Coastal Pollution Data Explorer: Metadata

Contents

OVERVIEW

These metadata are associated with the Coastal Pollution Data Explorer (CPDE), accessed at this link: https://arcg.is/0SqSO9. This document includes descriptions of the datasets, tables dictionaries, featured contaminants and metals, analytes included in each contaminant group, and literature sources.

Coastal Pollution Data Explorer (CPDE) is an interactive web-based interface where users can explore spatial and temporal trends of chemical, physical, biological, and toxicological data. Users can compare, analyze, graph, and download NOAA's National Centers for Coastal Ocean Science (NCCOS) contaminant data from 1986 to the present. CPDE includes data from NCCOS' Stressor Detection and Impacts science division, including the Monitoring and Assessment branch and Ecotoxicology branch data from 1986 to the present. The Monitoring and Assessment branch, particularly the long-term national Mussel Watch monitoring program, monitors the status and trends of chemical contaminants and biological stressors in coastal waters. The Ecotoxicology branch conducts research to evaluate and predict the effects of chemical contaminants and other environmental stressors on coastal ecosystems. Sampling for both programs includes tissue and sediment monitoring, emphasizing bivalves, which are region-dependent.

DATASETS DESCRIPTIONS

Following datasets are included in different parts of the CPDE and available for downloading. Provided below are the data field names, descriptions, and details for the different datasets available in the CPDE: Explore Data part of CPDE, Download Analytes Data part of CPDE, Infographic Reports part of CPDE.
A few quick notes about each part of the CPDE below:

- **"Explore Data" part of CPDE** includes 3 datasets of the National Mussel Watch Program - Legacy, Contaminants of Emerging Concern, and Trace Metals. In this part analytes in the dataset are grouped by the contaminant or metal and represented as totaled sums. Totaled sums may represent a subset of the contaminants in each group or sampling years, to preserve data continuity. Tissue data for Legacy and Metals was converted to wet weight for easier comparison to external guidelines (see "Chemical Descriptions & Available Thresholds"). Chemicals of emerging concern data for tissue samples are already received from the laboratories in wet weight units. Sediment data is always presented in dry weights. Additionally, all values below method detection limits (MDL) were qualified as undetected and were assigned a value of zero before sums and averages were calculated. Datasets are:
    - **Legacy contaminants** are chemicals once used in the U.S. but generally discontinued or banned. However, many persist in soil and water, breaking down slowly and continuing to impact wildlife long after their use has ended. Often originating from industrial activities, these pollutants remain in the environment for decades.
    - **Contaminants of Emerging Concern (CECs)** are chemicals and toxics that have been found in waterbodies that may cause ecological or human health impacts and are not currently regulated. Many of these compounds are manufactured to replace other banned chemicals. The scope and impact of these CECs are largely unknown and potentially vast.
    - **Trace metals** accumulate in both tissue and sediment, and although metals occur naturally in the environment, anthropogenic use of metal products, particularly since the industrial age, has resulted in excessive releases.
- **"Create Report" part of the CPDE** includes 3 datasets of the National Mussel Watch Program - Legacy, Contaminants of Emerging Concern, and Trace Metals mentioned above. Data provides the contamination summary for the area of interest and allows users to see all the contaminants and metals that have been sampled there since the start of the program. Similarly, to the explore part of the CPDE, datasets are grouped by the contaminant or metal and represented as totaled sums.
  When a CPDE user creates a report, the selected National Mussel Watch Program sample sites are compared to both regional and nationwide averages for the selected matrix. Comparative averages for Legacy and Metals data are derived from the Mussel Watch data from 2000-2010. This time span was selected because it represents the last ten years that Mussel Watch was able to complete comprehensive biennial nationwide surveys before the program began transitioning to the five-year rotational regional model currently used. These ten years of data therefore provide a robust national distribution of samples that were also predominantly analyzed by the same laboratory, reducing any potential imbalances induced by varying sampling designs or analytical procedures. Since the program didn't begin analyzing Contaminants of Emerging Concerns (CEC) until 2009, the Mussel Watch CEC dataset is comparatively limited both temporally and geographically. For this reason, all of the Mussel Watch Program's existing CEC data for each matrix is used for the comparative regional and national averages in the reports.
- **"Analyte Level Data" part of the CPDE** includes all the data from the long - term National Mussel Watch monitoring program, additional special studies from the NS&T Data Portal, and other data collected by NCCOS. Data is represented at individual analyte level. The "Download Analytes Data" part of the CPDE has all of the data in their original units to prevent data loss due to not having the required percent moisture/dry parameters to make the conversion for all of the samples. Please note, values provided by TDI-BI laboratory below the method detection limits (MDL) are included in the dataset to differentiate between parameters not detected by the analytical methods and parameters detected but not quantifiable. Users should adjust the downloaded data as needed for their purposes.

SUGGESTED CITATIONS

Please credit the Coastal Pollution Data Explorer (https://arcg.is/0SqSO9) when you use the data or create a data report.

Suggested citation: Morozova A.O., Buckel, C.A., Rider, M.M, Arzayus L.F., Apeti, D.A. 2023. "Coastal Pollution Data Explorer". ESRI Web Experience. US DOC NOAA NOS National Centers for Coastal Ocean Science (NCCOS). https://arcg.is/0SqSO9

DATA DICTIONARY

The following tables contain column descriptions for the datasets included in the CPDE.

Explore Data – Column Descriptions

Field	Description
OBJECTID	Unique numerical identifier for each feature/record in dataset/table
Site ID	Location label which defines the sampling site name
Fiscal Year	Fiscal year (October-September) the sample was collected
CPDE Explorer Group	Contaminant group
Unit	Units used to measure the result of a given parameter in a sample
Value	Measured result from the analyzed parameter
Region	Great Lakes, Gulf Coast, North Atlantic Coast, South Atlantic Coast, Pacific Coast, or International
Matrix	Sample matrix type
Scientific Name	Scientific name of matrix sampled
Study	Name of the study
Study Type	Type of study (long-term monitoring or a short-term special study).
Sample ID	An identifier used to uniquely identify a sample
General Location	Descriptive general location of the site, if needed
Specific Location	Descriptive specific location of the site, if needed
State	A state or territory where the sample was collected
Longitude	Longitude coordinates
Latitude	Latitude coordinates
Collaborator	Local, state, or federal agencies that collaborated on study design and implementation
Collection Date	A date when a sample was collected
Method	Analytical method used to process the sample
Laboratory	Laboratory where the sample was analyzed (see table below)
Parameter Count	How many parameters (analytes) are included in the contaminant group

Analyte Level Data – Column Descriptions

Field	Description
OBJECTID	Unique numerical identifier for each feature/record in dataset/table
Site ID	Location label which defines the sampling site name
Fiscal Year	Fiscal year (October-September) the sample was collected
CPDE SearchEngine Group	Contaminant group
Unit	Units used to measure the result of a given parameter in a sample
Value	Measured result from the analyzed parameter
Region	Great Lakes, Gulf Coast, North Atlantic Coast, South Atlantic Coast, Pacific Coast, or International
Matrix	Sample matrix type
Scientific Name	Scientific name of matrix sampled
Study	Name of the study
Study Type	Type of study (long-term monitoring or a short-term special study).
Sample ID	An identifier used to uniquely identify a sample
General Location	Descriptive general location of the site, if needed

Specific Location	Descriptive specific location of the site, if needed
State	A state or territory where the sample was collected
Latitude	Longitude coordinates
Longitude	Latitude coordinates
Collaborator	Local, state, or federal agencies that collaborated on study design and implementation
Collection Date	A date when a sample was collected
Method	Analytical method used to process the sample
Laboratory	Laboratory where the sample was analyzed (see table below)
Parameter Code	Abbreviated name of parameter (analyte)
Parameter	Name of parameter (analyte)
Qualifier	Data qualifier provided by the lab
Detection Limit	Lowest concentration a substance that can be reliably detected and reported with 99% confidence
Method Blank	Analyte-free matrix processed alongside samples to document contamination resulting from the analytical process

Laboratory Acronyms

Laboratories names are abbreviated in the Analyte Level Data. Please use the table below to identify the analytical laboratory associated with each sample.

Abbreviation	**Laboratory Full Description**
GERG	Geochemical and Environmental Research Group or the Department of Oceanography at Texas A&M University (TAMU), College Station, TX.
BATT	Battelle Laboratories, Duxbury, MA
SAIC	Scientific Applications International Corporation, LaJolla, CA
WC	Wadsworth Center, NY State Dept. of Health, Albany, NY (TDI-BI subcontractor)
TDI-BI	TDI-Brooks, College Station, TX
TERL	Trace Element Research Laboratory, Texas A&M University, College Station, TX (TDI-BI subcontractor)
HML	NOAA Ecotoxicology Branch, Charleston, SC
VIMS	Virginia Institute of Marine Science, Gloucester Point, VA
HRI	Health Research Inc., Menands, NY (TDI-BI subcontractor)
AXYS	SGS AXYS Analytical Services Ltd., Sidney, British Columbia
BAL	BAL Laboratory, Cranston, RI

METHODS

Since the majority of the data in the CPDE, and all of the data in the Explore section, originates from the Mussel Watch Program (MWP), the following samples distribution and methods sections are based on MWP protocols.

Samples Distribution

The Mussel Watch Program currently samples sediment and bivalves from a network of more than 400 sites along the North Atlantic Coast, South Atlantic Coast (including Puerto Rico), Pacific Coast (including Alaska and Hawaii), the Gulf Coast, and the Great Lakes on a 5-year rotating regional schedule. Sample distribution between the regions is:

1. Great Lakes: Zebra mussel (*Dreissena polymorpha*, Pallas 1771) and Quagga mussel (*Dreissena bugensis*, Andrusov, 1897), both which are invasive species (Hebert et al. 1989; Mills et al. 1993).
2. Gulf Coast: Eastern oyster (*Crassostrea virginica*, Gmelin, 1791).
3. North Atlantic Coast: Blue mussel (*Mytilus edulis*, Linnaeus 1758).
4. South Atlantic Coast: Eastern oyster (*Crassostrea virginica*, Gmelin, 1791); Mangrove oyster (*Crassostrea rhizophorae*, Guilding, 1828).
5. Pacific Coast, Alaska, & Hawaii: *Mytilus* species (i.e., *Mytilus galloprovinciallis, Mytilus trossulus, Mytilus californianus*, Conrad 1837); Hawaiian oyster (*Dendostrea sandvichensis*, Sowerby, 1871).

Field Methods

Originally, MWP sites were not randomly selected nor designed to target specific pollution sources. The sites were selected in locations with an abundant population of bivalves to allow repetitive sampling and to convey information about the degree of chemical contamination in the general area. Since 2010, the MWP has begun incorporating caged mussels into its sampling design to expand the geographical variety and extent of the program's data and to target areas of concern.

Sampling methods followed the MWP's standard field protocols. Wild oysters and mussels are collected by hand or dredged from intertidal to shallow subtidal zones. Caged-oysters were harvested from reference sites and deployed between 2-3 months. All bivalves were brushed clean, packed in iced containers and shipped to analytical laboratories within two days of collection. Sediment samples were collected using a modified Van-Veen sampler and preserved on ice or in refrigerators until shipped to the laboratory. The field activities were designed to have negligible impacts on the environment as called for by the NCCOS environmental compliance policy.

Analytical Methods

The laboratory at which analyses were conducted has changed over time and, although inter-laboratory calibrations have been conducted with low concern, this information is relevant. Analytical laboratory information is provided with the data in the “Download Analytes Data” part of the CPDE.

In the “Explore Data” part of the CPDE, tissue data for legacy organic contaminants and trace metals was converted to wet weight for easier comparison to external guidelines (see "Chemical Descriptions & Available Thresholds"). Contaminants of emerging concern data for tissue samples are already received from the laboratories in wet weight units. Sediment data is always presented in dry weights. Additionally, all values below method detection limit (MDL) were qualified as undetected and were assigned a value of zero before sums and averages were calculated. The “Download Analytes Data” part of the CPDE presents all of the data in their original units to prevent data loss due to not having the required percent moisture/dry parameters to make the conversion for all of the samples.

