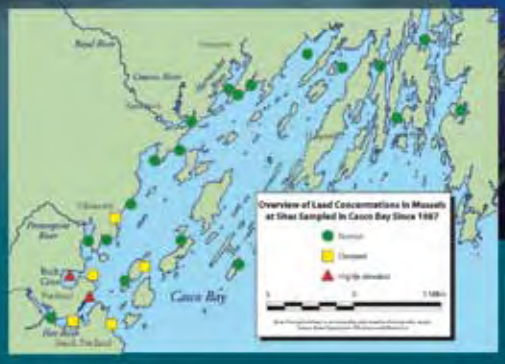
Toxic Pollution in Casco Bay

Sources and Impacts

Casco Bay Estuary Partnership



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The Casco Bay Estuary Partnership (CBEP) is a collaborative effort of state, federal, and local partners to preserve and protect the Bay's resources. For the past 16 years, CBEP has received significant annual federal funding to develop and implement a plan for the Bay's future. Since the *Casco Bay Plan* was adopted in 1996, the partners have been working together to meet the five goals stated in the plan, which include reducing toxics in the Bay, minimizing the loading of pathogens, toxics, and nutrients from stormwater, protecting the water quality of shellfish and swimming areas, protecting habitat, and promoting stewardship of the Bay's resources.

This report addresses the *Casco Bay Plan* goal "reduce toxic pollution in Casco Bay." The following objectives are stated in support of that goal:

- The accumulation of toxics in the sediment and biota shall be reduced.
- Seafood harvested from Casco Bay shall be acceptable for consumption
- Contamination in Casco Bay shall not have an adverse effect on the biological community

CBEP is working to reduce toxic pollution in Casco Bay through research, technical assistance, educational outreach, support for regulatory compliance, and through planning and assessment, including monitoring the levels of toxic chemicals over time.

Toxic Pollution in Casco Bay: Sources and Impacts complements and expands upon the information in the 2005 CBEP report, *State of the Bay.* In that document, CBEP reported on a series of environmental indicators, measures of environmental quality that can be reliably used to assess the current condition of Casco Bay and its watershed as well as temporal trends. Two of these indicators are directly related to toxic pollution: changing levels of toxic chemicals in the Bay's sediments over time; and levels of toxic chemicals in the tissues of blue mussels. In addition to expanding on these indicators, *Toxic Pollution in Casco Bay* details studies undertaken by CBEP and others on some of the sources of toxic chemicals entering the Bay and its watershed, on the impacts of toxic chemicals on Casco Bay wildlife, and on potential risks to human consumers of fish and shellfish. The report does not address groundwater pollution and drinking water issues. In the concluding chapter, the report explores the ways that CBEP and partner organizations are working to reduce the loading of toxic chemicals to the Bay and its watershed.

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C. Schlawe (bird)

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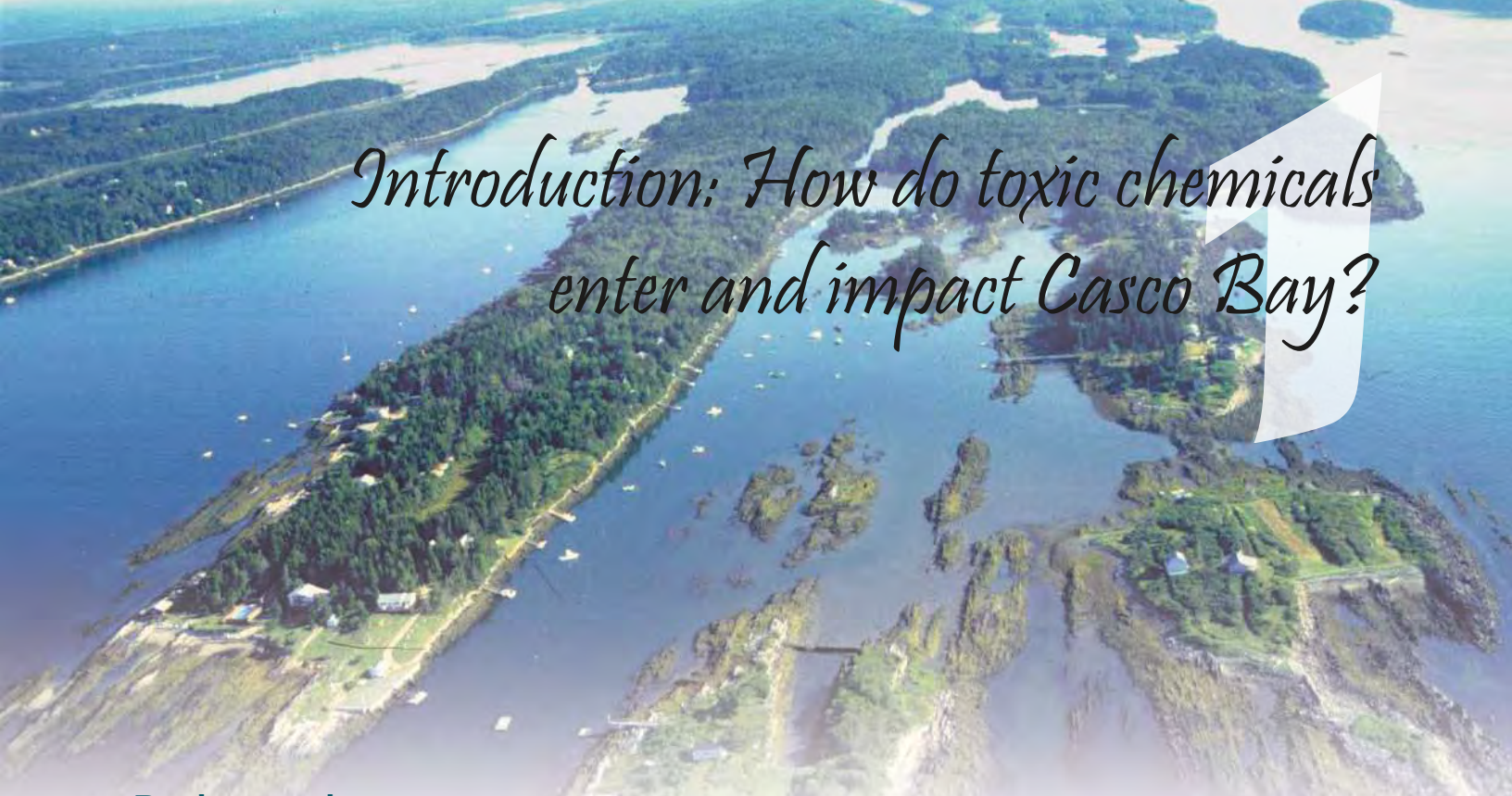


Introduction: How do toxic chemicals enter and impact Casco Bay?

Background

According to the Maine Department of Environmental Protection, the greatest stressors on estuarine and marine waters in Maine are bacteria and toxic chemicals (Maine DEP 2004). The toxic chemicals addressed in this report include two primary types of pollutants: organic chemicals and heavy metals. Organics are bonded forms of carbon, hydrogen and other atoms that occur either naturally or through human introduction. These organic chemicals slowly break down into hydrogen, oxygen, chlorine and other basic components but in the interim they and their interim metabolites (breakdown products) can be toxic to living organisms. Major pathways by which toxic chemicals enter the environment are illustrated in Figure 1-1.

Toxic organic chemicals found in Casco Bay and their primary sources include the following:

- **Polycyclic aromatic hydrocarbons (PAHs)** are the most common toxic contaminants in the Bay. They come primarily from combustion of fossil fuels and wood but also from fuel spills (Chapter 3).
- **Polychlorinated biphenyls (PCBs)** are potent carcinogens formerly used in electric transformers and other industrial applications. They were banned in the 1970s but they are still found in old landfills and dumps and are present at high levels in the Fore River. **Planar PCBs** are the most toxic form of PCBs. The source of these dioxin-like compounds is commercial PCB mixtures (Tanabe *et al.* 1987).
- **Pesticides** are largely carried from lawns and fields to water bodies via stormwater runoff. Although it has been banned since 1972, the pesticide DDT and its toxic breakdown products still persist in the environment.
- **Dioxins and furans** are formed when organic material is burned in the presence of chlorine. Incineration, pulp paper manufacturing, coal-fired utilities, diesel vehicles and metal smelting are all sources of dioxin in the environment (US EPA 2005). Although the pulp mill discharging into the Casco Bay stopped discharging pulp waste in 2000, dioxins and furans still reach the Bay via atmospheric deposition.
- **Butyltins** are toxic organometallic compounds, molecules in which metal is bonded to a carbon atom in an organic molecule. Butyltins get into the Bay's sediments primarily from marine anti-fouling paints.
- **Heavy metals** are dense metallic elements such as lead, mercury, arsenic, cadmium, silver, nickel, selenium, chromium, zinc and copper. Because they do not break down with time, metals delivered from point sources, stormwater runoff or atmospheric deposition can accumulate in the environment. In addition, metals can bind with organic chemicals forming organometallic compounds such as methyl mercury and butyltin, which can be highly toxic. Sources of heavy metals include vehicle emissions, industrial processes, coal combustion, weathering of metal pipes, and incineration (CBEP 1996).

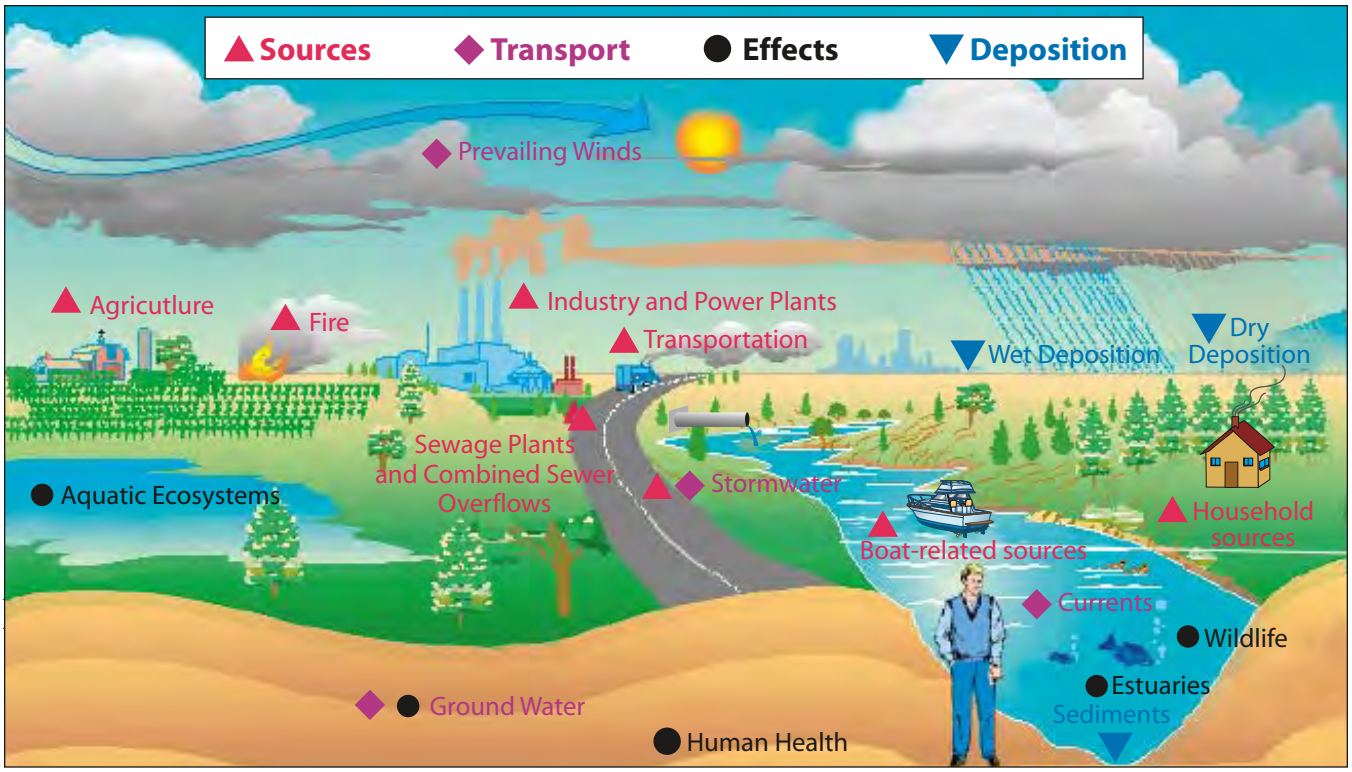


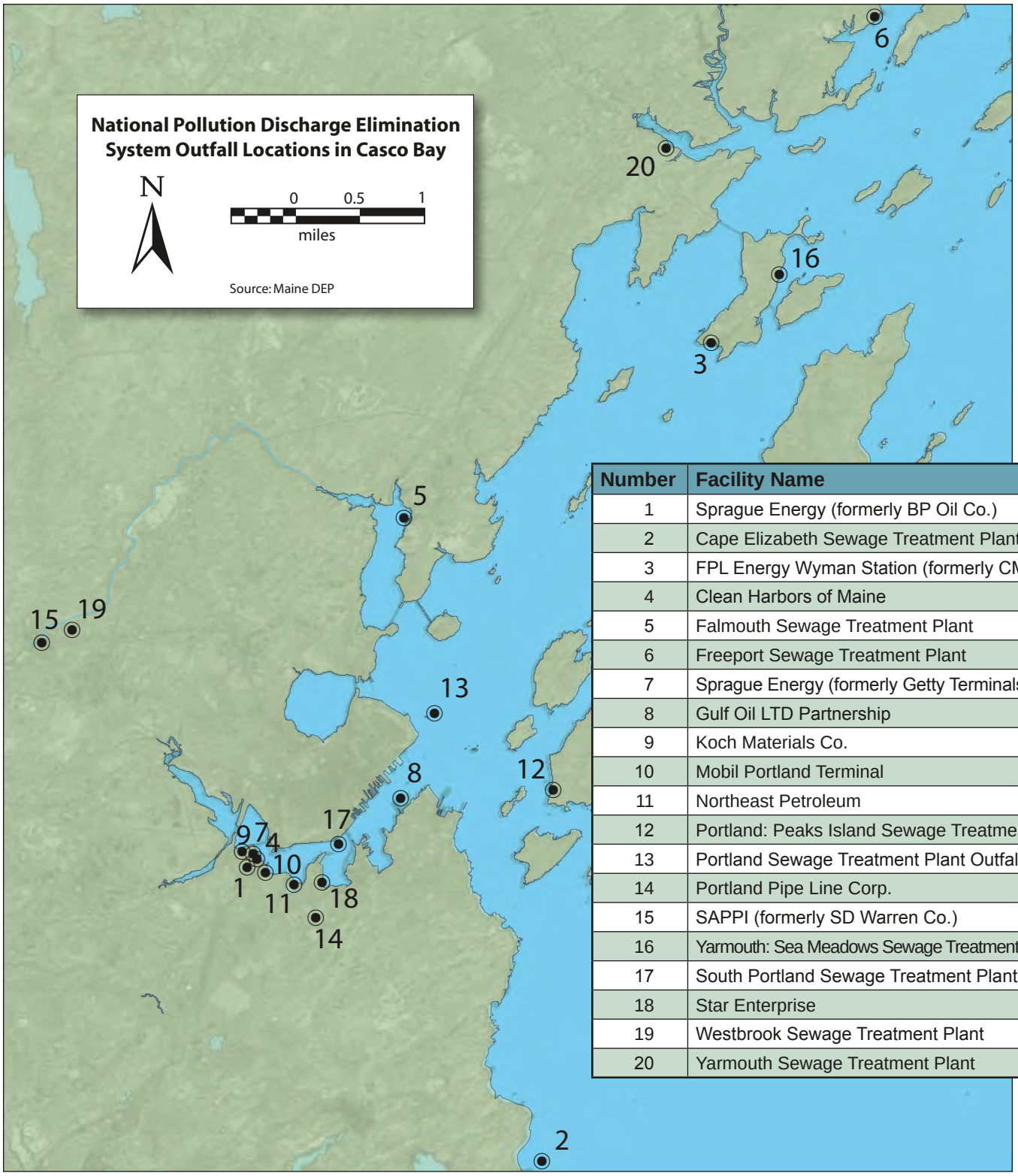
Figure 1-1. *Toxic Chemical Pathways. Major toxic chemical pathways including sources, transport mechanisms, deposition, and effects are illustrated. Sources of toxic chemicals include industrial and power plant discharges, transportation, agriculture, fires and incinerators, boats, and households. Whether the toxics are carried into the watershed by point sources such as pipes, smokestacks, and internal combustion engines, or are transported by wind, rain, and stormwater runoff, ultimately toxic chemicals are finding their way into freshwater and marine aquatic ecosystems. Adapted from National Science and Technology Council Committee on Environment and Natural Resources, Air Quality Research Subcommittee, 1999.*

Point Sources of Toxics to the Bay and the Watershed

Discharges to Casco Bay Waters

Prior to the passage of the federal Clean Water Act in 1970, water pollution from industrial sources had a major impact on the quality of water and sediments in Casco Bay and its watershed. Historic Sources of Pollution in Casco Bay (Hawes 1993) reviewed the "dirty" industrial past when pollutant discharges from railroad complexes, shipyards, tanneries, metal foundries, canneries, paint, textile and glass factories, along with human waste flowed into the watershed and its receiving waters. The electronics, petroleum, plastics and paper industries helped to contribute PCBs, PAHs, heavy metals and organic pollutants. By 1965, for example, the lower Presumpscot River was declared "dead" and living conditions for nearby residents "intolerable" (CBEP 1998).

As these major point source discharges were regulated and cleaned up in the decades following the Clean Water Act, it became clear that a legacy of toxic chemicals remained in the sediments of the watershed and the Bay itself (see Chapter 4). Today, a total of 49 point source discharges in Cumberland County are licensed by the State, through the National Pollutant Discharge Elimination System (NPDES) (US EPA 2006a). Among the major dischargers are: the sewage treatment plants in Portland, South Portland, Westbrook, Freeport, Falmouth and Yarmouth; the Central Maine Power Station on Cousins Island in Yarmouth; First Technology Control Devices in Standish; SAPPI Fine Paper (formerly SD Warren) in Westbrook and multiple oil-water stormwater separator discharges at oil terminals in South Portland. Smaller dischargers include industrial facilities, power plants and small sewage treatment plants. See Figure 1-2 for the locations of NPDES outfalls along the coast of Casco Bay.



Number	Facility Name
1	Sprague Energy (formerly BP Oil Co.)
2	Cape Elizabeth Sewage Treatment Plant
3	FPL Energy Wyman Station (formerly CMP)
4	Clean Harbors of Maine
5	Falmouth Sewage Treatment Plant
6	Freeport Sewage Treatment Plant
7	Sprague Energy (formerly Getty Terminals Corp.)
8	Gulf Oil LTD Partnership
9	Koch Materials Co.
10	Mobil Portland Terminal
11	Northeast Petroleum
12	Portland: Peaks Island Sewage Treatment Plant
13	Portland Sewage Treatment Plant Outfall
14	Portland Pipe Line Corp.
15	SAPPI (formerly SD Warren Co.)
16	Yarmouth: Sea Meadows Sewage Treatment Plant
17	South Portland Sewage Treatment Plant Outfall
18	Star Enterprise
19	Westbrook Sewage Treatment Plant
20	Yarmouth Sewage Treatment Plant

Figure 1-2: National Pollution Discharge Elimination System outfall locations along the coast of Casco Bay. *While licensed and monitored, point sources can still contribute toxic chemicals to the Bay. For example, sewage treatment plants are designed to treat total suspended solids (TSS) and biochemical oxygen demand (BOD). While there is some removal of metals as a side benefit, treatment plants can still contribute heavy metals (e.g., lead, cadmium, arsenic, zinc, silver, and mercury), as well as other toxic chemicals (CBEP 1996). While the levels of pollutants in effluent may meet water quality standards, over time, persistent pollutants can accumulate in the sediments. In addition to the discharges shown on the map, multiple urban combined sewer overflows (CSOs) continue to deliver toxic pollutants (PAHs from petroleum products and tires, for example) to local rivers and streams and ultimately to the Bay.*

Discharges to the Air

The 1970 federal Clean Air Act and amendments help to control pollution releases to the air by establishing ambient air quality standards and requirements for hazardous air pollutants. In Maine, industrial air emissions are licensed by the Maine DEP, which maintains an emissions inventory. Toxics or hazardous air pollutants (HAPs), if released in sufficient quantity, have the potential to cause cancer, respiratory disease or other serious health effects in humans and can have adverse effects on the environment. Toxic air pollutants can exist either as particles or in gaseous vapors. Particulate toxic air pollutants include heavy metals and PAHs. Vapors include benzene, toluene and xylene, found in gasoline; chloroform, from paper production; acrolein, from industrial processes and burning organic matter; perchloroethylene, used in dry cleaning; and methylene chloride, a volatile solvent used in industry (Maine DEP 2006).

Tracking Air Emissions in Maine

Maine DEP and US EPA track the loading of toxics to the atmospheric from local sources by developing air emissions inventories. Using standard protocols, estimations are usually made by multiplying "activity data" (*e.g.,* gallons of fuel burned) times an "emission factor" (*e.g.,* pounds of pollutant released per gallon of fuel burned). By convention, air emission inventories are often broken down into four major categories: Point Sources, Area Sources, Mobile Sources, and Biogenic Sources. A variety of techniques including direct measurement and modeling are used to estimate total emissions.

