12 (Recommended pharmaceutical indicator parameters from reviewed literature)

⁹⁹⁴ *Id.* at 62-63.

⁹⁹⁵ *Id.* at 62, citing Vajda, A.M., *et al.*, *Intersex and Other Forms of Reproductive Disruption in Feral White Sucker (Catostomus commersoni) Downstream of Wastewater Treatment Plant Effluent in Boulder, Colorado* (2003).

And EPA described a 1998 study that

documented widespread sexual disruption to wildlife from exposure to ambient (background) levels of “estrogenic constituents of sewage effluents” in rivers. The study correlated reproductive and developmental effects from exposure to hormonally active substances discharged from WWTPs. Intersex fish were found at all sites including the control site, suggesting that a low incidence of intersexuality may be natural. A much higher incidence of intersex fish was detected at sites impacted by sewage effluent, indicating that the effluent may be causing sexual disruption to wild fish.⁹⁹⁶

A later study, done in 2007, looked at PPCPs and other CECs in the Lower Columbia River in water column, suspended and bed sediment, juvenile salmon tissue, stomach contents, bile, and plasma, along with semi-permeable membrane devices.⁹⁹⁷ The report summarized its key findings as follows:

- PCBs, PAHs, organochlorine pesticides, and PBDEs were found at all sites.
- Most toxic contaminants were detected at low concentrations.
- The most frequently detected pesticides were atrazine, simazine, and metolachlor, which are suspected hormone disruptors. These pesticides were detected at quantifiable concentrations.
- Caffeine was present at all sites. Other frequently detected wastewater compounds were bisphenol A (a plasticizer), HHCB (a synthetic musk), trimethoprim (an antibiotic for people and fish), and anhydroerythromycin (a breakdown product of the antibiotic erythromycin, used for people and animals).

* * *

- The Willamette River is major source of toxic contaminants. Pesticides were found most often and at the highest concentrations at the Lower Willamette site, PAH and PBDE levels at the site were high, and several wastewater compounds were detected there, including the suspected hormone disruptors bisphenol A, HHCB, and tri(2-chloroethyl)phosphate.⁹⁹⁸

⁹⁹⁶ *Id.* at 62, citing Jobling, S., *et al.*, *Widespread Sexual Disruption in Wild Fish*, 32 *Environmental Science and Technology*, 2498-2506 (1998).

⁹⁹⁷ 2007 Columbia Sampling, *supra* n. 178 at 4.

⁹⁹⁸ *Id.* 35-36.

The concurrent sampling of salmon in the area supported a conclusion that “[t]he lower Columbia River is the most likely source of PBDE exposure for juveniles[.]”⁹⁹⁹ Harmful levels of PPCPs were found in salmon blood, as the report described:

To look for signs of exposure to pharmaceuticals and other wastewater compounds in juvenile salmon, blood samples were screened for vitellogenin, a yolk protein that indicates exposure to estrogen-like compounds, such as certain pharmaceuticals and personal care products. Normally vitellogenin is present only in adult female fish.

Vitellogenin was found in blood samples of juvenile salmon from the Lower Willamette and Confluence sites[.] This is consistent with the high number of detections of compounds at the Lower Willamette site, including bisphenol A and other compounds with known or suspected estrogenic activity.¹⁰⁰⁰

The known adverse effect of many pharmaceuticals and personal care products on aquatic designated uses meets the requirements of CWA Section 307(a)(1) for their inclusion on the Toxic Pollutants Lists and ensuring future regulation.

3. Polybrominated Diphenyl Ethers (PBDE)

Among the CECs, polybrominated diphenyl ethers (“PBDE”) are members of a broad class of brominated chemicals used as flame retardants; three main types are used in consumer products, two of which have been discontinued, leaving Deca-BDE as the sole form produced after 2004.¹⁰⁰¹ As described by NMFS in 2012, PBDEs:

have been used as additive flame-retardants in many products including electronics, textiles, and plastics. Additive flame-retardants can readily disassociate from the products they are added to and discharge into the environment. Due to the increase in fire regulations in many countries, the use of PBDEs has increased in the last few decades. PBDEs have been identified as a growing concern and have a ubiquitous distribution with increasing levels found

⁹⁹⁹ *Id.* at 49.

¹⁰⁰⁰ *Id.* at 49-50.

¹⁰⁰¹ Washington Department of Ecology/Washington State Department of Health, *Washington State Polybrominated Diphenyl Ether (PBDE) Chemical Action Plan: Final Plan* (Jan. 19, 2006) (hereinafter “PBDE Action Plan”) at viii.

in various matrices including surface water, sewage sludge, sediment, air, and biota (Hale et al. 2003, Hites 2004). PBDEs are structurally comparable to PCBs and share some similar toxicological properties (Hooper and McDonald 2000).

* * *

Although specific regional data is limited for PBDE levels, the environmental levels of a few PBDE congeners appear to have surpassed PCBs in some areas in North America (Hale et al. 2003, Ross et al. 2009). Recent studies have documented relatively high concentrations of PBDEs in Southern Resident killer whales (Krahn et al. 2007a, 2009, Mongillo 2009). Although PBDE levels in the whales are lower than PCBs or DDTs (Krahn et al. 2007a, 2009), concern is growing because PBDE exposure and accumulation will likely continue in the future increasing the risk to the health of the killer whales. Several other marine species have recently experienced an almost exponential increase in PBDE concentrations (e.g., Ikonomou et al. 2002, Lebeuf et al. 2004).¹⁰⁰²

NMFS reported that current levels of PBDE in the endangered Southern Resident killer whales have been found in the range of 199–2,745 ng/g wet weight as compared to “threat levels” determined for grey seals at concentrations of 170–460 ng/g lipid wet in blubber.¹⁰⁰³ NMFS has focused on the effect of PBDEs on threatened and endangered salmonids, which themselves are key prey of the Southern Resident killer whales. NMFS scientists have shown that Chinook salmon exposed to PBDEs caused “reduced survival during challenge with the pathogenic marine bacteria *Listonella anguillarum*” and altered macrophage function causing them to conclude that “important physiological functions of health and survival may be altered in fish from Puget Sound and the Columbia River exposed to BDE-47 and BDE-99.”¹⁰⁰⁴ NMFS has evaluated PBDE assimilation efficiency in juvenile Chinook, allowing for the ability to model

¹⁰⁰² NMFS Oregon Toxics BiOp, *supra* n. 718 at 81–82.

¹⁰⁰³ *Id.* at 540, Table 2.8.1.

¹⁰⁰⁴ Mary R. Arkoosh, et al., *Dietary Exposure to Individual Polybrominated Diphenyl Ether Congeners BDE-47 and BDE-99 Alters Innate Immunity and Disease Susceptibility in Juvenile Chinook Salmon*, 49 Environmental Science and Technology 6974–6981, 6979 (2015), available at <https://pubs.acs.org/doi/10.1021/acs.est.5b01076>.

contaminant bioaccumulation in exposed organisms and food webs.¹⁰⁰⁵ And NMFS scientists studied salmon fed five environmentally relevant concentrations of PBDE congeners finding that the most predominant found in salmon— BDE-47 (2,20,4,4'-tetrabromodiphenyl ether) and BDE-99 (2,20,4,40,5-pentabromodiphenyl ether)—affected thyroid hormones in the fish and concluding that PBDE-caused “changes in thyroid hormone levels occur that may have serious impacts on juvenile fish health and survival.”¹⁰⁰⁶

EPA has evaluated PBDEs because “some of the component congeners are persistent, bioaccumulative and toxic and intends to initiate a number of actions to limit the exposure and release of PBDE congeners and/or articles to which they have been added.”¹⁰⁰⁷ These proposed actions are pursuant to the Toxic Substances Control Act (“TSCA”) wherein EPA concluded:

Neurobehavioral effects was identified as the critical endpoint of concern for each of the four congeners (EPA, 2008a-d). The protocols for the studies were unique and did not conform to health effects test guidelines for neurotoxicity. For decaBDE, EPA also proposed that the data support a finding of “suggestive evidence of carcinogenic potential” (EPA, 2008d).¹⁰⁰⁸

With regard to non-human species, EPA cited Environment Canada’s conclusion:

[T]he greatest potential risks from PBDEs in the Canadian environment are the secondary poisoning of wildlife from the consumption of prey containing elevated concentrations of PBDEs and effects on benthic organisms that may result from elevated concentrations of certain PBDEs in sediments (Environment Canada, 2006). In a more recent report, Environment Canada has also concluded that decaBDE specifically is available for uptake in organisms and may accumulate to

¹⁰⁰⁵ Joseph P. Dietrich, *et al.*, *Assimilation Efficiency of PBDE Congeners in Chinook Salmon*, 40 *Environmental Science Technology* 3878–3886, 3884 (2015), available at <https://pubs.acs.org/doi/10.1021/es5057038>.

¹⁰⁰⁶ Mary R. Arkoosh, *et al.*, *Alteration of thyroid hormone concentrations in juvenile Chinook salmon (*Oncorhynchus tshawytscha*) exposed to polybrominated diphenyl ethers, BDE-47 and BDE-99*, 171 *Chemosphere* (2017) at 7, available at <https://www.sciencedirect.com/science/article/abs/pii/S0045653516317477?via%3Dihub>.

¹⁰⁰⁷ EPA, *Polybrominated Diphenyl Ethers (PBDEs) Action Plan* (Dec. 30, 2009) at 1.

¹⁰⁰⁸ *Id.* at 5.

high and potentially problematic levels in certain species such as birds of prey or mammalian predators (Environment Canada, 2009a).¹⁰⁰⁹

And, EPA described the state of science on these species:

Laboratory studies have shown that congeners associated with c-pentaBDE and octaBDE are capable of producing adverse effects in a variety of organisms including birds, mammals, and fish. In some cases these effects were observed at exposures levels similar to levels found in the environment. American kestrels and chickens exhibited adverse effects in laboratory studies when exposed to levels of c-pentaBDE and c-octaBDE similar to those which have been observed in monitoring studies conducted in San Francisco Bay and the Great Lakes (McKernan et al., 2009). Adverse effects included histopathological changes in immune organs, altered reproductive behavior and decreased embryo survival and decreased hatching rates. Zhang et al. (2009) reported that c-pentaBDE produced adverse reproductive and developmental effects in ranch mink. Zhang et al. (2009) also conducted biomonitoring of wild mink from the Great Lakes region and concluded that margins of safety for mink are small and that mink from the Hamilton Harbor exceeded the no observed effect concentrations. Timme-Laragy et al. (2006) reported that c-pentaBDE produced developmental and behavioral effects of fish embryos including spine curvature and hatching delay.¹⁰¹⁰

Citing “[a]vailable data also indicate that tetra-, penta-, and hexa-BDE are highly bioaccumulative” and “decaBDE likely contributes to the formation of bioaccumulative and/or potentially bioaccumulative transformation products such as lower brominated PBDEs in organisms and in the environment,”¹⁰¹¹ for species, EPA concluded:

The food chain is likely the largest contributor to environmental exposures with PBDE depositing in soil and water where fish and benthic organisms are initially exposed. Biomagnification occurs as predators up the food chain ingest the accumulated concentrations of PBDEs from their prey (Chen et al., 2007; Voorspoels et al., 2007; Shaw and Kannan, 2009; Stapleton and Baker, 2003). In some studies evidence has been provided that the concentrations of PBDE in biota have doubled every 3 to 6 years, the doubling time depending on species, life stage, and location. PBDE concentrations in marine biota in North America are the highest in the world and are increasing (Shaw and Kannan, 2009). After reviewing the available information, EPA has concluded that the extent of accumulation of congeners is directly related to PBDE levels in diet. Observed

¹⁰⁰⁹ *Id.* at 6.

¹⁰¹⁰ *Id.* at 6.

¹⁰¹¹ *Id.* at 7.

differences in PBDE congener profiles in marine mammals from California, Alaska, and the Gulf of Mexico indicate that diet is a significant source of PBDE exposure in marine wildlife (Shaw and Kannan, 2009).¹⁰¹²

Similarly, EPA concluded that for PBDE exposure via contaminated water, “populations identified with potentially high exposures are subsistence fishermen who consume PBDE-contaminated fish and Native Americans who reside in Arctic regions and consume whale and seal blubber (ATSDR, 2004).”¹⁰¹³

Washington State has issued a chemical action plan for PBDEs issued as a part of a strategy to reduce persistent, bioaccumulative toxics (Dec. 2000).¹⁰¹⁴ This plan found that “[t]here is already a reservoir of PBDEs in humans and in the environment,” noting that “PBDEs have been found in fish, polar bears, grizzly bears and Puget Sound orcas” as well as peregrine falcon, bald eagle, herring gull, and heron eggs; and harbor seals, ringed seals, and beluga whale.¹⁰¹⁵ In 2001 alone, almost 70,000 metric tons of PBDEs were produced globally, almost half of which was used in products sold in the U. S. and Canada,” and “[w]hile levels of PBDEs found in breast milk in the U.S. are not yet at a level of concern, levels in U.S. women are 10 to 100 times that found in women in Europe,” as Washington illustrated by the following graph.^{1016,}

¹⁰¹⁷

¹⁰¹² *Id.* at 7–8.

¹⁰¹³ *Id.* at 9.

¹⁰¹⁴ See Washington Department of Ecology, *Proposed Strategy to Continually Reduce Persistent, Bioaccumulative Toxics (PBTs) in Washington State* (Dec. 2000), available at <https://apps.ecology.wa.gov/publications/documents/0003054.pdf>; see also PBDE Action Plan, *supra* n. 1004 at vii.

¹⁰¹⁵ *Id.* at vii–viii, 11, 26, 28 (Table 8).

¹⁰¹⁶ *Id.* at viii.

¹⁰¹⁷ *Id.* at 13, Fig. 3.

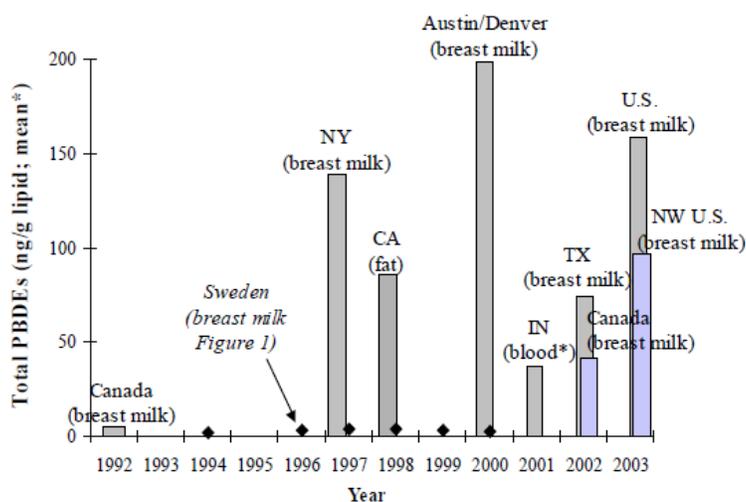


Figure 3. PBDEs in Breast Milk and Fat Samples Around the World⁵⁴

*Note: all total PBDE values are means, except levels for data from Indiana (IN) which are a median.

Ecology summarized the human health and environmental effects of PBDE as follows:

There are potentially serious health and environmental consequences as the amounts of PBDEs increase, such as neurotoxicity (i.e. effects to neurological development from exposures to unborn and newborn infants), leading to impacts on behavior, learning and memory. Other health effects may include bone malformations, reproductive impacts, and liver disorders.¹⁰¹⁸

Moreover, the state reported that Deca-BDE poses a unique problem in that “[l]aboratory studies indicate that the breakdown of Deca-BDE takes place through exposure to sunlight and through biological activity. Therefore, the Deca-BDE that is already in the environment is likely to be a long-term source of the more toxic forms of PBDEs long into the future.”¹⁰¹⁹ Ecology concluded that in evaluating the relevance of animal toxicity studies to human health implications,

[b]ody burden (i.e. accumulated amount of PBDEs in the body) is a better measure than daily intake when comparing rodent and human exposures. Body burdens will vary depending on the type of PBDE, the amount and duration of exposure, as well as on individual differences in absorption, metabolism and excretion. One recent report suggests that after adjusting for PBDE body burdens

¹⁰¹⁸ *Id.*; see also *id.* at 20–23 (summarizing animal toxicity studies).