Trace metals contaminant data is presented in micrograms per dry or wet gram of sample for tissue (µg/g dw or µg/g ww) and micrograms of contaminant per dry gram for sediment (µg/g dw). Contaminants of emerging concern and legacy contaminants are presented in nanograms of contaminant per dry or wet gram for tissue (ng/g dw or ng/g ww) and nanograms of contaminant per dry gram for sediment (ng/g dw). For years with replicate analyses (1986-1992 and newly established sites), the average of the replicate concentrations is presented.

Despite of the number of sites for a coastline as large as that of the U.S., relatively few species are required to determine a national contaminant perspective. For organic contaminants it is possible to compare across all sites because Mussel Watch species have a similar ability to bioaccumulate contaminants. For trace metals there are clear differences in bioaccumulation abilities between coastal mussels and oysters. Oysters have a greater affinity for zinc, copper and silver while mussels are better able to accumulate lead and chromium. Because of these differences, the different matrices (mussels, oysters, sediment, and fish) are presented separately in the Coastal Pollution Data Explorer.

CONTAMINANT GROUP'S DESCRIPTIONS

Metals

Aluminum	**Source:** Aluminum is widely distributed in the environment and is the most abundant metal in the Earth's crust (ATSDR, 2008). As a common crustal element not bioaccumulated to a significant degree in shellfish, aluminum was measured, in part, to help assess whether elevated concentrations of other metals were associated with tissue uptake or with ingestion of contaminated particles (LeBlanc et al., 2011). Aluminum is highly reactive and is never found as a free metal but rather combined with other elements, mostly oxygen, silicon, and fluorine. These compounds are typically found in soil, minerals, rocks, and clays. Aluminum occurs naturally in soil, water, and air. High levels in the environment can be caused by the mining and processing of aluminum ores or the production of aluminum metal, alloys, and compounds. Small amounts of aluminum are released into the environment from coal-fired power plants and incinerators. Products that contain aluminum include beverage cans, pots and pans, airplanes, siding and roofing, foil, explosives, fireworks, antacids, aspirin, food additives, antiperspirants, and cosmetics (ATSDR, 2008). **Toxicity:** Human exposure to aluminum is commonplace and typically occurs by the consumption of processed foods that include flour, baking powder, coloring agents, or anticaking agents (ATSDR, 2008). An average US adult consumes approximately 7-9 mg of aluminum per day. Occupational exposure is the primary cause of exposure to high levels of aluminum in humans. High levels of exposure can result in lung problems (largely addressed by wearing masks) and decreased nervous system function. Accretion of aluminum happens in humans with kidney disease, as it is not removed through the urinary tract like normal, and can sometimes result in the development of bone or brain diseases (ATSDR, 2008). **Status:** There is no US FDA recommended guideline for aluminum in seafood.
Antimony	**Source:** No information **Toxicity:** No information **Status:** Antimony is not regularly monitored by the Mussel Watch Program but the limited available data has been included in this dataset. For more information on this metal, please see the ATSDR Toxic Substances Portal (https://wwwn.cdc.gov/TSP/substances/ToxSubstance.aspx?toxid=58)
Arsenic	**Source:** Arsenic is found in high levels in the environment as a result of both natural sources and industrial production. It is naturally present in low concentrations in the Earth's crust and in rock, soil, water, and air and is highly prevalent in the bedrock of the Southern California Bight region. Additionally, 30% of atmospheric arsenic comes from volcanoes and other natural sources and a major source responsible for apparent elevated levels of arsenic in the Nation is natural crustal rock, which varies by region (Welch et al., 1988). Generally, with respect to trend analysis, continuous natural sources are associated with neither decreasing nor increasing trends (Kimbrough et al., 2008). Industrial products that contain arsenic include preserved wood, semiconductors, pesticides, defoliants, pigments, antifouling paints, and veterinary medicines. In the recent past, as much as 90% of arsenic in industrial production was used for wood preservation (ATSDR, 2007a). Atmospheric sources of arsenic include smelting, fossil fuel combustion, power generation, and pesticide application contribute to high environmental levels of arsenic (ATSDR, 2007a). **Toxicity:** Arsenic is an essential trace nutrient for many animals; however, dietary requirements for humans are unknown (Uthus,1992). Arsenic is toxic at high concentrations to fish, birds and plants. In animals and humans prolonged chronic exposure is linked to cancer (Goyer, 1986). Inorganic arsenic, the most toxic form, represents approximately 10% of total arsenic in bivalves (Edmonds and Francesconi, 1977, 1988, 1993; Phillips, 1990; FDA, 1993a). **Status:** Safety guidance levels for arsenic in fish and shellfish are no longer listed by the US FDA (FDA, 2011).
Barium	**Source:** No information **Toxicity:** No information **Status:** Barium is not regularly monitored by the Mussel Watch Program but the limited available data has been included in this dataset. For more information on this metal, please see the ATSDR Toxic Substances Portal (https://wwwn.cdc.gov/TSP/substances/ToxSubstance.aspx?toxid=57)
Beryllium	**Source:** No information **Toxicity:** No information **Status:** Beryllium is not regularly monitored by the Mussel Watch Program but the limited available data has been included in this dataset. For more information on this metal, please see the ATSDR Toxic Substances Portal (https://wwwn.cdc.gov/TSP/substances/ToxSubstance.aspx?toxid=33)
Cadmium	**Source:** Cadmium found in coastal and estuarine environments can be linked to both natural and non-point anthropogenic sources (Roesijadi, 1984). Cadmium occurs naturally in the Earth's crust as complex oxides and sulfides in ores (Plachy, 2003) and natural sources can be linked to river runoff from cadmium rich soils, leaching from bedrock, and upwelling from marine sediment deposits (Sokolova et al., 2005). Industrial sources and uses include zinc, lead, and copper production, electroplating and galvanizing, smelting, mining, fossil fuel burning, waste slag, and sewage sludge (ATSDR, 1999a; FDA, 1993b). In addition to abundant industrial applications, other products that contain cadmium include batteries, color pigment, plastics, and phosphate fertilizers. Generally, anthropogenic emissions exceed natural emissions and elevated cadmium levels are primarily located in freshwater-dominated estuaries (e.g., Mississippi Delta, Great Lakes, Chesapeake Bay). **Toxicity:** Cadmium is a non-essential and toxic element. Cadmium is toxic to fish, especially salmonid species and juveniles, and chronic exposure can result in reductions in growth. Cadmium has been shown to impair development and reproduction in several invertebrate species and osmoregulation in herring larvae (ATSDR, 1999a; Eisler, 1985). Respiration and consumption represent the two major exposure pathways for humans to cadmium with exposure to high levels resulting primarily from occupational exposure. **Status:** Safety guidance levels for cadmium in fish and shellfish are no longer listed by the US FDA (FDA, 2011).

Chromium	**Source:** Chromium is a naturally occurring metal found in rocks, animals, plants and soils in three main forms: chromium(0), chromium(III), and chromium(VI). Chromium is widely used in industries involved in electroplating, leather tanning, textile production, and the manufacture of chromium-based products including treated wood, tanned leather, stainless steel cookware, and metal-on-metal hip replacements (ATSDR, 2012a). Chromium can also be released into the environment from the burning of natural gas, oil, or coal (ATSDR, 2012a). **Toxicity:** Chromium is an essential trace nutrient but can be toxic at high levels. Exposure to chromium can result from inhalation,ingestion, and physical contact. Inhalation primarily occurs near industries using or manufacturing chromium or through cigarette smoke. Ingestion typically occurs through drinking or bathing water. The general population is most likely to be exposed to trace levels of chromium in food that is eaten, including fruits, vegetables, nuts, beverages, and meats, but exposure at high levels occurs primarily resulting from occupational exposure (ATSDR, 2012a). In humans, exposure to chromium most impacts the respiratory tract, specifically causing irritation to nasal lining, runny nose, and breathing problems. In animals, ingestion of chromium(VI) has been shown to cause negative effects on the stomach, small intestine, blood, and male reproductive system. The International Agency for Research on Cancer (IARC) has determined that chromium(VI) compounds are carcinogenic to humans. In animals, some studies show that exposure to high doses during pregnancy may cause miscarriage, low birth weight, and some changes in development of the skeleton and reproductive system (ATSDR, 2012a). **Status:** The EPA has established a maximum contaminant level of 0.1 mg/L for total chromium in drinking water and sought voluntary monitoring of chromium(VI) in drinking water by municipalities. OSHA has set a legal limit for atmospheric exposure over an 8-hour workday of 0.005 mg/m3 for chromium(VI), 0.5 mg/m3 for chromium(III), and 1.0 mg/m3 for chromium(0).
Cobalt	**Source:** No information **Toxicity:** No information **Status:** Cobalt is not regularly monitored by the Mussel Watch Program but the limited available data has been included in this dataset. For more information on this metal, please see the ATSDR Toxic Substances Portal (https://wwwn.cdc.gov/TSP/substances/ToxSubstance.aspx?toxid=64)
Copper	**Source:** Copper is a naturally occurring element that is ubiquitous in the environment. Anthropogenic sources include: mining, manufacturing, agriculture, sewage sludge, antifouling paint, fungicides, wood preservatives, and vehicle brake pads (ATSDR, 2004; Denier van der Gon et al., 2007). The U.S. ranks third in the world for utilization and second in production. **Toxicity:** Trace amounts of copper are an essential nutrient for plants and animals. High concentrations of copper can be toxic to aquatic organisms, with juvenile fishes and invertebrates much more sensitive than adults. Elevated levels of copper can impact aquatic organisms including the functioning of gills, reproduction, and development (Eisler, 1998). Although copper is not highly toxic to humans, chronic effects occur as a result of prolonged exposure to high doses and can cause eye irritation and damage to the digestive tract (ATSDR, 2004). **Status:** The EPA phase-out of chromated copper arsenate (CCA) wood preservatives and the 1980s restrictions on tributyltin marine antifouling paint has stimulated a transition to copper-based wood preservatives and marine antifouling paint. There is no recommended US FDA safety level for copper in fish and fish products (FDA, 2020).
Iron	**Source:** Iron is the second most abundant metal in the Earth's crust but tends to have low concentrations in water due to low solubility (Xing and Liu, 2011). Iron concentrations are generally low in natural freshwater bodies but contamination can occur from anthropogenic sources and result in eutrophication and algal blooms (Xing and Liu, 2011). Natural sources of iron in the environment are primarily from weathered rocks and soils and anthropogenic sources of iron input are primarily from fossil fuel combustion and incorporation of iron into foods and nutritional supplements (Liu et al., 2022). More recently, increasing usage of iron-based nanoparticles has raised concerns regarding their environmental behavior and ecological effects (Lei et al., 2018). **Toxicity:** Iron is an essential nutrient for humans, with adverse effects caused both by deficiency and exposure to high concentrations (EPA, 2006a). Iron deficiency, known as anemia, can cause fatigue, weakness, and cold extremities in humans. Conversely, exposure to high levels of iron in animals and humans typically results in injury to the gastrointestinal mucosa which can result in nausea, vomiting, abdominal pain, and diarrhea (Yuen and Becker, 2022). Exposure at these levels is typically caused by accidental ingestion (Lei et al., 2018). At the cellular level, exposure to high levels of iron can impair cellular metabolism in the heart, liver, and central nervous system (Yuen and Becker, 2022). In the environment, the toxicity of iron-based nanoparticles is increasingly observed (Lei et al., 2018). **Status:** There is no US FDA recommended guideline for iron in seafood.
Lead	**Source:** Lead is a ubiquitous metal that occurs naturally in the Earth's crust. Loadings of lead into coastal waters are primarily linked with wastewater discharge, river runoff, atmospheric deposition and natural weathering of rock. Lead can be found in air, soil and surface water (ATSDR, 2007b). Environmental levels of lead increased worldwide over the past century because of leaded gasoline use (ATSDR, 2007b). Significant reductions in source and load resulted from the regulation of lead in gasoline and lead based paints. High levels found in the environment are usually linked to anthropogenic activities such as manufacturing processes, paint and pigment, solder, ammunition, plumbing, incineration and fossil fuel burning. In the communications industry, lead is still used extensively as protective sheathing for underground and underwater cables, including transoceanic cable systems (USGS, 2008). **Toxicity:** Lead has no biological use and is toxic to many organisms, including humans. Exposure of fish to elevated concentrations of lead results in neurological deformities and black fins in fish (Mance, 1987). Lead primarily affects the nervous system, which results in decreased mental performance and inhibits typical mental development in humans. Exposure to lead may also cause brain and kidney damage and cancer (IARC, 2006). **Status:** Safety guidance levels for lead in fish and shellfish are no longer listed by the US FDA (FDA, 2011).