- **"Point Sources**" are facilities that emit pollutants above a certain threshold, from a stack, vent or similar discrete point of release. The State inventory is derived from summing the releases from each facility that reports. Point source estimates for an individual facility are generally the most accurate category, especially for the larger facilities.
- **"Area Sources"** are sources of air pollutants that are diffused over a wide geographical area or are estimated in the aggregate. Area sources include emissions from smokestacks, vents or other point pources, that in and of themselves are insignificant, but in aggregate may comprise important emissions. Examples would be emissions from small dry cleaners or home heating boilers or air toxics volatizing from house painting, chainsaws or lawnmowers.
- **"Mobile Sources"** are sources of air pollution from internal combustion engines used to propel cars, trucks, trains, buses, airplanes, ATV's, snowmobiles, boats, etc.
- **"Biogenic"** or background sources refers to the concentrations of Air Toxics that are from natural sources and man-made pollutants that are either still in the air from previous years emissions, or have been emitted outside the inventory area and then transported into the region. Maine DEP depends on US EPA to run models that determine releases from the natural sources (Maine DEP 2005).

Maine Air Toxics Initiative 2005 Inventory

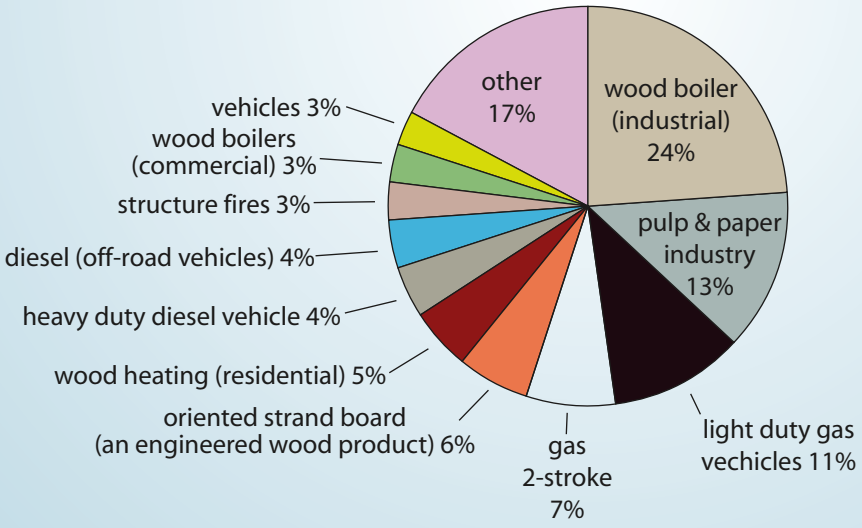
The most accurate, current emissions inventory of Hazardous Air Pollutants (HAPs) or Air Toxics for Maine, is the 2005 estimated emissions inventory that was compiled by the Maine Air Toxics Advisory Committee, a stakeholder group convened by the Maine DEP as part of the Maine Air Toxics Initiative (MATI). The Air Toxics Advisory Committee (ATAC) initially developed a complete HAP inventory for Maine. It was derived by assessing all of the available inventory data and "ground-truthed" based on field investigations, air toxics modeling results, ambient air monitoring programs, and input from the Maine Bureau of Health on the toxicity of various HAPs.

Information used to compile the HAP inventory included the US EPA National Emissions Inventory, data collected from under Maine's Chapter 137 Emissions Inventory of individual facilities that emit any of 217 pollutants above certain thresholds, and the Toxics Release Inventory (TRI). The federal Emergency Planning and Community Right-to-Know Act of 1986 as expanded by the Pollution Prevention Act of 1990 requires certain classes of companies that also employ more than 10 people, and that discharge one of 650 pollutants to the air, water, or land above certain thresholds, to report this information annually to the state and federal governments. US EPA then enters this information into the TRI database, which includes data from 1988 to the present. ATAC also compared emission results to the National Air Toxics Assessment and available ambient air monitoring data. From this information, emission sources that did not appear to be accurate were selected and revised as necessary. For the final MATI inventory, activity levels (amount of fuel burned, acres burned, etc.) are based on Maine specific data whenever possible (Maine DEP 2005). The MATI inventory has been used to assess the sources of emissions. (See Figure 1-3).

Maine Air Emissions Sources

Figure 1-3. The source of current air emissions in Maine can be assessed using the Maine Air Toxics Initiative inventory. It is important to note that the way that categories are lumped together greatly influences the relative ranking of source categories. The ranking is also greatly influenced by uncertainties in the inventory, particularly uncertainty with the emission factor for acrolein, a toxic organic chemical that is used in some industrial processes and can also enter the environment when organic matter such as wood, gasoline, and oil are burned. Total acrolein emissions could be 400% greater or 90% lower, if different emission factors were used for large wood combustion sources. Given these uncertainties, one possible ranking of sources is shown in the pie chart below. "Toxicity weighting" is an approach that accounts for the differing toxicity of air pollutants based on relative impact to human health (Maine DEP 2006). Note that many of the HAPs in the inventory (like acrolein) are primarily a concern due to human inhalation risks and that the toxicity weighting is not based on impacts to the ecosystem.

Sources of Maine Air Emissions
(Based on 2005 Estimated Toxicity-Weighted Emissions)

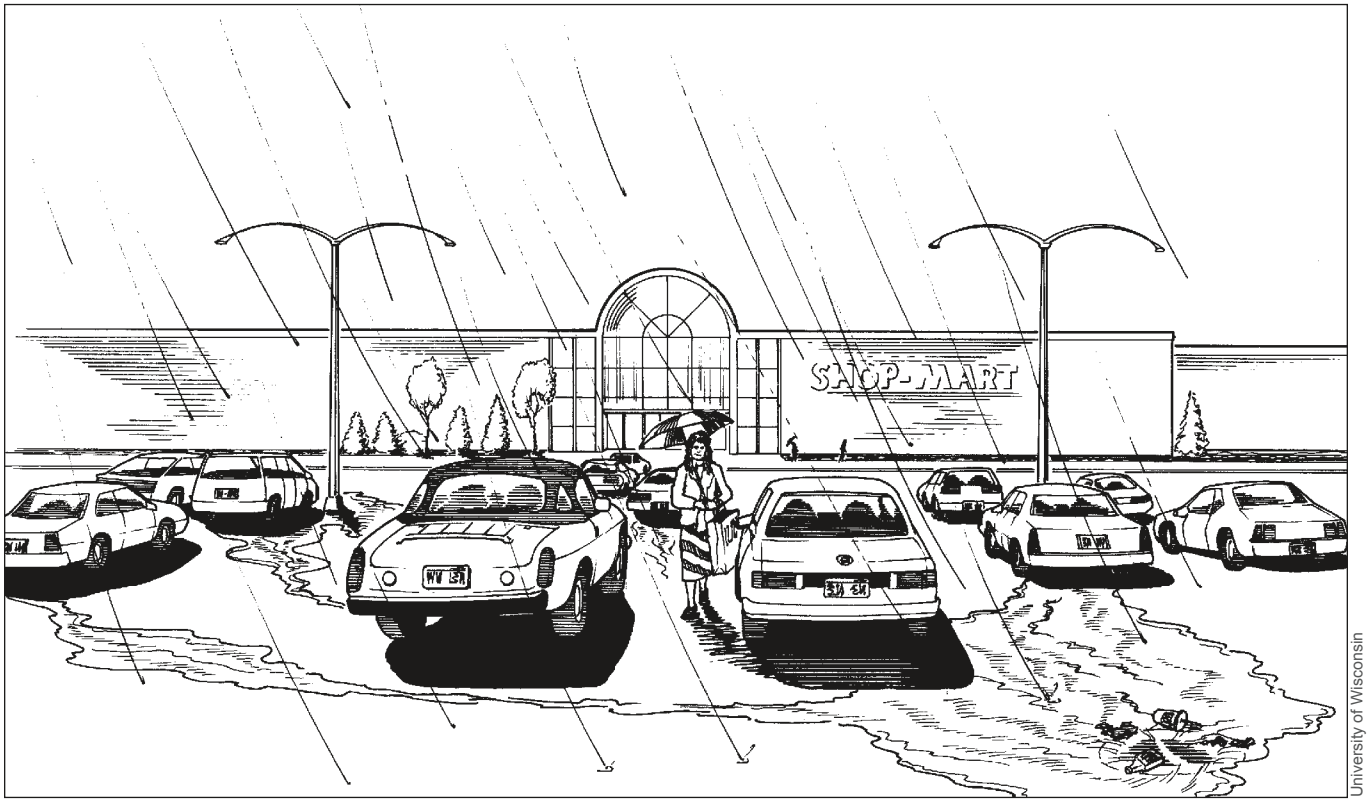


Nonpoint Sources of Toxics to the Bay and the Watershed

Today, nonpoint source pollution is a major contributor of toxic chemicals to the Bay and its watershed. A study undertaken by CBEP revealed that atmospheric deposition is likely the major source of the toxic heavy metal mercury and an important source of PAHs to the watershed and the Bay. Wet atmospheric deposition via precipitation and dry deposition via gases and particles also contribute other heavy metals such as cadmium, zinc, chromium and lead, which can serve as tracers of the sources of pollution (see Chapter 2).

Stormwater is also a major nonpoint source of toxic pollution to the Bay. As rainfall or snowmelt runs over paved or disturbed land surfaces, it picks up pollutants deposited to the ground surface from the atmosphere or local land-based sources and washes them into streams, rivers and eventually to the Bay. Metals and organic contaminants from construction sites, paved urban areas and roads, lawns and farms, underground storage tanks, and landfills adhere to the soil particles and organic matter carried in runoff water. Marinas and boating activities can also contribute toxic solvents and paints via stormwater runoff. And, when oil is spilled on roadways or directly into waterways, PAHs and other organic chemicals can impact wildlife and accumulate in the sediments (see Chapter 3).

Atmospheric deposition of pollutants carried by wind, rain, and snow is an important source of toxics to Casco Bay and its watershed. Above is a satellite image of a major snowstorm blanketing the east coast of the US (NASA Visible Earth 2006, http://veimages.gsfc.nasa.gov/4331/Sea_2002340.jpg).



University of Wisconsin

Figure 1-5. *Runoff from paved surfaces is a major nonpoint pollution source.*

Toxics in the Food Chain

Both toxic organic chemicals and some metals have the potential to increase in concentration as they move up the food chain from the algae and seagrasses that convert sunlight and carbon into food, to fish, birds and mammals, including humans (see Figure 1-6).

Since toxic chemicals tend to collect in sediments, the organisms that inhabit bottom sediments are exposed to the highest levels of contamination. These bottom-dwelling (benthic) organisms play a key role in the food chain, from the bacteria that recycle organic matter and release nutrients to the small crustaceans, worms and mollusks that are consumed by, for example, groundfish, lobsters and crabs. The benthic community in areas that are impacted by toxics lacks the expected diversity and abundance of animals found in clean, healthy bottom communities.

Moving up the food chain, fish that are exposed to toxics chemicals in the environment can experience altered biochemical, respiratory and immune function, developmental and structural abnormalities, cataracts, skin and gill diseases as well as both benign and malignant tumors (O'Connor and Huggett 1988). For example, PAHs have been shown to alter the egg maturation processes in fish (Nicolas 1999). Dietary exposure to mercury has been shown to cause neurological damage in Atlantic salmon (Berntssen *et al*. 2003). Consumption of worms contaminated by PAHs can cause flounder to develop tumors (McElroy *et al.* 1989). While a direct link with pollution has not been demonstrated, fishermen have observed liver tumors in fish caught off Casco Bay (CBEP 1996).

Mammals and birds that feed on benthic organisms or fish from contaminated fresh or salt water environments may absorb toxic pollutants, concentrating them in liver, fat, and muscle tissue (Chapters 6 and 7). Toxic organic chemicals have the potential to disrupt the normal activity of hormones (endocrine-disruption), causing cancer, adverse reproductive effects, birth and developmental effects, and effects on the immune systems (Shaw and DeGuise 2000, DeGuise *et al*. 2001). For example, susceptibility to massive viral epidemics has been observed in European harbor seals exposed to organic pollutants in their environment (Van Loveren *et al*. 2000).

In humans, the causal linkages between endocrine-disrupting organic chemicals and disease have been directly demonstrated in a few cases. PCB exposure to human fetuses *in utero* has been linked with neurological problems, and increased breast cancer risk has been linked with exposure to PCBs and the pesticides DDT and dieldrin (DeGuise *et al*. 2001). Dioxin, considered to be one of the most toxic substances ever identified, has the potential to cause severe reproductive and developmental problems and has been categorized most recently as "likely to be carcinogenic to humans" (NRC 2006) (see Chapter 8).

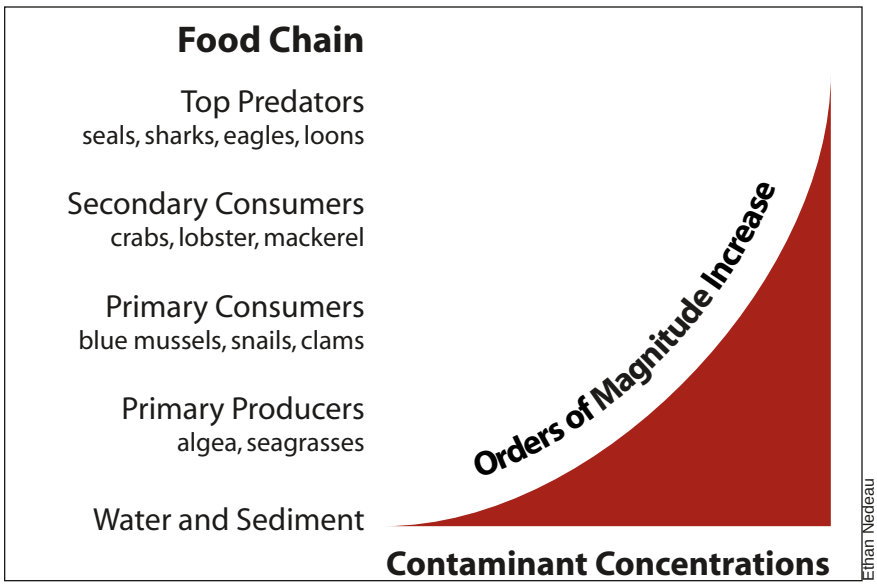


Figure 1-6: *This diagram illustrates the marine food chain, but the same processes occur in fresh water. When organisms are exposed to certain toxic chemicals through, for example, contact with contaminated water or sediment or by consuming prey, the chemicals can become sequestered in their tissues at a higher concentration than in the source (bioaccumulation). The concentration of toxics in organisms may become higher with each step up the food chain from the lowest to the highest links (biomagnification).*


Lee Doggett

In the contaminant-impacted inner Fore River, samples taken in 1989 included some hardy worm species. More sensitive organisms such as mollusks, crustaceans and other typical benthic invertebrates were absent. Even the few pollution-tolerant worms such as this Nephtys *had oil stuck to their "feet" (parapodia) (Doggett 2005).*

Casco Bay Water Bodies That Are Currently Impacted by Toxic Contaminants

According to the Maine DEP's 2004 Integrated Water Quality Monitoring and Assessment Report, elevated levels of toxic contaminants are most often found in harbor and port areas, near the mouths of rivers, in areas with high population density or where there is a legacy of pollutants in the sediments from past activities. Based on sediment analysis and mussel tissue testing, Maine DEP has identified three "Marine and Estuarine Areas of Concern for Toxic Contamination" in Casco Bay. They are the Fore River (1,230 acres), Back Cove (460 acres) and the Presumpscot River Estuary (620 acres) (Maine DEP 2004) (see Figure 1-7).

In addition, all fresh waters in Maine, including those in the Casco Bay watershed, are considered impaired by atmospheric deposition of mercury, resulting in elevated levels of mercury in fish (Maine DEP 2004). As a result of mercury accumulation in fish tissue, the State has issued fish consumption advisories with safe eating guidelines for all freshwater and some marine species. Elevated levels of PCBs, dioxins and DDT have also been identified in the tissues of some freshwater fish, resulting in additional limits to fish consumption for certain ponds and rivers. Fortunately, none of these fresh water bodies impacted by organic pollutants is in the Casco Bay watershed. PCBs and dioxins have been found in some saltwater fish in Maine, resulting in state-wide consumption advisories and safe eating guidelines for striped bass and bluefish. Due to elevated concentrations of dioxin, the State advises that consumers avoid any consumption of lobster tomalley, an organ that serves as the lobster's pancreas and liver, where contaminants can bioaccumulate (Maine CDC 2006).

Maine DEP

Because of the presence of certain toxic contaminants in fish tissues, state-wide consumption advisories and safe eating guidelines have been issued for all freshwater fish as well as for some marine fish, including the striped bass shown above (see Chapter 8).

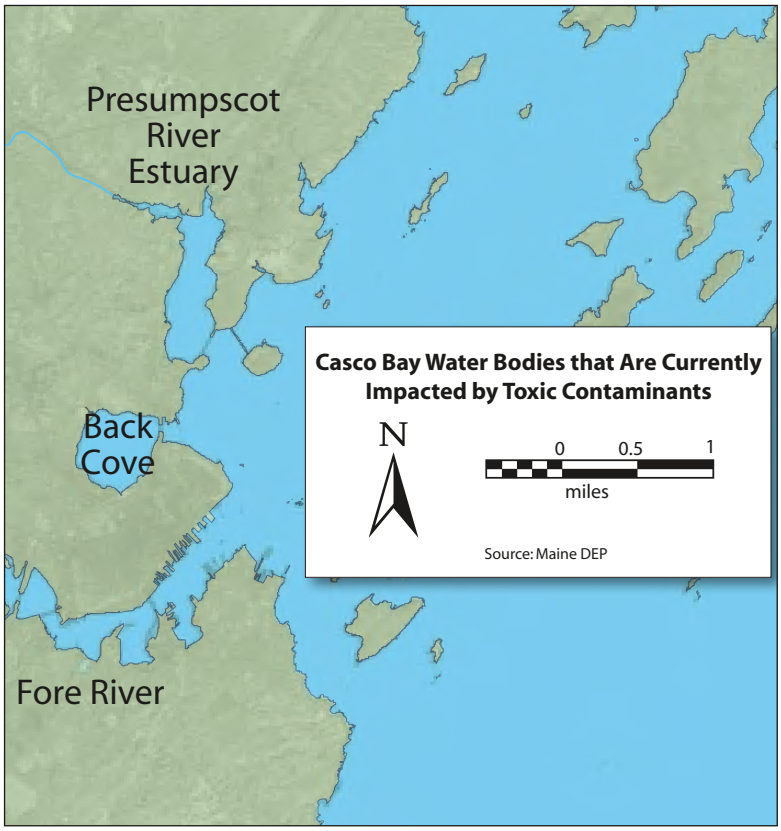


***Figure 1-7**. The Fore River, Back Cove and the Presumpscot River Estuary have been identified by Maine DEP as "Marine and Estuarine Areas of Concern for Toxic Contamination" in Casco Bay (Maine DEP 2004).*

Mike Bradley

Chris Taylor collects sediment samples from Casco Bay as part of the NCA and CBEP Monitoring Programs.

Monitoring Toxics in the Bay

CBEP and our partner organizations have been monitoring toxic contaminants along Maine's coast in recent years. These programs include: the National Coastal Assessment (NCA) (funded by the United States Environmental Protection Agency and administered in Maine by CBEP); the Maine Department of Environmental Protection Surface Water Ambient Toxics Monitoring program (SWAT); the Maine DEP Air Toxics Monitoring Program, including the Breathing Easier through Monitoring (BEAM) program in Portland and the two Mercury Deposition Network sites in the watershed, located in Freeport and Bridgton; the Gulf of Maine Council on the Marine Environment Gulfwatch mussel monitoring program, and CBEP Monitoring Program. CBEP and our partners are tracking the levels of organic chemicals and metals in the Bay's sediments (see Chapter 4), in lobsters, fish, clams and blue mussels (see Chapter 5), and in the precipitation that reaches the Bay (see Chapter 2). Other ongoing monitoring and research programs (such as studies by the Biodiversity Research Institute in Gorham, the Marine Environmental Research Institute in Blue Hill, and the Wise Laboratory of Environmental and Genetic Toxicology at the University of Southern Maine) are assessing the impacts of mercury and other toxic contaminants on Maine's birds and mammals (see Chapters 6 and 7).

A Report Overview

In the chapters that follow, *Toxic Pollution in Casco Bay: Sources and Impacts* describes studies undertaken by CBEP, our partners, state agencies, and research scientists on the sources of toxic chemicals that are entering the Bay and its watershed, on the impacts of toxic chemicals on Casco Bay area wildlife, and on potential risks to human consumers of fish and shellfish. The report also explores the ways that CBEP and our partner organizations are working to reduce the loading of toxics to the Bay and its watershed and to promote stewardship among all the citizens of Casco Bay. A glossary is provided which defines acronyms, abbreviations, and technical terms.

Steve Karpiak

References

Berntssen, M.H.G., A. Aatland, and R.D. Handy. 2003. Chronic dietary mercury exposure causes oxidative stress, brain lesions, and altered behavior in Atlantic salmon (*Salmo salar*) parr. *Aquatic Toxicology*. (65) 1. 55-72.

Casco Bay Estuary Partnership. 1996. rev. 2005. *Casco Bay Plan*. (http://www.cascobay.usm.maine.edu/CBUpdate.html) (October 24,2006).