¹⁰¹⁹ *Id.* at ix–x; see also *id.* at 35–40 (extended discussion of degradation of Deca-BDE).

between rodents and humans, high-end human exposures appear to be approaching toxic effects levels observed in animal studies, mainly for Penta-BDE associated congeners. A follow-up report suggests that the estimated daily intakes of women at the high end (95th percentile) of exposures currently exceed effects levels observed in animal studies for the most sensitive health endpoint (reproductive changes).¹⁰²⁰

In the aquatic environment, Ecology summarized studies as “indicat[ing] that PBDEs are ubiquitous in sediment and biota, and that their levels appear to be increasing rapidly.”¹⁰²¹ For example, the agencies reported that

PBDEs have been detected in sediment and soil in North America. Song et al. took sediment cores in 2001 and 2002 in Lake Superior at six locations away from lakeshores. In contrast to recent declining or level-off trends in PCB fluxes, the sedimentary records of PBDEs generally show a significant increase in recent years.¹⁰²²

Ecology also reported that

[t]emporal trends indicate increasing levels of PBDEs in animals. Ikonomou et al. measured the blubber of Arctic male ringed seals over the period 1981 to 2000. Mean total PBDE concentrations increased exponentially from approximately 0.6 µg/kg lipid in 1981 to 6.0 µg/kg lipid in 2000. Between 1989 and 1998, PBDE concentrations in tissue from harbor seals in San Francisco Bay doubled every 1.8 years. Lebeuf et al. measured PBDEs in blubber from beluga whales in the St. Lawrence Estuary in Canada for the period 1988 to 1999. Total PBDEs increased exponentially over the period, with a doubling period of no longer than three years.¹⁰²³

Despite its not being on the Toxic Pollutants Lists, EPA has placed a priority on investigating the presence of PBDEs in aquatic environments. For example, in a now decade-old EPA report on the Columbia River, the agency’s evaluation was limited to four toxic

¹⁰²⁰ *Id.* at 24 (internal citations omitted).

¹⁰²¹ *Id.* at 24.

¹⁰²² *Id.* at 26.

¹⁰²³ *Id.* at 27 (internal citations omitted).

contaminants—mercury, DDT, PCBs, and PBDEs—only the last of which is not on the Toxic Pollutants Lists. EPA noted:

These contaminants are of primary concern because (1) they are widely distributed throughout the Basin; (2) they may have adverse effects on wildlife, fish, and people; (3) they are found at levels of concern in many locations throughout the Basin; and (4) there is an opportunity to build on current efforts to reduce these contaminants within the Basin.”¹⁰²⁴

And EPA went on to say that

PBDEs, like PCBs, remain in the environment for a long time. PBDEs accumulate in all animals, but the concentrations continue to increase as an animal ages. However, unlike PCBs, EPA does not currently regulate PBDEs and only recently published a standard method for measuring PBDEs in environmental samples.¹⁰²⁵

EPA believes that discharges of treated sewage and disposal of sewage sludge onto land are both possible sources of PBDEs in Columbia River surface waters, citing “[a] study of PBDE contamination in the Canadian portion of the Columbia River found a correlation between high PBDE levels and areas where septic systems were concentrated near the River.”¹⁰²⁶ And EPA reported on a study by the Washington Department of Ecology on PBDE concentrations in sucker, mountain whitefish, and rainbow trout in the Spokane River in 1996, 1999, and 2005, concluding that “PBDE levels in these species are increasing in most reaches of the Spokane River. The most dramatic increases were found in mountain whitefish downstream from the Spokane metropolitan area,”¹⁰²⁷ as EPA illustrated by the following graph:¹⁰²⁸

¹⁰²⁴ EPA, *Columbia River Basin: State of the River for Toxics – January 2009* at 7 (2009) (hereinafter “Columbia Toxics Report”) available at

https://www.epa.gov/sites/default/files/documents/columbia_state_of_the_river_report_jan2009.pdf.

¹⁰²⁵ *Id.* at 26.

¹⁰²⁶ *Id.* at 26, citing Rayne, S., *et al.*, (2003), *Rapidly increasing brominated diphenyl ethers concentrations in the Columbia River System from 1992 to 2000*, 37(13) *Environmental Science and Technology* 2847-2854 (2003).

¹⁰²⁷ Columbia Toxics Report, *supra* n. 1024 at 27.

¹⁰²⁸ *Id.* at 27, fig. 5.17.

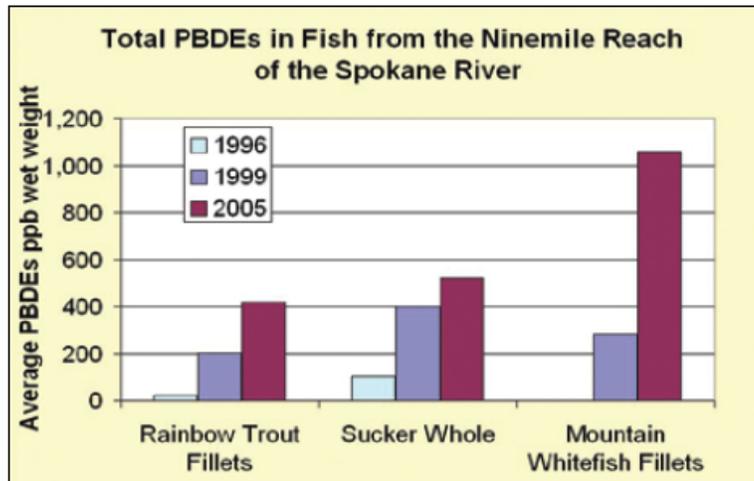


Figure 5.17: PBDE levels in Spokane River fish have increased since 1996.

EPA further concluded that studies

show that PBDEs are not only accumulating in larger fish but are being taken up by juvenile salmon as well.

In 2005, PBDEs were detected in all Asian clams collected from 36 stations throughout the Lower Columbia River. The Lower Columbia appears to be an important source of PBDEs for salmon on their migration to the ocean based on the difference in PBDE concentrations in juvenile salmon above and below Bonneville Dam (Figure 5.18).¹⁰²⁹

This finding was illustrated by a graph demonstrating significant contributions to the Lower Columbia River PBDE levels from the highly urbanized Willamette River.¹⁰³⁰

¹⁰²⁹ *Id.* at 27, citing Johnson, A., et al., *PBDE Flame Retardants in Washington Rivers and Lakes: Concentrations in Fish and Water, 2005-06*, Washington Department of Ecology Publication No. 06-03-027 (2006); 2007 Columbia Sampling, n. 178; Timothy Sherman, et al., *Corbicula fluminea: a potential freshwater bioaccumulative test species*, USACE Portland District and the Engineer Research Development Center (poster) (2007) (see Timothy Sherman, et al., *Corbicula fluminea as a Bioaccumulation Indicator Species: A Case Study at the Columbia and Willamette Rivers* (June 2009), available at <https://apps.dtic.mil/sti/pdfs/ADA507673.pdf>).

¹⁰³⁰ Columbia Toxics Report, *supra* n. 1024 at 27, fig. 5.18.

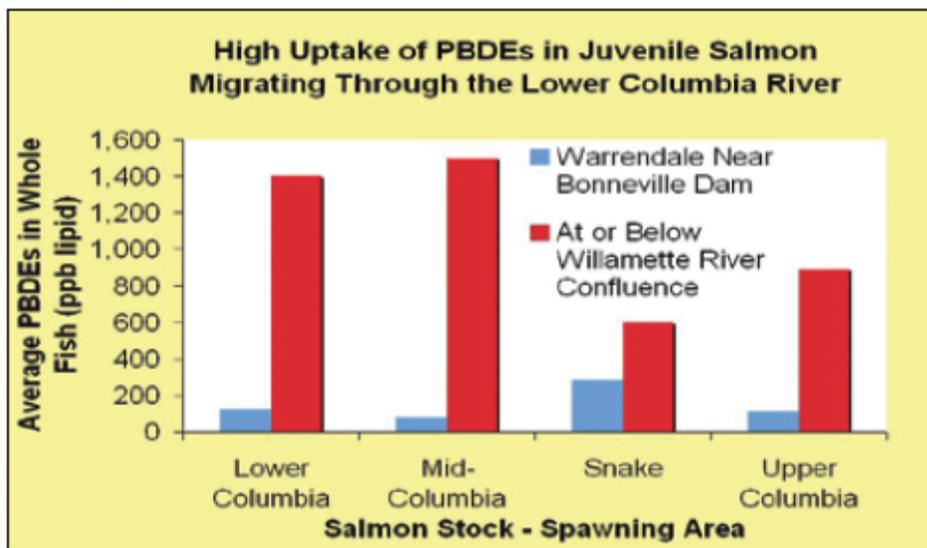


Figure 5.18: Migrating juvenile salmon, regardless of where they began their migration, consistently show higher levels of PBDEs when captured in the Lower Columbia River below the Bonneville Dam.

EPA also indicated that in addition to “rapid increases” where data have been collected, PBDEs are increasing in resident fish and predatory birds, such as bald eagle and osprey.¹⁰³¹ Yet PBDEs are notably absent from the list of TMDLs clean-up plans for toxics that EPA asserts have been completed or are under development to address contamination in the Columbia River Basin.¹⁰³²

EPA’s report on the Columbia River also demonstrates that PBDEs are not part of states’ monitoring plans but, rather, have been evaluated through special studies, possibly due to PBDEs’ not being on the Toxic Pollutants Lists. The following map shows the difference between Washington, which has sought information on PBDEs in the aquatic environment, as compared to Oregon and Idaho, which have not¹⁰³³:

¹⁰³¹ *Id.* at 29.

¹⁰³² *Id.* at 31.

¹⁰³³ *Id.* at 26.

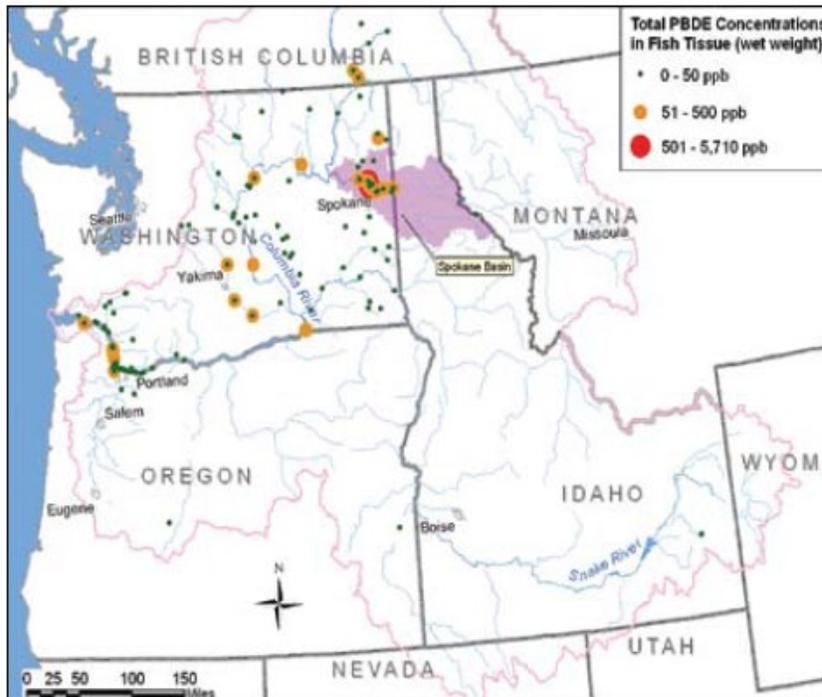


Figure 5.16: PBDEs are being detected and are increasing in fish in the Columbia River Basin. There is no information about PBDE levels in fish from waters that are unmarked on the map.

In a subsequent 2014 report on CECs in the Columbia River Basin, EPA reported that:

PBDE body burdens in mountain whitefish, a popular sport fish in the Upper Columbia River, were doubling every 1.6 years from 1992 to 2000—faster than anywhere else reported worldwide (Rayne et al. 2003). Since this finding, PBDEs have been monitored in water, sediment, and tissues throughout the lower Basin (Johnson et al. 2006, LCREP 2007, Nilsen et al. 2014, Alvarez et al. 2014), where they are ubiquitous. Several PBDE congeners biomagnified through several levels of the Columbia River food web (Nilsen et al. 2014), and concentrations in osprey eggs increased progressively from rural Umatilla to downstream of Portland (Henny et al. 2011). In a 2011 study, persistent, bioaccumulative, toxic chemicals, including flame retardants, were found in virtually all lower Columbia River resident fish (carp and largescale suckers) examined (Johnson and Friese 2012).¹⁰³⁴

Regarding the effects of PBDEs on designated uses, EPA cited studies showing that increasing concentrations of PBDE are adversely affecting wildlife including reproductive success in

¹⁰³⁴ Columbia CEC Strategy, *supra* n. 968 at 6–7.

osprey,¹⁰³⁵ changes in disease resistance in juvenile Chinook salmon,¹⁰³⁶ changes to bio-indicators in osprey,¹⁰³⁷ and effects on thyroid hormones of bald eagles.¹⁰³⁸

In 2017, EPA also cited PBDE as a threat to Columbia River species and water quality in a study sampling 42 sites along the Mid-Columbia River in 2008–2009.¹⁰³⁹ Levels of PBDE exceeded the screening values established for American kestrels in 5.5 percent of the reach studied.¹⁰⁴⁰ It also compared the mean concentrations of PBDEs in whole small fish for five mid-continent large river reaches, demonstrating that some U.S. rivers are far more contaminated by PBDEs than others.¹⁰⁴¹

Table 14. Comparison of small whole fish mean chemical concentrations (ng/g ww) for five Mid-continent large river reaches from Blocksom et al. (2010) and the MCR mean reported at reach-scale. Standard error of mean in parens.

River reach	DDT total	Dieldrin	Chlordane total	PCB total	PBDE total
Upper Mississippi	6.57 (0.37)	3.10 (0.18)	2.31 (0.23)	19.67 (0.93)	5.31 (0.39)
Impounded Mississippi	6.54 (0.37)	2.66 (0.11)	1.39 (0.14)	7.41 (0.84)	4.43 (0.28)
Missouri	5.47 (0.38)	3.19 (0.25)	4.54 (0.40)	7.41 (0.74)	12.72 (2.08)
Lower Missouri	6.14 (0.43)	3.59 (0.27)	5.14 (0.46)	8.43 (0.85)	14.18 (2.39)
Ohio	15.6 (0.62)	4.75 (0.32)	19.41 (0.73)	90.31(3.66)	28.1 (1.47)
Mid-Columbia	86.96 (3.00)	0.57 (0.01)	1.36 (0.11)	13.91 (0.30)	6.23 (0.12)

¹⁰³⁵ *Id.* at 8, citing C.J. Henny et al., *Polybrominated diphenyl ether flame retardants in eggs may reduce reproductive success of ospreys in Oregon and Washington, USA*, 18 *Ecotoxicology* 802-813 (2009) (hereinafter “Henny 2009”).

¹⁰³⁶ Columbia CEC Strategy, *supra* n. 968 at 12, citing M.R. Arkoosh, et al., *Disease Susceptibility Of Salmon Exposed To Polybrominated Diphenyl Ethers (PBDEs)* 98 *Aquatic Toxicology* 51-59 (2010).

¹⁰³⁷ *Id.* at 14, citing Henny 2009, *supra* n. 1035; C.J. Henny et al., *Wastewater dilution index partially explains observed polybrominated diphenyl ether flame retardant concentrations in osprey eggs from Columbia River Basin, 2008-2009*, 20 *Ecotoxicology* 682-697 (2011); and Furl C and Meredith C. *Perfluorinated Compounds in Washington Rivers and Lakes* (2010), available at <https://fortress.wa.gov/ecy/publications/summarypages/1003034.html>.

¹⁰³⁸ Columbia CEC Strategy, *supra* n. 968 at 15, citing L.S. Cesh LS, et al., *Polyhalogenated aromatic hydrocarbons and metabolites: Relation to circulating thyroid hormone and retinol in nestling bald eagles (Haliaeetus leucocephalus)* 29 *Environ Toxicol Chem.* 1301-1310 (2010).