Lithium	**Source:** No information **Toxicity:** No information **Status:** Lithium is not regularly monitored by the Mussel Watch Program but the limited available data has been included in this dataset. For more information on this metal, please see the EPA Technical Fact Sheet on Lithium (https://www.epa.gov/system/files/documents/2023-11/ucmr5-technical-fact-sheet-lithium-in-drinking-water.pdf)
Manganese	**Source:** Manganese is found in the environment both naturally and from anthropogenic sources including industrial production, pharmaceuticals, and gasoline (ATSDR, 2012b). Manganese occurs naturally in Earth's crust combined with other substances like oxygen, sulfur, and chlorine and is also a normal component of air, soil, water, and food. Humans are routinely exposed to low levels of manganese through drinking water, ground water, and soil. Anthropogenic sources of manganese include use in steel production to improve the hardness, stiffness, and strength of carbon steel, stainless steel, high-temperature steel, tool steel, cast iron, and superalloys. Manganese can also be found in products such as fireworks, dry-cell batteries, fertilizer, paints, medical imaging agents, cosmetics, and gasoline. Atmospheric manganese can result from industrial activities, mining, automobile exhaust, and cigarette smoke (ATSDR, 2012b). **Toxicity:** While manganese is an essential nutrient, it can have negative effects at high doses (ATSDR, 2012b). Typically, manganese enters the body through inhalation, ingestion, and dermal contact. Primarily, humans are exposed to manganese through manganese-containing nutritional supplements or food, especially grains, beans, nuts, and tea. Additionally, occupations such as welding or steel-making may increase the chances of high manganese exposure. High exposure to manganese often impacts the nervous system in the form of behavioral changes and slow, clumsy movements – referred to as "manganism". Additional observed effects include lung irritation resulting in pneumonia and negative changes in the male reproductive system. Typically, the concentrations required to produce these negative effects are twenty thousand times higher than concentrations normally found in the environment. **Status:** The EPA has established that exposure to manganese in drinking water at concentrations of 1 mg/L for 1 or 10 days is not expected to cause any adverse effects, and OSHA set a legal limit of 5 mg/m3 manganese in air averaged over an 8-hour work day (ATSDR, 2012b).
Mercury	**Source:** Mercury is found naturally as mercuric sulfide from forest fires, crustal ores, fossil fuels, and volcanoes. Anthropogenic sources of mercury are from mining, gold refining, coal fired power plants, and the wood pulp industry. Elevated levels occur as a result of human activity (ATSDR, 1999b). In the US, coal fired-electric turbines, municipal and medical waste incinerators, mining, landfills, and sewage sludge are the primary emitters of mercury into the air. **Toxicity:** Mercury is a highly toxic, non-essential trace metal that occurs naturally. Mercury is a human neurotoxin that also affects the kidneys and developing fetuses. The most common human exposure route for mercury is the consumption of contaminated food. Children, pregnant women, or women likely to become pregnant are advised to avoid consumption of swordfish, shark, king mackerel and tilefish and should limit consumption to fish and shellfish recommended by US FDA and US EPA (EPA, 1979; FDA, 2011). Mercury exposure can also cause reduced growth and reproduction rates in other phyla such as copepods (Eisler, 1987). **Status:** The US FDA has not established a safety level for mercury but has set a safety level of 1.0 ppm wet weight for methyl mercury, the form most likely to impact animals and humans (FDA, 2011).
Methyl Mercury	**Source:** No information **Toxicity:** No information **Status:** Methyl Mercury is not regularly monitored by the Mussel Watch Program but the limited available data has been included in this dataset. For more information on this metal, please see the ATSDR Toxic Substances Portal (https://wwwn.cdc.gov/TSp/ToxProfiles/ToxProfiles.aspx?id=115&tid=24)
Nickel	**Source:** Nickel is naturally occurring and widely distributed in the environment. It exists in alloy form in combination with other metals and as a soluble element. Naturally, nickel is derived from weathering rocks and soil and is transported to streams and rivers by runoff. River and stream input of nickel are the largest sources for oceans and coastal waters. Anthropogenic sources of nickel can be attributed to the presence of nickel in stainless steel, nickel-cadmium batteries, pigments, computers, wire, coinage, and electroplating (ATSDR, 2005a). Atmospheric sources are usually not significant, with the exception of in the Great Lakes, where the atmospheric input of nickel accounts for 60-80% of the total anthropogenic input to Lake Superior, and 20-70% of total inputs to Lakes Erie and Ontario (Nriagu et al., 1995). In Lakes Erie and Ontario, most of the nickel is derived from municipal and industrial wastewaters and thus is likely to be complexed with organic matter (Sweet et al., 1998). **Toxicity:** Nickel is a biologically essential trace element for animals and plants and is widely distributed in the environment at very low concentrations. Exposure to large doses of nickel can cause serious health effects, such as bronchitis, and long-term exposure can result in cancer. Food is the major source of human exposure to nickel (ATSDR, 2005a). **Status:** Safety guidance levels for nickel in fish and shellfish are no longer listed by the US FDA (FDA, 2011).
Selenium	**Source:** Selenium naturally occurs in the Earth's crust and is commonly found in rocks and soil (ATSDR, 2003). Weathering of rocks and soil can result in low levels of selenium in water and in the air as dust-like particles. Additionally, volcanic eruptions may release atmospheric selenium. Selenium is also commercially produced as a byproduct of copper refining or from burning coal or oil. Other products that selenium compounds can be found in include photographic devices, gun bluing, plastics, paints, anti-dandruff shampoos, vitamin and mineral supplements, fungicides, glass, drug preparation, and livestock feed supplements. Regardless of source, selenium is not often found in its elemental form in the environment, but rather combined with sulfide, silver, copper, lead, or nickel minerals (ATSDR, 2003). **Toxicity:** Selenium is an essential nutrient in low concentrations and is usually ingested by humans through drinking water, food, and air (ATSDR, 2003). Selenium is often contained in soils and varies by region, so depending on where food is grown, humans may be exposed to differing levels of selenium. Humans are not typically exposed to high selenium levels but this can vary with occupation, specifically for individuals employed in the metal industries, selenium-recovery processes, paint manufacturing, and special

	trades. When exposed at high levels, dizziness, fatigue, and irritation of mucous membranes can occur and even mild exposure over long time periods can result in brittle hair, deformed nails, and even loss of feeling and control in extremities (called selenosis). Conditions this extreme usually occur in areas with locally high levels of selenium where the population eats locally. There is no support that selenium causes cancer except for one specific form called selenium sulfide which has been shown to cause cancer in animals. **Status:** US FDA regulations allow a level of 50 ppb of selenium in bottled water and OSHA set the exposure limit for selenium compounds in the air for an 8-hour period at 0.2 mg selenium/m3 (ATSDR, 2003).
Silicon	**Source:** No information **Toxicity:** No information **Status:** Silicon (Si) is not regularly monitored by the Mussel Watch Program but the limited available data has been included in this dataset. For more information on this metal, please see the ATSDR Toxic Substances Portal (https://wwwn.cdc.gov/TSP/ToxProfiles/ToxProfiles.aspx?id=1483&tid=290)
Silver	**Source:** Silver is a naturally occurring element but elevated levels of contamination are typically from anthropogenic sources (ATSDR, 1990a). The natural wearing down of silver-bearing rocks and soil by the wind and rain releases large amounts of silver into the environment. Anthropogenically, products that contain silver are photographic technology, dental applications, electronics, silverware, jewelry, and solders. Photographic materials are the major anthropogenic source of silver that is released into the environment (ATSDR, 1990a). **Toxicity:** Silver is not an essential nutrient to humans and is mostly harmless at low levels and infrequent exposure (ATSDR, 1990a). Humans are generally exposed to silver through food and drinking water, and minimally through the air. Silver can cause some areas of the skin and other body tissues to turn gray or gray-blue in a condition known as "argyria". This condition can occur in humans who eat or breathe in silver compounds over a long period. Long or high exposure to silver compounds such as silver nitrate or silver oxide may cause breathing problems, lung and throat irritation, and stomach pain. These conditions typically manifest with occupational exposure. Skin contact with silver compounds has been found to cause mild allergic reactions, such as rash, swelling, and inflammation in some people. Tests in animals have shown that exposure to silver is only likely to be life threatening when very high amounts are swallowed, which is unlikely (ATSDR, 1990a). **Status:** Silver has been regulated in the United States since the 1960s, but initial criteria were conservative, as the realized risk of silver was not known at the time. The US EPA downgraded silver from a primary to a secondary maximum contaminant level in the 1990s due to its low realized risk from exposure (Purcell, 2009).
Thallium	**Source:** No information **Toxicity:** No information **Status:** Thallium is not regularly monitored by the Mussel Watch Program but the limited available data has been included in this dataset. For more information on this metal, please see the ATSDR Toxic Substances Portal (https://wwwn.cdc.gov/TSP/substances/ToxSubstance.aspx?toxid=49)
Tin	**Source:** Tin may be released to the environment from both natural and anthropogenic sources. Tin occurs in water in trace amounts naturally and can combine with other chemicals to form various inorganic and organic compounds. Sources of tin in coastal water and soil include manufacturing and processing facilities and mining. Tin has not been mined in the US since 1993 (USGS, 2008); however, Canadian tin mining occurs in the Great Lakes Region. Tin metal is mainly used as liner in cans for food and aerosols and is also used in paint, plastic, pesticide brass, bronze, and some soldering products (ATSDR, 2005b). Concentrations in unpolluted waters and the atmosphere are often near analytical detection limits. **Toxicity:** Humans are exposed to elevated levels of tin by eating from tin-lined cans and by consuming contaminated seafood (ATSDR, 2005b). Exposure to elevated levels of tin compounds by humans leads to liver damage, kidney damage, and cancer. **Status:** There is no US FDA recommended guideline for tin in seafood.
Uranium	**Source:** No information **Toxicity:** No information **Status:** Uranium is not regularly monitored by the Mussel Watch Program but the limited available data has been included in this dataset. For more information on this metal, please see the ATSDR Toxic Substances Portal (https://wwwn.cdc.gov/TSP/substances/ToxSubstance.aspx?toxid=77)
Vanadium	**Source:** No information **Toxicity:** No information **Status:** Vanadium is not regularly monitored by the Mussel Watch Program but the limited available data has been included in this dataset. For more information on this metal, please see the ATSDR Toxic Substances Portal (https://wwwn.cdc.gov/TSP/substances/ToxSubstance.aspx?toxid=50)
Zinc	**Source:** Zinc occurs naturally in the Earth's crust and can be released into the environment by natural rock weathering, but its anthropogenic sources far exceed its natural ones. Major industrial sources include electroplating, smelting, and drainage from mining operations (Mirenda, 1986). The greatest use of zinc is as an anti-corrosive coating for iron and steel products (sheet and strip steel, tube and pipe, wire, and wire rope). Canada is one of the largest producers and exporters of zinc. The United States is the largest customer for Canadian refined zinc and the automobile industry is the largest user of galvanized steel. **Toxicity:** Zinc is an essential nutrient and particles can be found in air, water, soil, and most foods. Human exposure to high doses of zinc may cause anemia or damage to the pancreas and kidneys (ATSDR, 2005c). However, zinc does not bioaccumulate in humans; therefore, toxic effects are uncommon and associated with excessively high doses. Fish exposed to low zinc concentrations can sequester it in some cases (McGeer et al., 2003). **Status:** There is no US FDA recommended safety level for zinc in fish and fish products.