Casco Bay Estuary Partnership. 2005. *State of the Bay* (http://www.cascobay.usm.maine.edu/SOTB.html) (May 16, 2006).

DeGuise, S., S.D. Shaw, *et al.* Consensus Statement: Atlantic Coast Contaminants Workshop 2000. *Environmental Health Perspectives.* 109 (12): 1301-1302. (http://www.ehponline.org/docs/2001/109p1301-1302deguise/EHP109p1301PDF.PDF) (December 19, 2006).

Doggett, L. Maine Department of Environmental Protection. May 10, 2005. Personal Communication.

Hawes, E.L. 1993. *Historic Sources of Pollution in Portland Harbor, 1840-1970 Including the Fore River, the Back Cove and South Portland Watersheds*. Casco Bay Estuary Partnership.

Maine Center for Disease Control and Prevention. 2006. *Environmental and Occupational Health Program: Fish and Game Guidelines*. (http://www.maine.gov/dhhs/eohp/fish/) (June 1, 2006).

Maine Department of Environmental Protection. *2004 Integrated Water Quality Monitoring and Assessment Report* (http://www.maine.gov/dep/blwq/docmonitoring/305b/index.htm#2004) (May 16, 2006).

Maine Department of Environmental Protection. 2005. *Draft Maine Air Toxics Priority List and Basis Statement.* (http://www.maine.gov/dep/air/toxics/mati-docs.htm) (October 7, 2005).

Maine Department of Environmental Protection. 2006. *Air Toxics An Overview*. (http://www.maine.gov/dep/air/toxics/overview.htm) (May 18, 2006).

McElroy, A.E., J.W. Farrington, and J.M. Teal. 1989. Bioavailability of Polycyclic Aromatic Hydrocarbons in the Aquatic Environment. In: *Metabolism of Polycyclic Aromatic Hydrocarbons* (U. Varanasi, ed.) CRC Press. Boca Raton, FL. pp. 1-39.

NASA Visible Earth. 2006. (http://veimages.gsfc.nasa.gov/4331/Sea_2002340.jpg) (Dec. 5, 2006).

National Research Council of the National Academies. 2006. *Health Risks from Dioxin and Related Compounds: Evaluation of the EPA Reassessment.* National Academies Press.

National Science and Technology Council Committee on Environment and Natural Resources. Air Quality Research Subcommittee. 1999. *The Role of Monitoring Networks in the Management of the Nation's Air Quality.* (http://esrl.noaa.gov/csd/AQRS/reports/monitoring.pdf) (December 19, 2006).

Nicolas, J-M. 1999. Vitellogenesis in fish and the effects of polycyclic aromatic hydrocarbon contaminants. *Aquatic Toxicology.* (45) 2-3. pp. 77-90.

O'Connor, J.M. and R. J. Huggett. 1988. Aquatic pollution problems, North Atlantic Coast, including Chesapeake Bay. *Aquatic Toxicology*. (11): 1-2. pp 163-190.

Shaw, S.D. and S. De Guise. 2000. Endocrine Disruptors in the Marine Environment: Impacts on Marine Wildlife and Human Health. *Proceedings of the Atlantic Coast Contaminants Workshop 2000*. Scientific Report of the Marine Environmental Research Institute: 192 pp.

Tanabe S., N. Kannan, N. Subramanian, S. Watanabe, and R. Tatsukawa. 987. Highly toxic coplanar PCBs: occurrence, source, persistency and toxic implications to wildlife and humans. *Environmental Pollution*. 47 (2); 147-163.

United States Environmental Protection Agency. 2005. *The Inventory of Sources and Environmental Releases of Dioxin-Like Compounds in the United States: The Year 2000 Update* (External Review Draft, March 2005; EPA/600/p-03/002A) (http://www.epa.gov/ncea/pdfs/dioxin/2k-update/) (June 1, 2006).

United States Environmental Protection Agency. 2006. *Water Discharge Permits (PCS)*. (http://www.epa.gov/enviro/html/pcs/pcs_query.html) (June 1, 2006).

Van Loveren, H., P.S. Ross, A.D.M.E. Osterhaus, and J.G. Vos. 2000. Contaminant-induced immunosuppression and mass mortalities among harbor seals. *Toxicology Letters*. pp. 112-113, 319-324.

2 Is atmospheric deposition a major contributor of PAHs and mercury to the Bay?

Background

The atmosphere serves as a source of toxic chemicals when particulate and gaseous pollutants released into the air are transferred to land and water surfaces through wet processes (such as precipitation and fog), and dry processes (via vapor or particles). Deposition to water bodies can be indirect (via runoff from the land) or directly to the water surface. Studies undertaken by the CBEP in 1991 and 1994 indicate that levels of heavy metals and organic pollutants are elevated above the normal background level throughout the Bay, including areas distant from point sources (see Chapter 4). In addition, elevated levels of methyl mercury have been detected in freshwater fish from water bodies throughout the state (see Chapters 6 and 8). This widespread distribution of toxic chemicals suggests that atmospheric deposition plays a major role in the delivery of toxic chemicals to the watershed and directly to the Bay. CBEP began a field monitoring program in 1998 to assess the magnitude of the atmospheric contribution of two important toxics: mercury and PAHs.

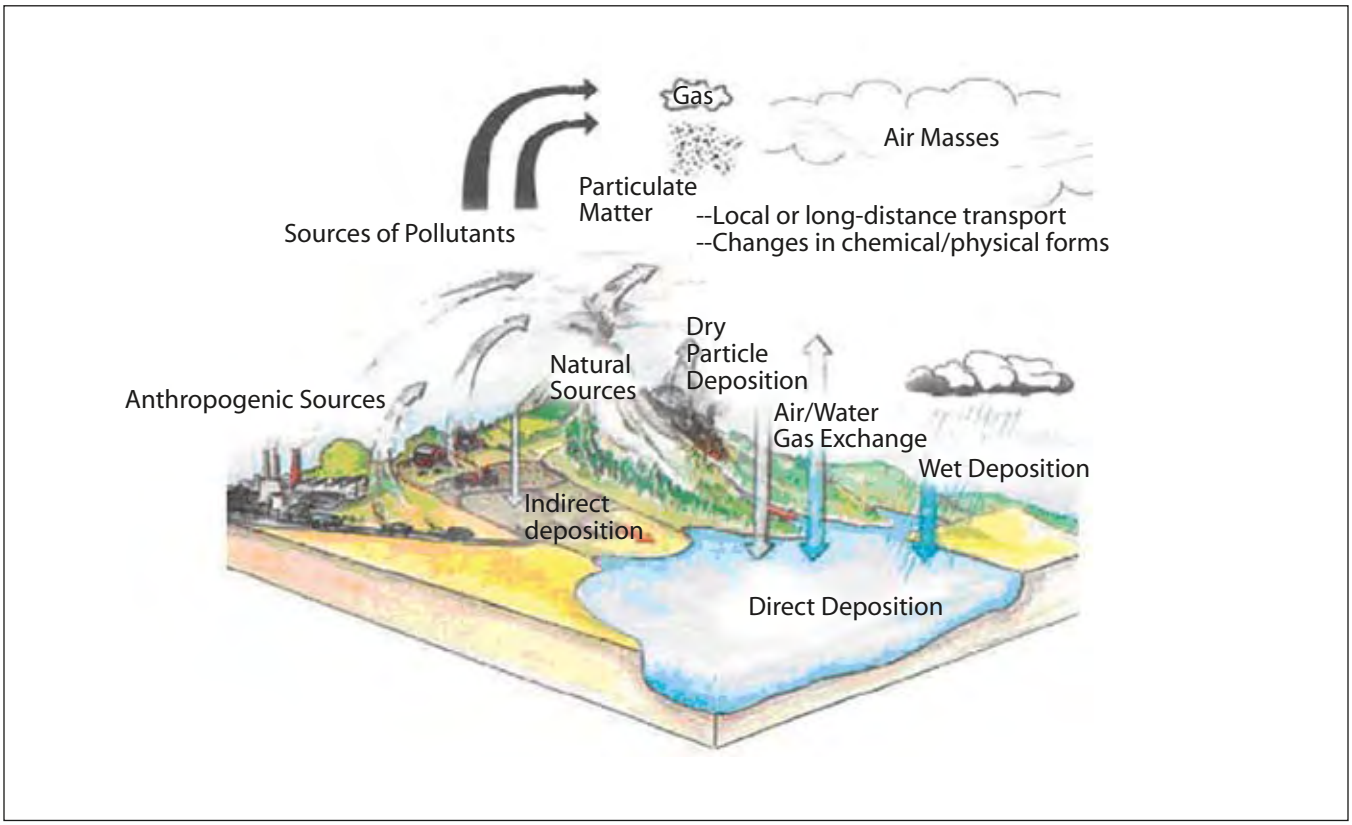


Figure 2-1. *Diagram of the sources, transport, and deposition of pollutants via the atmosphere (US EPA 2002).*

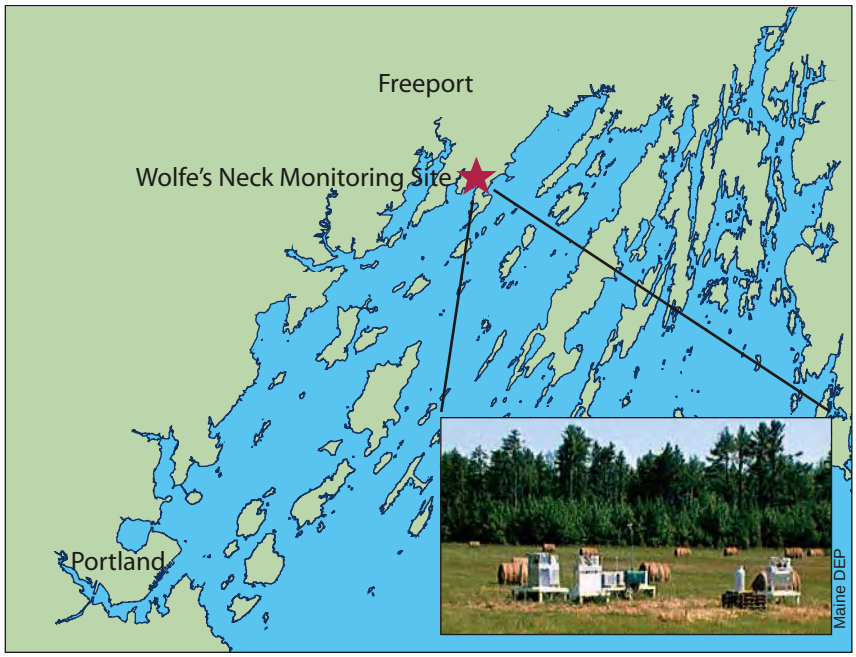


Figure 2-2. *Casco Bay Monitoring Site at Freeport*

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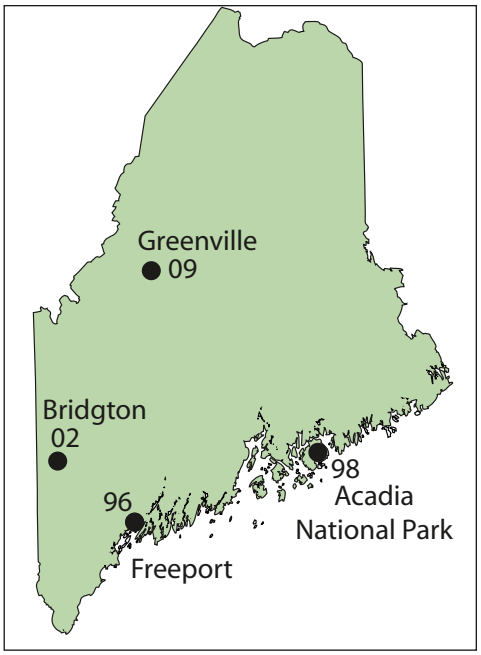


Figure 2-3. *There are four Mercury Deposition Network Sampling sites in Maine. ME96 is the Wolfe's Neck site in Freeport. ME02, also located in the Casco Bay watershed, is an inland site in Bridgton. ME 98 is another coastal site, located in Acadia National Park. ME09 is in Greenville.*

Mercury Deposition

Sources of mercury to the atmosphere include combustion of coal, oil, wood or natural gas, incineration of mercury-containing garbage, and industrial processes. Funded by a grant from US EPA, an atmospheric deposition monitoring station was established at Wolfe Neck Farm on the coast in Freeport, Maine (see Figure 2-2). A Mercury Deposition Network (MDN) sampler collected weekly samples of wet deposition (total precipitation and pollutant concentrations in the precipitation) of mercury from 1998-2001. Following the conclusion of the CBEP-funded study, DEP has continued data collection at the Freeport site. Data from other MDN sampling sites in Maine including Bridgton, which is an inland site located at the headwaters of the Casco Bay watershed, Greenville, and another coastal site in Acadia National Park is available for comparison to the Freeport data (see Figure 2-3).

The results of the mercury sampling were analyzed to determine whether atmospheric deposition is a significant source of mercury entering Casco Bay. Also, the study looked at how coastal Maine fits into the larger pattern of regional atmospheric deposition of mercury and whether there are annual or seasonal trends in wet deposition.

Wet deposition was determined by multiplying the weekly amount of precipitation collected at a site by the corresponding weekly average wet concentration of mercury. Annual deposition was calculated by summing the calculated weekly wet deposition amounts for that year. Dry deposition was inferred from pollutant concentrations in the ambient air or by assuming a ratio of dry deposition to wet deposition. For this study, 229 square miles was used for the surface area of Casco Bay and 985 square miles for the entire watershed surface area. Estimating wet and dry deposition to the Casco Bay watershed, based on the measurements available, can be highly uncertain. Contributing to the uncertainty in wet and dry deposition estimates are a number of issues, including:

- Uncertainty in the fraction of the toxic material deposited on water bodies and land surfaces in the Casco Bay watershed that ultimately reaches the Bay; and
- Year-to-year meteorological variability, which contributes to variability in annual deposition of metals and PAHs.

Results of the Mercury Monitoring

- Atmospheric deposition of mercury is the dominant source of mercury to Casco Bay when compared to loading from major point sources (see Figure 2-4).
- Mercury concentrations and deposition were generally higher in the spring and summer at Casco Bay. Snow and rain remove different fractions of air pollutants from the atmosphere and rain typically has higher concentrations of mercury.
- Large storm events can be a significant source of mercury deposition. One major storm during the period June 9-16, 1998 accounted for 21% of the total wet deposition for the year.
- Long-term monitoring data is critical in the assessment of trends in mercury deposition due to interannual variations in precipitation (see Figure 2-5).
- Wet deposition of mercury directly to the Bay surface area accounts for 10.5 to 16.4 lbs/yr. Estimates of dry deposition of mercury totaled 4.2 to 16.4 lbs/yr. (see Table 2-1). Total deposition from the atmosphere may be 85 to 92% of overall mercury loading directly to the Bay (Ryan *et al.* 2003). This estimate does not include the nonpoint source contribution of mercury to the Bay from runoff into rivers and streams that enter the Bay.

Table 2-1. Estimated Mercury discharges in the Casco Bay area.

Transport Process	Water Surface		Watershed Surface	
	Discharges (lb/yr)	% of Total	Discharges (lb/yr)	% of Total
Wet deposition	10.5-16.4	61-46	45.0–70.4	69-49
Dry deposition	4.2-16.4	24-46	18.0-70.4	27-49
Wastewater plants	2.55	15-8	2.55	4-2
Total	17.2-35.4	100	65.5-143	100

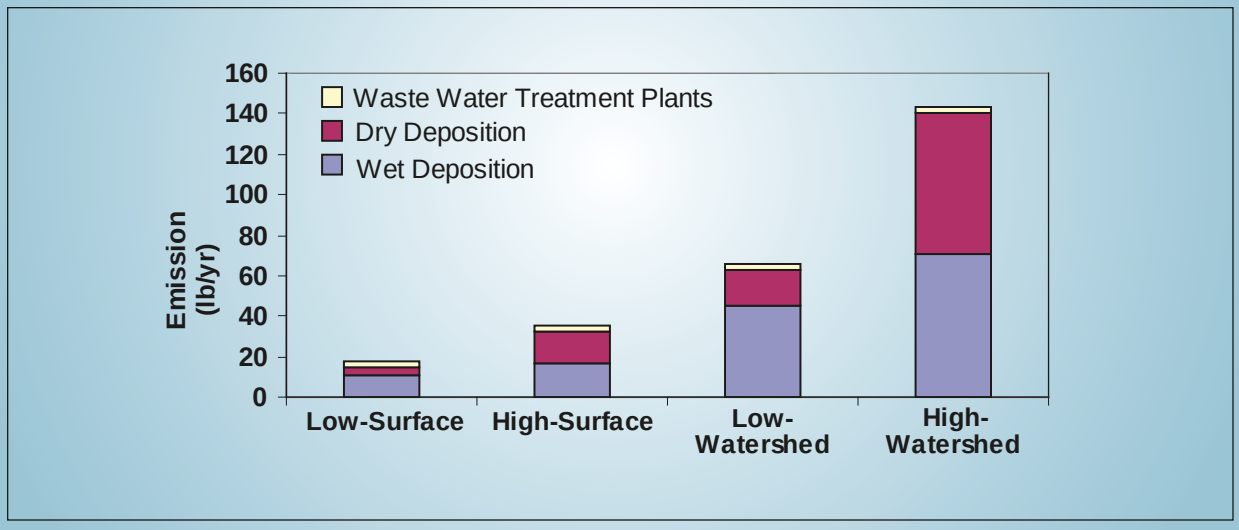


Figure 2-4. *Summary of wastewater treatment plant direct mercury discharges and dry and wet deposition of mercury to Casco Bay (in lb/yr). "Low" and "high" signify ranges in dry deposition estimates. "Surface" refers to the surface of Casco Bay while "watershed" refers to the entire watershed surface area (Ryan* et al. *2003). Low refers to the lowest estimated value, high to the highest estimated value.*

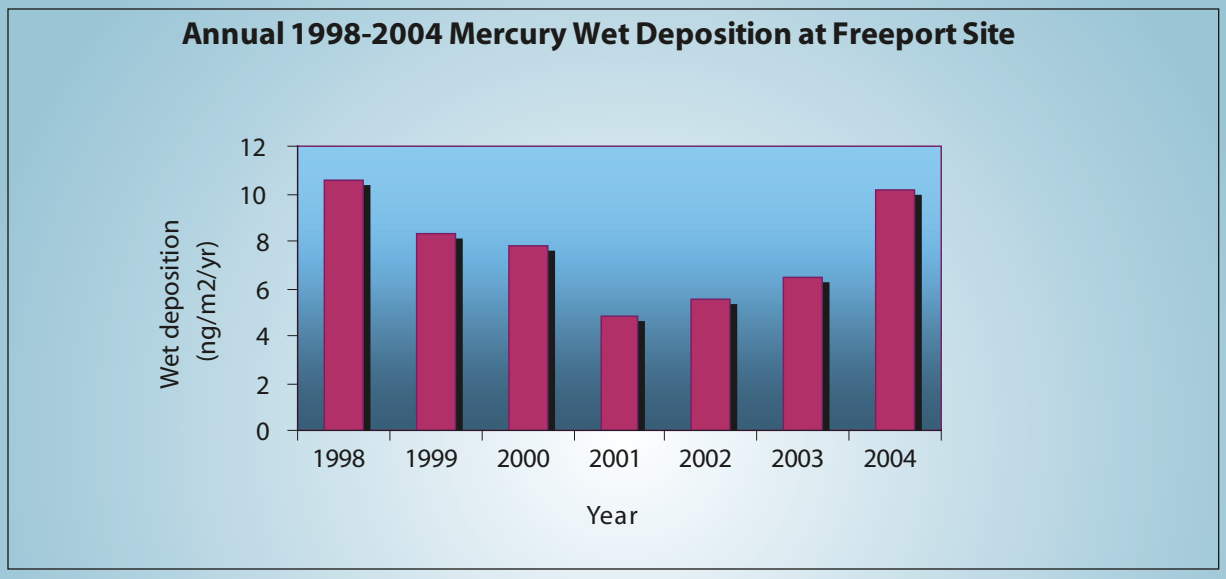


Figure 2-5. Assessing trends over time. *From 1998 to 2001, there appears to be a general trend of decreasing concentration of mercury deposited at the Freeport site. This apparent trend is the direct result of decreasing amounts of rainfall over the sampling period. In fact, the results of subsequent mercury monitoring conducted by Maine DEP in Freeport in 2002-2004 show an increasing amount of mercury deposition over that three-year period (Ryan* et al. *2003, Vanarsdale 2005). Clearly, long-term monitoring data is critical in the assessment of trends due to interannual variations in precipitation.*

Regional Mercury Air Pollution Patterns

Studying regional patterns of air pollution helps us to understand what is happening in Casco Bay. For example:

- Long-range transport of pollution in the Bay appears to be an important source of mercury. Wind trajectory analyses (studies of the movement of air masses) and source apportionment (studies that quantitatively identify the relative contributions of different source types to ambient air pollutant concentrations) indicate that polluted air masses from other regions (*e.g.,* coal-fired power plants) influence the air quality of the Casco Bay area. Local sources, such as vehicle emissions and industrial smokestacks, also likely contribute to pollution loading in the Bay.