¹⁰³⁹ EPA, *Mid-Columbia River Fish Toxics Assessment EPA Region 10 Report* (March 2017) (hereinafter “Mid-Columbia Report”).

¹⁰⁴⁰ *Id.* at 45. (The screening value was based on Environment Canada, *Canadian Environmental Protection Act, 1999 federal environmental quality guidelines for polybrominated diphenyl ethers (PBDEs) (2013)*, available at <https://www.ec.gc.ca/ese-ees/default.asp?lang=En&n=05DF7A37-1.>)

¹⁰⁴¹ Mid-Columbia Report, *supra* n. 1039 at 56, table 14.

In a further comparison with somewhat higher national PBDE levels for fish fillets evaluated for human health implications, EPA noted that concentrations of total PBDEs in the Mid-Columbia are “very similar (9.3 vs 11.6 ng/g)” to unpublished data from the National Rivers and Streams Assessment (NRSA) 2013–2014 study.¹⁰⁴²

For Puget Sound, the Washington Department of Ecology has reported particularly high levels of PBDEs:

PCBs and polybrominated diphenyl ethers (PBDEs) in whole body samples of individual summer/fall Chinook salmon from Puget Sound were 2 to 6 times more contaminated with PCBs and 5 to 17 times more contaminated with PBDEs than other populations of Chinook salmon from the Pacific West coastal areas.¹⁰⁴³

Notwithstanding this concern about PBDE contamination in Washington waters, the state processed data for only five CWA Section 303(d) assessment units, all on the Spokane River in the Columbia River basin.¹⁰⁴⁴ In each of these 303(d) findings that water quality standards have been violated, Ecology based its determination on the fact that the “Washington State Department of Health issued a fish consumption advisory for PBDEs in the Spokane River, from Upriver Dam to Nine Mile Dam.”¹⁰⁴⁵ In other words, the lack of a numeric criterion for PBDE in the state’s water quality standards ensured that Ecology does not complete a CWA assessment of its extensive monitoring on PBDEs found in the state’s waters.

Numerous states—including California, Connecticut, Hawaii, Illinois, Maine, Maryland, Massachusetts, Michigan, New Jersey, New York, Oregon, Pennsylvania, and Washington—and

¹⁰⁴² *Id.* at 56–57.

¹⁰⁴³ Final FCR Report, *supra* n. 199.

¹⁰⁴⁴ Washington Assessment Database, *supra* n. 272 (search results of Current Category 5 PBDE listings).

¹⁰⁴⁵ *See id.*, Listing ID: 97880, available at https://apps.ecology.wa.gov/approvedwqa/candidatepages/viewcandidatelistings.aspx?LISTING_ID=97880 (listing ID Nos. 97872, 97874, 97877, and 97882 are identical).

the European Union, individual countries, and international organizations have taken actions to ban or limit PBDEs.¹⁰⁴⁶ The well-known adverse effect of PBDEs, discussed in this petition and elsewhere in the scientific literature, meets the requirements of CWA Section 307(a)(1) for its inclusion on the Toxic Pollutants Lists.

4. The Synthetic Estrogen 17 α -Ethinylestradiol (“EE2”)

As noted above, EPA has defined endocrine-disrupting CECs to include synthetic estrogens and androgens and naturally-occurring estrogens. In its 2008 White Paper on CECs, EPA specifically called out 17 α -ethinylestradiol (“EE2”), citing

a multiyear study by Kidd et al. (2007), [in which] the authors showed that environmentally relevant concentrations of ethinylestradiol, EE2, caused reproductive failure and near collapse of a natural fathead minnow population in an experimental lake, and also had deleterious effects on the reproductive biology of the pearl dace. These direct effects resulting in loss of forage fish have led to cascading effects on the lake trout population due to lack of prey (Kidd, personal communication).¹⁰⁴⁷

EPA pointed out that, unlike some medicinal pharmaceuticals, “the synthetic steroids EE2 and trenbolone bind to (and act as agonists of) vertebrate estrogen and androgen receptors, respectively, with similar or greater affinity than the natural endogenous hormones, estradiol and testosterone.”¹⁰⁴⁸ Additionally, some pharmaceuticals, including EE2 and trenbolone, “are designed to be highly specific, and thus are extremely potent.”¹⁰⁴⁹ As a result of this “target specificity,” EPA states that “EE2 and trenbolone affect reproduction and development in fish at water concentrations in the very low ng/L (part-per-trillion) range (e.g., Ankley et al. 2003;

¹⁰⁴⁶ PBDE Action Plan, *supra* n. 1001 at 77–87.

¹⁰⁴⁷ EPA, *White Paper, Aquatic Life Criteria for Contaminants of Emerging Concern*, at Part II, Illustration of Recommendations Using Data for 17 α -ethinylestradiol (EE2) (June 3, 2008) (hereinafter “EE2 White Paper”) at 3.

¹⁰⁴⁸ *Id.* at 10.

¹⁰⁴⁹ *Id.* at 11.

Länge et al. 2001), well below effect concentrations for most chemicals for which current ALC [aquatic life criteria] have been derived.”¹⁰⁵⁰ Moreover, these two synthetic hormones are so potent that “effects observed in fish are at concentration levels below the methodological limit of detection for most laboratories even in laboratory test water, and even more so ambient water and effluents.”¹⁰⁵¹

EPA cites additional “studies from around the world [that] have shown an elevated occurrence of intersex fish downstream of municipal effluents containing natural and synthetic steroidal estrogens, including EE2 (WHO 2002).”¹⁰⁵² The White Paper includes an entire section on EPA’s illustration of how to use data based on EE2.¹⁰⁵³ The effects of EE2 are not hypothetical. As EPA points out, “toxicological effects of EE2 have been found both in the laboratory, the source of toxicological data for criteria development, and in the field, where criteria are used to enforce the regulatory authorities of the Clean Water Act.”¹⁰⁵⁴ Nor is the source of EE2 unknown to EPA:

[D]omestic sewage treatment plant (STP) effluents become the primary source of EE2 entering the aquatic environment (Damstra 2002). Kolpin et al. (2002) found EE2 in 5.7% of 139 streams monitored in the U.S. . . . Data collected from fish and surface waters downstream of STPs [sewage treatment plants] over the past decade have implicated steroidal estrogens as the primary constituents in domestic effluents leading to the occurrence of intersex fish (Gross-Sorokin et al. 2006).¹⁰⁵⁵

The known adverse effect of 17 α -ethynylestradiol on aquatic designated uses meets the requirements of CWA Section 307(a)(1) for its inclusion on the Toxic Pollutants Lists.

¹⁰⁵⁰ *Id.*

¹⁰⁵¹ *Id.*

¹⁰⁵² *Id.* at 22, citing T. Damstra, et al. (eds.), *Global assessment on the state-of-the-science of endocrine disruptors* (Chapter 4: Wildlife), World Health Organization (2002).

¹⁰⁵³ EE2 White Paper, *supra* n. 1047.

¹⁰⁵⁴ *Id.* at 4.

¹⁰⁵⁵ *Id.*

5. Organotins

“Organotins are organometallic compounds that exhibit complex environmental chemistry and toxicity,” and are used primarily as plasticizers and biocides.¹⁰⁵⁶ In 2011, NMFS published a paper noting that a 1975 handbook had identified more than 250 organotin compounds. Among them is the biocide tributyltin (“TBT”) that was commonly used on vessel hulls and for which EPA developed 304(a) recommended criteria in 2004. Triphenyltin (“TPT”) has also been widely used as an agricultural fungicide, as have fenbutatin, azocyclotin, and hexamethylditin that “end up in aquatic systems.”¹⁰⁵⁷ It has been estimated that 70 percent of organotins are used for the production of polyvinyl chloride (“PCV”) plastics, from which they leach into aquatic systems.¹⁰⁵⁸ NMFS reported in 2011:

The most common organotins in aquatic environments occur as triorganotins (e.g., TBT, TPT, trimethyltin [TMT], tripropyltin [TPrT], etc.), diorganotins (DBT, dimethyltin [DMT], diethyltin [DET], etc.), and monoorganotins such as monobutyltin (MBT), and monomethyltin (MMT). There are a very large number of potentially toxic organotins and many of these are found in the environment and are considered significant contaminants. Unfortunately, we know very little about the occurrence, bioaccumulation, and toxicity for most organotins. Organotin environmental chemistry is relatively complex because these compounds are often polar, ionizable, and hydrophobic.¹⁰⁵⁹

While use of TBT as an antifouling paint has been restricted, in the early 1980s through mid-1990s, water column concentrations in the 0.1–1.0 ng/mL range were found, as compared to the EPA recommended criteria of 0.07 ng/mL for freshwater and 0.007 ng/mL for marine water,

¹⁰⁵⁶ James P. Meador, *Organotins in Aquatic Biota: Occurrence in Tissue and Toxicological Significance* (2011), published in *Environmental Contaminants in Biota: Interpreting Tissue Concentrations*, 2nd edition, ed. W. Nelson Beyer & James P. Meador (Boca Raton: CRC, 2011) (hereinafter “Organotins in Biota”), available at <http://digitalcommons.unl.edu/usdeptcommercepub/552> at 256.

¹⁰⁵⁷ *Id.* at 256, 257.

¹⁰⁵⁸ *Id.* at 257, citing Cima *et al.*, *Organotin compounds in the environment*, ed. P. J. Craig, West Sussex: John Wiley and Sons (2003), at 101-149.

¹⁰⁵⁹ Organotins in Biota, *supra* n. 1056 at 259.

“certainly result[ing] in severe biological effects in many ecosystems.”¹⁰⁶⁰ Since then, as water column levels have improved, sediment concentrations have remained high and “sediment-associated TBT will likely continue to be a source and lead to elevated water and tissue concentrations.”¹⁰⁶¹

Bioaccumulation of organotins has proven difficult to assess but high bioconcentration factors (“BCF”) for aquatic invertebrates have been observed, such as a BCF of 15,000 for marine snails for TPrT and in the range of 2,000 to 95,000 for TBT.¹⁰⁶² As with some other toxic pollutants, “rate of uptake for TBT is highly variable among species” and similarly the rate of elimination is also highly variable, but NMFS generally concluded that “TBT (and likely other organotins) is very slowly eliminated from tissue.”¹⁰⁶³ NMFS summarized the body burden of marine mammals with regard to several organotins:

Marine mammals also appear to accumulate relatively high concentrations of organotins. Several recent studies and reviews demonstrate that numerous marine mammal species exhibit high levels in various tissues, including liver, blubber, and muscle. Tanabe (1999) found concentrations of TBT at high concentrations (35-2200 ng/g ww) in several different tissues of finless porpoise (*Neophocaena phocaenoides*) from waters around Japan, with similar high concentrations for DBT and MBT. A review article by Kajiwara et al. (2006) presents data for 11 marine mammals species from various locations (Japan, Great Britain, Mediterranean, United States, Indo-Pacific, and India) showing high concentrations of TBT in liver (mean values 20-820 ng/g ww, maximum = 1200 ng/g). A number of studies examined organotins in killer whales (*Orcinus orca*). Harino et al. (2008) found TBT concentrations in the range of 6-25 ng/g ww and far higher levels of DBT (16-556 ng/g) and MBT (16-152 ng/g) in the liver of this species (Table 7.2). They also report low levels of TPT (<i>58 ng/g) in blubber and liver, which was also noted by Kajiwara et al. (2006) who reported no detectable concentrations of TPT or DPT in killer whales.¹⁰⁶⁴

¹⁰⁶⁰ *Id.* at 260–261 citing K. Fent, *Ecotoxicology of organotin compounds*, 26 Crit. Rev. Toxicol. 1–117 (1996).

¹⁰⁶¹ Organotins in Biota, *supra* n. 1056 at 260.

¹⁰⁶² *Id.* at 264.

¹⁰⁶³ *Id.* at 265, 266.

¹⁰⁶⁴ *Id.* at 263.

In 2011, NMFS concluded that “[i]n all cases an organotin compound is far more toxic than its individual components,”¹⁰⁶⁵ and identified multiple types of toxic responses including: inhibition of cellular energy metabolism, endocrine disruption including imposex and intersex abnormalities, neurotoxicity, inhibition of ion pumps, inhibition of cytochrome P450, inhibition of intracellular enzymes, immune system impairment, reduced growth, shell chambering (excessive shell growth) in bivalves, maternal transfer to eggs and young, reproductive effects including impairment, behavioral alterations, as well as mortality.¹⁰⁶⁶

These and additional toxic responses are discussed in a 2017 analysis of data from 160 references that focused on TBT as an endocrine disruptor.¹⁰⁶⁷ This paper drew conclusions about the toxicity of TBT as well as the implications for adequate regulation of complex toxic compounds:

[A] more thorough evaluation of the available data clearly shows that TBT is highly toxic to a variety of aquatic taxa. Through a comparative analysis of the potency of TBT in various aquatic species, our review highlights the observation that fish are as sensitive, or more so, compared to molluscs when based on water exposure. This is an important conclusion because molluscs were long recognized as uniquely sensitive to this compound. TBT’s precise MeOA is still incompletely understood but may include link/cross-talk between PPARs (i.e., carbohydrate, lipid, protein metabolism), RXRs (i.e., development), thyroid (growth) and even sex determination and differentiation pathways; the latter pathways may be stronger affected by TBT exposure in species where environmental factors play a significant role in determining sex ratios (e.g., zebrafish).

Current screening and assessment methodologies are able to identify TBT as a potent endocrine disruptor with a high environmental risk. If those approaches were available when TBT was introduced to the market, it is likely that its use would have been regulated sooner, thus avoiding the detrimental effects on

¹⁰⁶⁵ *Id.* at 266.

¹⁰⁶⁶ *Id.* at 269-277.

¹⁰⁶⁷ Laurent Lagadic, *et al.*, *Tributyltin: Advancing the Science on Assessing Endocrine Disruption with an Unconventional Endocrine-Disrupting Compound*, 245 *Reviews of Environmental Contamination and Toxicology* 65–127, 67 (2017), available at https://link.springer.com/chapter/10.1007/398_2017_8.

marine gastropod populations and communities as documented over several decades.

This retrospective evaluation of TBT, a very potent endocrine disruptor in vertebrates and invertebrates, should serve as an example demonstrating how shortfalls within the framework of chemical toxicity evaluation can result in under-protective regulatory assessment. Nowadays, the assays included in the OECD Conceptual Framework, including those recently developed on gastropod molluscs would likely recognize TBT as a chemical of concern with respect to endocrine disruption, although its mechanism of action and potency across taxonomic groups would remain largely unknown. Reflective analysis of well-studied, but potentially misunderstood contaminants, such as TBT, provides important lessons that should serve as a guiding principle for future studies and refinements of assessment protocols.¹⁰⁶⁸

As demonstrated above, the known adverse effect of organotins on aquatic designated uses meets the requirements of CWA Section 307(a)(1) for its inclusion on the Toxic Pollutants Lists.

6. N-(1,3-dimethylbutyl)-N'-phenyl-p-phenylenediamine (6PPD-Quinone)

The story of N-(1,3-dimethylbutyl)-N'-phenyl-p-phenylenediamine (6PPD) (“6PPD-quinone”) illustrates the importance of EPA’s having a timely method of updating its Toxic Pollutants Lists in the future once it has taken actions to update the current lists. As described by the New York Times:

About 20 years ago, ambitious restoration projects had brought coho salmon back to urban creeks in the Seattle area. But after it rained, the fish would display strange behaviors: listing to one side, rolling over, swimming in circles. Within hours they would die — before spawning, taking the next generation with them. In some streams, up to 90 percent of coho salmon were lost.

* * *

On a rainy day in 2012, [the scientists] filled stainless steel containers with a translucent dark liquid coming out of the spout. This time, the salmon exhibited the bizarre symptoms and promptly died.

* * *

¹⁰⁶⁸ *Id.* at 105.

So the team brewed up a test concoction by soaking shredded tire tread in water. The salmon died.

They were getting closer to the answer, but the tire water still contained more than 2,000 chemicals.