Legacy Contaminants

Butyltins	**Source:** The parent butyltin compound is tributyltin and the other butyltin compounds are its less toxic transformation products. Tributyltin has had a variety of uses ranging from a biocide in antifouling paints to a catalyst and an ingredient in glass coatings (Bennett, 1996; Birchenough et al., 2002). In the late 1960s, tributyltin was incorporated into an antifouling polymer paint system, quickly becoming one of the most effective paints used on boat hulls (Birchenough et al., 2002). Beginning in 1989, the use of tributyltin as an antifouling agent was banned in the US on non-aluminum vessels smaller than 25 meters in length (Gibbs and Bryan, 1996). However, the continued use of tributyltin on ships and other antifouling paint applications increased the ubiquity of the compound in aquatic environments. Thus, tributyltin and its metabolites continue to be detected in many components of coastal and marine ecosystems in the US. **Toxicity:** Butyltins can be highly toxic in multiple forms as they naturally degrade in the environment. Tributyltin was first shown to have biocidal properties in the 1950's (Bennett, 1996; Evans, 1970). The presence of tributyltin in the environment has been linked to endocrine disruption (Batley, 1996; Strand et al., 2009). In the mid-1970s, the use of tributyltin was linked to abnormal shell development and poor weight gain in oysters, and more recently to an imposed condition (females developing male characteristics) in marine gastropod mollusks (Batley, 1996; Strand et al., 2009). **Status:** Beginning in 1989 the use of tributyltin as an antifouling agent was banned in the US on non-aluminum vessels smaller than 25 meters in length. There is no US FDA recommended safety level for butyltins in fish and fish products.
Chlordanes	**Source:** Chlordane belongs to a group of organic pesticides called cyclodienes. Technical chlordane was an insecticide used in the US from 1948-1983 for agricultural and urban settings to control insect pests. It was also the predominant insecticide for the control of subterranean termites. Agricultural uses were banned in 1983 and all uses were banned by 1988. These compounds are some of the most ubiquitous contaminants measured by the Mussel Watch Program. Removal from both soil and water sources is primarily by volatilization and particle-bound runoff. In air chlordane degrades as a result of photolysis and oxidation. Chlordane exists in the atmosphere primarily in the vapor-phase but the particle-bound fraction is important for long-range transport. Chlordane is prevalent in the Arctic due to the grasshopper effect and distributed in the food web (Hargrave et al., 1992). Chlordane binds to dissolved organic matter further facilitating its transport in natural waters. **Toxicity:** Human exposure to chlordane can occur through eating crops from contaminated soil fish and shellfish from contaminated waters or breathing contaminated air. Chlordane can enter the body by being absorbed through the skin inhalation and ingestion. At high levels chlordane can affect the nervous system, digestive system, brain and liver and is also carcinogenic. Chlordane is highly toxic to invertebrates and fish (FDA, 2011). **Status:** The insecticide, Chlordane, was banned for use in 1988 in the United States. The US FDA has established a safety level of 0.3 ppm wet weight for both chlordane and heptachlor/ heptachlor epoxide in all fish (FDA, 2011).
Chlorpyrifos	**Source:** Chlorpyrifos is a white, crystal-like solid with a strong odor that has been used as a pesticide in the United States since 1965 in both agricultural and non-agricultural settings (ATSDR, 1997; EPA, 2022a). In 2000, there was a voluntary agreement to eliminate, phase out, and modify certain uses of chlorpyrifos, specifically private homeowner usage except for roaches, discontinuing uses on tomato plants and lowering the maximum residue level of grapes (EPA, 2022a). **Toxicity:** Exposure to chlorpyrifos may occur through exposure to pesticides by eating food contaminated with chlorpyrifos, breathing in fumes, or getting them on skin or eyes (NPIC, 2023). Exposure can also occur through contaminated groundwater, which can happen if products containing chlorpyrifos were used or mixed for application near a well for termite control (NPIC, 2023). Once it is inside the body, chlorpyrifos itself is not toxic but when the body attempts to break it down, it creates a toxic form (NPIC, 2023). Typical symptoms may include sweating, headache, nausea, and dizziness, while more severe exposure can cause vomiting, abdominal muscle cramps, muscle twitching, tremors, weakness, and loss of coordination (ATSDR, 1997; NPIC, 2023). Generally, the body is able to excrete the contaminant within a few days (NPIC, 2023). Studies have not shown that chlorpyrifos is a carcinogen (ATSDR, 1997). **Status:** As of 2022, chlorpyrifos is no longer used on food and animal feed crops, although it may still be used on non-fruit bearing trees (NPIC, 2023).
Chlorobenzenes	**Source:** Chlorobenzenes belong to the family of organic halogen compounds and are widely used as degreasers, chemical intermediates and solvents for pesticide formulations, adhesives, paints, polishes, dyes and drugs. For example, pentachloroanisole comes from the biomethylation of pentachlorophenol, a chemical used as a general biocide, fungicide, bactericide, herbicide, molluscicide, algaecide and insecticide by agriculture and other industries including textiles, paints, oil drilling and forestry (Canada, 2012). Although chlorobenzenes are not banned, their production has decreased by 60% since the peak in 1960 due primarily to regulations on DDT, in which it was used as part of the manufacturing process (ATSDR, 1990b). **Toxicity:** There is inadequate evidence to classify chlorobenzenes as carcinogens, however, animal studies indicate that livers, kidneys and the central nervous system are affected by exposure to chlorobenzenes (ATSDR, 1990b). **Status:** Hexachlorobenzene has not been manufactured in the United States (as an end-product) since its last registered use as a pesticide was voluntarily canceled in 1984. Small amounts of hexachlorobenzene are still released to the environment by industrial activities as fugitive and stack emissions, or in wastewater.
DDTs	**Source:** Total DDTs (dichlorodiphenyltrichloroethanes) analyzed historically are the sum of six compounds which are ortho and para forms of DDT and its transformation products DDE and DDD the latter being the most predominant form found in the environment. Technical DDT the insecticide was composed of up to 14 compounds of which 65-80% was the active ingredient pp'-DDT (44'-DDT). The next major component op'-DDT (24'-DDT) (15-21%) is nearly inactive as an insecticide. DDT was used worldwide as an insecticide for agricultural pests and mosquito control (ATSDR, 2022). **Toxicity:** Due to its persistence and hydrophobic nature, DDT bioaccumulates in organisms. Organochlorine pesticides are typically neurotoxins and DDT has been shown to interfere with the endocrine system (Rogan and Chen, 2005). DDT and its metabolite DDE were specifically linked to eggshell thinning in birds (Lincer, 1975).

	Status: Its use in the United States was banned in 1972 but it is still used in some countries today. DDT was banned due to its environmental persistence, bioaccumulation and toxicity to non-target organisms. The US FDA has established a safety level of 5 ppm wet weight for DDT and DDE in all fish (FDA, 2011).
Dieldrins	**Source:** Dieldrins were widely used as insecticides in the 1960s for the control of termites around buildings and general crop protection from insects. In 1970, all uses of dieldrins were banned due to concern that they could cause severe aquatic environmental change and potential carcinogenicity (EPA, 1980). The ban was lifted in 1972 to allow limited use of dieldrins, primarily for termite control. All uses of dieldrins were finally banned in 1989 (EPA, 1990). **Toxicity:** Exposure to dieldrins occurs through ingestion of contaminated water and food products, including fish and shellfish, and through inhalation of indoor air in buildings treated with these insecticides. Acute and long-term human exposures are associated with central nervous system intoxication (ATSDR, 2002). Because dieldrins can build up in the body and are slow to leave, health effects can occur from long periods of exposure to smaller amounts. Aldrin and dieldrin are carcinogenic to animals and classified as likely human carcinogens. **Status:** Because of concerns about damage to the environment and potentially to human health, EPA banned all uses of aldrin and dieldrin in 1974, except to control termites. In 1987, EPA banned all uses. The US FDA has established a safety level of 0.3 ppm wet weight for aldrin and dieldrin in all fish (FDA, 2011).
Endosulfans	**Source:** Technical grade endosulfan is a mixture of two isomers (Endosulfan I and II) and Endosulfan sulfate is a product of oxidation and can be found in technical grade endosulfan. Endosulfan was a restricted-application pesticide, used to treat certain crops against aphids, beetles, leafhoppers, white flies, etc. (ATSDR, 2015a). Endosulfan can be found in the environment in the atmosphere, soil and water. Endosulfans can travel long distances by air and may be broken down by sunlight. In soil endosulfans attach to soil particles limiting its movement further. **Toxicity:** The general population is exposed to Endosulfans through diet, breathing contaminated air, ingesting contaminated water, or being in contact with contaminated soil or plants (ATSDR, 2015a). In humans, endosulfan appears to accumulate in the liver, kidneys, and brain and are typically excreted within a few days or weeks. Endosulfan accumulation has been shown to primarily affect the nervous system. Exposure to high levels of endosulfans can induce hyperactivity and convulsion. There is no evidence to suggest that endosulfans can cause cancer or any other disease in humans (ATSDR, 2015a). **Status:** The use of endosulfans was restricted to certain crops before its phase-out by 2016 (ATSDR, 2015a).
HCHs	**Source:** Technical grade HCH (hexachlorocyclohexane) contains the alpha, beta, gamma, delta, and epsilon forms of HCH. Almost all of the insecticidal properties are found in gamma-HCH (lindane) which is used as an insecticide on fruit, vegetables and forest crops. It is also found in lotion, cream or shampoo as a prescription to treat head and body lice and scabies (ATSDR, 2005d). **Toxicity:** All of the isomers are toxic to animals to varying degrees and are persistent in the environment. The Department of Health and Human Services (DHHS), International Agency for Research on Cancer (IARC) and the EPA vary in their classification of HCH as a human carcinogen. However, technical HCH, alpha-HCH, and beta-HCH are listed by all three as at least possible human carcinogens (ATSDR, 2005d). In 2015, based on a review of the most recent data on lindane, the IARC modified its classification from "probably carcinogenic to humans" to "known to cause human cancer" (IARC, 2015). In sediments and water, HCH can be broken down into less toxic substances by algae, bacteria, and fungi, but it is a slow process. HCH has been shown to accumulate in the fatty tissue of fish. **Status:** In 2009 the Stockholm Convention on Persistent Organic Pollutants implemented an international ban on the use of lindane in agriculture but allowed a 5-year extension for its use in the treatment of head lice and scabies (UNEP, 2009). The United States did not ratify the convention; however, the EPA requested the voluntary cancellation of the last agricultural uses of lindane in 2006 (EPA, 2006b).
Mirex	**Source:** Mirex was commercially introduced in the United States in 1959 for use in pesticide formulations and as an industrial fire retardant (ATSDR, 2020). Mirex was used in products including rubber, plastic, paints, paper, and electrical goods. In the 1960s, mirex was commonly used to control fire ants in southern States. Mirex was banned for use in the United States in 1978, except for use on pineapples until stocks on hand were exhausted (ATSDR, 2020). **Toxicity:** Exposure to mirex typically occurs through dermal contact with contaminated soil and ingestion of local wildlife or contaminated food, as mirex bioaccumulates in organisms and adheres to soil particles which can contaminate crops (ATSDR, 2020). Due to their limited solubility in water and nonvolatile nature, exposure through drinking water and inhalation is unlikely. The primary consequences of exposure to mirex in animals can be seen in the liver, kidneys, selected developmental endpoints, and the thyroid. Specific negative consequences include decreased glycogen storage in the liver, increased glomerulosclerosis and proteinuria in the kidneys, ocular lesions in newborns, and increases in cystic follicles in the thyroid. Decreased fertility, testicular atrophy, reproductive failure, and marked developmental toxicity have been observed following exposure to mirex. Mirex has also been classified as a carcinogen to animals (ATSDR, 2020). **Status:** Mirex has not been manufactured or used in the United States since 1978. Mirex breaks down slowly in the environment and any detected concentrations are probably due to residual chemicals rather than any new sources.
PAHs	**Source:** PAHs (polycyclic aromatic hydrocarbons) are formed from the fusing of benzene rings during the incomplete combustion of organic materials. PAHs are found in creosote, soot, petroleum, coal and tar, and are the only organic contaminants measured by the Mussel Watch Program that have natural sources (forest fires and volcanoes) in addition to anthropogenic sources (automobiles emissions, home heating, coal fired power plants). PAHs can also enter the aquatic environment by means of discharge from industrial and wastewater treatments plants (ATSDR, 1995) or can volatilize from an oil spill if they have a small molecular weight. **Toxicity:** Made up of a suite of hundreds of compounds, PAHs exhibit a wide range of toxicities. While many aquatic organisms like fish can metabolize PAHs, marine invertebrates, such as oysters, are less able to efficiently metabolize them and as such can be better indicators of overall environmental exposure (Neff, 1985). The PAH contents of plants and animals may be much higher than PAH contents of soil or water in which they live (ATSDR, 1995). A number of the PAHs that bioaccumulate in aquatic