- In many of the years sampled, the Freeport site in Casco Bay had the highest rate of mercury deposition of the four sites in Maine. Coastal sites tend to receive more rainfall, contributing to the higher rates of wet deposition of mercury at the Freeport and Acadia sites, which are on the coast (see Figure 2-6). There may also be local coastal sources of mercury contributing significantly to coastal wet deposition.

- Within Maine, annual wet deposition rates of mercury were similar to or slightly higher than those reported in nearby states (see Figure 2-7). If precipitation is uniform, then similar levels of wet deposition indicate similar levels of air emissions (lb/acre) in each state, implying that Maine is neither a source nor a sink for mercury (Ryan *et al.* 2003).

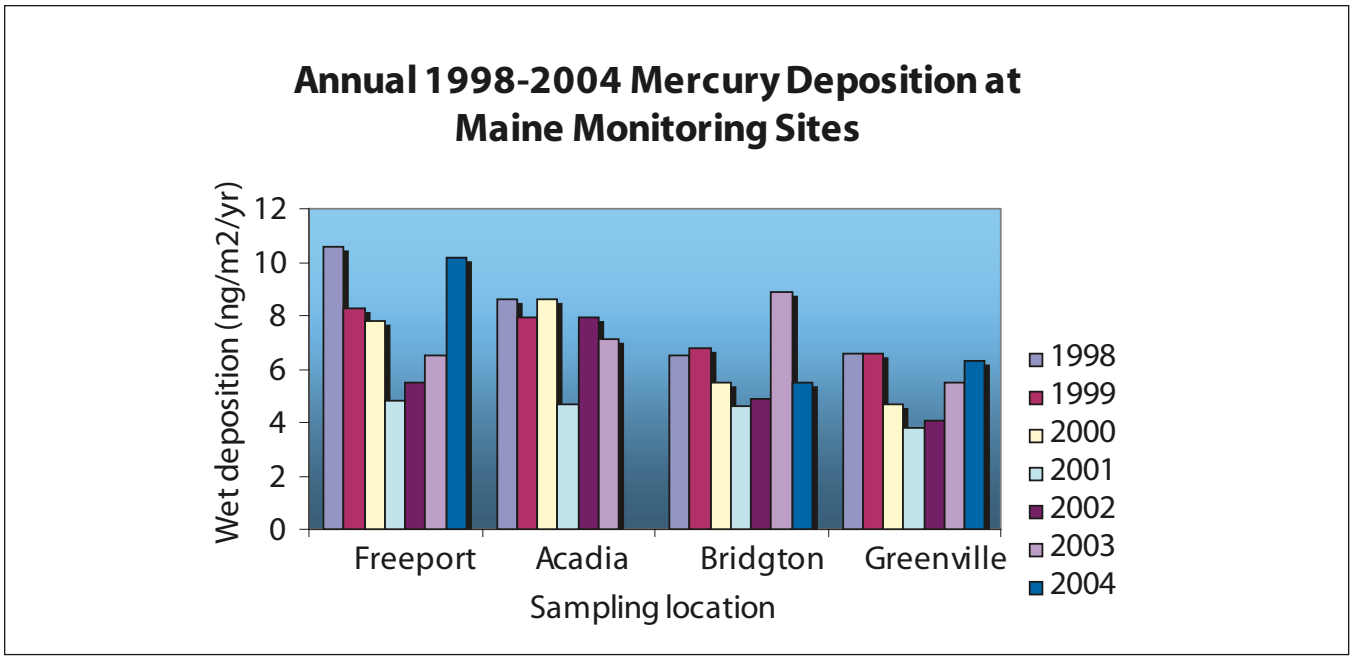


Figure 2-6. *Coastal sites tend to receive more rainfall, contributing to the higher rates of wet deposition of mercury seen at the Freeport and Acadia sites in many of the years during the sampling period.*

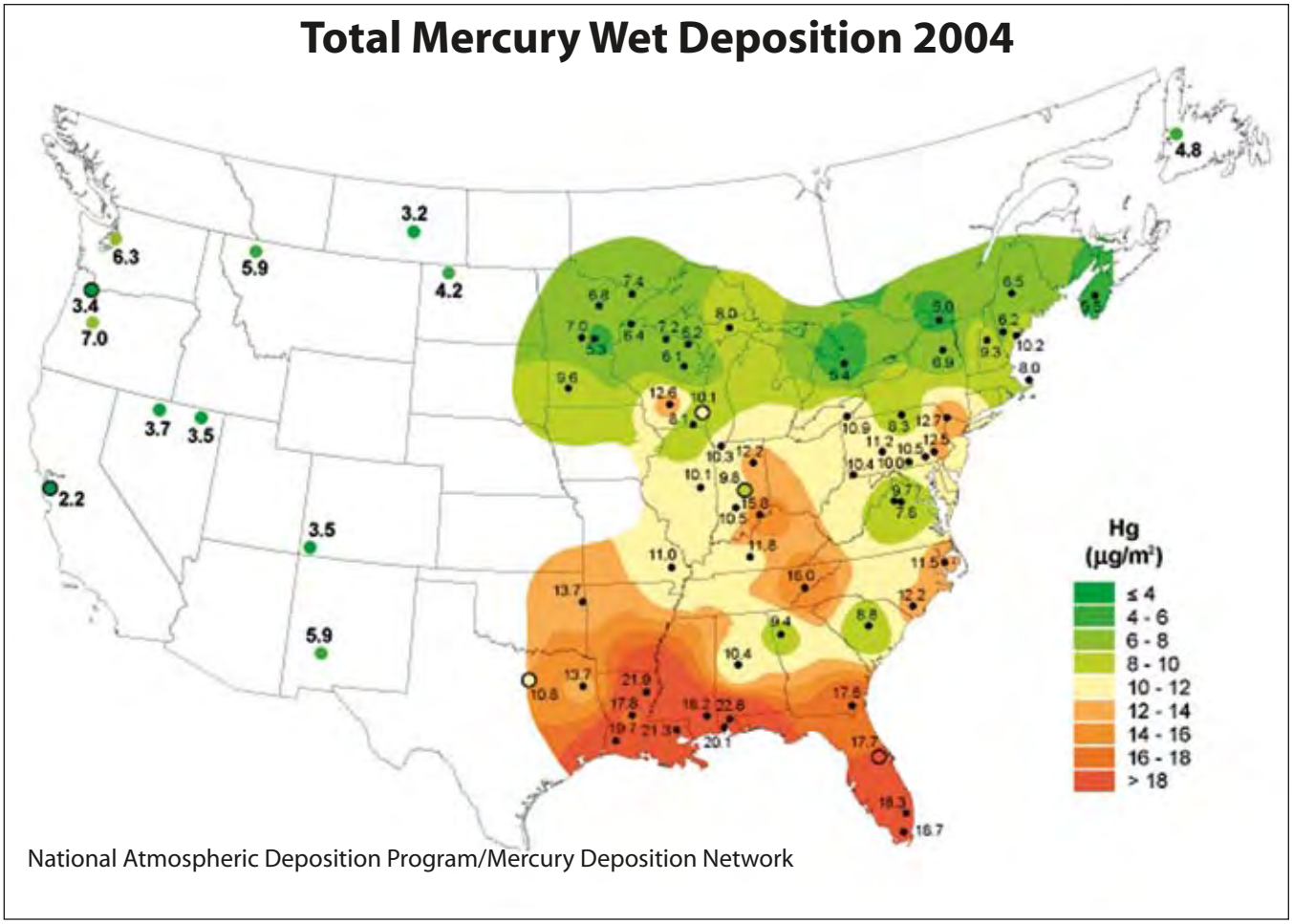


Figure 2-7. *Total Mercury Wet Deposition 2004 for Eastern USA (NADP/MDN 2006)*

Trace Metal Analysis

In 2002 a trace metal sampling train was added to the mercury (MDN) sampler at the Freeport site. With assistance from DEP and a grant from US EPA, weekly integrated wet deposition samples were collected for the trace metals selenium, arsenic, cadmium, chromium, copper, manganese, magnesium, nickel, lead and zinc. Trace metals found in wet deposition samples are useful as markers for different emission sources and can be used to verify changes in pollutant loading. For example:

- Antimony is found in flame retardants and indicates waste incineration as a source.
- Selenium/arsenic/zinc are indicators of coal combustion as a source.
- Vanadium/nickel are indicators of oil combustion as a source.
- Beryllium is an indicator of coal combustion as a source.
- Cadmium is an indicator of incineration as a source.
- Manganese is an indicator of cement/steel production as a source.

Good correlations between metals indicate similar sources or source regions. Analysis of trace metal data collected from 2003 to 2004 indicated that there was a good correlation among zinc, lead, cadmium, and chromium, and among the metals zinc, lead, arsenic and selenium. Wind trajectory analysis showed that concentrations of arsenic, selenium, mercury, cadmium, chromium and magnesium were highest when the wind was from the west. Concentrations of copper and zinc were lowest when the wind was from the south or southeast, suggesting that these metals are from different sources (Wu *et al*. 2006). CBEP is undertaking a follow up study on this and conducting further analyses of the trace metals data to help identify sources of the metals.

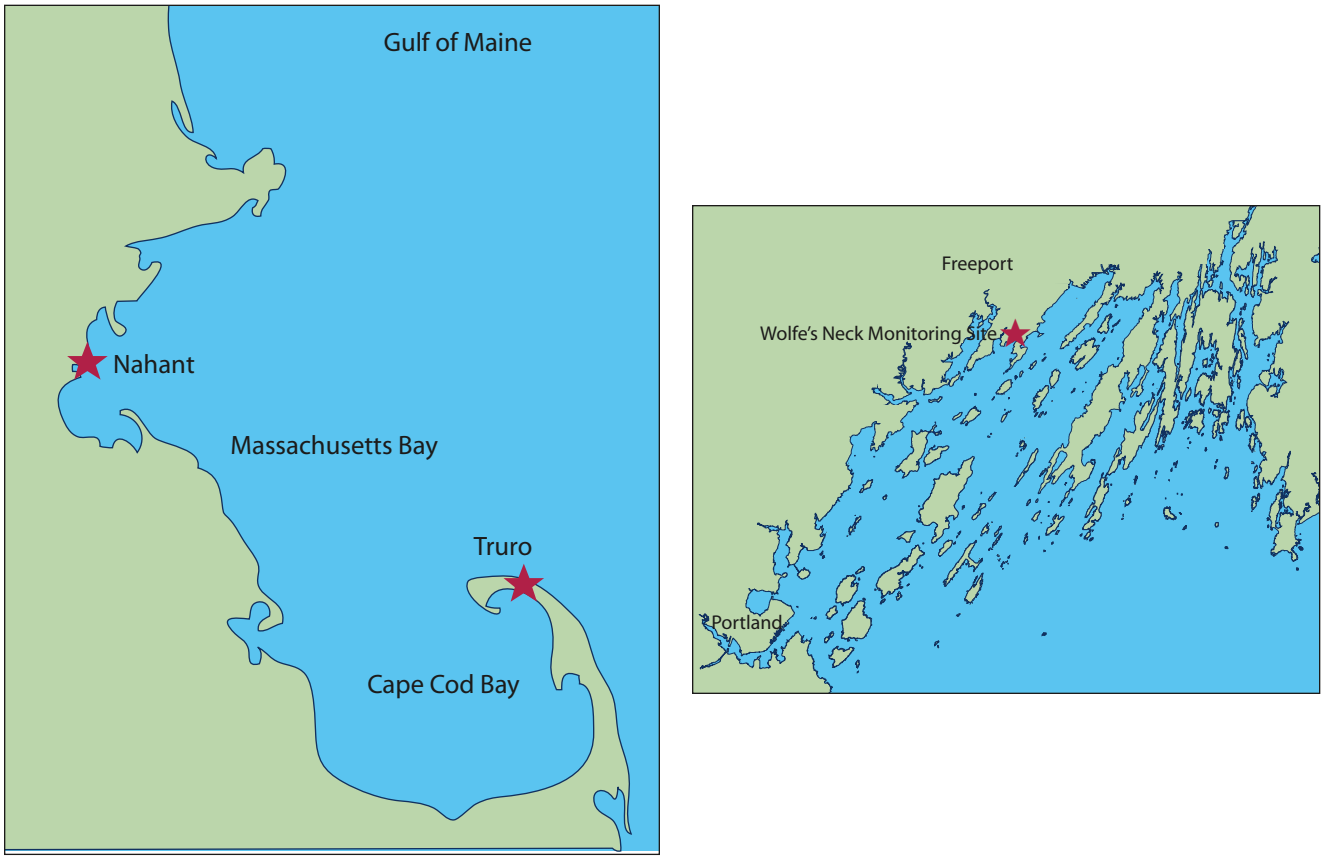


Figure 2-8. *Location of PAH monitoring sites in Massachusetts Bay and Casco Bay (Golomb 2001b).*

Polycyclic Aromatic Hydrocarbons (PAHs)

PAHs, the most common toxic pollutants found in the sediments of the Bay, enter the atmosphere primarily from the incomplete combustion of fossil fuels such as coal, oil and natural gas, and from wood burning. Airborne PAHs in the dry phase (aerosols or gases) are short-lived, surviving only tens of hours at most. Thus, in dry air, PAHs are deposited close to emission sources. When carried by raindrops and snow, PAHs can survive up to hundreds of hours and can travel thousands of miles from distant industrial sources (Mackay *et al*. 1992).

Table 2-2. PAHs measured in the Golomb et al. 2001b study

Acenaphthelene
Fluorene
Phenanthrene
Anthracene
Fluoroanthene
Pyrene
Benzo(a)anthracene
Chrysene
Benzo(b)fluoroanthene
Benzo(k)fluoroanthene
Benzo(a)pyrene
Perylene
Indeno(1,2,3-c,d)pyrene
Dibenz(a,h)anthracene
Benzo(g,h,i)perylene
Coronene

In 1998, CBEP funded a study of the wet and dry atmospheric contribution of 16 types of PAHs (see Table 2-2). The research was conducted at Wolfe's Neck in Freeport by Dr. Dan Golomb and associates from the University of Massachusetts/Lowell. Between March 1998 and February 2000, 41 dry deposition and 32 wet deposition samples were collected at the Wolfe Neck, Freeport site. The monitoring study estimated that the annual wet deposition of the 16 measured PAHs was 91 $\mu g/m^2$/yr. The Freeport data were compared to data from monitoring stations at Nahant and Truro on Massachusetts Bay (see Figure 2-8). The lower estimated wet deposition of 78.5 $\mu g/m^2$/yr at Nahant suggests that upper air trajectories and precipitating clouds carry somewhat more wet deposition to Freeport (Golomb *et al*. 2001a). The dominant species of PAHs reaching Freeport via wet deposition were phenanthrene (32.3%), fluorene (14.2%) and fluoranthene (9.1%). The composition of species reaching the Nahant site was quite similar, suggesting that the origin of the PAHs arriving at these two sites via wet deposition is similar (Golomb *et al*. 2001b).

Major sources of dry atmospheric deposition of PAHs are jet exhaust, gasoline fueled vehicles, diesel fueled vehicles, wood combustion and others in that order (Golomb *et al*. 2001b). Dry deposition varies from week to week, with greater deposition during the heating season. The estimated dry deposition at Freeport was 81.5 $\mu g/m^2$/yr, far less than the 832 $\mu g/m^2$/yr measured in Nahant. This suggests that dry deposition is due to local emission sources, which are far fewer and more distant around Wolfe's Neck than around Nahant, which is located about 10 kilometers from Logan International Airport, close to Boston and several industrial suburbs. Wolfe's Neck is about 30 km from the metropolitan Portland area and Portland International Airport, and there are no industrial suburbs in the vicinity of the monitoring site (Golomb *et al.* 2001b). The dominant PAH species deposited via dry deposition in Freeport were fluoroanthene (22%), pyrene (17.9%), benzo (b and k) fluoroanthene (11.5%) and acenaphthylene (11.5%). The dominant species deposited in Nahant were benzo (b and k) fluoroanthene (14.1%), fluoranthene (13.8 %), phenanthrene (12.7%), anthracene (11.5%), and pyrene (10.4%). These differences further support the idea that different local sources are involved (Golomb *et al*. 2001b).

Using the results of the weekly wet and dry deposition sampling in Freeport, the total direct annual atmospheric input of PAHs to the surface of Casco Bay was calculated to be 64 kg PAHs/yr. While data is not available on the contribution of PAHs from other nonpoint sources, atmospheric PAHs have been estimated to represent 30-56% of total input of PAHs to the estuary (Richardson *et al*., 2003)

Summary/Conclusions

The Casco Bay atmospheric deposition studies indicate that the atmosphere is the major contributor of mercury and the likely source of 30% or more of the PAHs that enter the coastal ecosystem. Pollutants can be deposited from nearby sources or can travel from other regions of the country via wind and precipitation. Further studies will be necessary to assess the locations of the major sources contributing toxic pollutants to the Bay and its watershed via the atmosphere. See Chapter 9 for a discussion of efforts to reduce local sources of atmospheric loading of toxic chemicals.

References

Golomb, D., E. Barry, G. Fisher, M. Koleda, P. Varanusupakul. University of Massachusetts Lowell. 2001a. *Atmospheric Deposition of Polycyclic Aromatic Hydrocarbons at Casco Bay.* Casco Bay Estuary Partnership.

Golomb, D., E. Barry, G. Fisher, P. Varanusupakul, M. Koleda, T. Rooney. 2001b. Atmospheric deposition of polycyclic aromatic hydrocarbons near New England coastal waters, *Atmospheric Environment,* (35) 6245-6258. (http://www.cascobay.usm.maine.edu/pdfs/Golumb) (December 20, 2006).

Kennicutt, M.C., T.L. Wade, B.J. Presley. 1992. Texas A&M University. *Assessment of Sediment Contamination in Casco Bay.* Casco Bay Estuary Partnership.

Mackay, D., Shiu, W-Y, Ma, K-C 1992. *Illustrated Handbook of Physical-Chemical Properties and Environmental Fate of Organic Chemicals.* Lewis Publishers. Boca Raton, FL.

National Atmospheric Deposition Program/Mercury Deposition Network. 2006. MDN Concentration and Deposition Maps. (http://nadp.sws.uiuc.edu/mdn/maps/) (June 6, 2006).

Richardson, C., D. Saball, A. Johnson, D. Wright, D. Gould, A. Vanarsdale, J. Weiss, B. Bayley-Smith, E. Doering. 2003. *Estimating Estuarine Pollutant Loading from Atmospheric Deposition Using Casco Bay, Maine as a Case Study.* Casco Bay Estuary Partnership. (http://www.cascobay.usm.maine.edu/pdfs/estimate.pdf) (December 20, 2006).

Ryan, P.A., H.R. Hafner, S.G. Brown. 2003. *Deposition of Air Pollutants to Casco Bay.* Casco Bay Estuary Partnership. (http://www.cascobay.usm.maine.edu/pdfs/SONOMA.pdf) (December 20, 2006).

United States Environmental Protection Agency. 2001. *Frequently Asked Questions About Atmospheric Deposition: A Handbook for Managers*. EPA-453/R-01-009. (http://www.epa.gov/oar/oaqps/gr8water/handbook/airdep_sept_2.pdf) (September 7, 2006).

Vanarsdale, A. 2005. United States Environmental Protection Agency. Personal Communication.

Wu, J., S. Brown, H. Hafner. 2006. *Casco Bay Trace Metal Validation and Preliminary Analysis.* Casco Bay Estuary Partnership.

How do oil spills impact Casco Bay?

Maine DMR

Oil spreading up the Fore River from the Julie N *oil tanker spill in September, 1996.*

Background

Spilled oil threatens many types of coastal habitat areas, including sheltered beaches where there is little wave action to disperse spilled oil, tidal flats where oil may seep into the muddy sediments, and salt marsh areas where oil may damage sensitive root systems. Animals and plants can be impacted by direct physical contact with the oil. For example, filter-feeding shellfish and bird eggs can be smothered by oil. The feathers of birds or the fur of seals lose their insulating properties when coated with oil, leading to the danger of death from cold. Birds can also drown when their feathers become matted with oil. Oil can destroy food resources, directly killing prey species and also tainting the way they taste and smell and making them unacceptable as food. If ingested, oil can damage the digestive system. Oil vapors have the potential to damage the nervous system of animals, as well as damaging their lungs and liver.

USF&WS

Birds can drown when their feathers are matted with oil after a serious spill.

The more volatile components of oil may evaporate rapidly, leaving the heavier components of crude oil, such as PAHs, to persist longer in the environment. These persistent toxic chemicals have the potential to cause more subtle, long-term effects such as reproductive problems in birds (US EPA 1999). Benthic invertebrates exposed long-term to elevated levels of PAHs in the sediments may experience impacts including inhibited reproduction and death (US EPA 2005). For fish, exposure to polluted sediments containing multiple toxins including PAHs can result in cancerous lesions, fin erosion, liver abnormalities, reproductive problems, cataracts and suppression of the immune system (Fabacher *et al.* 1991; Weeks and Warinner 1984, 1986; O'Conner and Huggett 1988; Nicolas 1999).

Factors that Affect the Severity of Oil Spills

Polycyclic aromatic hydrocarbons (PAHs), are the most widespead toxic pollutants in Casco Bay (CBEP 1996). They are found in fossil fuels and can enter the Bay directly when oil is spilled into marine waters. Oil, whether in the form of crude, unrefined oil, or fuel oil tends to spread horizontally into a slick on the surface of water. The surface tension, specific gravity, and viscosity of the particular type of oil spilled affect the ability of the oil to spread.