* * *

Dr. Tian's "aha!" moment came one morning. Guessing that the mystery chemical had transformed from a substance originally added to the tire, he looked for a compound whose carbon and nitrogen molecules matched, ignoring the oxygen and hydrogen, since the latter are more likely to be altered when a chemical transforms. In an Environmental Protection Agency report on tire rubber, he found a match: an antioxidant called 6PPD.¹⁰⁶⁹

As explained in the abstract of the paper that surfaced this ubiquitous pollutant, 6PPD-quinone had not been identified before 2020:

In U.S. Pacific Northwest coho salmon (*Oncorhynchus kisutch*), stormwater exposure annually causes unexplained acute mortality when adult salmon migrate to urban creeks to reproduce. By investigating this phenomenon, we identified a highly toxic quinone transformation product of N-(1,3-dimethylbutyl)-N'-phenyl-p-phenylenediamine (6PPD), a globally ubiquitous tire rubber antioxidant. Retrospective analysis of representative roadway runoff and stormwater-affected creeks of the U.S. West Coast indicated widespread occurrence of 6PPD-quinone (<0.3 to 19 micrograms per liter) at toxic concentrations (median lethal concentration of 0.8 ± 0.16 micrograms per liter). These results reveal unanticipated risks of 6PPD antioxidants to an aquatic species and imply toxicological relevance for dissipated tire rubber residues.¹⁰⁷⁰

A subsequent study found that "6PPD-Q also was more toxic than previously calculated and should be categorized as a "very highly toxic" noting that "[a]mong the 'very highly toxic' chemicals for which we have [aquatic life criteria] ALC, the toxicity of 6PPD-quinone is similar to that of the most toxic of 12 chemicals, all with LC50s <1 ppb."¹⁰⁷¹ Despite the urgent need

¹⁰⁶⁹ Catrin Einhorn, The New York Times, *How Scientists Tracked Down a Mass Killer (of Salmon) Something was decimating the salmon that had been restored to creeks around Puget Sound*, available at <https://www.nytimes.com/2020/12/03/climate/salmon-kill-washington.html>.

¹⁰⁷⁰ Zhenyu Tian, et al., *A ubiquitous tire rubber-derived chemical induces acute mortality in coho salmon*, 371 (6525) *Science* 185-189 (Dec. 3, 2020); Erratum for the Report (Feb. 18, 2022).

¹⁰⁷¹ Zhenyu Tian, et al., *6PPD-Quinone: Revised Toxicity Assessment and Quantification with a Commercial Standard*, available at <https://pubs.acs.org/doi/10.1021/acs.estlett.1c00910>; *id.* at Supporting Information, available at <https://pubs.acs.org/doi/pdf/10.1021/acs.estlett.1c00910#notes1>.

for regulatory limits on 6PPD-quinone, EPA’s failure to have placed it on the Toxic Pollutants Lists undermines the likelihood that the agency will take the steps such a listing would prompt. Moreover, the story of 6PPD-quinone’s identification as a highly toxic agent serves to underscore the importance of EPA’s updating the Toxic Pollutants Lists on a regular basis.

7. Microplastics

Microplastics are plastic particles from 5 mm to 1 nm¹⁰⁷² that are added intentionally or unintentionally to consumer products,¹⁰⁷³ or are the degraded result of larger plastic articles.¹⁰⁷⁴ “This micropollutant is spread in all types of environments and is serving as a ‘minor but efficient’ vector for carrier contaminants such as pesticides, pharmaceuticals, metals, polychlorinated biphenyls (PCBs), and polycyclic aromatic hydrocarbons (PAHs).”¹⁰⁷⁵ Ubiquitous in the environment, microplastics have been found in surface water, seabed sediments, beaches, freshwater, wastewater effluent, sea ice, organisms, food products including wild and farmed seafood,¹⁰⁷⁶ and bottled water.¹⁰⁷⁷ The smallest microplastics, termed

¹⁰⁷² EPA, *Microplastics Research*, available at <https://www.epa.gov/water-research/microplastics-research>. EPA defines “nanoplastics” as smaller than 1 nm. *Id.*

¹⁰⁷³ Microplastics are intentionally added to many products, including fertilizers, plant products, cosmetics, household and industrial detergents, paints, and gas and oil products. See European Chemicals Agency, *Microplastics*, available at <https://echa.europa.eu/hot-topics/microplastics>. See also Parichehr Hanachi *et al.*, *Abundance and properties of microplastics found in commercial fish meal and cultured common carp (Cyprinus carpio)*, 26 *Environmental Science and Pollution Research* 23777 (June 17, 2019), available at <https://link.springer.com/article/10.1007/s11356-019-05637-6>.

¹⁰⁷⁴ NOAA, National Ocean Service, Facts, *What are microplastics?* (last updated Jan. 26, 2023) (hereinafter “NOAA Microplastics”) available at <https://oceanservice.noaa.gov/facts/microplastics.html>; see also Silvia D. Martinho *et al.*, *Microplastic Pollution Focused on Sources, Distribution, Contaminant Interactions, Analytical Methods, and Wastewater Removal Strategies: A Review* (hereinafter “A Review of Microplastics”) *International Journal of Environmental Research and Public Health* (May 19, 2022) at 5610, available at <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9104288/>.

¹⁰⁷⁵ *Id.*

¹⁰⁷⁶ See *e.g.*, Britta R. Baechler *et al.*, *Microplastic occurrence and effects in commercially harvested North American finfish and shellfish: Current knowledge and future directions*, 5(1) *Limnology and Oceanography Letters* 113 (Nov. 5, 2019) (hereinafter “Microplastics in Fish”) available at <https://aslopubs.onlinelibrary.wiley.com/doi/10.1002/lol2.10122>.

¹⁰⁷⁷ A Review of Microplastics, *supra* n. 1074 At 3.

“nanoplastics,” can be absorbed into animal guts, eventually settling into tissues and organs, and entering the bloodstream.¹⁰⁷⁸ While microplastics can enter the food chain by direct ingestion at all trophic levels, they may bioaccumulate through prey consumption.¹⁰⁷⁹ In animals, microplastics have a wide range of effects including diminishing major organ efficacy, causing chronic inflammation, decreasing fertility and growth, harming fish gills, neurotoxicity, intestinal dysfunctions, impaired hormonal functions, inhibited fetal development, developmental disorders, behavioral changes, oxidative and hepatic stress, damage to DNA, and reduced fecundity.¹⁰⁸⁰

Plastic’s ability to bond with chemicals makes consumption of microplastics an entry pathway for toxic pollutants into animals.¹⁰⁸¹ “The properties of microplastics directly influence the adsorption process (e.g., polymer size, shape, and density, hydrophobic properties, and the surface-to-volume ratio)” and “[t]he higher the adsorption capacity for a given pollutant of a specific microplastic, the higher the harmful potential of the consortium microplastic/pollutant.”¹⁰⁸² “The longer a plastic particle remains in the aquatic environment, the more concentrated the contaminants can become as they accumulate on the particle surface over time[.]”¹⁰⁸³ Not only do microplastics absorb other toxic pollutants but the plastic material itself contains chemicals as part of the manufacturing process.¹⁰⁸⁴

¹⁰⁷⁸ See generally, *Microplastics in Fish*, *supra* n. 1076.

¹⁰⁷⁹ *Id.* at 117.

¹⁰⁸⁰ See generally *id.*

¹⁰⁸¹ *Id.*; see also *A Review of Microplastics*, *supra* n. 1074.

¹⁰⁸² *Id.* at 5.

¹⁰⁸³ EPA, *A Summary of Literature on the Chemical Toxicity of Plastics Pollution to Aquatic Life and Aquatic-Dependent Wildlife* (Dec. 2016) (hereinafter “EPA Microplastics Literature”) 17, available at https://www.epa.gov/sites/default/files/2017-02/documents/tfw-trash_free_waters_plastics-aquatic-life-report-2016-12.pdf.

¹⁰⁸⁴ *Microplastics in Fish*, *supra* n. 1076 at 117 (“Additives and monomers, including bisphenol A (BPA), organotoxins, and phthalates, with established biologically harmful properties such as

Seafood consumption is a common source for human microplastic exposure, posing a heightened risk for people who consume high levels of fish and shellfish,¹⁰⁸⁵ making microplastics an environmental justice concern.¹⁰⁸⁶ Scientists have calculated that people absorb tens of thousands of microplastic particles—each person, each year.¹⁰⁸⁷ Retention of microplastics is related to their size:

Once ingested, >90% of MPs were reported to be excreted in feces, especially large particles >150 µm; however, smaller particles may be absorbed systematically. It has been reported that MPs 0.1–10 µm in size can cross the blood-brain barrier and the placenta, particles <150 µm can cross gastrointestinal epithelium, and particles <2.5 µm can enter the systemic circulation through endocytosis.¹⁰⁸⁸

reproductive toxicity, mutagenicity, and carcinogenicity, are used to manufacture plastics. If microplastics are ingested, these compounds can be released from the polymer and absorbed by predators.”) (internal citations omitted). *See also*, Kurunthachalam Kannan & Krishnamoorthi Vimalkumar, *A Review of Human Exposure to Microplastics and Insights Into Microplastics as Obesogens* (hereinafter “Microplastic Human Exposure”), 12 *Frontiers in Endocrinology* 724989 (Aug. 18, 2021) available at <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8416353/> (“Exposure of human cell lines to [microplastic] MP additives such as phthalates, bisphenols, and organotins causes adverse effects through the activation of nuclear receptors, peroxisome proliferator-activated receptors (PPARs) α , β , and γ and retinoid X receptor (RXR), leading to oxidative stress, cytotoxicity, immunotoxicity, thyroid hormone disruption, and altered adipogenesis and energy production.”); EPA Microplastics Literature, *supra* n. 1083 at 14–15.

¹⁰⁸⁵ Madeleine Smith *et al.*, *Microplastics in Seafood and the Implications for Human Health*, 5 *Current Environmental Health Reports* 375–386 (Aug. 16, 2018) at 375, available at <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6132564/>.

¹⁰⁸⁶ Seaweed consumption is also a source of microplastic consumption. *See* Microplastic Human Exposure, *supra* n. 1084 at 9.

¹⁰⁸⁷ *Id.* at 4 (“One study reported a per capita intake of MPs through ingestion of food, water, and dust and inhalation of air of 74,000–121,000 items annually. Another study estimated an annual per capita MP intake of 39,000–52,000 items, including 37–1000 from sea salt, 4000 from tap water, and 11,000 from shellfish. A probabilistic lifetime exposure model predicted a MP intake rate of 184 ng/capita/d for children and 583 ng/capita/d for adults, through nine different exposure sources. Mass (or weight)-based estimates of annual MP ingestion were reported to be 15–287 g/person.”) (internal citations omitted); *id.* at 9 (“Globally, humans may ingest an average of 0.1–5 g/week of MPs up to 1 mm in size, or 74,000–121,000 particles per year. However, the authors of that study noted that this could be an underestimate of actual exposures.”) (internal citations omitted).

¹⁰⁸⁸ *Id.* at 10.

EPA cites field and laboratory studies that show “[f]ish have also been found to ingest plastic and have elevated tissue concentrations of chemicals associated with plastics.”¹⁰⁸⁹

For aquatic species, microplastics are a particular hazard because they look like small food particles¹⁰⁹⁰ and when consumed in lieu of food, can lead to nutritional deficiency and intestinal blockage.¹⁰⁹¹ Microplastics pose a particular risk to filter feeding fish that consume plankton.¹⁰⁹² Fish exposed to microplastics have lower amounts of protein and minerals, and often have impaired liver function, than those fed a diet with no microplastic.¹⁰⁹³ In the marine environment, new research is finding “a change in microbial community structure in response to increasing [nanoplastic particle] NP exposure, possibly due to polystyrene as a new source of organic-based material within the mesocosms,” where microbes are key to ecosystem maintenance, the marine food web, and biogeochemical processes.¹⁰⁹⁴ A newly published study looking at exposures of fish to microplastics, plastic microfibers, and natural microparticles, found:

¹⁰⁸⁹ EPA Microplastics Literature, *supra* n. 1083 at 33.

¹⁰⁹⁰ Christina J. Thiele, *et al.*, *Microplastics in fish and fishmeal: an emerging environmental challenge?*, 11 *Nature* 2045 (2021), available at <https://doi.org/10.1038/s41598-021-81499-8>.

¹⁰⁹¹ NOAA, Marine Debris Program, Office of Response and Restoration, *Bite Size Plastic: How Marine Wildlife Snack on Our Trash*, available at <https://blog.marinedebris.noaa.gov/bite-size-plastic-how-marine-wildlife-snack-our-trash>.

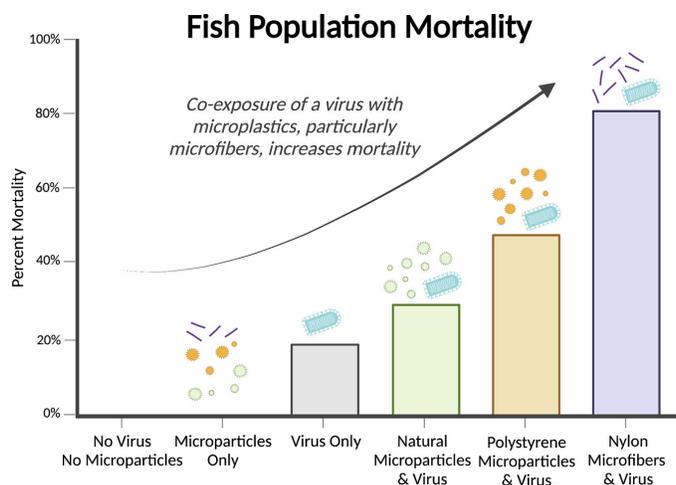
¹⁰⁹² Alonzo Alfaro-Nunez *et al.*, *Microplastic pollution in seawater and marine organisms across the Tropical Eastern Pacific and Galapagos*, 11 *Scientific Reports* 6424 (2021) available at <https://www.nature.com/articles/s41598-021-85939-3>.

¹⁰⁹³ Xing Lu, *et al.* *Chronic exposure to high-density polyethylene microplastic through feeding alters the nutrient metabolism of juvenile yellow perch (Perca flavescens)*, 9 *Animal Nutrition* 143 (June 2022), available at <https://www.sciencedirect.com/science/article/pii/S2405654522000245>

¹⁰⁹⁴ EPA, Science Inventory, M. Giroux *et al.*, *Effects of Polystyrene Nanoplastic on Benthic Microbial Communities*, SETAC North Atlantic Chapter, 28th Annual Meeting 2022, Groton, CT, June 27 - 28, 2022, available at https://cfpub.epa.gov/si/si_public_record_report.cfm?dirEntryId=355141&Lab=CEMM&showcriteria=2&timstype=&timssubtypeid=&epanumber=&ombcat=Any&datebeginpublishedpresented=&dateendpublishedpresented=&datebeginupdated=&dateendupdated=&deid=&personname=&personid=&role=Any&journalname=&journalid=&publishername=&publisherid=&sortby=pubDate&count=25&searchall=Microplastics.

Mortality increased significantly when fish were co-exposed to virus and microplastics, particularly microfibers, compared to virus alone. This presents the unique finding that microplastics (not natural microparticulate matter) may have a significant impact on population health when presented with another stressor. Further, we found that mortality correlated with host viral load, mild gill inflammation, immune responses, and transmission potential.¹⁰⁹⁵

This result was illustrated as follows:¹⁰⁹⁶



For wildlife, microplastics can pose a serious risk to seabirds due to their consumption of plastic both firsthand and secondhand when they eat fish that also contain microplastics and related chemicals.¹⁰⁹⁷ For example, EPA cited a study on short-tailed shearwaters (*Puffinus tenuirostris*) in which PBDE congeners were not found in the prey of short-tailed shearwaters but were found in the plastic particles within the bird's digestive tracts, suggesting that ingested plastic was the source of PBDEs.¹⁰⁹⁸ A similar finding was made for high concentrations of chromium and silver in fledgling flesh-footed shearwaters (*Puffinus carneipes*), and the level for

¹⁰⁹⁵ Meredith Evans Seeley, *et al.*, *Microplastics exacerbate virus-mediated mortality in fish*, 866 *Science of the Total Environment* 161191 (2023), available at <http://dx.doi.org/10.1016/j.scitotenv.2022.161191>.