	and terrestrial organisms are toxic and some, including benzo(a)pyrene, benz(a)anthracene, chrysene, benzo(b)fluoranthene, benzo(k)fluoranthene, dibenzo(a,h)anthracene, and indeno(1,2,3-c,d)pyrene, are likely carcinogens (ATSDR, 1995). Toxic responses to PAHs in aquatic organisms include reproduction inhibition, mutations, liver abnormalities and mortality. Exposure to aquatic organisms results from oil spills, boat exhaust and urban runoff. Human exposure to PAHs can come as a result of being exposed to smoke from forest fires, automobile exhaust, home heating using wood, grilling, and cigarettes. **Status:** There is no US FDA recommended safety level for PAHs in fish and fish products (EPA, 2000).
PBDEs	**Source:** Brominated flame retardants (BFRs), such as polybrominated diphenyl ethers (PBDEs) and polybrominated biphenyls (PBBs), are a group of chemicals with 209 possible unique congeners used in firefighting materials and in consumer and household products to reduce flammability (ATSDR, 2015b). A subset of these congeners was analyzed in this study (19 PBBs and 51 PBDEs). Commercially, three types of PBDE industrial mixtures have been available: pentabromodiphenyl ether (penta-BDE), octabromodiphenyl ether (octa-BDE), and decabromodiphenyl ether (deca-BDE) mixtures (EPA, 2014a). As the products that contain these compounds age and degrade or are discarded, PBDEs leach into the environment. PBDEs have become ubiquitous in the environment and are detected in materials including household dust, human breast milk, sediment, and wildlife (ATSDR, 2015b). The less brominated PBDEs, like tetra-, penta-, and hexa-BDE, demonstrate a high affinity for lipids and tend to bioaccumulate in animals and humans. In contrast, highly brominated PBDEs like deca-BDE tend to absorb more into sediment and soil. **Toxicity:** The toxicology of PBDEs is not well understood, but PBDEs have been associated with tumors, neurodevelopmental toxicity, and thyroid hormone imbalance (Siddiqi et al., 2003). Some PBDE congeners have hepatotoxic and mutagenic effects, while others may act as estrogen receptor agonists in vitro (Meerts et al., 2001). **Status:** Due to their ubiquitous distribution, persistence, and potential for toxicity, the manufacturing of the penta- and octa-BDE mixtures began to be phased out in 2004, and the deca- mixture in 2013 (EPA, 2014a; Schreder and La Guardia, 2014).
PCBs	**Source:** Polychlorinated Biphenyls (PCBs) are synthetic organic compounds that have been used in numerous applications including electrical transformers and capacitors, hydraulic and heat transfer fluids, pesticides and in paints (ATSDR, 2000). PCBs have a biphenyl ring structure (two benzene rings with a carbon-to-carbon bond) and a varying number (1-10) of chlorine atoms. There are 209 individual PCB compounds or congeners possible. PCBs were manufactured in the US between 1929 and 1977. In the US, a single manufacturer produced all PCBs and the commercial products were referred to as Aroclors, which are mixtures of PCB congeners. Approximately 65% of PCBs manufactured in the US were used in electrical applications (Eisler and Belisle, 1996). Other applications were lubricants, hydraulic fluids, paints, adhesives, plasticizers, and flame retardants (Kimbrough et al., 2008). Improper disposal and leakage are responsible for their original environmental introduction. Current pollution sources include volatilization and runoff from landfills, leaks from old electrical equipment, and dredging of contaminated sediments (WHO and IPCS, 1993). **Toxicity:** PCBs have been linked to many health issues including adversely affecting reproduction, growth, metabolism and survival in animals (Eisler and Belisle, 1996). The main human exposure route for PCBs is through eating contaminated seafood and meats. PCBs are associated with skin ailments, neurological, and immunological responses and at high doses can decrease motor skills and cause memory loss. Other effects can include hepatotoxicity, immunotoxicity, neurotoxicity, low birth weight, and teratogenicity (Eisler and Belisle, 1996). Exposure to PCBs in fish has been linked to reduced growth, reproductive impairment, and vertebral abnormalities (Eisler and Belisle, 1996). PCBs have also been shown to cause cancer in laboratory animals and are likely carcinogens in humans (ATSDR, 2000). The main human exposure route for PCBs is through eating contaminated seafood and meats which is the reason for many consumption advisories. **Status:** The import, manufacture, and sale of PCBs was made illegal in the United States in the late 1970s. The US FDA safety level for PCBs in all fish (edible portions) is 2 ppm wet weight, irrespective of which mixture of PCBs is present at the residue (FDA, 2011). The US EPA Recreational and Subsistence Fishery Screening Values are 20 ng/g ww and 2.45 ng/g ww, respectively (EPA, 2000).

Contaminants of Emerging Concern (CEC)

Antibiotic (PPCP)	**Source:** Environmental detections of pharmaceuticals and personal care products (PPCPs) include a wide spectrum of therapeutic and consumer-use compounds such as prescription and over-the-counter medications, hormones, synthetic fragrances, detergents, disinfectants, insect repellants, and antimicrobial agents. In 2009, an estimated 3.9 billion prescriptions were written for the top 300 pharmaceuticals in the U.S. (Lundy, 2010). Pharmaceutical companies produce over 22.6 million kg (50 million pounds) of antibiotics annually in the U.S., with approximately 60% for human use and 40% for animal agriculture use (Levy, 1998). There are numerous pathways by which PPCPs are introduced into the environment, although the primary routes include wastewater discharge or improper disposal of unused drugs (Daughton and Ternes, 1999). **Toxicity:** Because pharmaceuticals are designed to have a biological effect, the major environmental concerns associated with PPCPs are their potential ecotoxicity and unintentional human health impacts. Potential impacts of PPCPs in the environment include abnormal physiological effects, impaired reproduction, and increased cancer rates (Boyd and Furlong, 2002). According to the U.S. EPA, many CECs, including PPCPs, are suspected to be endocrine disruptors, which alter the normal functions of hormones, resulting in various health effects (Ankley et al., 2008). **Status:** No information
Antidepressant (PPCP)	**Source:** Environmental detections of pharmaceuticals and personal care products (PPCPs) include a wide spectrum of therapeutic and consumer-use compounds such as prescription and over-the-counter medications, hormones, synthetic fragrances, detergents, disinfectants, insect repellants, and antimicrobial agents. In 2009, an estimated 3.9 billion prescriptions were written for the top 300 pharmaceuticals in the U.S. (Lundy, 2010). Pharmaceutical companies produce over 22.6 million kg (50 million pounds) of antibiotics annually in the U.S., with approximately 60% for human use and 40% for animal agriculture use (Levy, 1998). There are numerous pathways by which PPCPs are introduced into the environment, although the primary routes include wastewater discharge or improper disposal of unused drugs (Daughton and Ternes, 1999).

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Antidiabetic (PPCP)	**Source:** Environmental detections of pharmaceuticals and personal care products (PPCPs) include a wide spectrum of therapeutic and consumer-use compounds such as prescription and over-the-counter medications, hormones, synthetic fragrances, detergents, disinfectants, insect repellants, and antimicrobial agents. In 2009, an estimated 3.9 billion prescriptions were written for the top 300 pharmaceuticals in the U.S. (Lundy, 2010). Pharmaceutical companies produce over 22.6 million kg (50 million pounds) of antibiotics annually in the U.S., with approximately 60% for human use and 40% for animal agriculture use (Levy, 1998). There are numerous pathways by which PPCPs are introduced into the environment, although the primary routes include wastewater discharge or improper disposal of unused drugs (Daughton and Ternes, 1999). **Toxicity:** Because pharmaceuticals are designed to have a biological effect, the major environmental concerns associated with PPCPs are their potential ecotoxicity and unintentional human health impacts. Potential impacts of PPCPs in the environment include abnormal physiological effects, impaired reproduction, and increased cancer rates (Boyd and Furlong, 2002). According to the U.S. EPA, many CECs, including PPCPs, are suspected to be endocrine disruptors, which alter the normal functions of hormones, resulting in various health effects (Ankley et al., 2008). **Status:** No information
Antihistamine (PPCP)	**Source:** Environmental detections of pharmaceuticals and personal care products (PPCPs) include a wide spectrum of therapeutic and consumer-use compounds such as prescription and over-the-counter medications, hormones, synthetic fragrances, detergents, disinfectants, insect repellants, and antimicrobial agents. In 2009, an estimated 3.9 billion prescriptions were written for the top 300 pharmaceuticals in the U.S. (Lundy, 2010). Pharmaceutical companies produce over 22.6 million kg (50 million pounds) of antibiotics annually in the U.S., with approximately 60% for human use and 40% for animal agriculture use (Levy, 1998). There are numerous pathways by which PPCPs are introduced into the environment, although the primary routes include wastewater discharge or improper disposal of unused drugs (Daughton and Ternes, 1999). **Toxicity:** Because pharmaceuticals are designed to have a biological effect, the major environmental concerns associated with PPCPs are their potential ecotoxicity and unintentional human health impacts. Potential impacts of PPCPs in the environment include abnormal physiological effects, impaired reproduction, and increased cancer rates (Boyd and Furlong, 2002). According to the U.S. EPA, many CECs, including PPCPs, are suspected to be endocrine disruptors, which alter the normal functions of hormones, resulting in various health effects (Ankley et al., 2008). **Status:** No information
Cardiovascular (PPCP)	**Source:** Environmental detections of pharmaceuticals and personal care products (PPCPs) include a wide spectrum of therapeutic and consumer-use compounds such as prescription and over-the-counter medications, hormones, synthetic fragrances, detergents, disinfectants, insect repellants, and antimicrobial agents. In 2009, an estimated 3.9 billion prescriptions were written for the top 300 pharmaceuticals in the U.S. (Lundy, 2010). Pharmaceutical companies produce over 22.6 million kg (50 million pounds) of antibiotics annually in the U.S., with approximately 60% for human use and 40% for animal agriculture use (Levy, 1998). There are numerous pathways by which PPCPs are introduced into the environment, although the primary routes include wastewater discharge or improper disposal of unused drugs (Daughton and Ternes, 1999). **Toxicity:** Because pharmaceuticals are designed to have a biological effect, the major environmental concerns associated with PPCPs are their potential ecotoxicity and unintentional human health impacts. Potential impacts of PPCPs in the environment include abnormal physiological effects, impaired reproduction, and increased cancer rates (Boyd and Furlong, 2002). According to the U.S. EPA, many CECs, including PPCPs, are suspected to be endocrine disruptors, which alter the normal functions of hormones, resulting in various health effects (Ankley et al., 2008). **Status:** No information
Chemotherapy (PPCP)	**Source:** Environmental detections of pharmaceuticals and personal care products (PPCPs) include a wide spectrum of therapeutic and consumer-use compounds such as prescription and over-the-counter medications, hormones, synthetic fragrances, detergents, disinfectants, insect repellants, and antimicrobial agents. In 2009, an estimated 3.9 billion prescriptions were written for the top 300 pharmaceuticals in the U.S. (Lundy, 2010). Pharmaceutical companies produce over 22.6 million kg (50 million pounds) of antibiotics annually in the U.S., with approximately 60% for human use and 40% for animal agriculture use (Levy, 1998). There are numerous pathways by which PPCPs are introduced into the environment, although the primary routes include wastewater discharge or improper disposal of unused drugs (Daughton and Ternes, 1999). **Toxicity:** Because pharmaceuticals are designed to have a biological effect, the major environmental concerns associated with PPCPs are their potential ecotoxicity and unintentional human health impacts. Potential impacts of PPCPs in the environment include abnormal physiological effects, impaired reproduction, and increased cancer rates (Boyd and Furlong, 2002). According to the U.S. EPA, many CECs, including PPCPs, are suspected to be endocrine disruptors, which alter the normal functions of hormones, resulting in various health effects (Ankley et al., 2008). **Status:** No information
Contemporary Use Pesticides	**Source:** Contemporary Use Pesticides (CUPs) include the class of current use pesticides and contemporary industrial by-product chemicals, such as octachlorostyrene. Primary examples of CUPs include organophosphates, neonicotinoids, pyrethroids, n-methyl carbamates, and insect growth regulator hormones (EPA, 2011). CUPs are generally a group of semi-volatile chemicals that span multiple chemical classes and can be analyzed concurrently. These pesticides are typically more water-soluble than legacy organochlorine pesticides, such as DDT and chlordane, and often do not bioaccumulate in organisms. It has been estimated that in 2007, over 565 million kg of