- Surface tension refers to the degree of attraction between the surface molecules in a liquid. This attraction is decreased by heat so oil spreads more rapidly in warmer weather.
- Specific gravity refers to the density of a substance in comparison to water. Since oil has a lower specific gravity than water, it floats in the surface, where it can be spread by wind and currents. As the lighter components of oil evaporate, leaving the heavier substances, oils and tars may sink and coat rocks and sediments on the bottom.
- Viscosity refers to the thickness or resistance to flow of a liquid. The more viscous or thicker the oil, the less likely it is to spread (US EPA 1999).

Weathering of Spilled Oil

The severity of an oil spill is also affected by natural environmental processes (weathering).These chemical, physical, and biological processes are illustrated in figure 3-1 (Zhu *et al*. 2001). In addition to spreading oil over the water surface, which is influenced by viscosity and surface tension as discussed above, weathering includes processes of dispersion, emulsification, evaporation, photooxidation and biodegradation.

- **Dispersion**: When the water column is agitated, oil can break into droplets that are dispersed throughout the water column (US EPA 1999). Also, interaction of the oil with fine (micron-sized) particles on the surface can reduce its adhesion to sediments or rocks, resulting in the formation of oil droplets that disperse into the water column (Owens 1999).
- **Emulsification**: Waves can further disperse oil droplets into an emulsion, a thick, sticky mixture of water trapped in viscous oil that can linger in the environment for years (US EPA 1999).
- **Evaporation** is the most significant weathering process right after a spill occurs, removing the volatile substances in the oil mixture. For crude oil, this can include 20-50% of the oil spilled. For Number 2 fuel oil, the volatile components may be about 75% of the oil mixture. Gasoline and kerosene are made up 100% of volatile components.
- **Photooxidation** occurs when sunlight transforms complex high molecular weight petroleum compounds into simpler compounds which are more soluble in water and potentially more available to vulnerable biological organisms (Zhu *et al.* 2001).
- **Biodegradation**: The process of biodegradation of petroleum occurs when microorganisms consume the hydrocarbons in oil as food, a process that is enhanced by warmer water temperatures (US EPA 1999). When oil is spilled onto or washed onto the beach, it can be biodegraded or can enter the sediment through adsorption to soil particles. There it may migrate through the sediments and/or eventually be released.

Also, oil that is spread up onto a beach may be buried under the sand during the next tidal cycle, then subsequently uncovered and released into the ocean.

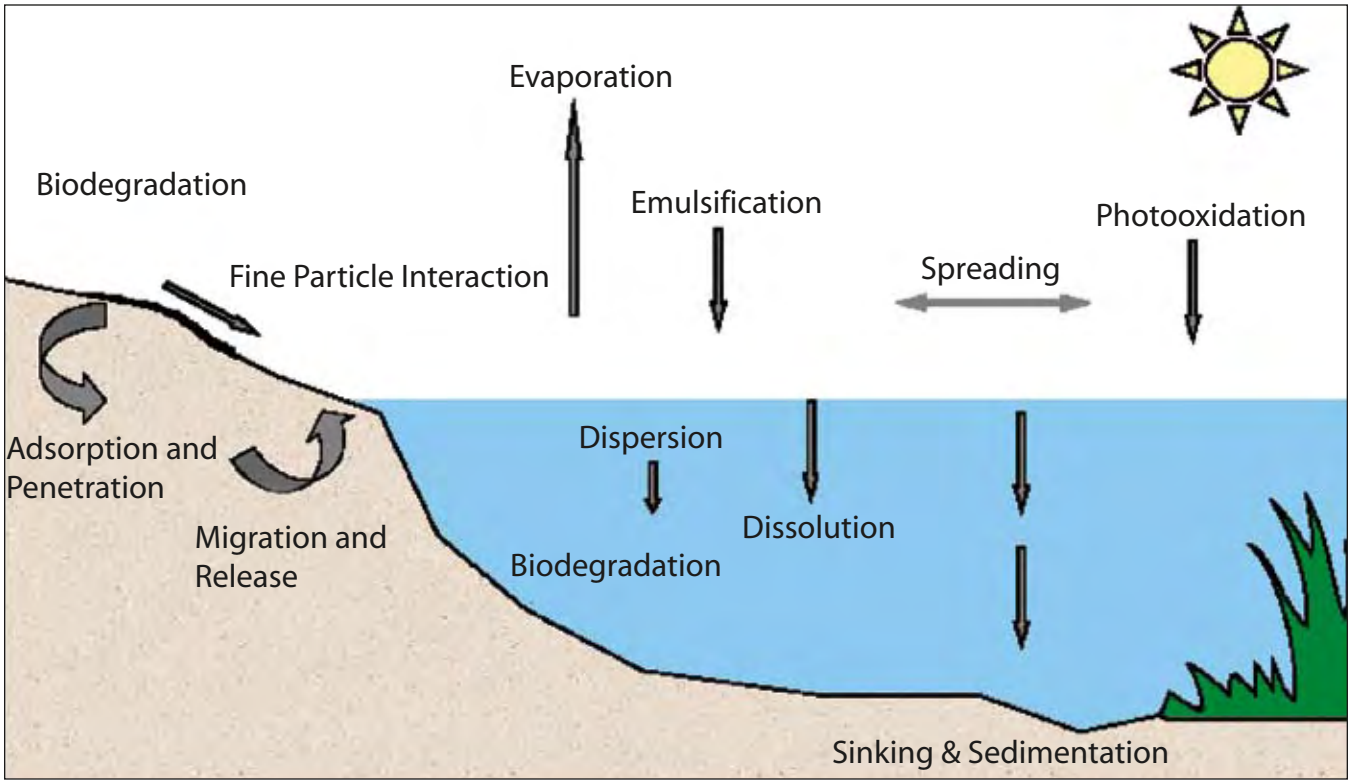


Figure 3-1 *Major weathering processes after an oil spill (Zhu* et al., *2001).*

Oil Spills: Small and Major

There are typically about 70 fuel spills to surface waters reported each year in Maine, averaging 20 gallons per spill (CBEP 1996). While the cumulative effect of many small spills is damaging, major spills can have both immediate and devastating impacts as well as leaving a legacy of toxics in the sediments and in the tissues of animals that inhabit them. The *Exxon Valdez* spill in 1989 captured public attention in Maine and throughout the world when 11 million gallons of crude oil poured into Alaska's Prince William Sound, killing thousands of seabirds and marine mammals (Zhu 2001).

The *Julie N* Oil Spill

Eight years after the *Exxon Valdez* disaster, Portland experienced a much smaller but still dramatic spill. On September 27, 1996, the oil tanker *Julie N*, heavily laden with 200,000 barrels of fuel oil, struck the south side of the former Million Dollar Bridge (now called the Casco Bay Bridge) linking Portland Harbor and South Portland. A total of 179,634 gallons of heavy fuel oil and Number 2 diesel oil spilled into the water. The oil was carried by winds and tides into the upper Fore River and Stroudwater Marsh area, including Long Creek. While 78% of the oil was recovered through containment and cleanup efforts, it is estimated that over 38,000 gallons remained in the environment. While flushing and hot water washing could be used to clean some areas, approximately 8 miles of marsh were coated with oil.

It was determined that the least destructive approach for the sensitive marsh environment was to leave the marshes to slowly recover naturally, through burial in the sediments, evaporation and breakdown by bacteria, photooxidation, and wave action (Maine DEP 2006).

Damage Assessment

Assessment of the damage caused by the *Julie N* spill was undertaken on behalf of the State of Maine and the National Oceanic and Atmospheric Administration's Natural Resource Damage Assessment Program. The studies included an investigation of the impacts to marine vegetation, animal communities, sediments, birds and water quality as well as loss of human uses of the resources.

Oil "fingerprinting" was used to identify the presence of PAHs from spilled *Julie N* fuel in the water and sediments of the Fore River. The same technique was used to correlate PAHs found in the tissues of marine organisms with the fuel from the *Julie N*. The studies showed that Julie N fuel-derived PAHs had accumulated in the flesh of lobsters and soft-shelled clams in the Fore River, and in scallops from Eastern Point (Portland) to Cape Elizabeth. The highest body burdens were found in blue mussels collected in the Fore River, where total PAH concentrations were 10 to 30 times higher than in mussels sampled in 1994, prior to the spill. Over 1600 birds were soiled by *Julie N* oil (Maine DEP 1998).

Tom Jewell

In 1996 the Julie N *oil tanker, pictured above, spilled 179,634 gallons of fuel oil into the Fore River after striking the former Million Dollar Bridge while entering the harbor.*

Human uses of the marine resources were also impacted by the spill. For example, temporary closure of Portland Harbor to vessel traffic resulted in loss of revenue from sport fishing, whale watching, tour boats and ferries. Harvesting of marine fish and shellfish was closed or restricted by the Maine Department of Marine Resources in portions of Casco Bay and the Fore River from the day of the spill until the fishery was finally fully reopened in mid-November, 1996.

Legal Settlement

Ultimately, the *Julie N* legal settlement under the federal Damage Assessment and Restoration Program generated $1,000,000 to lessen the overall impact of the spill on the ecology of the Bay (Mauseth and Csulak 2003). The funds were used to reduce the discharges of oil and grease into the Fore River area, to enhance habitat in Scarborough Marsh for bird species impacted by the spill, and to protect land used for marine bird nesting (DEP 2006). In addition, funds were used to create a trail along the Fore River. Opened on the fifth anniversary of the spill, the scenic trail includes interpretive signs that describe the ecosystem and the impacts of the spill to this fragile area.

Portland Trails

Trail along the Fore River paid for with Julie N *settlement funds includes interpretive signs that describe the ecosystem and impacts of the spill (Portland Trails website www.trails.org) .*

Other Recent Spills in Casco Bay

Smaller spills happen several times each year in Casco Bay. For example, on April 7, 2003, a tank truck spilled 8,000–10,000 gallons of jet fuel, much of which reached the intertidal salt marshes of Pleasantdale Cove in the Fore River estuary. Fortunately, long-term damage to the marsh was limited by the highly volatile nature of jet fuel, which largely evaporated in the days following the spill (Maine DEP 2003). The potential exists for a spill of millions of gallons of oil, far more serious than the *Julie N* spill. Each year, more than 100 oil tankers offload oil in Portland, Maine. The tanker *Braer*, which was an occasional visitor to Portland in the early 1990's, ran aground off the coast of Scotland and spilled 25 million gallons of fuel into marine waters in 1993 (http://www.cascobay.com/environ/responder.htm). This was one of the largest spills in history (Rowland 2000).

Maine DEP

One of the Maine DEP's oil recovery barges is the Netepenawesit *(the Indian translation is "He Who Watches"). The barge has its tanks loaded with water and its JBF 500 skimming system deployed.*

Limiting the Impact of Oil Spills

Good marine vessel management can prevent spills. If spills do happen, containment and cleanup are key approaches. The State of Maine Marine Oil Spill Contingency Plan (Maine DEP 1997) includes roles and responsibilities, cleanup strategies, and wildlife rehabilitation approaches. Cleanup techniques include the containment of spilled product, use of mechanical recovery methods such as oil skimming vessels and skimming units, and use of absorbent materials such as absorbent boom and absorbent pads. When specific authorization is given, additional cleanup alternatives such as the use of dispersants and in-situ burning can be done. In 2002, the State installed permanent moorings for the attachment of oil containment booms in order to rapidly block off the Fore River and protect its sensitive marshes from a future spill. In addition, the State maintains two 210,000 gallon (or 5,000 barrel) oil recovery barges ready for deployment. The barges are shallow draft allowing them to operate in areas close to shore, such as the Fore River. One barge is moored in South Portland (the *Aucocisco*) and the other barge is moored in Bucksport (the *Netepenawesit*). Each barge includes a JBF 500 dynamic inclined plain skimming system. This system makes each of the barges a complete clean-up unit capable of skimming oil and pumping it directly into the barge's storage tanks.

The privately owned 208 foot *Marine Responder*, stationed permanently in Portland Harbor, is one of the world's largest and most sophisticated oil cleanup vessels. This 12 million dollar ship is on call to minimize the impact of spills in the New England area. After traveling to the site of a large spill, the ship sends out a smaller workboat which tows a 400 foot boom from the rear deck of the *Responder*. The boom forms a J-shaped loop to contain the oil while a skimmer pumps the oil into holding tanks on the ship. Each tank can hold 42,000 gallons of oil. Oil from the tanks is then pumped to barges, which carry to oil to shore for disposal (*Casco Bay Online* 2006).

Preparing for Another Spill

Both contingency planning for response to oil spills and damage assessment following a spill require a clear understanding of the environmentally valuable and vulnerable areas along the coast. This is especially true for Casco Bay, which has the largest volume of oil transport in New England. Resources currently available to provide this background information for oil spill response and assessment activities include:

- **Coastal Waterbird Surveys**: Coastal waterbird surveys conducted during the 1980's were used to help determine the number of birds impacted by the *Julie N* oil spill in the Fore River in 1996. As a result, the settlement for damage relating to this spill was aimed at helping to increase the waterbird population in Casco Bay. Aerial waterbird surveys conducted in Casco Bay in 2000 by the Maine Department of Inland Fisheries and Wildlife and the U.S. Fish and Wildlife Service, with the assistance of funding from the Casco Bay Estuary Partnership, will help to guide response efforts in the event of a future spill. For example, knowing the locations of bird habitat areas can help guide efforts to install booms to avoid oiling, to haze birds from a threatened site, or to avoid using bird colony locations as staging areas during oil cleanup.

- **Fringing Marsh Assessments**: Casco Bay is fringed by many small areas of intertidal salt marsh which are highly vulnerable in the event of an oil spill. These marshes serve as important habitat for invertebrates and fish. Juvenile marine species such winter flounder and hake use the marsh habitat, as do migratory species such as eels and alewife, and transient species like Atlantic herring and striped bass. Recently, nine fringing salt marshes along Casco Bay were studied by scientists from the University of New England and Wells National Estuarine Research Reserve. The study assessed the value of these small marshes to fish, invertebrate and plant production, and well as their value as buffers against sea level rise and coastal erosion. The results of the study confirmed that these small marshes play an important role in the Bay's food web and in maintaining a diverse assemblage of plant and animal species in Casco Bay (Morgan *et al.* 2005). The data gathered will improve our baseline knowledge for assessment of natural resource damage in these fragile areas in the event of a future spill (Maine DEP 2004).

- **Environmental Vulnerability Index Map**: Maine Department of Environmental Protection has developed an "Environmental Vulnerability Index Map" as a tool to guide oil spill contingency planning and response. The map (see Figure 3-2) illustrates important coastal resources that could be adversely impacted by a spill. These include the fringing marshes and bird habitat areas described in the sections above, as well as fish runs, shellfish beds, threatened and endangered species habitat, marine worm and eelgrass areas, and important human resources (*e.g.,* aquaculture lease sites, lobster dealers, and conservation lands). In the event of a future oil spill, containment and cleanup efforts will be targeted at vulnerable resource areas identified in the map.

Summary/Conclusions

In the short-term, spilled oil can threaten the survival of coastal birds and other organisms directly impacted by the oil itself. Longer term, the toxic PAHs in spilled fuel can linger in the environment, leading to wildlife health impacts including reproductive problems, tumors, and suppression of the immune system. While spills both small and large can and sometimes do happen, coastal managers are planning for rapid response and have developed tools to limit environmental damage in the event of another major spill in the Bay.

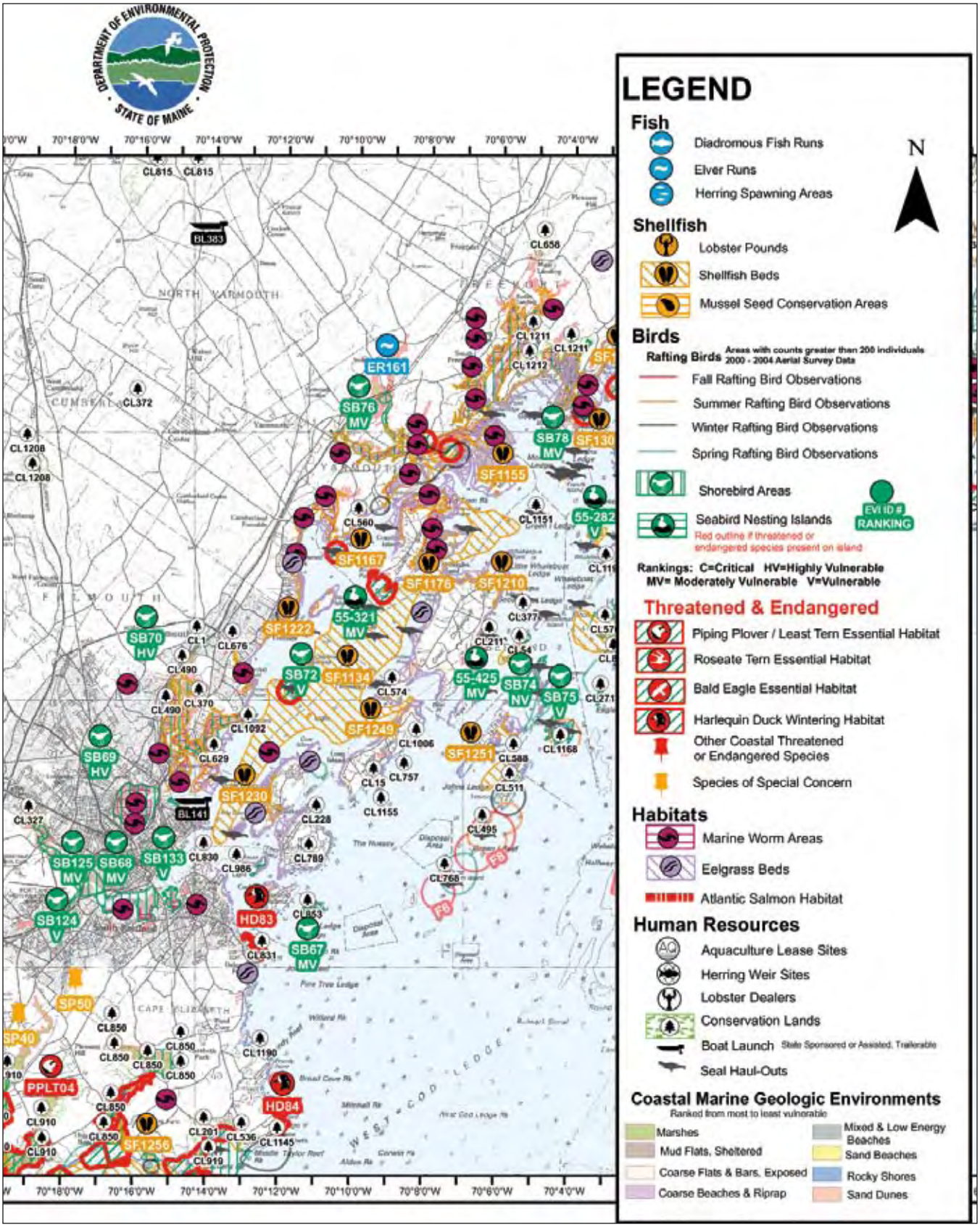


Figure 3-2: *A portion of the Maine DEP Environmental Vulnerability Index Map showing coastal resources at risk from marine oil spills, focusing on the Portland area. Not all resources in any specific area are shown. These maps are intended to provide information solely for marine spill contingency planning (Maine DEP 2006).*

References

Casco Bay Estuary Project. 1996. *Casco Bay Plan.*

Casco Bay Online. 2006. *An Inside Look at the Maine Responder*. (www.cascobay.com/environ/responder.htm) (May 8, 2006).

Fabacher, D.L., J.M. Besser, C.J. Schmitt, J.C. Harshbarger, P.H. Peterman, and J.A. Lebo. 1991. Contaminated sediments from tributaries of the Great Lakes: chemical characterization and cancer-causing effects in medaka (*Oryzias latipes*). *Arch. Environ. Contam. Toxic.* 20:17-35.

Maine Department of Environmental Protection. 1997. *Marine Oil Spill Contingency Plan.* (http://mainegov-images.informe.org/dep/rwm/publications/pdf/contplan.pdf) (March 8, 2006).

Maine Department of Environmental Protection. 1998. Julie N *Preassessment Data Report*. Prepared for Maine Department of Environmental Protection, Maine Department of Conservation, Inland Fish and Wildlife, Department of Marine Fisheries, National Oceanic and Atmospheric Administration, United States Department of the Interior. Industrial Economics, Inc.

Maine Department of Environmental Protection. 2003. *Assessment of the Effects of the South Portland Four-Star Fuel Spill on the Salt Marshes in Pleasantdale Cove.* P-263-2003. Normandeau Associates.

Maine Department of Environmental Protection. 2004. *Integrated Water Quality Monitoring and Assessment Report.*

Maine Department of Environmental Protection 2006. *After the Oil Spill: Natural Resources Damage Assessment Restoration Plans for the 1996* Julie N *Oil Spill.* (http://www.Maine.gov/dep/rwm/JulieN/story2.htm) (February 14, 2006).

Mauseth, G.S. and F.G. Csulak. 2003. *Damage assessment and restoration following the* Julie N *oil spill: A case study.* Proceedings of the International Oil Spill Conference, Vancouver, B.C., April 2003.