¹⁰⁹⁶ *Id.* at 1.

¹⁰⁹⁷ EPA Microplastics Literature, *supra* n. 1083 at 33.

¹⁰⁹⁸ *Id.*

both metals was positively correlated with the mass of plastic ingested by the birds.¹⁰⁹⁹ A recent study of wild seabirds found environmentally-relevant microplastic concentrations and mixtures in wild seabirds—northern fulmars (*Fulmarus glacialis*) and Cory’s shearwaters (*Calonectris borealis*):

The amount of microplastics in the gut was significantly correlated with gut microbial diversity and composition: microplastics were associated with decreases in commensal microbiota and increases in (zoonotic) pathogens and antibiotic-resistant and plastic-degrading microbes.¹¹⁰⁰

Microplastics are known to block gastrointestinal tracts of small birds and can also create lacerations and irritation internally.¹¹⁰¹

EPA has taken no action to regulate microplastics.¹¹⁰² Even as EPA has delayed, at least five U.S. states are working to reduce microplastics in surface water, stormwater, and treated sewage.¹¹⁰³ For example, California has established microplastics regulations and monitoring requirements for drinking water.¹¹⁰⁴ The state’s plan focuses on pollution prevention by stopping plastic waste at its source, pathway interventions, and outreach and education to inform

¹⁰⁹⁹ *Id.*

¹¹⁰⁰ Gloria Fackelmann *et al.*, *Current levels of microplastic pollution impact wild seabird gut microbiomes*, 7 *Nature Ecology & Evolution* 698-706 (May 2023), available at <https://www.nature.com/articles/s41559-023-02013-z>.

¹¹⁰¹ Canadian Wildlife Health Cooperative, *Wildlife Ingestion of Microplastics*, at 2, available at http://www.cwhc-rcsf.ca/docs/fact_sheets/Wildlife%20ingestion%20of%20microplastics.pdf.

¹¹⁰² The Microbead-Free Waters Act of 2015 constitutes the only federal regulation of microplastics. 21 U.S.C. § 331(ddd)(1) (prohibiting the manufacture or introduction or delivery for introduction into the interstate commerce of rinse-off cosmetic that contains intentionally-added plastic microbeads)

¹¹⁰³ See Darcy Metzler, Simone Simbeck, *Microplastics: Global buzz and concern spur increased regulation*, Haley Aldrich, (Apr. 28, 2022), available at <https://www.haleyaldrich.com/resources/articles/microplastics-global-buzz-and-concern-spur-increased-regulation/>.

¹¹⁰⁴ *Id.*

the public and industries about microplastics and their risks.¹¹⁰⁵ Finally, the state plans to standardize a statewide monitoring approach of microplastics, and create risk thresholds.

F. Metals not on the Priority Pollutants Lists for Which Evidence Demonstrates a Need for 304(a) Criteria

While it is generally assumed that EPA has adopted Section 304(a) recommended criteria for all metals that pose a threat to human health or aquatic species, EPA should challenge this assumption in evaluating what toxic pollutants should be on updated Toxic Pollutants Lists. Two examples are the metals cobalt and manganese, the latter of which is discussed in section IV, *supra*.

Cobalt is essential to batteries that power cellphones and electric cars, most of which is currently mined in the Democratic Republic of the Congo under inhumane circumstances.¹¹⁰⁶ Possibly as a result of this fact, mining for cobalt has restarted in the State of Idaho,¹¹⁰⁷ following EPA's placement of extensive areas that were previously mined under the Superfund program.¹¹⁰⁸ For purposes of Superfund remediation, EPA established a cobalt water quality clean-up level at 0.086 mg/L (86 µg/L) in 2007 after having established a more protective level

¹¹⁰⁵ California Ocean Protection Council, *California Takes Decisive Action to Reduce Microplastics Pollution: State Adopts a First-in-Nation Approach to Protecting Ocean and Human Health*, available at <https://www.opc.ca.gov/2022/02/california-takes-decisive-action-to-reduce-microplastics-pollution-state-adopts-a-first-in-nation-approach-to-protecting-ocean-and-human-health/>.

¹¹⁰⁶ See, e.g., Nicholas Niarchos, *The Dark Side of Congo's Cobalt Rush* (May 24, 2021) available at <https://www.newyorker.com/magazine/2021/05/31/the-dark-side-of-congos-cobalt-rush>; Dionne Searcey and Eric Lipton, *Hunt for the 'Blood Diamond of Batteries' Impedes Green Energy Push* (Nov. 29, 2021), available at <https://www.nytimes.com/2021/11/29/world/congo-cobalt-albert-yuma-mulimbi.html>.

¹¹⁰⁷ See, e.g., Oregon Public Broadcasting, *In Idaho, America's first, and only, cobalt mine in decades is opening* (Oct. 8, 2022), available at <https://www.opb.org/article/2022/10/08/in-idaho-america-s-first-and-only-cobalt-mine-in-decades-is-opening/>; Michael Holtz, *Idaho is Sitting on One of the Most Important Elements on Earth* (Jan. 24, 2022) (hereinafter "Cobalt Mining in Idaho"), available at <https://www.theatlantic.com/science/archive/2022/01/cobalt-clean-energy-climate-change-idaho/621321/>.

¹¹⁰⁸ EPA, Superfund Site: *Blackbird Mine, Lemhi County, ID Cleanup Activities*, available at <https://cumulis.epa.gov/supercpad/SiteProfiles/index.cfm?fuseaction=second.Cleanup&id=1000256#bkground>

of 0.038 mg/L in 2003.¹¹⁰⁹ Both permitted and unpermitted discharges have already taken place. EPA issued an NPDES permit to Noranda Mining for the Blackbird Mine near Cobalt, Idaho in 1990, with no cobalt monitoring required or effluent limits for the metal.¹¹¹⁰ New activities have been the subject of state enforcement actions.¹¹¹¹

Cobalt is not on the Toxic Pollutants Lists and EPA has no recommended criteria for it.¹¹¹² Cobalt does, however, have a long history of adverse effects to aquatic life as explained in a 2015 paper led by the USGS:

This paper presents a 30+ year record of changes in benthic macroinvertebrate communities and fish populations associated with improving water quality in mining-influenced streams. Panther Creek, a tributary to the Salmon River in central Idaho, USA suffered intensive damage from mining and milling operations at the Blackbird Mine that released copper (Cu), arsenic (As), and cobalt (Co) into tributaries. From the 1960s through the 1980s, no fish and few aquatic invertebrates could be found in 40 km of mine-affected reaches of Panther Creek downstream of the metals contaminated tributaries, Blackbird and Big Deer Creeks.

Efforts to restore water quality began in 1995, and by 2002 Cu levels had been reduced by about 90%, with incremental declines since. Rainbow Trout (*Oncorhynchus mykiss*) were early colonizers, quickly expanding their range as areas became habitable when Cu concentrations dropped below about 3X the U.S. Environmental Protection Agency's biotic ligand model (BLM) based chronic aquatic life criterion. Anadromous Chinook Salmon (*O. tshawytscha*) and steelhead (*O. mykiss*) have also reoccupied Panther Creek. Full recovery of salmonid populations occurred within about 12-years after the onset of restoration efforts and about 4-years after the Cu chronic criteria had mostly been met, with recovery interpreted as similarity in densities, biomass, year class strength, and condition factors between reference sites and mining-influenced sites. Shorthead

¹¹⁰⁹ See EPA, *Explanation of Significant Differences for the Record of Decision for the Blackbird Mine Lemhi County, Idaho* (May 31, 2012) at 6, available at <https://semspub.epa.gov/work/10/100002633.pdf>.

¹¹¹⁰ EPA, *Authorization to Discharge Under the National Pollutant Elimination System, Noranda Mining, Inc., Blackbird Mine, Permit No. ID-002525-9* (April 30, 1990).

¹¹¹¹ See e.g., Idaho Department of Environmental Quality, *In the Matter of Idaho Cobalt Company, Consent Order* (draft, undated) available at <https://www2.deq.idaho.gov/admin/LEIA/api/document/download/16289>

¹¹¹² Note that Canada has guidelines for cobalt. See Environment Canada, *Federal Environmental Quality Guidelines Cobalt* (May 2017), available at <https://www.ec.gc.ca/ese-ees/default.asp?lang=En&n=92F47C5D-1>.

Sculpin (*Cottus confusus*) were slower than salmonids to disperse and colonize. While benthic macroinvertebrate biomass has increased, species richness has plateaued at about 70 to 90% of reference despite the Cu criterion having been met for several years. Different invertebrate taxa had distinctly different recovery trajectories. Among the slowest taxa to recover were *Ephemerella*, *Cinygmula* and *Rhithrogena* mayflies, Enchytraeidae oligochaetes, and *Heterlimnius* aquatic beetles. Potential reasons for the failure of some invertebrate taxa to recover include competition, and high sensitivity to Co and Cu.¹¹¹³

As noted by USGS, the elevation of cobalt levels in this mined area—on average 16 µg/L, compared to background levels of 0.2 µg/L¹¹¹⁴, and compared to EPA’s clean-up level of 86 µg/L—resulted in the failure of some mayfly species to recover. Clearly, the EPA clean-up level is not sufficiently protective of aquatic life. More recently, a team of scientists calculated a chronic criterion for cobalt using EPA aquatic life criteria protocols, generating a value of 7.13 µg/L at hardness 100 mg/L.¹¹¹⁵ The authors did not include a bioavailability or hardness adjustment, both of which would be lower in the soft water streams common in mountainous areas such as the Idaho Cobalt Belt. Regardless, these criteria are far more protective than the 86 µg/L EPA established for the clean-up of the Blackbird Mine. Future regulatory actions—as of a year ago six have been proposed in this area¹¹¹⁶—based on that clean-up level would fail to protect aquatic life.

¹¹¹³ Chris Mebane, *et al.*, *Recovery of a mining-damaged stream ecosystem*, 3(1) *Elementa: Science of the Anthropocene*, 000042 (2015), available at <https://doi.org/10.12952/journal.elementa.000042>.

¹¹¹⁴ *Id.* at 8; see also W.A. Stubblefield, *et al.*, *Acute and chronic toxicity of cobalt to freshwater organisms: using a species sensitivity distribution approach to establish international water quality standards*, 39(4) *Environmental Toxicology and Chemistry* 799-811 (2020) at 800, available at <https://doi.org/10.1002/etc.4662>.

¹¹¹⁵ *Id.* at 809.

¹¹¹⁶ See Cobalt Mining in Idaho, *supra* n. 1107 (“At least six mining companies have applied for permits from the U.S. Forest Service to operate in the region.”).

The known adverse effects of cobalt on aquatic designated uses and the effects of manganese on human health meet the requirements of CWA Section 307(a)(1) for its inclusion on the Toxic Pollutants Lists.

G. Current Use Pesticides the Use of Which the National Marine Fisheries Service and/or the U.S. Fish and Wildlife Service Have Determined Poses Jeopardy to Aquatic or Aquatic-Dependent Threatened and Endangered Species

Since 2008, as the result of litigation, NMFS has issued numerous biological opinions pursuant to Section 7 of the Endangered Species Act (“ESA”) on the effects of various current-use pesticides on the continued existence of some ESA-listed species and their designated critical habitat by EPA’s registration under the Federal Insecticide, Fungicide, and Rodenticide Act (“FIFRA”) of those pesticides.¹¹¹⁷ Of the pesticide biological opinions, NMFS has concluded that registration or reregistration of the following pesticides poses jeopardy to the continued existence of these species: diflubenzuron, fenbutatin oxide, propargite, oryzalin, pendimethalin, trifluralin, 2,4-D, diuron, chlorothalonil, bensulide, dimethoate, ethoprop, methidathion, naled, phorate, phosmet, carbaryl, carbofuran, and methomyl.¹¹¹⁸ Based on the findings of NMFS that

¹¹¹⁷ NMFS, *Pesticide Consultations*, available at <https://www.fisheries.noaa.gov/national/consultations/pesticide-consultations> (hereinafter “NMFS BiOp Website”).

¹¹¹⁸ *Id.*; see also 2009 NMFS Carbaryl BiOp, *supra* n. 756; NMFS, *National Marine Fisheries Service Endangered Species Act Section 7 Consultation Biological Opinion Environmental Protection Agency Registration of Pesticides Containing Azinphos methyl, Bensulide, Dimethoate, Disulfoton, Ethoprop, Fenamiphos, Naled, Methamidophos, Methidathion, Methyl parathion, Phorate and Phosmet 772-775* (August 31, 2010) available at http://www.nmfs.noaa.gov/pr/pdfs/final_batch_3_opinion.pdf; NMFS, *National Marine Fisheries Service Endangered Species Act Section 7 Consultation Biological Opinion Environmental Protection Agency Registration of Pesticides 2,4-D, Triclopyr BEE, Diuron, Linuron, Captan, and Chlorothalonil 773-774* (June 30, 2011) available at http://www.nmfs.noaa.gov/pr/pdfs/consultations/pesticide_opinion4.pdf; NMFS, *National Marine Fisheries Service Endangered Species Act Section 7 Consultation Final Biological Opinion Environmental Protection Agency Registration of Pesticides Oryzalin, Pendimethalin, Trifluralin 640-641* (May 31, 2012) available at http://www.nmfs.noaa.gov/pr/pdfs/consultations/pesticides_batch5opinion.pdf; NMFS, *National Marine Fisheries Service Endangered Species Act Section 7 Consultation Biological Opinion Environmental Protection Agency Registration of Pesticides Containing*

waterborne exposure to these current use pesticides poses jeopardy to aquatic species, these pollutants should be placed on the Toxic Pollutant List.

In addition, as discussed in sub-section VII.A.3 *supra*, NMFS had determined in 2008 that the pesticides chlorpyrifos, diazinon, and malathion pose jeopardy to salmonids, a decision that was superseded by a new jeopardy opinion in 2017, and a new non-jeopardy opinion in 2022 based on “additional species protections or “conservation measures” negotiated between the two federal agencies and the registrants for the three pesticides.¹¹¹⁹ The need for these conservation measures demonstrates the toxicity of the compounds.

In 1989, the FWS finalized consultation on 112 pesticides for numerous species across the country.¹¹²⁰ This biological opinion found the following 70 pesticides cause jeopardy for one or more ESA-listed aquatic or aquatic-dependent species evaluated: acephate, aldicarb, aminopyridine, atrazine, guthion, benomyl, bensulide, bifenox, toxaphene, captan, carbaryl, carbofuran, carbophenothion, chlorothalonil, chlorpyrifos, cleothocarb, copper sulfate, 2,4-D, diazinon, dichlorvos, dicofol, dicotophos, diflubenzuron, dimethoate, disulfoton, diuron, endosulfan, endrin, EPN, ethion, ethoprop, parathion, fenamiphos, fenitrothion, fensulfothion,

Diflubenzuron, Fenbutatin Oxide, and Propargite (Jan. 7, 2015) available at https://media.fisheries.noaa.gov/dam-migration/63806567_pesticides_biop_7_1_7_2015.pdf; NMFS, *National Marine Fisheries Service Endangered Species Act Section 7 Consultation Biological Opinion Environmental Protection Agency Registration of Pesticides Containing bromoxynil and prometryn* (June 25, 2021) available at <https://www.fisheries.noaa.gov/resource/document/biological-opinion-bromoxynil-and-prometryn>; NMFS, *National Marine Fisheries Service Endangered Species Act Section 7 Consultation Biological Opinion Environmental Protection Agency Registration of Pesticides Containing Metolachlor and 1,3-Dichloropropene* (June 25, 2021), available at <https://www.fisheries.noaa.gov/resource/document/biological-opinion-metolachlor-and-13-dichloropropene>.

¹¹¹⁹ NMFS BiOp Website, *supra* n. 1117; see also 2022 Chlorpyrifos BiOp, *supra* n. 748.