	current-use pesticides were used in the USA (EPA, 2011). Among pesticides, herbicides accounted for 40% of total usage and insecticides accounted for 17% (EPA, 2011). While agricultural application accounts for over 60% of pesticides used, urban usage is increasing (EPA, 2011). Pesticides enter the environment seasonally through surface run-off, pesticide drift, direct discharge, and atmospheric long-range transport (USGS, 1999; Federighi, 2008). **Toxicity:** Octachlorostyrene is a by-product of industrial processes involving aluminum refining and combustion of chlorinated compounds. Listed in the EPA priority list of most bioaccumulative compounds, octachlorstyrene is highly toxic and extremely persistent when released to the environment (Chu et al. 2003). **Status:** Octachlorostyrene is included in this study as it has been found in the environment at increasing concentrations, particularly in industrial areas (Chu et al. 2003).
Contrast Agent (PPCP)	**Source:** Environmental detections of pharmaceuticals and personal care products (PPCPs) include a wide spectrum of therapeutic and consumer-use compounds such as prescription and over-the-counter medications, hormones, synthetic fragrances, detergents, disinfectants, insect repellants, and antimicrobial agents. In 2009, an estimated 3.9 billion prescriptions were written for the top 300 pharmaceuticals in the U.S. (Lundy, 2010). Pharmaceutical companies produce over 22.6 million kg (50 million pounds) of antibiotics annually in the U.S., with approximately 60% for human use and 40% for animal agriculture use (Levy, 1998). There are numerous pathways by which PPCPs are introduced into the environment, although the primary routes include wastewater discharge or improper disposal of unused drugs (Daughton and Ternes, 1999). **Toxicity:** Because pharmaceuticals are designed to have a biological effect, the major environmental concerns associated with PPCPs are their potential ecotoxicity and unintentional human health impacts. Potential impacts of PPCPs in the environment include abnormal physiological effects, impaired reproduction, and increased cancer rates (Boyd and Furlong, 2002). According to the U.S. EPA, many CECs, including PPCPs, are suspected to be endocrine disruptors, which alter the normal functions of hormones, resulting in various health effects (Ankley et al., 2008). **Status:** No information
Flame Retardants	**Source:** Alternative flame retardants (AFRs) are added to a wide variety of industrial and consumer products such as textiles, rugs, furniture, and plastics (de Wit, 2002). There are several groups of chemicals characterized as AFRs including hexabromocyclododecanes (HBCDs) and chlorinated organophosphate chemicals (CPP). Although brominated, HBCDs are classified here as an “alternative flame retardant” because they were initially introduced as an alternative to brominated flame retardants such as PBBs and PBDEs, but have since been banned themselves. HBCDs are primarily used in household consumer products such as upholstery, polystyrene, and textiles (de Wit, 2002). The chlorinated organophosphate flame retardants such as tris(1,3-dichloroisopropyl)phosphate (TDCPP) are mainly used as additives in textiles. As additives, chlorinated organophosphate flame retardants tend to leach into water and air over time. In the environment, TDCPP can accumulate in animal fat tissues (Andresen et al., 2004). The brominated flame retardants 2-ethylhexyl tetrabromobenzoate (TBB) and 2-ethylhexyl 3,4,5,6-tetrabromophthalate (TBPH) and their metabolites have anti-androgenic and anti-thyroid hormonal activities properties (Klopcic et al., 2016). The chemicals TBB and TBPH were introduced as replacements for the PBDEs and functionally reduce flammability in products like electronic devices, textiles, plastics, coatings, and polyurethane foams. **Toxicity**: HBCDs are ubiquitous in the environment, but their ecotoxicity is not well understood. In June 2022, EPA released a final revised risk determination for HBCD which finds that HBCD presents an unreasonable risk of injury to human health and the environment (EPA, 2022b). **Status:** HBCDs are banned globally under the Stockholm Convention on Persistent Organic Pollutants since 2013; however, the US were not signatories to the convention and continued manufacturing HBCD until 2018 (EPA, 2022c).
Gastrointestinal (PPCP)	**Source:** Environmental detections of pharmaceuticals and personal care products (PPCPs) include a wide spectrum of therapeutic and consumer-use compounds such as prescription and over-the-counter medications, hormones, synthetic fragrances, detergents, disinfectants, insect repellants, and antimicrobial agents. In 2009, an estimated 3.9 billion prescriptions were written for the top 300 pharmaceuticals in the U.S. (Lundy, 2010). Pharmaceutical companies produce over 22.6 million kg (50 million pounds) of antibiotics annually in the U.S., with approximately 60% for human use and 40% for animal agriculture use (Levy, 1998). There are numerous pathways by which PPCPs are introduced into the environment, although the primary routes include wastewater discharge or improper disposal of unused drugs (Daughton and Ternes, 1999). **Toxicity:** Because pharmaceuticals are designed to have a biological effect, the major environmental concerns associated with PPCPs are their potential ecotoxicity and unintentional human health impacts. Potential impacts of PPCPs in the environment include abnormal physiological effects, impaired reproduction, and increased cancer rates (Boyd and Furlong, 2002). According to the U.S. EPA, many CECs, including PPCPs, are suspected to be endocrine disruptors, which alter the normal functions of hormones, resulting in various health effects (Ankley et al., 2008). **Status:** No information
Hormone (PPCP)	**Source:** Environmental detections of pharmaceuticals and personal care products (PPCPs) include a wide spectrum of therapeutic and consumer-use compounds such as prescription and over-the-counter medications, hormones, synthetic fragrances, detergents, disinfectants, insect repellants, and antimicrobial agents. In 2009, an estimated 3.9 billion prescriptions were written for the top 300 pharmaceuticals in the U.S. (Lundy, 2010). Pharmaceutical companies produce over 22.6 million kg (50 million pounds) of antibiotics annually in the U.S., with approximately 60% for human use and 40% for animal agriculture use (Levy, 1998). There are numerous pathways by which PPCPs are introduced into the environment, although the primary routes include wastewater discharge or improper disposal of unused drugs (Daughton and Ternes, 1999). **Toxicity:** Because pharmaceuticals are designed to have a biological effect, the major environmental concerns associated with PPCPs are their potential ecotoxicity and unintentional human health impacts. Potential impacts of PPCPs in the environment include abnormal physiological effects, impaired reproduction, and increased cancer rates (Boyd and Furlong, 2002). According to the U.S. EPA, many CECs, including PPCPs, are suspected to be endocrine disruptors, which alter the normal functions of hormones, resulting in various health effects (Ankley et al., 2008). **Status:** No information

Neurological (PPCP)	**Source:** Environmental detections of pharmaceuticals and personal care products (PPCPs) include a wide spectrum of therapeutic and consumer-use compounds such as prescription and over-the-counter medications, hormones, synthetic fragrances, detergents, disinfectants, insect repellants, and antimicrobial agents. In 2009, an estimated 3.9 billion prescriptions were written for the top 300 pharmaceuticals in the U.S. (Lundy, 2010). Pharmaceutical companies produce over 22.6 million kg (50 million pounds) of antibiotics annually in the U.S., with approximately 60% for human use and 40% for animal agriculture use (Levy, 1998). There are numerous pathways by which PPCPs are introduced into the environment, although the primary routes include wastewater discharge or improper disposal of unused drugs (Daughton and Ternes, 1999). **Toxicity:** Because pharmaceuticals are designed to have a biological effect, the major environmental concerns associated with PPCPs are their potential ecotoxicity and unintentional human health impacts. Potential impacts of PPCPs in the environment include abnormal physiological effects, impaired reproduction, and increased cancer rates (Boyd and Furlong, 2002). According to the U.S. EPA, many CECs, including PPCPs, are suspected to be endocrine disruptors, which alter the normal functions of hormones, resulting in various health effects (Ankley et al., 2008). **Status:** No information
Pain/ Anti-Inflammatory (PPCP)	**Source:** Environmental detections of pharmaceuticals and personal care products (PPCPs) include a wide spectrum of therapeutic and consumer-use compounds such as prescription and over-the-counter medications, hormones, synthetic fragrances, detergents, disinfectants, insect repellants, and antimicrobial agents. In 2009, an estimated 3.9 billion prescriptions were written for the top 300 pharmaceuticals in the U.S. (Lundy, 2010). Pharmaceutical companies produce over 22.6 million kg (50 million pounds) of antibiotics annually in the U.S., with approximately 60% for human use and 40% for animal agriculture use (Levy, 1998). There are numerous pathways by which PPCPs are introduced into the environment, although the primary routes include wastewater discharge or improper disposal of unused drugs (Daughton and Ternes, 1999). **Toxicity:** Because pharmaceuticals are designed to have a biological effect, the major environmental concerns associated with PPCPs are their potential ecotoxicity and unintentional human health impacts. Potential impacts of PPCPs in the environment include abnormal physiological effects, impaired reproduction, and increased cancer rates (Boyd and Furlong, 2002). According to the U.S. EPA, many CECs, including PPCPs, are suspected to be endocrine disruptors, which alter the normal functions of hormones, resulting in various health effects (Ankley et al., 2008). **Status:** No information
PFASs	**Source:** Per- and polyfluoroalkyl substances (PFAS) are a group of fluorine-containing compounds used in industrial processes related to surface protection/coatings, fire-fighting foam, insecticides, and commercial polymer manufacturing (ATSDR, 2018). Typically, PFAS enter the aquatic environment through aqueous effluent from fire training/fire response sites, industrial sites, wastewater treatment plants, and runoff from the land application of contaminated biosolids (ATSDR, 2018). This class of chemicals appears to accumulate in the environment and, because of their widespread use, they are becoming ubiquitous in sediment and tissue samples in coastal habitats (Chen et al., 2012; CDC, 2018). **Toxicity:** When they are taken up by organisms, PFAS are suspected to be endocrine disruptors and can cause developmental problems in animals (Grun and Blumberg, 2009). Perfluorooctane sulfonic acid (PFOS) is one of the most toxic PFAS contaminants, according to available toxicological data. It has been linked to liver damage, cancer, and immune system suppression in humans (CDC, 2018). Thus, this class of CECs has garnered increasing interest in the past 10-15 years. **Status:** While the manufacturing of PFOS and PFOA has been phased out in the US, the EPA and several states have started developing health-based guidelines for PFOS and PFOA in drinking water (Corder et al., 2018). There are thousands of PFAS pollutants, but only a few are becoming more routinely monitored in the environment.
Respiratory (PPCP)	**Source:** Environmental detections of pharmaceuticals and personal care products (PPCPs) include a wide spectrum of therapeutic and consumer-use compounds such as prescription and over-the-counter medications, hormones, synthetic fragrances, detergents, disinfectants, insect repellants, and antimicrobial agents. In 2009, an estimated 3.9 billion prescriptions were written for the top 300 pharmaceuticals in the U.S. (Lundy, 2010). Pharmaceutical companies produce over 22.6 million kg (50 million pounds) of antibiotics annually in the U.S., with approximately 60% for human use and 40% for animal agriculture use (Levy, 1998). There are numerous pathways by which PPCPs are introduced into the environment, although the primary routes include wastewater discharge or improper disposal of unused drugs (Daughton and Ternes, 1999). **Toxicity:** Because pharmaceuticals are designed to have a biological effect, the major environmental concerns associated with PPCPs are their potential ecotoxicity and unintentional human health impacts. Potential impacts of PPCPs in the environment include abnormal physiological effects, impaired reproduction, and increased cancer rates (Boyd and Furlong, 2002). According to the U.S. EPA, many CECs, including PPCPs, are suspected to be endocrine disruptors, which alter the normal functions of hormones, resulting in various health effects (Ankley et al., 2008). **Status:** No information
Stimulant (PPCP)	**Source:** Environmental detections of pharmaceuticals and personal care products (PPCPs) include a wide spectrum of therapeutic and consumer-use compounds such as prescription and over-the-counter medications, hormones, synthetic fragrances, detergents, disinfectants, insect repellants, and antimicrobial agents. In 2009, an estimated 3.9 billion prescriptions were written for the top 300 pharmaceuticals in the U.S. (Lundy, 2010). Pharmaceutical companies produce over 22.6 million kg (50 million pounds) of antibiotics annually in the U.S., with approximately 60% for human use and 40% for animal agriculture use (Levy, 1998). There are numerous pathways by which PPCPs are introduced into the environment, although the primary routes include wastewater discharge or improper disposal of unused drugs (Daughton and Ternes, 1999). **Toxicity:** Because pharmaceuticals are designed to have a biological effect, the major environmental concerns associated with PPCPs are their potential ecotoxicity and unintentional human health impacts. Potential impacts of PPCPs in the environment include abnormal physiological effects, impaired reproduction, and increased cancer rates (Boyd and Furlong, 2002). According to the U.S. EPA, many CECs, including PPCPs, are suspected to be endocrine disruptors, which alter the normal functions of hormones, resulting in various health effects (Ankley et al., 2008).