Morgan P.A., M. Dionne, R. MacKenzie, and L. Curci. 2005. *Ecological functions and values of fringing salt marshes susceptible to oil spills in Casco Bay, Maine.* Prepared for Maine Oil Spill Advisory Council. Maine Sea Grant, Orono, Maine.

Nicolas, J.-M. 1999. Vitellogenesis in fish and the effects of polycyclic aromatic hydrocarbon contaminants. *Aquatic Toxicology*. 45:2-3. pp. 77-90.

O'Conner, J.M. and R.J. Huggett. 1988. Aquatic pollution problems, North Atlantic coast, including Chesapeake Bay. *Aquatic Toxicology*. 11:163-190.

Owens, E.H. 1999. The interaction of fine particles with stranded oil. *Pure Appl. Chem*. 71 (1): 83-93.

Rowland, H. (Ed). 2000. *International Oil Spill Statistics 1999*, NY: Aspen Law & Business. New York.

United States Environmental Protection Agency. 1999. *Understanding Oil Spills and Oil Spill Response*. EPA 540-K-99-007.

United States Environmental Protection Agency. 2005. *Information on the Toxic Effects of Various Chemicals and Groups of Chemicals.* (http://www.epa.gov/R5Super/ecology/html/toxprofiles.htm#pahs) (February 8, 2006).

Weeks, B.A. and J.E. Warinner. 1984. Effects of toxic chemicals on macrophage phagocytosis in two estuarine fishes. *Mar. Environ. Res.* 14:327-35.

Weeks, B.A. and J.E. Warinner. 1986. Functional evaluation of macrophages in fish from a polluted estuary. *Vet. Immun. Immunopathol.* 12:313-20.

Zhu, X., A.D. Venosa, M.T. Suidan, and K. Lee. 2001. *Guidelines for the Bioremediation of Marine Shorelines and Freshwater Wetlands*. United States Environmental Protection Agency. (http://www.epa.gov/oilspill/pdfs/bioremed.pdf) (February 14, 2006).

4 What are the levels of toxic chemicals in the sediments of Casco Bay?

Steve Karpiak

Healthy bottom communities have a diverse assemblage of organisms. This healthy shallow water soft bottom benthic community includes eelgrass and multiple species of mollusks and small crustaceans. An impacted community would include fewer species and a predominance of pollution tolerant organisms.

Background

When scientists first analyzed the surface layer of bottom sediments in Casco Bay in 1980, they were surprised to find a wide array of toxic contaminants present, including organic chemicals and heavy metals. These chemicals found their way to the Bay via multiple pathways, including rivers, stormwater runoff, point sources (*e.g.,* outfall pipes), small and large oil spills, and atmospheric deposition. Once in the aquatic environment, many toxic chemicals are hydrophobic (*i.e.,* they do not readily dissolve in water) and can become attached to sediment particles. Unless transported away by currents, the contaminated particles settle to the bottom and remain in the sediments where they may break down chemically over time or become buried under newer layers of sediment. Even when clean sediments are deposited on top of contaminated sediments, dredging and biological activity (such as burrowing and deposit feeding) can bring the contaminants back to the surface.

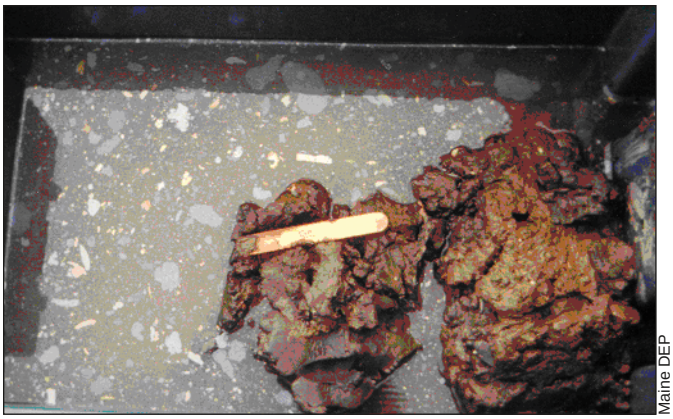
Bottom-dwelling (benthic) animals that are exposed to contaminated sediments can suffer adverse effects. These benthic organisms play an important role in the food chain, recycling organic matter and serving as a food source for groundfish (*e.g.,* flounder, cod, and haddock), lobsters and crabs. By ingesting benthic organisms that live and feed on contaminated sediments, fish and large crustaceans may experience inhibited growth and reproduction, disease vulnerability and even death (EPA 2006). Humans who consume seafood contaminated by toxic chemicals can also potentially be at risk. For example, the presence of dioxins in Casco Bay, largely a byproduct of pulp and paper mills, has resulted in elevated dioxin concentrations in the liver (tomalley) of lobsters (see Chapter 8). Toxic contamination can have an impact at the ecosystem level as well. Highly polluted areas experience shifts in the density and composition of the benthic animal community, with fewer species present and a predominance of hardy, pollution tolerant organisms.

Table 4-1: Analytes Measured During Both the CBEP 1991-1994 and 2001-2002 Sediment Studies

(Wade and Sweet 2005)

PAHs	PCB Congeners (varying configurations of chemical structure)	Pesticides	Trace Metals	Butyltins	Dioxins/ furans	Planar PCBs
9-naphthalene	2,4'-Dichlorobiphenyl (congener 8)	Aldrin	Silver	TBT (tributyltin)	TCDF	PCB 77
1-Methylnaphthelene	2,2',5-Trichlorobiphenyl (congener 18)	Alpha-chlordane	Arsenic	DBT (dibutyltin)	2,3,7,8-TCDF	PCB 126
2,6-Dimehtylnaphthalene	2,4,4'-Trichlorobiphenyl (congener 28)	2,4'-DDT	Cadmium	MBT (monobutyltin)	1,2,3,7,8-PeCDF	PCB 169
2,3,5-Trimethylnaphthalene	2,2',3,5'-Tetrachlorobiphenyl (congener 44)	4,4'-DDT	Chromium	Total butyltin	2,3,4,7,8-PeCDF	
Acenaphthylene	2,2',5,5'-Tetrachlorobiphenyl (congener 52)	2,4'-DDE	Copper		1,2,3,4,7,8-HxCDF	
Acenaphthene	2,3',4,4'-Tetrachlorobiphenyl (congener 66)	4,4'-DDE	Mercury		1,2,3,6,7,8-HxCDF	
Biphenyl	3,3",4,4'-Tetrachlorobiphenyl (congener 77)	2,4'-DDD	Nickel		2,3,4,6,7,8-HxCDF	
Fluorene	2,2',4,5,5'-Pentachlorobiphenyl (congener 101)	4,4'-DDD	Lead		1,2,3,7,8,9-HxCDF	
Anthracene	2,3,3',4,4'-Pentachlorobiphenyl (congener 105)	Dieldrin	Selenium		1,2,3,4,6,7,8,-HpCDF	
1-Methylphenanthrene	2,3',4,4',5-Pentachlorobiphenyl (congener 118)	Endosulfan I	Zinc		1,2,3,4,7,8,9-HpCDF	
Dibenzothiophene	3,3',4,4',5-Pentachlorobiphenyl (congener 126)	Endosulfan II	Iron		OCDF	
Fluoranthene	2,2',3,3',4,4'-Hexachlorobiphenyl (congener 128)	Endosulfan sulfate			2,3,7,8-TCDD	
Pyrene	2,2',3,4,4',5'-Hexachlorobiphenyl (congener 138)	Endrin			1,2,3,7,8-PeCDD	
Benzo[a]anthracene	2,2',4,4',5,5'-Hexachlorobiphenyl (congener 153)	Hexachlorobenzene			1,2,3,4,7,8-HxCDD	
Chrysene	2,2',3,3',4,4',5-Heptachlorobiphenyl (congener 170)	Heptachlor			1,2,3,6,7,8-HxCDD	
Benzo[b]fluoroanthene	2,2',3,4,4',5,5'-Heptachlorobiphenyl (congener 180)	Heptachlor epoxide			1,2,3,7,8,9-HxCDD	
Benzo[k]fluoroanthene	2,2',3,4',5,5',6-Heptachlorobiphenyl (congener 187)	Lindane			1,2,3,4,6,7,8-HpCDD	
Benzo[a]pyrene	2,2',3,3',4,4',5,6-Octochlorobiphenyl (congener 195)	Mirex			OCDD	
Indeno[1,2,3-cd]pyrene	2,2',3,3',4,4',5,5',6-Nonachlorobiphenyl (congener 206	Trans-nonchlor				
Dibenz[a,h]anthracene	2,2,3,3',4,4',5,5',6,6'-Decachlorobiphenyl (congener 209)	Toxaphene				
Benzo[g,h,i]perylene	Total PCBs	Total pesticides				

Monitoring the Sediments in the Bay


Maine DEP

Sediment pollution can result from past industrial activities. At the site of a former coal gas works plant, which operated in Portland for almost a century, coal tar can still be seen oozing into the Fore River estuary. Remediation is underway at the site (Doggett 2006).

With the publication of *Troubled Waters—A Report on the Environmental Health of Casco Bay* (Hauge 1988), there was a growing awareness that toxic contaminant levels were elevated in the Bay's sediments. Concern over these toxic pollutants and their impacts on the health of the Bay's ecosystem was the impetus for the Maine DEP and the Governor of Maine to submit a nomination package to the US EPA's National Estuary Program in 1989. In 1990, the Casco Bay Estuary Project, now the Casco Bay Estuary Partnership (CBEP), was established, receiving significant federal and state funding. One of the first major studies undertaken by the CBEP was the 1991 baseline assessment of sediment contamination levels at 65 sites in the Bay. The study used state-of-the art analytical and statistical methods. Sampling sites selected were intended to provide good areal coverage of the Bay, to assess sediments of different ages and textures (including erosional features), and to provide a good representation of various bottom communities (Kennicutt *et al*. 1994). The site selection also considered water depth, circulation patterns and historical data, *i.e.*, areas where there was a known "dirty history" such as industrial facilities and point discharges (see Figure 4-1).

Sampling site locations were designated as either Cape Small (CS), East Bay (ES), IB (Inner Bay), Outer Bay (OB), Shallow Water (SW), or West Bay (WB) (see figure 4-1). Undisturbed surface samples were collected using either grab samplers or by hand and were analyzed for heavy metals, polycyclic aromatic hydrocarbons (PAHs), aliphatic hydrocarbons (hydrocarbons lacking a benzene ring, such as plant-derived waxes), polychlorinated biphenyls (PCBs) and pesticides (Kennicutt *et al.* 1992). In 1994, 28 of the original sites and 5 new sites were analyzed for butyltins (organometallic compounds), dioxins and furans, and planar PCBs, the most toxic PCB conformation (*i.e.,* spatial arrangement of atoms and bonds) (Wade *et al.* 1995). See Table 4-1 for a list of analytes measured as part of the studies.

Results of the 1991 and 1994 Sediment Sampling Studies

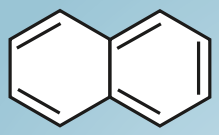
The results of the 1991 and 1994 sediment studies indicated that the most widespread contaminants in the Bay are petroleum and its byproducts, especially PAHs derived from high-temperature combustion processes. Geographically, the contaminants are found in highest concentration near sources such as the mouths of rivers, highly populated areas, and point source outfalls. Some regional differences are also explained by sediment accumulation patterns (Kennicutt *et al*. 1994).

The following data is summarized from Kennicutt *et al*. 1992 and Kennicutt *et al*. 1994:

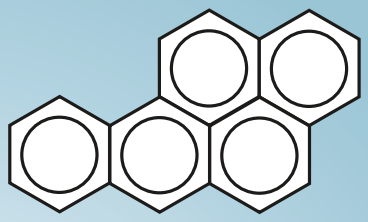
- **PAHs:** PAHs were found at all the sites sampled in the Bay. The predominant PAHs were highly condensed ring structures indicating a pyrogenic (combustion) source associated with urbanized and industrialized locations. High molecular weight four-ring and larger PAHs made up over 60% of the PAHs in the Casco Bay sediments (see Box on p. 34). The Inner Bay had the highest level of PAHs, especially sediments from the Fore River and Portland area, where levels of PAHs were high and comparable to other contaminated estuaries (see Figures 4-2a, 4-3a, and 4-4a) (Macauley *et al*. 1994, USEPA 1997).

  A statistical technique called principle components analysis was used to assess the regional influences of various sources of contaminants (weathered petroleum, fresh diesel fuel, pyrogenic hydrocarbons (from combustion), and biogenic material (such as natural plant waxes of land-based and aquatic origin). Not surprisingly, Inner Bay and shallow water sites nearest to Portland were characterized by higher inputs of low molecular weight PAHs from weathered petroleum than other parts of the Bay, probably from stormwater runoff and point sources associated with urban activities (see Figure 4-3a).

- **PCBs:** Total PCBs, based on the sum of 20 PCB congeners (varying configurations of chemical structure) were highly elevated in the Inner Bay near Portland. Generally the lowest values were in Cape Small and West Bay (see Figure 4-5a).
- **Pesticides:** Total DDT (DDT plus its breakdown products DDD and DDE) were highest in the Inner Bay near Portland and lowest in Cape Small and West Bay. The pesticide chlordane was highest at the Inner Bay sites and the lowest at West Bay and Cape Small. The other organic pesticides (see the list in Table 4-1) were near or below the detection limit of the analytical method [0.25 ng/g or ppb (parts per billion)]. None of the pesticides was highly elevated. (See Figure 4-6a)
- **Trace Metals:** Trace metals are naturally found in sediments. To correct for the natural background level of metals, all samples were normalized to iron. The distribution of metals is strongly influenced by the grain size of the sediments, which was also measured as part of this study. Trace metal levels were generally highest in the Inner Bay. While some of the cadmium, lead, silver, zinc, and mercury values detected in the Casco Bay sediments were elevated above background, likely by human activities, few of the samples were highly elevated above background (See Figure 4-7a).



Naphthalene, an example of a low molecular weight PAH, has two benzene rings.



Benzo[a]pyrene, an example of a high molecular weight PAH

Low and High Molecular Weight PAHs in Casco Bay

PAHs are environmentally persistent organic compounds that are strongly held to solid particles, both suspended in the water and in bottom sediments. Chronic exposure to PAHs can result in cancer and other serious health impacts. In the aquatic environment, PAHs are easily mobilized into the base of the food web by benthic organisms. The toxicity of PAHs tends to increase with increased molecular weight in aquatic systems (US EPA 2006, Eisler 1987). PAHs are often divided into two categories in the aquatic environment—the less toxic, less persistent low molecular weight PAHs and the more toxic, more persistent high molecular weight PAHs.

Low molecular weight PAHs are typically derived from weathered petroleum (biodegraded oil) and diesel fuel that enter the Bay via fuel spills or urban runoff (see Chapter 3). Examples are naphthalene and acenaphthene. Generally the solubility of PAHs decreases with increasing molecular weight. When in the marine environment, PAHs tend to stick to solid particles and settle into the sediments.

High molecular weight PAHs have a highly condensed molecular ring structure (4 rings or larger) that indicates a pyrogenic (combustion) source associated with urbanized and industrialized locations. These PAHs may come from particles in car exhaust, municipal and industrial combustion sites, and coal tar, and may be carried to the Bay via stormwater runoff and atmospheric deposition (see Chapter 2). They include, for example, Benzo[a]pyrene, $C_{20}H_{12}$, a five-ring polycyclic aromatic hydrocarbon that is mutagenic and highly carcinogenic. Benzo[a]pyrene is a product of incomplete combustion and is found, for example, in vehicle exhaust fumes (especially from diesel engines).

The following data is summarized from Wade *et al*. 1995:

- **Butyltins, dioxins and furans, and planar PCBs:** Butyltins, dioxins and furans, and planar PCBs (the most toxic conformation of PCBs) were found throughout Casco Bay. In general, conentrations were in the low range compared to similar estuarine areas, with the highest concentrations near likely sources of contamination. The Inner Bay had the highest concentrations of these contaminants, due to inputs from the Fore and Presumpscot Rivers. Tributyltin (TBT) and its breakdown products dibutyltin (DBT) and monobutyltin (MBT) were at the highest concentrations near marinas and other areas where boats concentrate, since they primarily come from marine anti-fouling paints (see Figure 4-8a). Dioxins and furans and especially 2,3,7,8-TCDD (tetrachlorodibenzodioxin), a potent toxic dioxin, were found in highest concentrations 10 miles downstream of the paper mill in Westbrook. Elevated dioxin/furan concentrations were also noted in East Bay, possibly due to transport from the Androscoggin River or local combustion sources (Wade *et al*. 1995) (see Figure 4-9a). For planar PCBs, the spatial distribution was similar to that measured in 1991 for total PCBs, with the highest levels in the Inner Bay and the lowest in West Bay and Cape Small. In general, total planar PCBs increased with increasing concentration of dioxin and furan. Falmouth Foreside had the highest concentration of planar PCB (see Figure 4-10a).

Average PAH Compositions in Sediments by Region Within Casco Bay Based on 1991 Sampling

The map shows the sampling sites in each of the designated sections of the Bay: Cape Small, East Bay, Inner Bay, Outer Bay, Shallow Water, and West Bay.
(Kennicut *et. al* 1992)

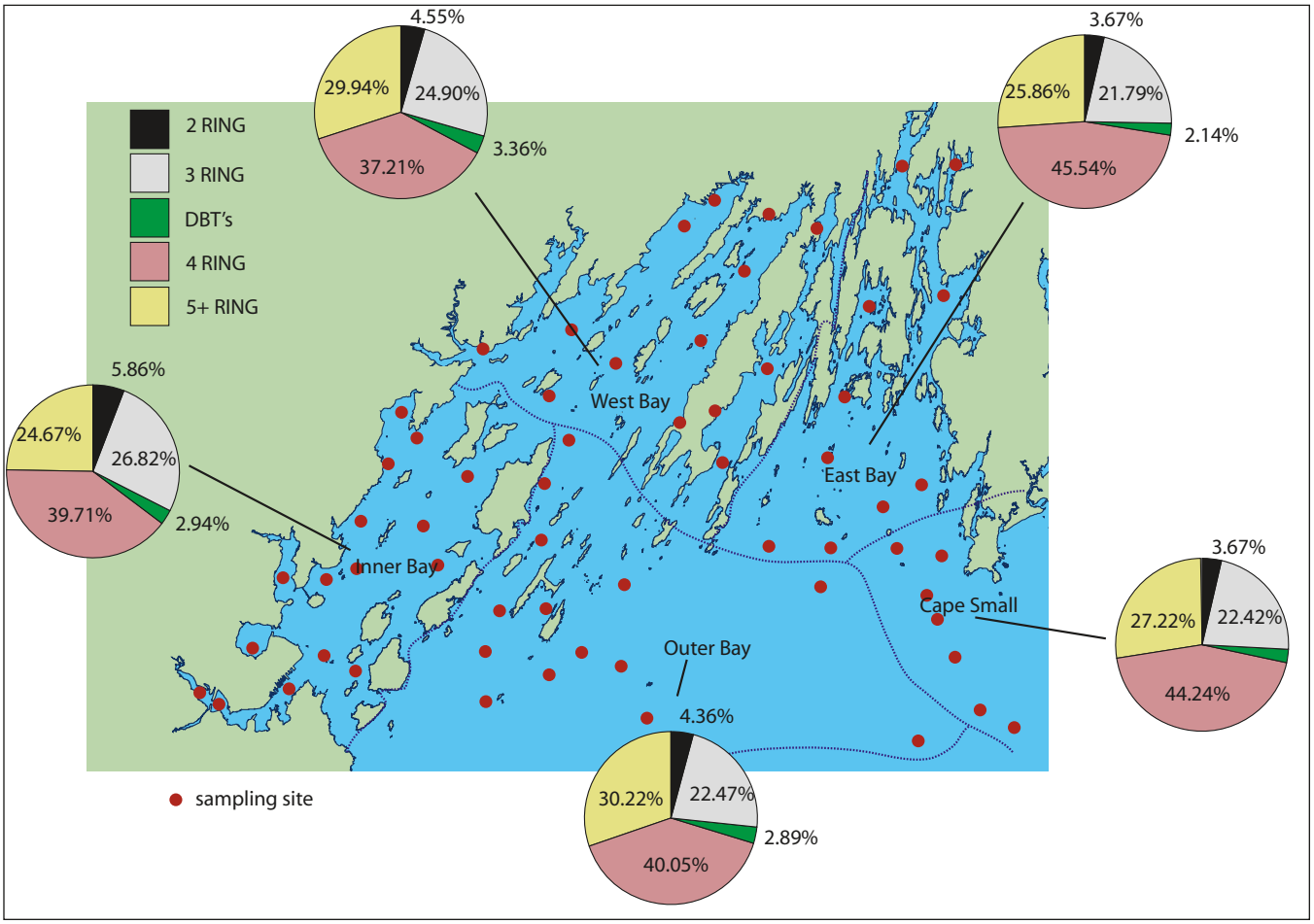


Figure 4-1: *The predominance of High Molecular Weight PAHs indicates that most of the PAHs delivered to the Bay come from post-combustion sources (hydrocarbon fuels burned at high temperature). The Inner Bay area around Portland has the highest percentage of Low Molecular Weight PAHs, likely from weathered petroleum that entered the bay via fuel spills or urban runoff (Kennicutt* et al. *1992).*

Changes in Toxic Contamination over Time: 1991/1994 versus 2000/2001

This section expands on the sediment toxics indicator (contaminant concentration change over time) reported in *State of the Bay* (CBEP 2005). In summer of 2000 and 2001, in partnership with US EPA's National Coastal Assessment, CBEP resampled the sediments at the original sites in Casco Bay for PAHs, PCBs, pesticides, metals, butyltins, dioxins/furans and planar PCBs (see Table 4-1). Scientists from Texas A&M University compared the results of the 1991/1994 sampling data to the 2000/2001 data. They concluded that most toxic chemicals have decreased or stayed the same over time, indicating that pollution control strategies are working in Casco Bay. See Figures 4-2a through 4-10b for a comparison of toxic contaminant levels in 1991/1994 and 2000/2001 samples.