¹¹²⁰ FWS, *U.S. Fish and Wildlife Service Biological Opinion on Selected Pesticides* (June 14, 1989, revised September 14, 1989) available through National Service Center for Environmental Publications (NSCEP), <http://www.epa.gov/nscep/index.html>.

fenthion, fenvalerate, fenofos, isofenphos, malathion, mancozeb, methidathion, methomyl, methoprene, methyl parathion, mevinphos, naled, nytrapyrin, oxamyl, oxydemeton-methyl, oxyfluorfen, paraquat dichloride, pendimethalin, permethrin, phorate, phosmet, profenfos, propachor, propargite, propazine, pyrethrin, SSS-tributyl phosphorotrithioate, sulprofos, temephos, terbufos, terbutryn, theodicarb, thiophanate-methyl, trichlorfon, and trifluralin.

Without repudiating the jeopardy findings and reasonable and prudent alternatives (“RPA”) issued in the 1989 biological opinion, FWS has subsequently explained the serious limitations of that assessment. In a letter to EPA written over a decade later, concerning use of pesticides in the State of Texas, FWS noted the following limitations of its published opinion: (1) a majority of pesticides used “have had inadequate or no consultation,” (2) “critical habitat has been insufficiently addressed,” and (3) “no current mechanism exists for updating pesticide protection measures for recently listed species, critical habitat, or listed species that previously have undergone consultation.”¹¹²¹ FWS concluded that its 1989 biological opinion is “outdated since [it] represent[s] consultations for only 19 Texas species and 125 pesticide active ingredients.”¹¹²² Moreover, FWS stated that the RPAs set out in the 1989 opinion provide inadequate protection because “new information in the spray drift/runoff literature indicates that buffer zones and other protection measures provided in the 1989 Biological Opinion should be revised.”¹¹²³ In short, the 1989 BiOp is not protective.

¹¹²¹ Letter from David C. Frederick, Supervisor, FWS, to Gregg Cooke, Regional Administrator, EPA, Re: *EPA’s Noncompliance in Texas on National Pesticide Consultations 2* (June 28, 2001).

¹¹²² *Id.*

¹¹²³ *Id.*

FWS also sharply criticized EPA’s use of a quotient model for assessing the risks of pesticides because the model

cannot currently account for sublethal effects by pesticides on listed species such as endocrine disruption, abnormal behavioral changes, olfactory interference in anadromous fish spp., etc. Such sublethal effects from pesticide applications “may affect” listed species and therefore constitute harm as part of take as defined in the ESA. Since pesticide protection measures contained in the 1989 Biological Opinion have been based in part on use of the quotient model, the Service believes that: a) the biological opinion must be revised to provide more accurate protection measures for listed species, and b) the current process used by EPA for reaching “may affect” determinations for listed species must be re-evaluated including the role of mathematical models.¹¹²⁴

With regard to another pesticide use, EPA engaged in informal ESA consultation with NMFS on the use of atrazine in the Chesapeake Bay watershed.¹¹²⁵ In response, NMFS declined to concur that atrazine is not likely to adversely affect ESA-listed species and concluded that “NMFS is aware that atrazine has reached concentrations of up to 98 µg /L in surface waters of the Chesapeake Bay watershed and peak concentrations may be substantially higher” noting that “[t]oxicity data suggest these concentrations are likely to adversely affect listed species in the Chesapeake Bay watershed due to either direct toxicity, or habitat associated impacts.”¹¹²⁶ NMFS critiqued EPA’s evaluation of the hazards of atrazine because the “the acute threshold of 100 ug/L used by EPA was based on an LC50 study that was 74-fold less sensitive than the median lethal concentration available for another surrogate species in EPA’s ECOTOX

¹¹²⁴ *Id.* at 3.

¹¹²⁵ Letter from James H. Lecky, Director, Protected Resources, NMFS, to Arthur-Jean Williams, EPA, Re: *Request for Endangered Species Act Section 7 Informal Consultation on the Environmental Protection Agency's Re-Registration and Use of Atrazine in the Chesapeake Bay Watershed, September 1, 2006* (May 20, 2007), available at https://media.fisheries.noaa.gov/dam-migration/63806506atrazine_letter_epa.pdf; see also NMFS, *Technical Appendix The National Marine Fisheries Service's Technical Review of the Environmental Protection Agency's Pesticides Effects Determination for Atrazine on Federally Listed Species in the Chesapeake Bay Watershed* (undated), available at https://media.fisheries.noaa.gov/dam-migration/63806507atrazine_appendix.pdf.

¹¹²⁶ *Id.* at 2.

database” and “the threshold of 65 ug/L used by EPA for chronic exposure was 135-fold less sensitive than 0.5 ug/L, the concentration of atrazine that impairs fish reproductive and behavioral endpoints.”¹¹²⁷ NMFS concluded: “The studies reviewed by NMFS suggest that adverse effects likely occur at concentrations of atrazine well below 65 and 100 ug/L. Consequently, the actual risk to listed species of atrazine use in the Chesapeake Bay watershed may be significantly underestimated in the current assessment.”¹¹²⁸

The FWS also raised concerns with regard to EPA’s proposed reregistration of the pesticide atrazine.¹¹²⁹ FWS criticized EPA’s risk assessment because it did not: (1) include sublethal effects of pesticides, (2) used inappropriate surrogate species to evaluate effects on threatened and endangered species, (3) failed to consider the toxicological effects of “inert” ingredients and adjuvants (which increase pesticide effectiveness), (4) failed to consider the potential for bioaccumulation, and (5) failed to evaluate mixtures of chemicals including other pesticides, thereby underestimating the potential for ecological impacts.¹¹³⁰ FWS specifically addressed the failure of EPA’s atrazine evaluation to consider the pesticide’s effects on amphibians, pointing out that EPA’s risk ranges were “not based on risks to amphibians”¹¹³¹ despite recent research specifically evaluating the risk to amphibians from that pesticide. Likewise, there was no evaluation of the risk of bioaccumulation in amphibians, despite recent studies’ demonstrating that effect.¹¹³² The agency also noted that because amphibian larvae

¹¹²⁷ *Id.*

¹¹²⁸ *Id.*

¹¹²⁹ Letter from Everett Wilson, Chief, Division of Environmental Quality, FWS, to Kimberly Nesci Lowe, Chemical Review Manager, Information and Resources and Services Division, EPA, *USF&WS Comments on Ecological Risk Assessment of Atrazine for Re-Registration* (June 27, 2002)

¹¹³⁰ *Id.* at 2-5.

¹¹³¹ *Id.* at 4.

¹¹³² *Id.* at 5.

subsist on algae and other phytoplankton, adverse impacts to aquatic plants “could have adverse effects to amphibians. This is particularly important because of the dramatic decline in amphibian populations worldwide[.]”¹¹³³ Making an observation that is highly relevant to the water quality standards program, FWS also pointed out that

[a]quatic systems that have fish often lack amphibians and vice versa. In Murphy et al. (2000) several citations are listed for the predatory effects on amphibians by fish[.] The text states that “many anuran species cannot coexist with such predatory fish.” Therefore, an aquatic community with amphibians is likely to be much different than an aquatic community with fish.¹¹³⁴

EPA also engaged in an informal consultation with NMFS on the use of racemic metolachlor in Idaho, Washington, California, and Oregon. In response to EPA’s conclusion that racemic metolachlor is not likely to adversely affect ESA-listed species in these Western states, NMFS disagreed and concluded that “[t]oxicity data suggest these [measured fish] concentrations are likely to adversely affect listed salmonids either directly, or indirectly via habitat associated impacts.”¹¹³⁵ To the best of Petitioners’ knowledge, EPA never initiated consultation with NMFS as requested by the latter agency. There is no completed biological opinion and there is every reason to believe that levels of racemic metolachlor allowed by FIFRA registration label have and continue to result in “salmonid prey are likely to be exposed at higher concentrations than estimated by EPA and that adverse effects are likely for sensitive

¹¹³³ *Id.* at 4.

¹¹³⁴ *Id.*

¹¹³⁵ Letter from James H. Lecky, Director, Protected Resources, NMFS, to Arthur-Jean Williams, EPA, Re: *Request for Endangered Species Act Section 7 Informal Consultation on the Environmental Protection Agency’s Re-Registration and Use of Racemic Metolachlor in Idaho, Washington, California, and Oregon in relation to listed Pacific salmon and steelhead* (July 13, 2007), available at https://media.fisheries.noaa.gov/dam-migration/63806561racemic_metolachlor_letter.pdf; see also NMFS, *Technical Appendix The National Marine Fisheries Service’s (NMFS) Technical Review of the Environmental Protection Agency’s Pesticides Effect determinations for Racemic Metolachlor on Federally Listed Salmonid Species in the Pacific Northwest and California* (undated), available at https://media.fisheries.noaa.gov/dam-migration/63806560racemic_metolachlor_appendix.pdf.

taxa.”¹¹³⁶ And, EPA did not respond to NMFS’s request for an assessment for pesticides “commonly co-located in surface waters with racemic metolachlor . . . includ[ing] atrazine, s-metolachlor, and the commonly applied chloroacetanilides, acetochlor and alachlor.”¹¹³⁷

Finally, EPA sought to initiate formal consultation on the pesticides clomazone¹¹³⁸ and fomesafen¹¹³⁹ related to EPA’s proposed FIFRA registration. In both instances, NMFS concluded that “EPA’s request for formal consultation appears premature.”¹¹⁴⁰ NMFS went on to detail the extensive information needed to complete these consultations. There is no evidence that EPA provided sufficient information for these consultations to have been completed.

All the aforementioned pesticides for which FWS and NMFS have determined are a hazard to aquatic life meet the requirements of CWA Section 307(a)(1) for their inclusion on the Toxic Pollutants Lists.

H. Pollutants for Which Toxic Criteria in the NTR Have Not Been Updated Since 1992 for Which EPA Has Subsequently Published Updated Recommended Criteria

The National Toxics Rule (“NTR”) was EPA’s response to some states’ failing to adopt numeric criteria following the 1987 amendments to the CWA.¹¹⁴¹ The numeric toxic criteria promulgated in the NTR by EPA apply to the following states in whole or in part: Rhode Island,

¹¹³⁶ *Id.* at 3.

¹¹³⁷ *Id.* at 5.

¹¹³⁸ See Letter from Angela Somma, Chief, Protected Resources, NMFS, to Arthur-Jean Williams, EPA, Re: *Request for Endangered Species Act Section 7 Consultation for Registration of Pesticide Products Containing Clomazone throughout the United States and its Affiliated Territories* (May 22, 2009), available at <https://media.fisheries.noaa.gov/dam-migration/63806519clomazoneresponse.pdf>.

¹¹³⁹ See Letter from Angela Somma, Chief, Protected Resources, NMFS, to Arthur-Jean Williams, EPA, Re: *Request for Endangered Species Act Section 7 Consultation on the Environmental Protection Agency’s Review of their Registration of Pesticide Products Containing the Active Ingredient Fomesafen* (May 22, 2009), available at https://media.fisheries.noaa.gov/dam-migration/63806532fomesen_response.pdf.

¹¹⁴⁰ *Id.* at 1.

¹¹⁴¹ NTR Rules, *supra* n. 94.

Vermont, District of Columbia, Florida, Kansas, California,¹¹⁴² Nevada, and Alaska.¹¹⁴³ These criteria are limited to pollutants on the Toxic Pollutants Lists.¹¹⁴⁴ As a consequence, whenever EPA updates or adds to its 304(a) criteria for pollutants not on the Toxic Pollutants Lists, *see supra* sub-sections VII.A and B, the states covered by the NTR continue to not have numeric criteria for those pollutants. Pollutants that are not included in the NTR for this reason are the following non-priority pollutants (with their latest dates of recommended criteria publication): aluminum (2018), ammonia (freshwater 2013), carbaryl (2012), diazinon (2005), nonylphenol (2005), parathion (1995), and tributyltin (2004), along with the older criteria from the 1980s for ammonia (saltwater), chloride, chlorine, chlorpyrifos, demeton, guthion, iron, malathion, methoxychlor, and mirex. EPA determined all of these toxic pollutants were of sufficient hazard to aquatic life across the nation to warrant the publication of recommended criteria but has taken no action to add to the Toxic Pollutants Lists to ensure that they would apply to states that have failed to adopt 304(a) criteria.

I. Other Persistent, Bioaccumulative, and Toxic Chemicals Have Been Identified Through EPA Regulatory Programs

In addition to toxic chemicals identified under CERCLA and TRI, discussed *supra*, EPA has identified toxic pollutants of concern in carrying out other laws specific to managing

¹¹⁴² EPA promulgated the California Toxics Rule in 2000, consisting of numeric aquatic life criteria for 23 priority toxic pollutants; numeric human health criteria for 57 priority toxic pollutants; and a compliance schedule provision based on an Administrator's determination the criteria were necessary after a state court decision left California "without numeric water quality criteria for many priority toxic pollutants as required by the Clean Water Act, necessitating this action by EPA." Water Quality Standards; Establishment of Numeric Criteria for Priority Toxic Pollutants for the State of California, 65 Fed. Reg. 31681 (May 18, 2000).

¹¹⁴³ 40 C.F.R. § 131.36(d).

¹¹⁴⁴ 40 C.F.R. § 131.36(a) ("Scope. This section is not a general promulgation of the section 304(a) criteria for priority toxic pollutants but is restricted to specific pollutants in specific States."); *id.* § 131.36(b)(1) ("EPA's Section 304(a) criteria for Priority Toxic Pollutants.").

hazardous chemicals, namely the Toxic Substances Control Act (“TSCA”) and the Resource Conservation and Recovery Act (“RCRA”). There are likely numerous pollutants identified in these programs that likewise should be evaluated for placement on the Toxics Pollutants Lists. One example is nonylphenol, discussed in sub-section VII.A.11 *supra*, about which EPA says in the context of TSCA, “[Nonylphenols and Nonylphenol Ethoxylates] NP/NPE chemicals are highly toxic to aquatic life and have a wide variety of industrial and consumer uses that could lead to environmental releases.”¹¹⁴⁵

After Congress amended TSCA in 2016, EPA began implementing its new requirements.¹¹⁴⁶ First, it issued the following list of 10 chemicals under evaluation: 1,4-dioxane, 1-bromopropane, asbestos, carbon tetrachloride, cyclic aliphatic bromide cluster, methylene chloride, N-methylpyrrolidone, pigment violet 29, tetrachloroethylene (perchloroethylene), and trichloroethylene.¹¹⁴⁷ EPA subsequently issued final rules for five persistent, bioaccumulative, and toxic chemicals that EPA determined met the criteria for expedited action under TSCA.¹¹⁴⁸ After issuing final rules for these five toxics on January 6, 2021, EPA invited further public comment to “immediately review” these chemicals that “are

¹¹⁴⁵ EPA, Assessing and Managing Chemicals under TSCA, *Fact Sheet: Nonylphenols and Nonylphenol Ethoxylates*, available at <https://www.epa.gov/assessing-and-managing-chemicals-under-tsca/fact-sheet-nonylphenols-and-nonylphenol-ethoxylates>.

¹¹⁴⁶ See EPA, *Table of Milestones under the Amended Toxic Substances Control Act*, available at <https://www.epa.gov/chemicals-under-tsca/table-milestones-under-amended-toxic-substances-control-act>.

¹¹⁴⁷ See EPA, News Releases from Headquarters › Chemical Safety and Pollution Prevention (OCSPP), *EPA Names First Chemicals for Review Under New TSCA Legislation*, available at <https://www.epa.gov/archive/epa/newsreleases/epa-names-first-chemicals-review-under-new-tsca-legislation.html>.

¹¹⁴⁸ EPA, *Proposed Rule Regulation of Persistent, Bioaccumulative, and Toxic Chemicals Under TSCA Section 6(h)*, available at <https://www.regulations.gov/document/EPA-HQ-OPPT-2021-0202-0001>. These chemicals are 2,4,6-tris(tert-butyl)phenol (2,4,6-TTBP) (CASRN 732-26-3); decabromodiphenyl ether (decaBDE) (CASRN 1163-19-5); phenol, isopropylated phosphate (3:1) (PIP (3:1)) (CASRN 68937-41-7); pentachlorothiophenol (PCTP) (CASRN 133-49-3); and hexachlorobutadiene (HCBD) (CASRN 87-68-3). Of these, only the latter is on the Priority Pollutants List.

toxic and remain in the environment for long periods of time and can build up or accumulate in the body.”¹¹⁴⁹ In response, a distinguished list of academics, scientists, and clinicians responded by telling EPA that the agency “must address . . . all pathways of exposure and release” of these highly persistent toxic chemicals.¹¹⁵⁰ This group of scientists noted specifically that EPA’s rules on decabromodiphenyl ether (“decaBDE”) and hexachlorobutadiene (“HCB”) are “in marked contrast to the international consensus achieved under the Stockholm Convention by science and policy experts for [their] global elimination.”¹¹⁵¹ The commenters point to EPA’s contention in developing the HCB rules that it “presumes compliance” with the CWA is “inadequate to meet EPA’s requirement to reduce exposure to the extent practicable and in addition to being in direct contrast to the stated position of EPA leadership.”¹¹⁵² For other pollutants, EPA would not even be able to presume compliance with the CWA because EPA has failed to address them under this important statute.