	Status: No information
Surfactants	**Source:** Surfactants, such as alkylphenols, are a class of chemicals used in detergents and as surfactants in industrial processes. Some household detergents (i.e., laundry soaps) also include surfactants. The most common sources of surfactants to aquatic systems are wastewater and septic system discharges (Ying et al., 2002). These compounds tend to be persistent in the environment, have a strong affinity for suspended particles, and are well preserved in bottom sediments (Ying et al., 2002). In the environment, alkylphenol ethoxylate surfactants biodegrade into more environmentally stable metabolites, such as the alkylphenol n-ethoxylates, alkylphenoxy acetic, alkylphenoxy polyethoxy acetic acids, and alkylphenols (EPA, 2014b). **Toxicity:** The compounds 4-nonylphenol (4-NP) and 4-n-octylphenol (4-OP) are degradation products of 4-nonylphenol mono-ethoxylate (NP1EO) and 4-nonylphenol di-ethoxylate (NP2EO), which are byproducts of the parent alkylphenol polyethoxylate. These degradation products are reported to be more toxic than the parent compounds and act as hormone mimics (Ying et al., 2002). Surfactants are shown to have estrogenic endocrine-disrupting effects on vertebrate organisms, and they have been linked to severe decreases in lobster larval survival and juvenile lobster hormonal changes (Laufer et al., 2013). **Status:** 4-NP, 4-OP, NP1EO and NP2EO were included in the EPA New Use Rules list of 15 toxic AP compounds (EPA, 2014b)

LISTS OF ANALYTES

Asterisks indicate contaminants that are included in the group sums in the Explore Data and Create Report parts of the CPDE.

Legacy Contaminants Groups

Butyltins
Dibutyltin*
Monobutyltin*
Tetrabutyltin*
Tributyltin*

Chlordanes
Alpha-Chlordane*
Cis-Nonachlor*
Gamma-Chlordane*
Heptachlor*
Heptachlor-Epoxide*
Oxychlordane*
Trans-Nonachlor*

Chlorobenzenes
1,2,3,4-Tetrachlorobenzene*
1,2,3,5-Tetrachlorobenzene
1,2,4,5-Tetrachlorobenzene*
Hexachlorobenzene*
Pentachloroanisole*
Pentachlorobenzene*

DDTs
2,4'-DDD*
2,4'-DDE*
2,4'-DDT*
4,4'-DDD*
4,4'-DDE*
4,4'-DDT*
DDMU

Dieldrins
Aldrin*
Dieldrin*
Endrin Aldehyde
Endrin Ketone
Endrin*

Endosulfans
Endosulfan I*
Endosulfan II*
Endosulfan Sulfate*

HCHs
Alpha-Hexachlorocyclohexane*
Beta-Hexachlorocyclohexane*
Delta-Hexachlorocyclohexane*
Gamma-Hexachlorocyclohexane*

Chlorpyrifos
Chlorpyrifos*

Mirex
Mirex*

PCBs								
Dichlorobiphenyls	PCB19*	PCB41_64*	PCB65*	PCB89_90	PCB112*	PCB138_160*	PCB160_158*	PCB187_182*
Heptachlorobiphenyls	PCB20	PCB42*	PCB66*	PCB91*	PCB114	PCB138_164_163*	PCB162*	PCB188*
Hexachlorobiphenyls	PCB21_20_33*	PCB42_59_37*	PCB66_80*	PCB92*	PCB114_122*	PCB140*	PCB164_163	PCB189*
Nonachlorobiphenyls	PCB22*	PCB43*	PCB67*	PCB94*	PCB114_131_122*	PCB141*	PCB165	PCB191*
Octachlorobiphenyls	PCB22_51*	PCB44*	PCB68_41_64*	PCB95*	PCB116_117*	PCB141_179*	PCB165_131*	PCB192_172*
Pentachlorobiphenyls	PCB23*	PCB45*	PCB69	PCB96_103*	PCB118*	PCB142_146_161*	PCB166*	PCB193
Tetrachlorobiphenyls	PCB24*	PCB46*	PCB70*	PCB97*	PCB118_106	PCB143*	PCB167*	PCB194*
Trichlorobiphenyls	PCB24_27*	PCB46_69_73*	PCB70_76	PCB97_125_86*	PCB118_108*	PCB144*	PCB169*	PCB195*
PCB1*	PCB25*	PCB47	PCB71*	PCB99*	PCB119*	PCB146*	PCB170_190*	PCB195_208*
PCB2*	PCB26*	PCB47_48_75*	PCB72*	PCB100*	PCB120_83*	PCB147*	PCB171*	PCB196
PCB3*	PCB27*	PCB48_47	PCB74	PCB101	PCB121_93_95*	PCB148_145*	PCB172*	PCB196_203*
PCB4_10*	PCB28*	PCB48_75_47*	PCB74_61*	PCB101_84_90*	PCB123*	PCB149	PCB173*	PCB197*
PCB6*	PCB28_31*	PCB49*	PCB76	PCB101_90*	PCB124*	PCB149_123*	PCB174*	PCB198
PCB7	PCB29*	PCB50*	PCB76_70*	PCB101_90_89	PCB126*	PCB149_139*	PCB175*	PCB199*
PCB7_9*	PCB30*	PCB51*	PCB77*	PCB102_98*	PCB128*	PCB150*	PCB176*	PCB200*
PCB8_5*	PCB31*	PCB52*	PCB78*	PCB103	PCB128_167*	PCB151*	PCB176_137*	PCB201*
PCB9	PCB33	PCB53*	PCB79*	PCB104*	PCB129*	PCB152*	PCB177*	PCB201_173_157*
PCB11*	PCB33_53_20*	PCB54*	PCB81*	PCB105*	PCB129_126*	PCB153	PCB178*	PCB202*
PCB12*	PCB34*	PCB55*	PCB82*	PCB105_127*	PCB130*	PCB153_132_168*	PCB179*	PCB203_196*
PCB13*	PCB35*	PCB56*	PCB83*	PCB106_107*	PCB132*	PCB153_168*	PCB180*	PCB204*
PCB14*	PCB36*	PCB56_60*	PCB84*	PCB107*	PCB132_168	PCB154	PCB180_193*	PCB205*
PCB15*	PCB37*	PCB58*	PCB84_89_90_101	PCB107_108	PCB134_133*	PCB155*	PCB181*	PCB206*
PCB16	PCB38*	PCB59*	PCB85*	PCB108_107_123	PCB135*	PCB156*	PCB183*	PCB207*
PCB16_32*	PCB39*	PCB60*	PCB86*	PCB109*	PCB136*	PCB156_171_202*	PCB184*	PCB208*
PCB17*	PCB40*	PCB61	PCB87_115*	PCB110*	PCB136_154*	PCB157*	PCB185*	PCB209*
PCB18*	PCB40_57*	PCB62*	PCB88*	PCB110_77*	PCB137*	PCB158*	PCB186_178*	
PCB18_17	PCB41	PCB63*	PCB89_113*	PCB111_115_87*	PCB138_158	PCB159*	PCB187*	

Contaminants of Emerging Concern (CEC) Groups

Surfactants
4-n-octylphenol*
4-nonylphenol*
NP1EO*
NP2EO*

Flame Retardants
Alpha-Hexabromocyclododecane*
Beta-Hexabromocyclododecane*
BTBPE*
Gamma-Hexabromocyclododecane*
TBB*
TBPH*
TCEP*
TCPP*
TDCPP*

PBBs
PBB 1 [2-MonoBB]
PBB 2 [3-MonoBB]
PBB 3 [4-MonoBB]
PBB 4 [2,2'-DiBB]
PBB 7 [2,4-DiBB]
PBB 9 [2,5-DiBB]
PBB 10 [2,6-DiBB]
PBB 15 [4,4'-DiBB]
PBB 18 [2,2',5-TriBB]
PBB 26 [2,3',5-TriBB]
PBB 30 [2,4,6-TriBB]
PBB 31 [2,4',5-TriBB]
PBB 49 [2,2',4,5'-TetraBB]
PBB 52 [2,2',5,5'-TetraBB]
PBB 53 [2,2',5,6'-TetraBB]
PBB 77 [3,3',4,4'-TetraBB]
PBB 80 [3,3',5,5'-TetraBB]
PBB 103 [2,2',4,5',6-PentaBB]
PBB 155 [2,2',4,4',6,6'-HexaBB]

Contemporary Use Pesticides		
Ametryn*	Diazinon*	Octachlorostyrene*
Atrazine*	Diazinon-Oxon*	Parathion-Ethyl*
Azinphos-Methyl*	Dimethoate*	Parathion-Methyl*
Baygon	Disulfoton Sulfone*	Permethrin*
Captan*	Disulfoton*	Permethrin, cis-*
Carbaryl	Ethion*	Permethrin, trans-*
Carbofuran	Fenitrothion*	Perthane*
Chlorothalonil*	Fonofos*	Phorate*
Chlorpyrifos Oxon	Gluthion	Phosmet*
Cyanazine*	Hexazinone*	Pirimiphos-Methyl*
Cypermethrin*	Malathion*	Quintozene*
Cypermethrin-A*	Maloxon	Simazine*
Cypermethrin-B*	Methamidophos	Tecnazene*
Cypermethrin-C*	Methidathion	Terbufos*
Dacthal*	Methoxychlor*	Trifluralin
DEET*	Metribuzin*	
Desethylatrazine*	Molinate	

PBDEs		
BDE 1 [2-MonoBDE]*	BDE 37 [3,4,4'-TriBDE]*	BDE 155 [2,2',4,4',6,6'-HexaBDE]*
BDE 2 [3-MonoBDE]*	BDE 47 [2,2',4,4'-TetraBDE]*	BDE 166 [2,3,4,4',5,6-HexaBDE]*
BDE 3 [4-MonoBDE]*	BDE 49 [2,2',4,5'-TetraBDE]*	BDE 181 [2,2',3,4,4',5,6-HeptaBDE]*
BDE 4 [2,2'-DiBDE]*	BDE 49_71 [2,2',4,5'-TetraBDE_2,3',4',6-TetraBDE]*	BDE 183 [2,2',3,4,4',5',6-HeptaBDE]*
BDE 7 [2,4-DiBDE]*	BDE 66 [2,3',4,4'-TetraBDE]*	BDE 190 [2,3,3',4,4',5,6-HeptaBDE]*
BDE 8 [2,4'-DiBDE]*	BDE 71 [2,3',4',6-TetraBDE]*	BDE 194 [2,2',3,3',4,4',5,5'-OctaBDE]*
BDE 10 [2,6-DiBDE]*	BDE 75 [2,4,4',6-TetraBDE]*	BDE 195 [2,2',3,3',4,4',5,6-OctaBDE]*
BDE 11 [3,3'-DiBDE]*	BDE 77 [3,3',4,4'-TetraBDE]*	BDE 196 [2,2',3,3',4,4',5,6'-OctaBDE]*
BDE 12 [3,4-DiBDE]*	BDE 85 [2,2',3,4,4'-PentaBDE]*	BDE 197 [2,2',3,3',4,4',6,6'-OctaBDE]*
BDE 13 [3,4'-DiBDE]*	BDE 99 [2,2',4,4',5-PentaBDE]*	BDE 198_199_203_200 [OctaBDE]*
BDE 15 [4,4'-DiBDE]*	BDE 100 [2,2',4,4',6-PentaBDE]*	BDE 201 [2,2',3,3',4,5',6,6'-OctaBDE]*
BDE 17 [2,2',4-TriBDE]*	BDE 116 [2,3,4,5,6-PentaBDE]*	BDE 202 [2,2',3,3',5,5',6,6'-OctaBDE]*
BDE 25 [2,3',4-TriBDE]*	BDE 118 [2,3',4,4',5-PentaBDE]*	BDE 204 [2,2',3,4,4',5,6,6'-OctaBDE]*
BDE 28 [2,4,4'-TriBDE]*	BDE 119 [2,3',4,4',6-PentaBDE]*	BDE 205 [2,3,3',4,4',5,5',6-OctaBDE]*
BDE 30 [2,4,6-TriBDE]*	BDE 126 [3,3',4,4',5-PentaBDE]*	BDE 206 [2,2',3,3',4,4',5,5',6-NonaBDE]*
BDE 32 [2,4',6-TriBDE]*	BDE 138 [2,2',3,4,4',5'-HexaBDE]*	BDE 207 [2,2',3,3',4,4',5,6,6'-NonaBDE]*
BDE 33 [2',3,4-TriBDE]*	BDE 153 [2,2',4,4',5,5'-HexaBDE]*	BDE 208 [2,2',3,3',4,5,5',6,6-NonaBDE]*
BDE 35 [3,3',4-TriBDE]*	BDE 154 [2,2',4,4',5,6'-HexaBDE]*	BDE 209 [2,2',3,3',4,4',5,5',6,6'-DecaBDE]*