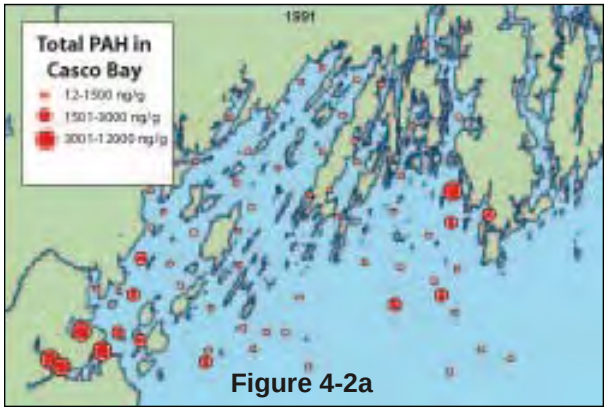


Figure 4-2a

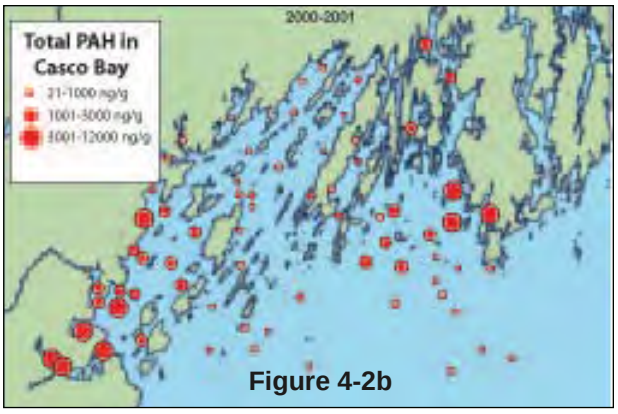


Figure 4-2b

Total PAHs: *For sites with total PAHs elevated above 2000 ng/g (or ppb), 10 sites were higher in 2000/2001 and 2 were higher in 1991, indicating that total PAH increased at some sites and decreased at others (Wade and Sweet 2005).*

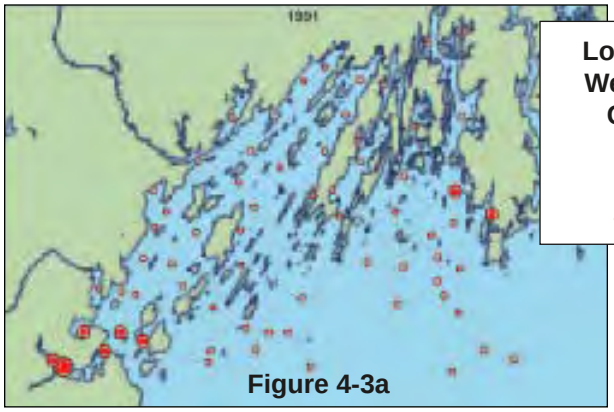


Figure 4-3a

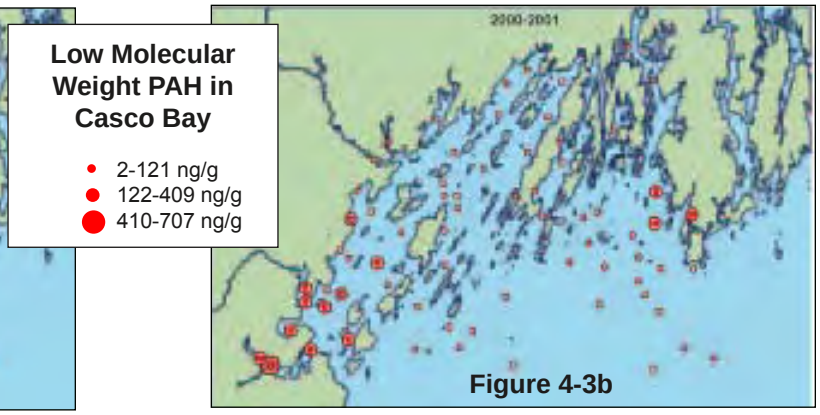


Figure 4-3b

Total Low Molecular Weight PAHs: *Total low molecular weight PAHs (the sum of naphthalene, acenaphthylene, acenaphthene, fluorene, and anthracene) generally decreased between sampling periods (Wade and Sweet 2005).*

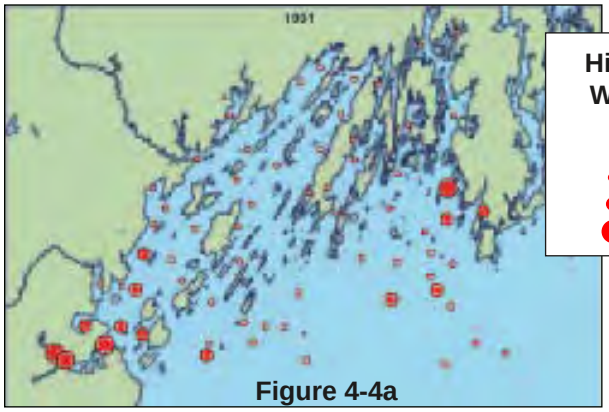


Figure 4-4a

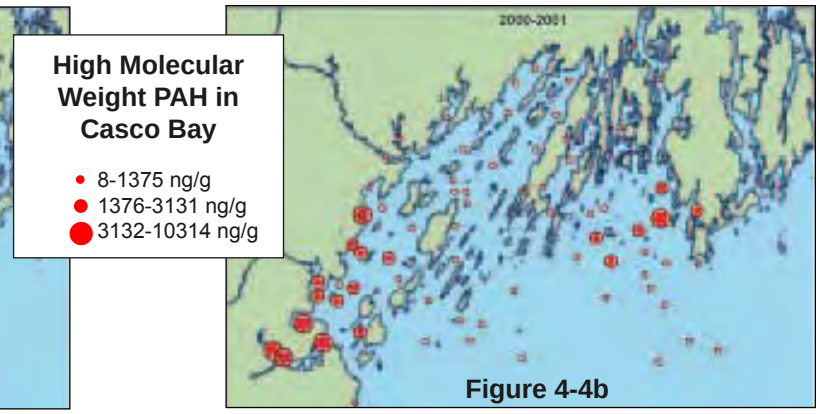


Figure 4-4b

Total high molecular weight PAHs: *The total high molecular weight PAHs (the sum of fluoranthene, pyrene, benzo(a)anthracene, chrysene, benzo(b)fluoroanthene, benzo(a)pyrene, indeno(1,2,3,-c,d)pyrene, dibenzo(a,h)anthracene, and benzo (g,h,i)pyrelene) increased at most of the sites over the time period. This suggests that the increased use of fossil fuels has been balanced by environmental controls that lower PAH inputs to the Bay (Wade and Sweet 2005).*

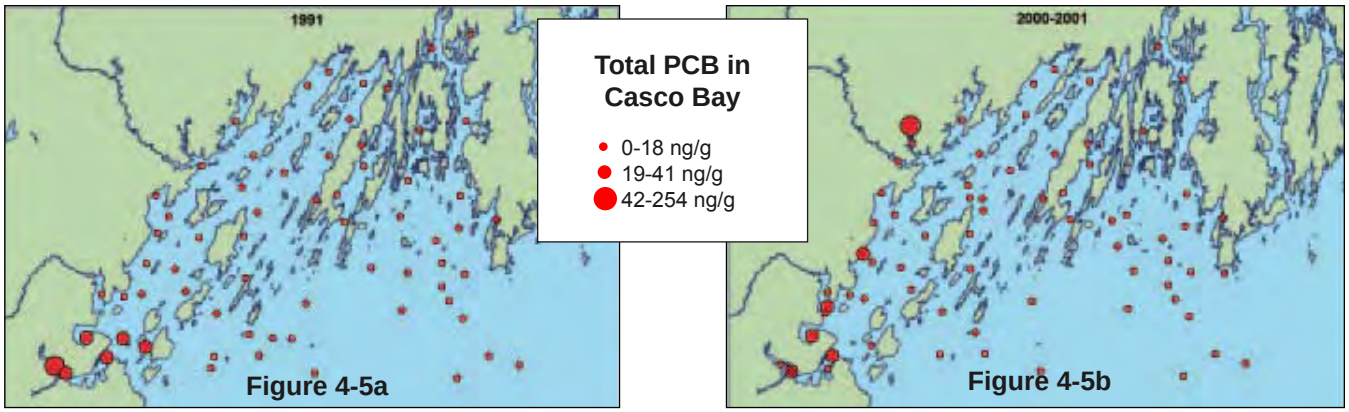


Figure 4-5a Figure 4-5b

Total PCBs: *Total PCB concentrations for the sum of 20 PCB congeners were generally lower in 2000/2001 than in 1991. Of 65 sites sampled, only 8 had higher concentrations in 2000/2001 (Wade and Sweet 2005). Manufacture of PCBs has been banned in the United States since 1977. Of the estimated 1.2 million tons of PCBs manufactured before the ban, it has been estimated that 65% is still in use in electrical equipment, 31% is in the environment, and 4% has been degraded or incinerated (Tanabe 1988). While residual PCBs are still entering the waters of the Bay from runoff and atmospheric deposition, the ban appears to be effectively decreasing levels in the Bay's sediments (Wade and Sweet 2005).*

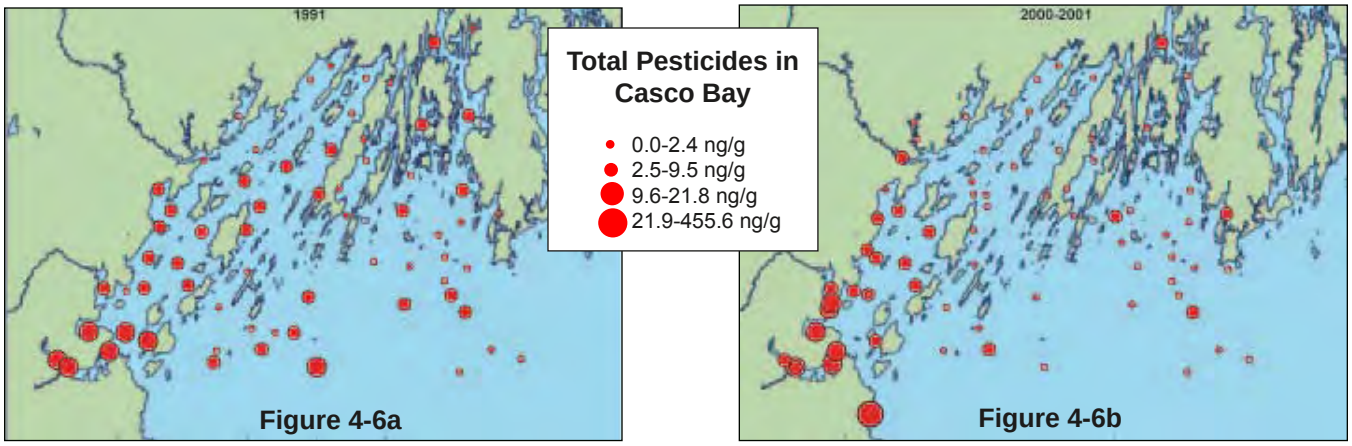


Figure 4-6a Figure 4-6b

Total Pesticides: *Total pesticide concentrations for 2000/2001 were generally lower compared to 1991. The most significant of the pesticides making up the total were DDTs. Of 59 sites sampled, only 10 had higher total DDT concentrations in 2000/2001, the rest were lower than in 1991. This is not surprising since the pesticides tested have been banned in the United States for decades. For example, use of DDT was discontinued in 1972. While they have long half-lives (on the order of 10 to 20 years for half of the total concentration to break down) these contaminants should slowly decrease in the environment as a result of the ban (Wade and Sweet 2005).*

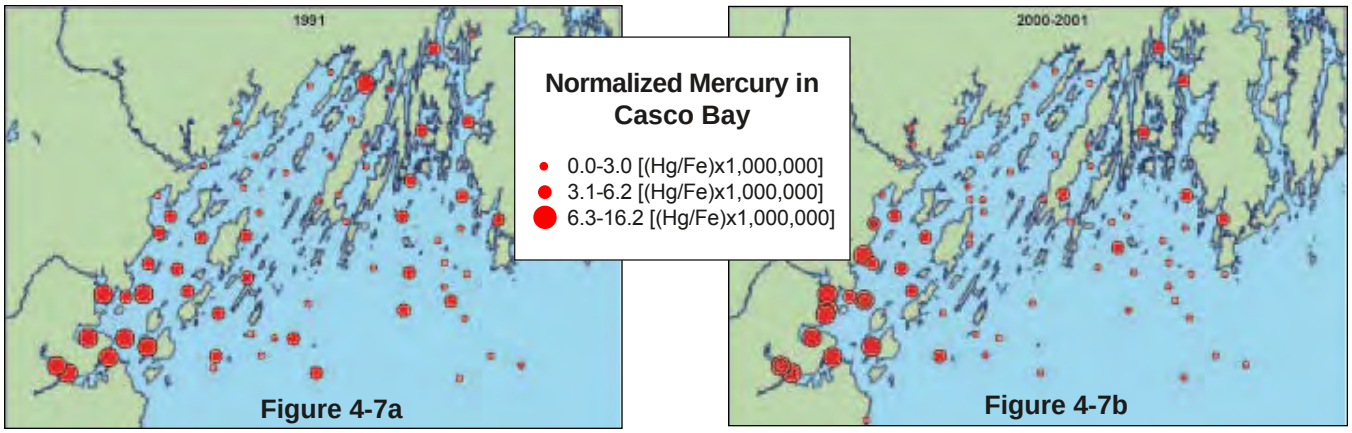


Figure 4-7a Figure 4-7b

Metals: *This figure illustrates the decline in mercury concentrations over the study period. Between 1991 and 2000/2001, there were decreasing concentrations at the majority of sites for cadmium, chromium, mercury, nickel, and selenium. There was no apparent change between 1991 and 2000/2001 in arsenic, copper, lead and zinc. Silver was the only metal that increased in concentration at most sites during the study period (Wade and Sweet 2005).*

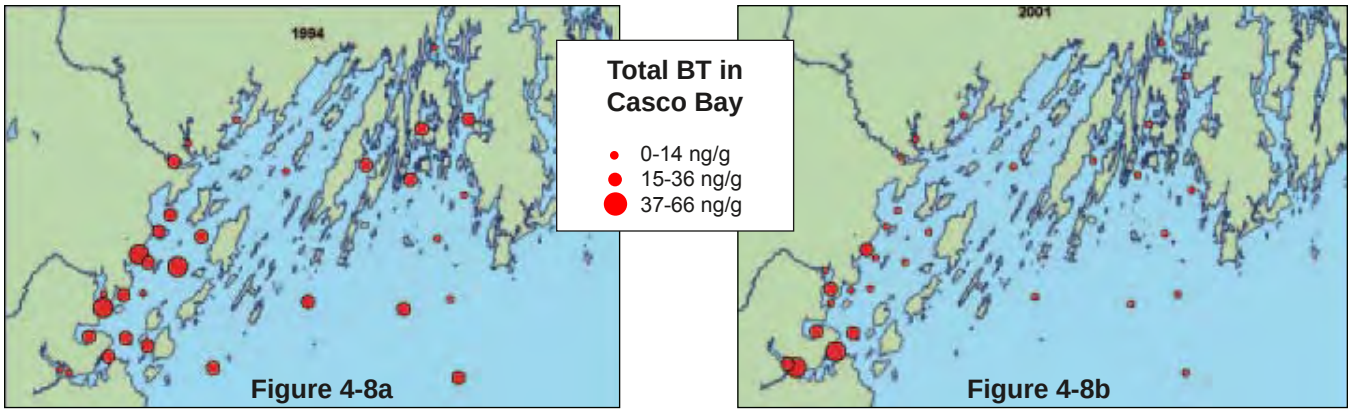


Figure 4-8a Figure 4-8b

Butyltins: *Concentrations of total butyltin (the sum of TBT, DBT and MBT) were lower in 2000/2001 at 23 of the 29 sites sampled, indicating a general decrease across the Bay. Only six sites were higher in total butyltin in 2000/2001. Of these, five were in the Inner Bay area. TBT is an ingredient in marine anti-fouling paints. The overall decline of TBT concentrations in the Bay's sediments reflects the effectiveness of the federal and Maine laws which now ban the use of paints with TBT for all uses except for vessels longer than 25 meters or having aluminum hulls (Maine DEP 1999). The continued use of TBT paints on large commercial vessels may explain the presence of elevated concentrations of TBT in the sediments of Inner Bay sites (Wade and Sweet 2005).*

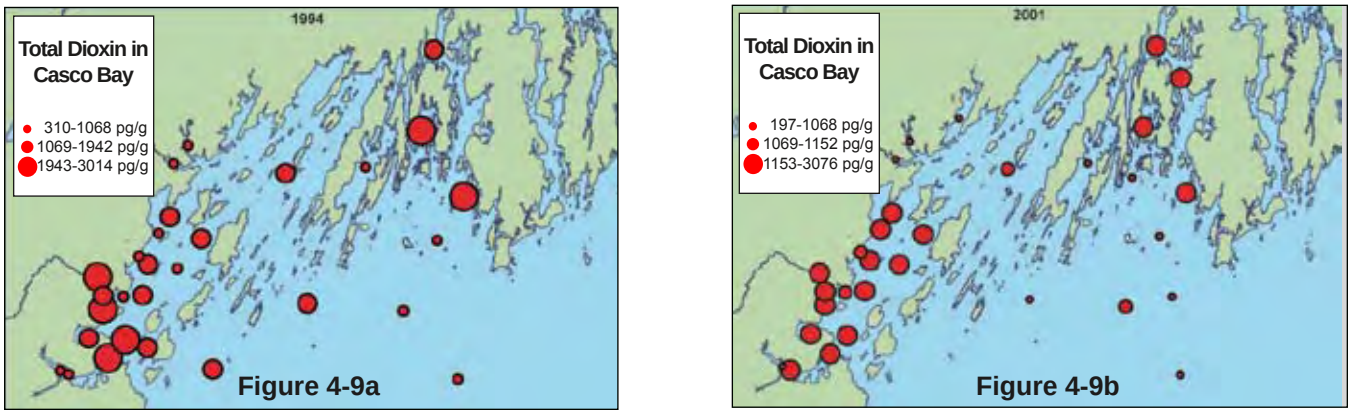


Figure 4-9a Figure 4-9b

Dioxins and Furans: *Differences in concentration were analyzed for 19 dioxin/furans for the 1994 and 2000/2001 sampling periods. Total dioxins for both sampling periods are shown above. Six compounds were higher in 2000/2001, 7 remained about the same, and 3 were higher in concentration in 2000/2001. There was no systematic increase or decrease of dioxins and furans. In the long term, regulations lowering the production of these toxic chemicals should lead to a decrease of concentrations in the environment (Wade and Sweet 2005). With the cessation of the pulping operation at the Westbrook paper mill in 1999 Westbrook, a major source of dioxin has been eliminated.*

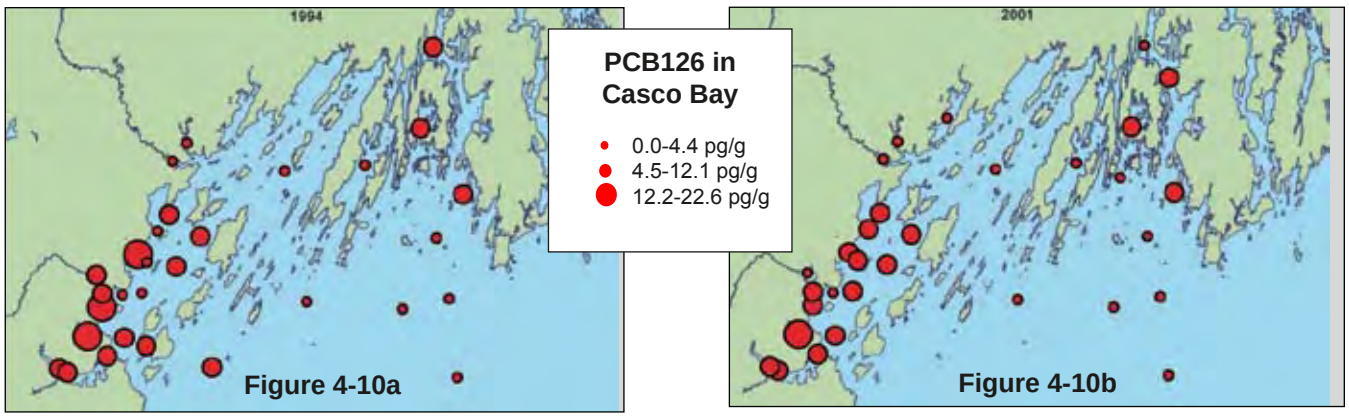


Figure 4-10a Figure 4-10b

Planar PCBs: *Planar PCB 77 showed no overall change between 1994 and 2000/2001. Planar PCB 126 concentrations generally decreased from 1994 to 2001, as illustrated above. The third planar PCB sampled, PCB 169, was not detected in enough samples to observe a change (Wade and Sweet 2005).*

Toxicity of Casco Bay Sediments

The following summary is based on the analysis of the 1991/1994 and 2000/2001 data (Wade and Sweet 2005).