J. Pollutants Listed in Appendix C to the *NRDC v. Train* Settlement

In the 1976 *NRDC v. Train* settlement, EPA agreed to the following:

In addition to those pollutants to which regulations must be established pursuant to subsection (a) of this paragraph 4, the Administrator shall also identify the categories or category of point sources which are discharging to navigable waters or introducing into treatment works . . . which are publicly owned the pollutants listed in Appendix C to this agreement.¹¹⁵³

¹¹⁴⁹ *Id.*

¹¹⁵⁰ *Comments from Academics, Scientists and Clinicians on the Regulation of Persistent, Bioaccumulative, and Toxic Chemicals under Section 6(h) of the Toxic Substances Control Act* (May 17, 2021), available at https://prhe.ucsf.edu/sites/g/files/tkssra341/f/wysiwyg/2021%2005%2017_%20PBT%20rule%20proposal_UCSF%20PRHE_comments%20and%20appendices_EPA.pdf.

¹¹⁵¹ *Id.* at 2.

¹¹⁵² *Id.* at 6.

¹¹⁵³ *NRDC v. Train* Settlement, *supra* n. 22, at ¶ 4b.

The chemicals listed in Appendix C are as follows: acetone, n-alkanes (C[10]-C[30]), biphenyl, chlorine, dialkyl ethers, dibenzofuran, diphenyl ether, methylethyl ketone, nitrites, secondary amines, styrene, and terpenes.¹¹⁵⁴ Of these, EPA has promulgated ELGs for three of the pollutants: acetone (2), methylethyl ketone (1), and nitrites (1).¹¹⁵⁵ To Petitioners' knowledge, with the exception of the ELGs established for these three pollutants, EPA has not identified the categories or category of point sources that are directly or indirectly discharging the pollutants listed in Appendix C to the nation's waters. These chemicals should be added to the Toxic Pollutants Lists.

K. Pollutants Included in National Effluent Guidelines

EPA's national effluent limitation guidelines do, in some instances, include toxic pollutants that are not on the Toxic Pollutants Lists.¹¹⁵⁶ EPA does not treat these pollutants as

¹¹⁵⁴ *Id.*, Appendix C.

¹¹⁵⁵ See ELG Database, *supra* n. 512 (Pollutant search set for Appendix C pollutants).

¹¹⁵⁶ Individual pollutants covered by at least one ELG in EPA's Effluent Limitations Guidelines and Standards (ELG) Database are listed under "Pollutant" in EPA's Pollutant Search online database available at <https://owapps.epa.gov/elg/>. Toxic pollutants not on the Toxic Pollutants Lists but included in the ELG database include the following: 1,3-dichloropropene; 2,4-D; 2,4-D salts and esters; 2,4-DC salts and esters; 3,4,5-Trichlorocatechol; 3,4,5-Trichloroguaiacol; 3,4,6-Trichlorocatechol; 3,4,6-Trichloroguaiacol; 4,5,6-Trichloroguaiacol; Acephate; Acetone; Acetonitrile; Acetophenone; Acifluorfen; Alachlor; Aldicarb; .alpha.-Terpineol; Aluminum; Ametryn; Ammonia; Aniline; AOX (Adsorbable organic halides); Atrazine; Azinphos-methyl (Guthion); Barium; Benfluralin; Benomyl and Carbendazim; Benzoic acid; Bolstar; Bromacil; Bromacil lithium salt; Bromide; Bromomethane; Bromoxynil; Bromoxynil octanoate; Busan 40 (Potassium-N-hydroxymethyl-N-methyldithiocarbamate); Busan 85 (Potassium dimethyldithiocarbamate); Butachlor; Capafol; Cabam-S (sodium dimethyldithiocarbamate); Carbaryl; Carbazol; Carbofuran; Chloromethane; Chloroneb; Chlorothalonil; Chlorpyrifos; Cobalt; Cyanazine; Dazomet; DCPA (dimethyl 2,3,5,6-tetrachloroterphthalate); DEF (S.S.S-tributyl phosphorotrithioate); Dichlorvos; Diethylamine; Dimethyl sulfoxide; Dinoseb; Dioxathion; Disulfoton; Diuron; Endothall salts and esters; Ethalfluralin; Ethanol; Ethion; Ethyl acetate; Fenarimol; Fensulfotion; Fention; Fenvalerate; Fluoride; Fluorine; Gold; Indium; Iron; Isobutyraldehyde; Isopropalin; Isopropanol; Isopropyl acetate; Isopropyl ether; KN Methyl (Potassium N-methyldithiocarbamate); Linuron; Malathion; Manganese; MCPA (2-methyl-4-chlorophenoxyacetic acid) salts and esters; Merphos; Methamidophos; Methanol; Methomyl; Methoxychlor; Methyl cellosolve (2-methoxyethanol); Methyl ethyl ketone; Methyl formate; Metribuzin; Mevinphos; Molybdenum; Nebam; Nebonate; Naled; n-Amyl acetate; n-Butyl acetate; n-Decane; n-Octadecane; Norfurlazon; o-Cresol; o-Dichlorobenzene; Organo-tin pesticides; Palladium; Parathion; Parathion ethyl; Parathion methyl; PCNB

“toxic” pollutants but, rather, as “nonconventional” pollutants.¹¹⁵⁷ With few exceptions (e.g., ammonia, aluminum) these pollutants are not subject to water quality-based permitting because they are not on the Toxic Pollutants Lists, yet they have been deemed of sufficient concern to EPA as toxic to aquatic life and/or human health to have been included in one or more ELGs. If a chemical is of concern in one industrial sector, it should be treated with the same concern in another sector.

L. Federal Agency Toxic Constituent Identifications

There are multiple sources that identify toxic pollutants for which there is extensive evidence on toxicity and prevalence in U.S. waters that EPA must consider when adding constituents to its Toxic Pollutants Lists. Two of these are discussed below and, as is evident from the USGS effort, these are dependent, in turn, upon multiple other national and international sources. Neither of these two constituent monitoring screening evaluations discussed here address toxic residues in aquatic animal tissue.

1. USGS National Water-Quality Assessment Constituent Prioritization

(Pentachloronitrobenzene); p-Cresol; Pedimethalin; Permethrin; Phorate; Phosmet; Platinum; Prometon; Prometryn; Pronomide; Propachlor; Propanil; Propazine; Pyrethrin I and II; Pyradine; Radium 22; Surfactants, anionic; Simazine; Stirofos; Sulfide; Tantalum; TCDF (2,3,7,8-Tetrachlorodibenzofuran); TCMTB ((Benzothiazol-2-ylthio)methyl thiocyanate; Tebuthurion; Terbacil; Terbufos; Terbutylazine; Terbutryn; Tetrachlorocatechol; Tetrachloroguaicol; Tetrahydrofuran; Tin; Titanium; Total phenolics; Total phenols; Total residual chlorine; Trans-1,2-dichloroethylene; Triadmefon; Tribromomethane; Trichlorophenol; Trichlorosyringol; Triethylamine; Trifluralin; Tungsten; Uranium; Vanadium; Vapam (sodium Methylthiocarbamate); Xylene; Ziram (zinc dimethyldithiocarbamate).

¹¹⁵⁷ See EPA, Effluent Guidelines, Learn About Effluent Guidelines, *Pollutant Types*, available at https://19january2021snapshot.epa.gov/eg/learn-about-effluent-guidelines_.html (“EPA has identified 65 pollutants and classes of pollutants as ‘toxic pollutants’, of which 126 specific substances have been designated ‘priority’ toxic pollutants. All other pollutants are considered to be ‘nonconventional.’”).

In 2013, the USGS published its evaluation of 2,541 constituents for the purpose of prioritizing them for national- and regional-level ambient monitoring of water and sediment.¹¹⁵⁸ These constituents were organized in the following groups: volatile organic compounds in water; pesticides in water or sediment; pharmaceuticals and hormones in water or sediment; trace elements and other inorganic constituents in water or sediment; cyanotoxins in surface water; lipophilic organic compounds in sediment; disinfection by-products in water; high-production-volume chemicals in water; wastewater-indicator and industrial compounds in water; and radionuclides in water.¹¹⁵⁹ They were then evaluated for “(1) the likelihood of a constituent to occur in the matrix of interest, and (2) the likelihood of that constituent to have adverse effects on human health or aquatic life, or both.”¹¹⁶⁰ As a result, “a constituent that could occur widely in the environment and that is likely to affect human health or aquatic life would be considered a high priority for ambient monitoring.”¹¹⁶¹ The result was three tiers of constituents.

USGS developed the list of constituents to screen by looking at the following sources for effects to human health: EPA drinking water standards and advisories; EPA’s Contaminant Candidate List, a list of 103 compounds (“not currently subject to proposed or promulgated national primary drinking-water regulations, but are known or anticipated to occur in public water systems and may require future regulation under the Safe Drinking Water Act”); EPA’s Unregulated Contaminant Monitoring Rule (“UCMR”) monitoring data; Guidelines for Canadian

¹¹⁵⁸ See USGS, *Prioritization of Constituents for National- and Regional-Scale Ambient Monitoring of Water and Sediment in the United States* (June 1, 2013) (hereinafter “USGS Prioritization”) available at <https://www.usgs.gov/publications/prioritization-constituents-national-and-regional-scale-ambient-monitoring-water-and>.

¹¹⁵⁹ *Id.* at 3. Of these, all were evaluated for both human health and aquatic life except for volatile organic compounds, cyanotoxins, disinfection by-products, and radionuclides, all of which were evaluated for human health alone. *Id.* at 5.

¹¹⁶⁰ *Id.* at 4–5.

¹¹⁶¹ *Id.* at 5.

Drinking Water Quality are published by Health Canada; State of California maximum contaminant levels and public health goals; EPA’s Mid-Atlantic Risk Assessment User’s Guide for CERCLA sites; Centers for Disease Control and Prevention (CDC) National Health and Nutrition Examination Survey (documenting chemicals detected in human blood or urine); Canada’s Domestic Substances List Program (“a substance is “toxic” if it enters the environment in a quantity or concentration or under conditions that (1) may have an immediate or long-term harmful effect on the environment or its biological diversity, (2) may constitute a danger to the environment upon which life depends, or (3) may constitute a danger to human life or health in Canada”); USGS’s National Water-Quality Assessment (“NAWQA”) Potential Endocrine Disruptors List, which relies “mainly on three references: the Institute for Environment and Health (2005), Global Water Research Coalition (2003), and BKH Consulting Engineers (2000)” to identify 22 constituents with the “highest potential for exposure and endocrine disruption” of 108 “identified as having evidence of endocrine disruption and high exposure concern”¹¹⁶²; the Harvard School of Public Health, Industrial Chemicals with Neurotoxic Effects (identifies 203 industrial constituents that “cause neurodevelopmental disorders, particularly during early fetal development”¹¹⁶³); and the State of California Proposition 65 List (a 285 subset of 796 compounds for which there is some information on toxicity).¹¹⁶⁴

For aquatic life, the screening list was developed to focus on freshwater constituents derived from the following sources: EPA’s 304(a) recommended criteria; Canadian Water-

¹¹⁶² *Id.* at 8. These 22 compounds are as follows: acetochlor, alachlor, atrazine, bisphenol A, bromomethane, 4-chloro-2-methylphenol, cyfluthrin, diazinon, 3,4-dichloroaniline, 2,4-dichlorophenoxyacetic acid (2,4-D), dicofol, dieldrin, dimethoate, diuron, α -endosulfan, β -endosulfan, linuron, malathion, 4-tert-octylphenols (branched), parathion-methyl, simazine, and tetrachloroethene.

¹¹⁶³ *Id.* at 9.

¹¹⁶⁴ *Id.* at 7–9.

Quality Guidelines for the Protection of Aquatic Life; the Great Lakes Water Quality Agreement of 1978; European Commission priority list of endocrine disruptors (the Commission determined 147 of a candidate list of 553 suspected endocrine disruptors were “likely to be persistent in the environment or produced in high volumes” and for its screening list, USGS chose 118 constituents for which there was “clear evidence of endocrine disrupting activity” or “some evidence indicating potential activity”)¹¹⁶⁵; and the Canadian Domestic Substances List—substances that are inherently toxic to the environment.¹¹⁶⁶ Additional information was sourced from “international concern, and that also may be present in water or sediment in the United States, as evidenced by treaties or collaborative programs.”¹¹⁶⁷

The results of the screening process identified 1,081 constituents of high priority—602 for water and 686 for sediment¹¹⁶⁸—while USGS identified 436 for water and 246 for sediment that it determined were not the highest priority for ambient monitoring “because of resource limitations but could be of interest to other programs that focus on targeted sampling of contaminated sites, such as animal feeding operations, landfills, or wastewater effluent discharge points.”¹¹⁶⁹

2. EPA’s Contaminant Candidate List and Unregulated Contaminant Monitoring Rule

Pursuant to the Safe Drinking Water Act (“SDWA”), as amended in 1996, EPA carries out a monitoring program for unregulated contaminants, including the development of a

¹¹⁶⁵ *Id.* at 10.

¹¹⁶⁶ *Id.* at 9–10.

¹¹⁶⁷ *Id.* at 10.

¹¹⁶⁸ *Id.* at 21–51.

¹¹⁶⁹ *Id.* at 20.