PFASs
11-Chloroeicosafluoro-3-oxaundecane-1-sulfonic acid*
4,8-dioxa-3Hperfluorononanoate*
4:2 Fluorotelomer sulfonic acid*
6:2 Fluorotelomer sulfonic acid*
8:2 Fluorotelomer sulfonic acid*
Hexafluoropropylene oxide-dimer acid*
N-ethylperfluorooctane sulfonamidoacetic acid*
N-ethylperfluorooctanesulfonamide*
N-ethylperfluorooctanesulfonamidoethanol*
N-methylperfluorooctane sulfonamidoacetic acid*
N-methylperfluorooctanesulfonamide*
N-methylperfluorooctanesulfonamidoethanol*
Perfluoro(2-((6-chlorohexyl)oxy) ethanesulfonic acid)*
Perfluorobutane sulfonic acid*
Perfluorobutanoic acid*
Perfluorodecane sulfonic acid*
Perfluorodecanoic acid*
Perfluorododecanesulfonic acid*
Perfluorododecanoic acid*
Perfluoroheptane sulfonic acid*
Perfluoroheptanoic acid*
Perfluorohexane sulfonic acid*
Perfluorohexanoic acid*
Perfluorononane sulfonic acid*
Perfluorononanoic acid*
Perfluorooctane sulfonamide*
Perfluorooctane sulfonic acid*
Perfluorooctanoic acid*
Perfluoropentane sulfonic acid*
Perfluoropentanoic acid*
Perfluorotetradecanoic acid*
Perfluorotridecanoic acid*
Perfluoroundecanoic acid*

Antidepressant (PPCP)
10-hydroxy-amitriptyline*
Alprazolam*
Amitriptyline*
Citalopram*
Clonidine*
Desmethyldiltiazem*
Diazepam*
Fluoxetine*
Meprobamate*
Norfluoxetine*
Oxazepam*
Paroxetine*
Sertraline*
Venlafaxine*
Venlafaxine

Cardiovascular (PPCP)
Amlodipine*
Atenolol*
Atorvastatin*
Dehydronifedipine*
Digoxigenin*
Digoxin*
Diltiazem*
Enalapril*
Furosemide*
Gemfibrozil*
Hydrochlorothiazide*
Metoprolol*
N-Desmethyldiltiazem*
Norverapamil*
Propranolol*
Rosuvastatin*
Simvastatin*
Triamterene*
Valsartan*
Verapamil*
Warfarin*

Antihistamine (PPCP)
Diphenhydramine*
Fluocinonide*
Fluticasone propionate*
Promethazine*

Chemotherapy (PPCP)
Amphetamine*
Amsacrine*
Busulfan*
Cyclophosphamide*
Daunorubicin*
Doxorubicin*
Etoposide*
Melphalan*
Teniposide*
Trenbolone acetate*
Trenbolone*

Gastrointestinal (PPCP)
Cimetidine*
Ranitidine*

Pain/Anti-Inflammatory (PPCP)
2-Hydroxy Ibuprofen*
Acetaminophen*
Betamethasone*
Codeine*
Colchicine*
Hydrocortisone*
Ibuprofen*
Methylprednisolone*
Naproxen*
Oxycodone*
Prednisolone*
Prednisone*
Propoxyphene*

Hormone (PPCP)
17a-Dihydroequilin*
17a-estradiol*
17a-Ethynyl estradiol*
17B-estradiol*
Allyl Trenbolone*
Androstenedione*
Androsterone*
Bisphenol-A*
Desogestrel*
Diethylstilbestrol*
Drospirenone*
Equilenin*
Equilin*
Estriol*
Estrone*
Medroxyprogesterone Acetate*
Mestranol*
Norethindrone*
Norgestimate*
Norgestrel*
Progesterone*
Tamoxifen*
Testosterone*

Stimulant (PPCP)
1,7-Dimethylxanthine*
Benzoylecgonine*
Caffeine*
Cocaine*
Cotinine*
Paraxanthine*

Respiratory (PPCP)
Albuterol*
Hydrocodone*
Theophylline*

Contrast Agent (PPCP)
Diatrizoic acid*
Iopamidol*

Antidiabetic (PPCP)
Glipizide*
Glyburide*
Metformin*

Neurological (PPCP)
Benztropine*
Carbamazepine*

Antibiotic (PPCP)					
4-Epianhydrochlortetracycline [EACTC]*	Cefotaxime*	Erythromycin*	Norfloxacin*	Sulfachloropyridazine*	Thiabendazole*
4-Epianhydrotetracycline [EATC]*	Chlortetracycline [CTC]*	ErythromycinH20*	Ofloxacin*	Sulfadiazine*	Triclocarban*
4-Epichlortetracycline [ECTC]*	Ciprofloxacin*	Flumequine*	Ormetoprim*	Sulfadimethoxine*	Triclosan*
4-Epioxytetracycline [EOTC]*	Clarithromycin*	Isochlortetracycline [ICTC]*	Oxacillin*	Sulfamerazine*	Trimethoprim*
4-Epitetracycline [ETC]*	Clinafloxacin*	Lincomycin*	Oxolinic Acid*	Sulfamethazine*	Tylosin*
Anhydrochlortetracycline [ACTC]*	Clotrimazole*	Lomefloxacin*	Oxytetracycline [OTC]*	Sulfamethizole*	Virginiamycin M1*
Anhydrotetracycline [ATC]*	Cloxacillin*	Metronidazole*	Penicillin G*	Sulfamethoxazole*	Zidovudine*
Azathioprine*	Demeclocycline*	Miconazole*	Penicillin V*	Sulfanilamide*	
Azithromycin*	Doxycycline*	Minocycline*	Roxithromycin*	Sulfathiazole*	
Carbadox*	Enrofloxacin*	Moxifloxacin*	Sarafloxacin*	Tetracycline [TC]*	

Additional Analytes Groups

Ancillary
- Sample dry weight
- Sample percent dry weight
- Sample percent wet weight
- Sample wet weight

Tissue
- Percent lipid dry weight
- Percent lipid wet weight

Carbon
- Total Inorganic Carbon
- Total Organic Carbon

Grain size
- Clay - Chemistry Sample
- Fines - Chemistry Sample
- Gravel - Chemistry Sample
- Particle size
- Sand - Chemistry Sample
- Silt - Chemistry Sample
- Sorting coefficient

Sewage
- Clostridium perfringens - Count 1
- Clostridium perfringens - Count 2
- Clostridium perfringens - dry
- Clostridium perfringens - Mean
- Clostridium perfringens - Moisture
- Clostridium perfringens - wet

Standards
- PCB198
- PCB103

Fluorescent aromatic compounds
- Fluorescent aromatic compounds [high weight]
- Fluorescent aromatic compounds [low weight]

Energetics
- 1,3,5-Trinitrobenzene
- 1,3-Dinitrobenzene
- 1-Chloro-3-nitrobenzene
- 2,2',6,6'-tetranitro-4,4'-azoxytoluene
- 2,4,6-Trinitrophenylmethylnitramine
- 2,4,6-Trinitrotoluene
- 2,4-Dinitrotoluene
- 2,6-Dinitrotoluene
- 2-Amino-4,6-dinitrotoluene
- 2-Nitrotoluene
- 3-Nitrotoluene
- 4-Amino-2,6-dinitrotoluene
- 4-Nitrotoluene
- Hexahydro-1,3,5-trinitro-1,3,5-triazine
- Nitrobenzene
- Octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine
- Pentaerythritol tetranitrate

Polychlorinated Dioxins and Furans
- 1,2,3,4,6,7,8-Heptachlorodibenzofuran
- 1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin
- 1,2,3,4,7,8,9-Heptachlorodibenzofuran
- 1,2,3,4,7,8-Hexachlorodibenzofuran
- 1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin
- 1,2,3,6,7,8-Hexachlorodibenzofuran
- 1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin
- 1,2,3,7,8,9-Hexachlorodibenzofuran
- 1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin
- 1,2,3,7,8-Pentachlorodibenzofuran
- 1,2,3,7,8-Pentachlorodibenzo-p-dioxin
- 2,3,4,6,7,8-Hexachlorodibenzofuran
- 2,3,4,7,8-Pentachlorodibenzofuran
- 2,3,7,8-Tetrachlorodibenzofuran
- 2,3,7,8-Tetrachlorodibenzo-p-dioxin
- Octachlorodibenzofuran
- Octachlorodibenzo-p-dioxin

Alkanes
- 2,6,10-Trimethyldodecane (i-C15)
- 2,6,10-Trimethyltridecane (i-C16)
- n-Decane
- n-Docosane
- n-Dodecane
- n-Dotriacontane
- n-Eicosane
- n-Heneicosane
- n-Hentriacontane
- n-Heptacosane
- n-Heptadecane
- n-Heptatriacontane
- n-Hexacosane
- n-Hexadecane
- n-Hexatriacontane
- n-Nonacosane
- n-Nonadecane
- n-Nonane
- n-Nonatriacontane
- n-Octacosane
- n-Octadecane
- n-Octatriacontane
- Norpristane
- n-Pentacosane
- n-Pentadecane
- n-Pentatriacontane
- n-Tetracontane
- n-Tetracosane
- n-Tetradecane
- n-Tetratriacontane
- n-Triacontane
- n-Tricosane
- n-Tridecane
- n-Tritriacontane
- n-Undecane
- Phytane [i-C20]
- Pristane [i-C19]
- Unresolvable Complex Mixture

Alkylphenol Ethoxylates
- 4-nonylphenol Decaethoxylate
- 4-nonylphenol Diethoxylate
- 4-nonylphenol Ethoxylate
- 4-nonylphenol Heptaethoxylate
- 4-nonylphenol Hexaethoxylate
- 4-nonylphenol Nonaethoxylate
- 4-nonylphenol Octaethoxylate
- 4-nonylphenol Pentaethoxylate
- 4-nonylphenol Tetraethoxylate
- 4-nonylphenol Triethoxylate
- Nonylphenol Polyethoxylate
- NP11EO
- NP12EO
- NP13EO
- NP14EO
- NP15EO
- NP16EO
- Octylphenol diethoxylate
- Octylphenol ethoxylate
- Octylphenol monoethoxylate
- Octylphenol pentaethoxylate
- Octylphenol tetraethoxylate
- Octylphenol triethoxylate

Nutrients
- Ammonium
- Nitrate
- Nitrate+Nitrite
- Nitrite
- Organic Nitrogen
- Orthophosphate
- Silica
- Total Nitrogen
- Total Phosphorous
- Urea

Biomarkers
- Aryl hydrocarbon hydroxylase

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