Contaminant Candidate List” (“CCL”) every five years.¹¹⁷⁰ These unregulated contaminants are defined as “contaminants that are currently not subject to any proposed or promulgated national

¹¹⁷⁰ SDWA 42 U.S.C. § 300g-1(b)(1)(B); *see also* EPA, *Drinking Water Contaminant Candidate List (CCL) and Regulatory Determination*, available at <https://www.epa.gov/ccl> (hereinafter “EPA CCL Website”). Note, in addition, that EPA is required to issue a list of no more than 30 unregulated contaminants to be monitored by public water systems. These Unregulated Contaminant Monitoring Rules include the following: 64 Fed. Reg. 50556–50620 (Sept. 17, 1999) (pertaining to 2,4-dinitrotoluene; 2,6-dinitrotoluene; acetochlor; DCPA mono-acid degradate; DCPA di-acid degradate; 4,4'-DDE; EPTC; molinate; MTBE; nitrobenzene; perchlorate; terbacil; 1,2-diphenylhydrazine; 2-methylphenol; 2,4-dichlorophenol; 2,4-dinitrophenol; 2,4,6-trichlorophenol; diazinon; disulfoton; diuron; Fonofos; Linuron; nitrobenzene; Prometon; terbufos); 72 Fed. Reg. 368–398 (Jan. 4, 2007) (pertaining to Dimethoate; Terbufos sulfone; 2,2',4,4'-tetrabromodiphenyl ether (BDE-47); 2,2',4,4',5'-pentabromodiphenyl ether (BDE-99); 2,2',4,4',5,5'-hexabromobiphenyl (HBB); 2,2',4,4',5,5'-hexabromodiphenyl ether (BDE-153); 2,2',4,4',6-pentabromodiphenyl ether (BDE-100); 1,3-dinitrobenzene; 2,4,6-trinitrotoluene (TNT); Hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX); Acetochlor; Alachlor; Metolachlor; Acetochlor ethane sulfonic acid (ESA); Acetochlor oxanilic acid (OA); Alachlor ethane sulfonic acid (ESA); Alachlor oxanilic acid (OA); Metolachlor ethane sulfonic acid (ESA); Metolachlor oxanilic acid (OA); N-nitroso-diethylamine (NDEA); N-nitroso-dimethylamine (NDMA); N-nitroso-di-n-butylamine (NDBA); N-nitroso-di-n-propylamine (NDPA); N-nitroso-methylethylamine (NMEA); N-nitroso-pyrrolidine (NPYR)); 77 Fed. Reg. 26072–26101 (May 2, 2012) (pertaining to 1,2,3-trichloropropane; 1,3-butadiene; chloromethane (methyl chloride); 1,1-dichloroethane; bromomethane (methyl bromide); chlorodifluoromethane (HCFC-22); bromochloromethane (halon 1011); 1,4-dioxane; vanadium; molybdenum; cobalt; strontium; chromium; chromium-6; chlorate; perfluorooctanesulfonic acid (PFOS); perfluorooctanoic acid (PFOA); perfluorononanoic acid (PFNA); perfluorohexanesulfonic acid (PFHxS); perfluoroheptanoic acid (PFHpA); perfluorobutanesulfonic acid (PFBS); 17-β-estradiol; 17-α-ethynylestradiol (ethinyl estradiol); 16-α-hydroxyestradiol (estriol); equilin; estrone; testosterone; 4-androstene-3,17-dione); 81 Fed. Reg. 92666–92692 (Dec. 20, 2016) (pertaining to germanium; manganese; alpha-hexachlorocyclohexane; chlorpyrifos; dimethipin; ethoprop; oxyfluorfen; profenofos; tebuconazole; total permethrin (cis- & trans-); tribufos; HAA5 (includes: dibromoacetic acid, dichloroacetic acid, monobromoacetic acid, monochloroacetic acid, trichloroacetic acid); HAA6Br (includes: bromochloroacetic acid, bromodichloroacetic acid, dibromoacetic acid, dibromochloroacetic acid, monobromoacetic acid, tribromoacetic acid); HAA9 (includes: bromochloroacetic acid, bromodichloroacetic acid, chlorodibromoacetic acid, dibromoacetic acid, dichloroacetic acid, monobromoacetic acid, monochloroacetic acid, tribromoacetic acid, trichloroacetic acid); 1-butanol; 2-methoxyethanol; 2-propen-1-ol; butylated hydroxyanisole; o-toluidine; quinoline); and 86 Fed. Reg. 73131–73157 (Dec. 27, 2021) (pertaining to 11-chloroeicosafluoro-3-oxaundecane-1-sulfonic acid (11Cl-PF3OUdS); perfluorodecanoic acid (PFDA); 1H, 1H, 2H, 2H-perfluorodecane sulfonic acid (8:2 FTS); perfluorododecanoic acid (PFDoA); 1H, 1H, 2H, 2H-perfluorohexane sulfonic acid (4:2 FTS); perfluoroheptanesulfonic acid (PFHpS); 1H, 1H, 2H, 2H-perfluorooctane sulfonic acid (6:2 FTS); perfluoroheptanoic acid (PFHpA); 4,8-dioxa-3H-perfluorononanoic acid (ADONA); perfluorohexanesulfonic acid (PFHxS); 9-chlorohexadecafluoro-3-oxanone-1-sulfonic acid (9Cl-PF3ONS); perfluorohexanoic acid (PFHxA); hexafluoropropylene oxide dimer acid (HFPO-DA) (GenX); perfluorononanoic acid (PFNA); nonafluoro-3,6-dioxaheptanoic acid (NFDHA); perfluorooctanesulfonic acid (PFOS); perfluoro (2-ethoxyethane) sulfonic acid (PFEESA); perfluorooctanoic acid (PFOA); perfluoro-3-methoxypropanoic acid (PFMPA); perfluoropentanesulfonic acid (PFPeS); perfluoro-4-methoxybutanoic acid (PFMBA); perfluoropentanoic acid (PFPeA); perfluorobutanesulfonic acid

primary drinking water regulations, but are known or anticipated to occur in public water systems.”¹¹⁷¹ After a final CCL is published, EPA determines whether it will regulate at least five contaminants from the list in what is termed a “regulatory determination,” based on whether the contaminant may have an adverse effect on human health, it is known to occur or substantially likely to occur in public water systems, and regulation of the contaminant “presents a meaningful opportunity for health risk reductions.”¹¹⁷²

To date, EPA has completed five CLLs and four related regulatory determinations.¹¹⁷³ In its first CCL, denominated “CCL 1,” EPA identified 50 chemicals and 10 microbial contaminants that present a potential public health concern in drinking water¹¹⁷⁴ and decided not to regulate any of the eight chemical constituents evaluated in its regulatory determination.¹¹⁷⁵

(PFBS); perfluoroundecanoic acid (PFUnA); perfluorobutanoic acid (PFBA); n-ethyl perfluorooctanesulfonamidoacetic acid (NEtFOSAA); perfluorotetradecanoic acid (PFTA); n-methyl perfluorooctanesulfonamidoacetic acid (NMeFOSAA); perfluorotridecanoic acid (PFTrDA), lithium).

¹¹⁷¹ EPA, Contaminant Candidate List (CCL) and Regulatory Determination, Basic Information on the CCL and Regulatory Determination, *What is the drinking water CCL?*, available at <https://www.epa.gov/ccl/basic-information-ccl-and-regulatory-determination>.

¹¹⁷² *Id.* at *What happens to contaminants on the CCL?*; *id.* at *What criteria does EPA consider to make a regulatory determination?*; SDWA 42 U.S.C. § 300g-1(b)(1)(A), (B)(ii).

¹¹⁷³ EPA CCL Website, *supra* n. 1172.

¹¹⁷⁴ 63 Fed. Reg. 10274–10287 (March 2, 1998).

¹¹⁷⁵ 68 Fed. Reg. 42897–42906 (July 18, 2003). The regulatory determinations for these eight contaminants provide EPA will significant information about their toxicity for the purpose of adding them to the Toxic Pollutants Lists. See EPA, *Contaminant Candidate List Regulatory Determination Support Document for Aldrin and Dieldrin* (July 2003), available at https://www.epa.gov/sites/default/files/2014-09/documents/support_cc1_aldrin-dieldrin_ccl_regdet.pdf; EPA, *Health Effects Support Document for Aldrin/Dieldrin* (Feb. 2003), available at https://www.epa.gov/sites/default/files/2014-09/documents/support_cc1_aldrin-dieldrin_healtheffects.pdf; EPA, *Contaminant Candidate List Regulatory Determination Support Document for Manganese* (July 2003), available at https://www.epa.gov/sites/default/files/2014-09/documents/support_cc1_magnese_ccl_regdet.pdf; EPA, *Health Effects Support Document for Manganese* (Feb. 2003), available at https://www.epa.gov/sites/default/files/2014-09/documents/support_cc1_magnese_healtheffects_0.pdf; EPA, *Drinking Water Health Advisory for Manganese* (Jan. 2004), available at https://www.epa.gov/sites/default/files/2014-09/documents/support_cc1_magnese_dwreport_0.pdf; EPA, *Candidate Contaminant List Regulatory Determination Support Document for Metribuzin* (July 2003), available at https://www.epa.gov/sites/default/files/2014-09/documents/support_cc1_metribuzin_ccl_regdet.pdf; EPA, *Health Effects Support Document for*

In its CCL 2, EPA merely carried forward the remaining 51 constituents from CCL 1,¹¹⁷⁶ chose 11 chemical contaminants to evaluate and decided not to regulate any of them.¹¹⁷⁷ For CCL 3, EPA identified 104 chemicals or chemical groups¹¹⁷⁸ and decided not to regulate four of them and to postpone a determination on a fifth.¹¹⁷⁹ In 2016, EPA announced its CCL 4 with 97 chemicals¹¹⁸⁰ and in 2021 issued its regulatory determination to not regulate six chemicals and to

Metribuzin (Feb. 2003), available at https://www.epa.gov/sites/default/files/2014-09/documents/support_cc1_metribuzin_healtheffects.pdf; EPA, *Contaminant Candidate List Regulatory Determination Support Document for Naphthalene* (July 2003), available at https://www.epa.gov/sites/default/files/2014-09/documents/support_cc1_naphthalene_ccl_regdet.pdf; EPA, *Health Effects Support Document for Naphthalene* (Feb. 2003), available at https://www.epa.gov/sites/default/files/2014-09/documents/support_cc1_naphthalene_healtheffects.pdf¹¹⁷⁶ 70 Fed. Reg. 9071–9077 (Feb. 24, 2005); EPA, *Contaminant Candidate List Regulatory Determination Support Document for Sodium* (July 2003), available at https://www.epa.gov/sites/default/files/2014-09/documents/support_cc1_sodium_ccl_regdet.pdf; EPA, *Drinking Water Advisory: Consumer Acceptability Advice and Health Effects Analysis on Sodium* (Feb. 2003), available at https://www.epa.gov/sites/default/files/2014-09/documents/support_cc1_sodium_dwreport.pdf; EPA, *Contaminant Candidate List Regulatory Determination Support Document for Sulfate* (July 2003), available at https://www.epa.gov/sites/default/files/2014-09/documents/support_cc1_sulfate_dwreport.pdf; EPA, *Drinking Water Advisory: Consumer Acceptability Advice and Health Effects Analysis on Sulfate* (Feb. 2003), available at https://www.epa.gov/sites/default/files/2014-09/documents/support_cc1_sulfate_healtheffects.pdf; EPA, *Candidate Contaminant List Regulatory Determination Support Document for Hexachlorobutadiene* (July 2003), available at https://www.epa.gov/sites/default/files/2014-09/documents/support_cc1_hexachlorobutadiene_ccl_regdet.pdf; EPA, *Health Effects Support Document for Hexachlorobutadiene* (July 2003), available at https://www.epa.gov/sites/default/files/2014-09/documents/support_cc1_hexachlorobutadiene_healtheffects.pdf.

¹¹⁷⁷ 72 Fed. Reg. 24015–20458 (May 1, 2007). As with the regulatory determinations for CCL 1, those for CCL 2 contain relevant information for adding the following pollutants to the Toxic Pollutants Lists: boron; dimethyl tetrachloroterephthalate (DCPA) mono- and di-acid degradates; 1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene (DDE); 1,3-dichloropropene; 2,4- and 2,6-dinitrotoluene; s-ethyl dipropylthiocarbamate (EPTC); fonofos; terbacil; and 1,1,2,2-tetrachloroethane. See EPA, *Regulatory Determinations Support Document for Selected Contaminants from the Second Drinking Water Contaminant Candidate List (CCL 2)* (June 2008), available at https://www.epa.gov/sites/default/files/2014-09/documents/report_cc12-reg2_supportdocument_full.pdf¹¹⁷⁸ 74 Fed. Reg. 51850–51862 (Oct. 8, 2009).

¹¹⁷⁹ 81 Fed. Reg. 13–19 (Jan. 4, 2016). EPA’s support documents for these regulatory determinations provide information for use in adding pollutants to the Toxic Pollutants Lists. See EPA, *Regulatory Determination 3 Support Document* (Dec. 2015) (pertaining to 1,3-dinitrobenzene, dimethoate, terbufos, and terbufos sulfone), available at <https://www.regulations.gov/document/EPA-HQ-OW-2012-0155-0029>.

¹¹⁸⁰ 81 Fed. Reg. 81099–81114 (Nov. 17, 2016).

regulate two, PFOS and PFOA.¹¹⁸¹ Finally, EPA completed CCL 5 in 2022 that includes 66 chemicals and three chemical groups.¹¹⁸²

Notwithstanding a 27-year history of choosing to not add any new chemicals to regulate under the SDWA but two, the evaluations of toxic contaminants contained in these efforts are relevant to adding pollutants to the Toxic Pollutants Lists under the CWA. EPA's SDWA regulatory determinations are based on public water systems, not private drinking water wells where many people, particularly poor and rural people, obtain their drinking water, and do not address protecting human health from contaminants in fish and shellfish tissue. Moreover, pursuant to the SDWA, EPA factors the cost and feasibility of treatment technologies into its regulatory decision-making on preventing risk to human health, unlike the CWA. And, finally, the SDWA is unlike the CWA in that it seeks only to protect human health from treated drinking water from public systems, and has no role in protecting aquatic and aquatic-dependent fish and wildlife.

VIII. RELIEF REQUESTED BY THIS PETITION

For the reasons explained in this petition, NWEA and the Center request that EPA undertake rulemaking pursuant to the Administrative Procedure Act, 5 U.S.C. § 553, to place the

¹¹⁸¹ 86 Fed. Reg. 12272–12291 (March 3, 2021). The regulatory determinations for CCL 4 provide information for use in adding pollutants to the Toxic Pollutants Lists. *See*, EPA determinations in Docket No. EPA-HQ-OW-2019-0583 (pertaining to perfluorooctanesulfonic acid (PFOS); perfluorooctanoic acid (PFOA); 1,1-dichloroethane; acetochlor; methyl bromide (bromomethane); metolachlor; nitrobenzene; and RDX) *available at* <https://www.regulations.gov/search?filter=EPA-HQ-OW-2019-0583>.

¹¹⁸² 87 Fed. Reg. 68060–68050 (Nov. 14, 2022).

pollutants and chemical families listed in Attachments A, B,¹¹⁸³ and C¹¹⁸⁴ on the Toxics Pollutant List and/or the Priority Pollutant List.¹¹⁸⁵

In addition, for the reasons detailed above, Petitioners hereby also petition EPA to take the following additional actions as follows:

- To establish by rule a method by which EPA will (1) propose changes to and accept public input on the Toxic Pollutant List and Priority Pollutant List every three years; (2) commit to revise the lists upon completion of this triennial review; and (3) make determinations pursuant to CWA Section 307(b)(1) to identify pollutants that are not susceptible to treatment by publicly owned treatment works and are therefore likely to pass through such facilities, or to interfere with the operation of such treatment works; and
- For pollutants identified in this petition, make determinations pursuant to CWA Section 307(b)(1) to identify pollutants that are not susceptible to treatment by publicly owned treatment works and are therefore likely to pass through such facilities, or to interfere with the operation of such treatment works, for both those with only secondary treatment and those with advanced secondary and/or tertiary treatment operations.

CONCLUSION

As this petition has demonstrated, the Toxic Pollutants Lists are key to how EPA and the states carry out both the technology-based and the water quality-based approaches of the Clean Water Act. EPA's 47-year delay in updating these lists to reflect the wide range of toxic

¹¹⁸³ The Endocrine Disruption Exchange ("TEDX"), *TEDX List of Potential Endocrine Disruptors* (2011), available at <https://endocrinedisruption.org/interactive-tools/tedx-list-of-potential-endocrine-disruptors/search-the-tedx-list#sname=&searchfor=any&sortby=chemname&action=search&searchcats=all&sortby=chemname>; see also *id.* at *Methods*, available at <https://endocrinedisruption.org/interactive-tools/tedx-list-of-potential-endocrine-disruptors/methodology>.

¹¹⁸⁴ USGS Prioritization, *supra* n. 1158, at 21, Table 1 ("Constituents identified as having high priority (Tier 1) for national- or regional-scale ambient monitoring of water or sediment in the United States").

¹¹⁸⁵ Some of these pollutants may already be on one list, but not the other, because the Toxic Pollutant List contains chemical families and categories that may have become ambiguous with the passage of time but then included only one or a few chemicals from that family on the Priority Pollutant List. Through this Petition, Petitioners ask that all the pollutants and chemical families included in the above list be added to both the Toxics Pollutant List and the Priority Pollutant List unless they are already on those lists.

pollutants found in water, sediment, fish, and wildlife imperils the health of people, the food web, and species, including jeopardizing the most vulnerable—threatened and endangered species and people already subject to environmental injustices.

Respectfully submitted,



Nina Bell, Executive Director
Northwest Environmental Advocates
P.O. Box 12187
Portland, OR 97212
nbell@advocates-nwea.org

Brett Hartl, Government Affairs Director
Hannah Connor, Environmental Health Deputy Director
Center for Biological Diversity
1411 K St. NW, Suite 1300
Washington, DC 20005
BHartl@biologicaldiversity.org
hconnor@biologicaldiversity.org

Dated this day, the 31st of July 2023.

Attachments: Attachment A – List of Pollutants and Chemical Families
Attachment B – TEDX List
Attachment C – USGS Tier 1 Constituents
Attachment D – List of Attachments
CD with attachments