- **PAHs:** While highly elevated above natural background levels, the PAH concentrations seen in the sediments of the inner part of the Bay were between the levels identified by the National Status and Trends Program as Effects Range Low (ERL, possible biological effects = 4,022 ppb) and Effects Range Median (ERM, probable biological effects = 44,792 ppb) (Long *et al*. 1995). The majority of PAHs detected in the Bay's sediments are high molecular weight, combustion-related and sequestered in fine particles.
- **PCBs:** PCB concentrations at almost all sites were below the toxic response threshold (ERL = 22.7 ppb). The exception was the Fore River site sampled in 1991, where the PCB concentration exceeded the ERM (180 ppb dry weight) (Long *et al*. 1995).
- **Pesticides:** Concentrations of pesticides were low compared to concentrations considered toxic (ERL for total DDT = 1.58 ppb (Long *et al*. 1995).
- **Metals:** The concentrations of metals in Casco Bay are lower than levels known to cause harmful effects to organisms. Even in the few areas with elevated metal levels in Casco Bay, the concentrations are lower than the highly contaminated sediments in urban areas like Long Island Sound and Boston Harbor. Silver, cadmium, lead, zinc and mercury concentrations in the Bay indicate that metals resulting from anthropogenic (human) activities have been deposited in a few areas, but at levels that are unlikely to cause toxic effects.
- **Butyltins, dioxins and furans, and planar PCBs:** These chemicals were not present at toxic concentrations. In general, the highest concentrations of these toxic chemicals were found near known sources. For example, elevated butyltin concentrations (a constituent of marine anti-fouling paints) were found near boat anchorages and marinas, while dioxins and furans were found in elevated concentrations downstream of pulp and paper mills (Wade and Sweet 2005). Despite relatively low concentrations of 2,3,7,8-TCDD (a potent toxic dioxin) in most of the Bay, the elevated levels found in lobster tomalley from Casco Bay (Mower 1994) indicate that dioxin is available to organisms in the food chain and is being bioaccumulated (see Chapter 1) (Wade *et al.* 1995).

Steve Karpiak

2004 Portland Harbor/Fore River Study

In addition to the Casco Bay-wide sediment studies described above, sites in Portland Harbor and the Fore River were sampled in 2004 for PAHs and the heavy metals cadmium, chromium, copper, lead, mercury, nickel, silver, and zinc. This sampling was conducted by Friends of Casco Bay (FOCB), supported by a Natural Resource Damage Assessment grant and funds from the CBEP. Sites were selected based on the need for future dredging as well as past "dirty history," including the *Julie N* oil spill, industrial uses, proximity to combined sewer overflows (CSOs), and drainage from the Jetport and Maine Mall.

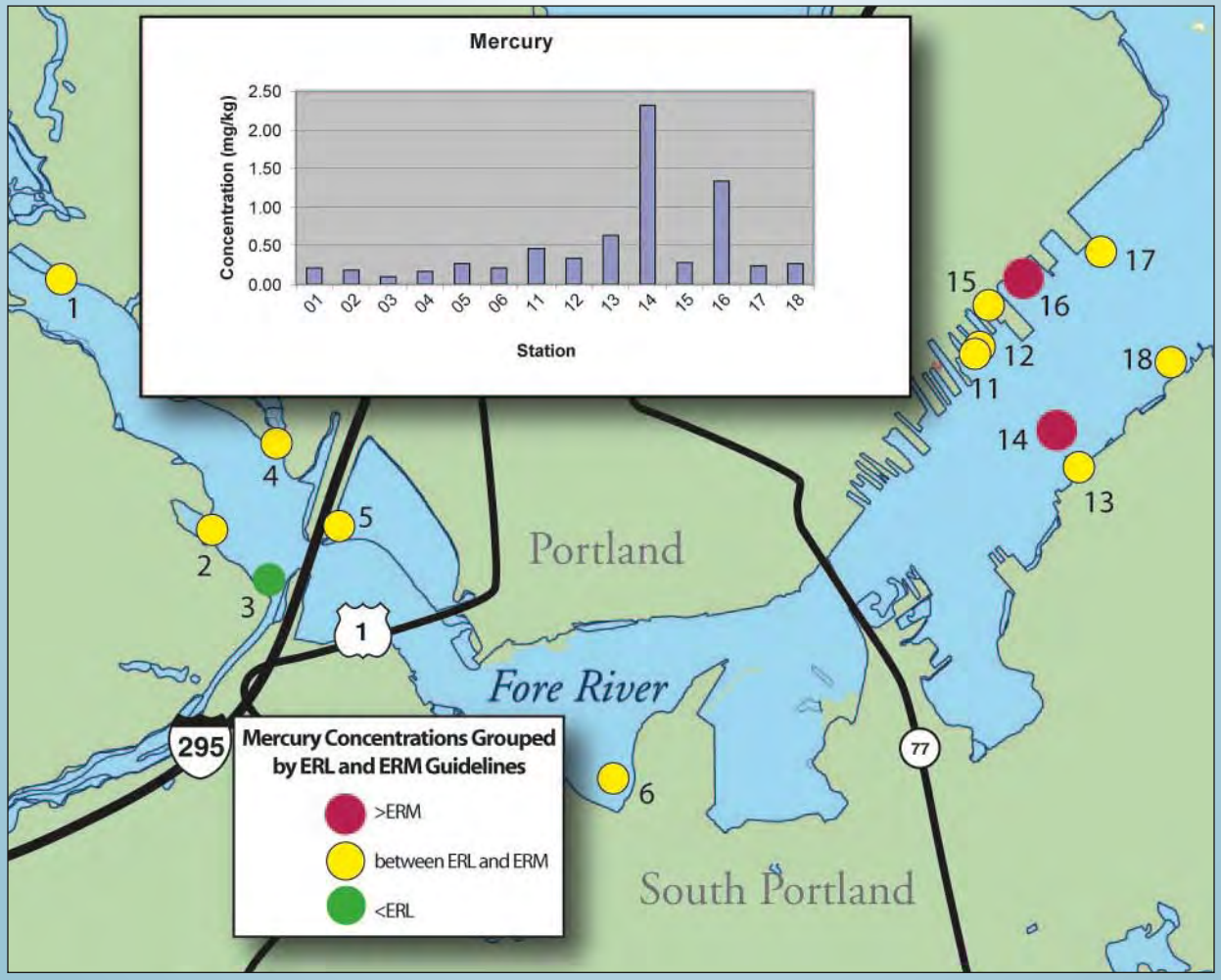
Friends of Casco Bay

Friends of Casco Bay scientist Peter Milholland and volunteer Pam Joy use a grab sampler to collect sediment samples for the Portland Harbor/Fore River study conducted in 2004.

Results

- **Metals:** Mean sediment concentrations were slightly elevated above the ERL (possible biological effects) for cadmium, lead, nickel, silver and zinc for several of the 18 sites sampled. Mercury concentration exceeded the ERL at most sites and was elevated above the ERM (probable biological effects) at two sites: (14) and the Maine State Pier (Station 16). Copper concentrations were elevated above the ERL at 4 sites and exceeded the ERM at the Maine State Pier (Station16) (FOCB 2005a).

Figure 4-9: Mercury Concentrations in the Fore River Grouped by ERL and ERM Concentrations

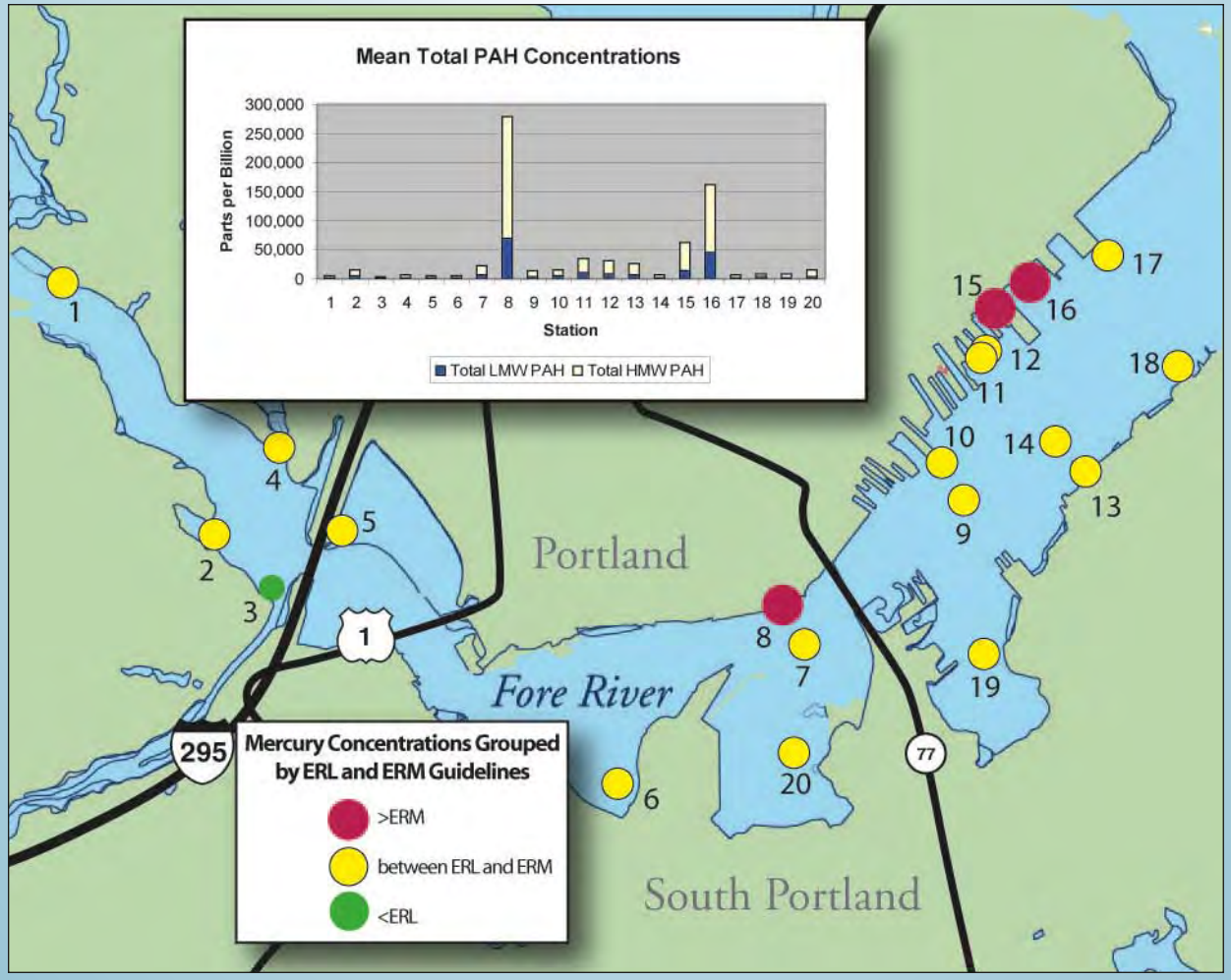


- **PAHs:** Total PAH concentrations at all but one of the 20 sites sampled were elevated beyond the ERL concentration (possible biological effects), while the Gas Works/China Clay Docks (Station 8) and two sites near large CSOs, the Maine State Pier (Station16) and the Casco Bay Ferry Terminal (Station 15), exceeded the ERM concentration (probable biological effects) established by the NOAA Status and Trends program (Long *et al.* 1995).

  The ratio of low molecular weight PAHs to high molecular weight PAHs can be used as a way to "fingerprint" the likely source of pollution. Low molecular weight PAHs are generally from pre-combustion sources such as oil spills, while high molecular weight PAHs are associated with post-combustion products, entering the marine environment via stormwater runoff and atmospheric deposition. The Casco Bay Ferry Terminal site (Station 15), for example, had a "fingerprint" suggesting primarily post-combustion sources, likely from the CSO at the site (FOCB 2005b).

This sampling study has provided baseline data on the current status of the Harbor and Fore River sediments and will be valuable as future dredging needs and potential dredging impacts are evaluated.

Figure 4-10: Total PAH Concentrations in the Fore River Grouped by ERL and ERM Concentrations



Summary/Conclusions

The levels of toxic chemicals in the sediment of Casco Bay are not likely to pose a biological threat to resident biological organisms in most areas of the Bay. However, PAHs and PCBs are elevated in some parts of the Inner Bay, exceeding the thresholds believed to cause biological impacts. While the levels of sediment contamination are low in much of the Bay, toxic pollutants have the potential to become concentrated in higher predator organisms through the processes of biomagnification and bioaccumulation (see Chapter 1). For example, the elevated levels of dioxin in lobster tomalley and PCBs in the tissues of bluefish and striped bass are a result of these processes (see Chapter 8).

CBEP's sediment studies suggest that the levels of many of the toxic pollutants found in the sediments of Casco Bay are declining over time. This is likely the result of successful federal, state and local environmental control strategies, including bans on the manufacture and use of certain chemicals (*e.g.,* DDT, PCBs), regulations which limit the use or release of toxic chemicals (*e.g.,* TBT, dioxin), and ongoing efforts to reduce toxic chemical releases from point and non-point sources. For further discussion of efforts to reduce the loading of toxics to the Bay, see Chapter 9.

References

Casco Bay Estuary Partnership. 2005. *State of the Bay*.

Doggett, L. 2006. Maine Department of Environmental Protection. Personal Communciation.

Eisler, R. 1987. Polycyclic aromatic hydrocarbon hazards to fish, wildlife, and invertebrates: a synoptic review. *U.S. Fish Wildlife Service*. *Biol. Rep.* 85(1.11).

Friends of Casco Bay. 2005a. *Toxic Contaminants in the Surficial Sediments of the Fore River, Maine. Presentation at the State of the Bay Conference,* November 3, 2005, South Portland, Maine. Mike Doan, presenter.

Friends of Casco Bay. 2005b. *Sediment PAH Concentrations from the 2004 Study of Portland Harbor.*

Hauge, P. 1988. *Troubled Waters: A Report on the Environmental Health of Casco Bay.* Conservation Law Foundation/Island Institute. 71 pp.

Kennicutt, II, M.C., T.L. Wade, and B.J. Presley. 1992. Texas A & M University. *Assessment of Sediment Contamination in Casco Bay.* Casco Bay Estuary Project.

Kennicutt II, M.C., T.L. Wade, B.J. Presley, A.G. Requejo, J. M. Brooks, and G.J. Denoux. 1994. Sediment contaminant impacts in Casco Bay, Maine: Inventories, Sources, and Potential for Biological Impact. *Environmental Science and Technology*. 28 (1) 1 -15.

Long, E.R., D.D. MacDonald, S.L. Smith, and F.D. Calder. 1995. Incidence of adverse biological effects within ranges of chemical concentrations in marine and estuarine sediments. *Environmental Management* 19(1): 81-97.

Macauley, J.M., J.K. Summers, V.D. Engle, P.T. Heitmuller, G.T. Brooks, M. Babikow, and A.M. Adams. 1994. *Statistical Summary: EMAP- Estuaries Louisianian Province—1992*. U.S. Environmental Protection Agency, EPA/620/R-94-002.

Mower, B. 1994. *Dioxin Monitoring Program.* Maine Department of Environmental Protection.

Tanabe, S. 1988 PCB problems in the future: Foresight from current knowledge. *Environmental Pollution* 50: 5-28.

United States Environmental Protection Agency. 1997. *The Incidence and Severity of Sediment Contamination in Surface Waters of the United States. Volume 1: National Sediment Quality Survey*. U.S. Environmental Protection Agency. EPA 823-R-97-006.

United States Environmental Protection Agency. 2006. *Information on the toxic effects of various chemicals and groups of chemicals.* (http://www.epa.gov/R5Super/ecology/html/toxprofiles.htm) (August 31, 2006).

Wade, T.L., T.J. Jackson, L. Chambers, and P. Gardinali. 1995. Texas A & M University. *Assessment of Contaminants in Sediments from Casco Bay*. Casco Bay Estuary Project.

Wade, T.L. and S.T. Sweet. 2005. Texas A & M University. *Assessment of Sediment Contamination in Casco Bay.* Casco Bay Estuary Project.

How are blue mussels serving as an indicator organism in Casco Bay?

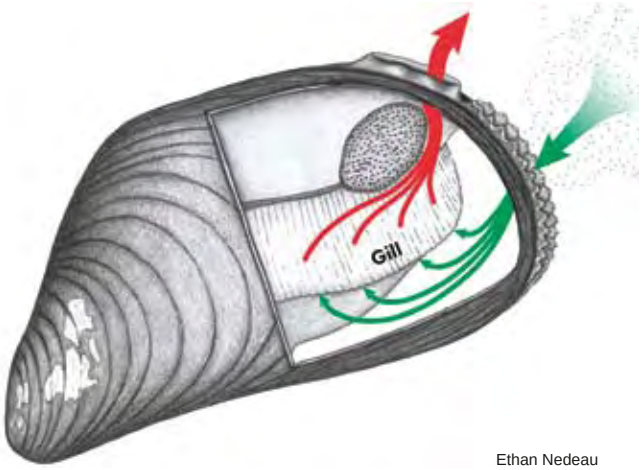
5

Sonny McAlpin

Background

The impact of toxic chemicals on the health of natural resources and the humans who use them has been a serious concern for in the United States since the 1960's, when the devastating effects of DDT were first publicized. We now know that toxic chemicals, including metals, pesticides and other organic chemicals are found at varying concentration levels everywhere in our environment. A key question for health and resource managers is whether or not they are present at levels that can cause toxic effects in humans and other organisms (Maine DEP 2005). Regularly monitoring toxic chemicals levels in a common resident organism that serves as a "sentinel" or "indicator" organism can be an effective environmental management tool.

The common blue mussel, *Mylius edulis,* is an ideal indicator species for marine environments. It is sedentary as an adult and relatively long-lived, accumulating contaminants from the local environment as it feeds and through surface contact with the sediments (see Figure 5-1). The mussel is commonly found throughout the coastal areas of the Gulf of Maine, making it useful for regional as well as local contaminant assessment (GOMC 2004). In Maine, blue mussels are found in dense beds in the intertidal zone (between the high and low tide lines), where they can serve as good indicators of sediment contamination. Because they are primary consumers at the base of the food chain, elevated levels of toxic contaminants in the tissues of mussels may suggest that higher level consumers like fish and humans may be at risk from contaminants in the ecosystem.



Ethan Nedeau

Figure 5-1. *The common blue mussel serves as an excellent indicator of environmental contamination. As the mussel breathes and feeds, its gill filters out and retains particles, including contaminants, which can be digested and assimilated into its tissues.*

Monitoring Blue Mussels in Maine's Coastal Waters

In 1987, Maine DEP began a major long-term study (the Marine Environmental Monitoring Program) to assess the levels and locations of toxic contaminants along the coast, using the common blue mussel as the indicator species. Because regional and national programs also sample mussels (NOAA's Mussel Watch and the Gulf of Maine Council on the Marine Environment's Gulf Watch) these larger data sets help to provide a context for assessing the relative conditions in Maine. The goals of Maine DEP's blue mussel sampling program included:

- Defining background (or baseline) levels of toxic chemicals in Maine mussels and
- Determining what levels pose a health risk to humans and/or marine life

Sampling included mussels from 24 "reference sites" thought to represent a relatively unimpacted background condition, free from industrial and anthropogenic influences. These sites were used to describe normal background (baseline) levels. Normal was defined as plus or minus 2 standard deviations around the mean of concentrations found in mussels collected at the reference stations. Because the concentration of toxic chemicals in mussel tissue varies with season, age of mussels, location in the intertidal zone and reproductive state, the time of collection was standardized to an "index period" from late August to early October, with mussels selected from the low intertidal or shallow subtidal zone. The Maine DEP program divided the Maine coast into 8 regions, each reflecting an ecological system such as a large estuary, coastal or intertidal regime. One of the regions selected was Casco Bay, a semi-enclosed system with a deeply indented coastline and many islands (Maine DEP 2005).

Mussels were sampled for the metals aluminum (Al), arsenic (As), cadmium (Cd), copper (Cu), iron (Fe), nickel (Ni), lead (Pb), zinc (Zn), silver (Ag), and mercury (Hg) as well as pesticides, dioxins and furans, PAHs (polycyclic aromatic hydrocarbons), and PCBs (polychlorinated biphenyls) at multiple sites in Casco Bay. Since 1996, CBEP has supplemented the Maine DEP's Marine Environmental Monitoring Program and Surface Water Ambient Toxics Monitoring programs by sampling at additional Casco Bay sites.

Based on the mussel sampling data, the Marine Environmental Monitoring Program has established normal baseline reference concentrations for metals in mussels, with the exception of arsenic. Arsenic is compared to elevated levels as reported in NOAA (1988). Organics (PAHs, pesticides, and PCBs) are also compared to NOAA's 1998 reported elevated levels (Maine DEP 2005).

Key Results of Maine DEP and CBEP Mussel Sampling in Maine

Blue mussel soft tissue data is now available from approximately 65 sites sampled along the Maine coast during the period 1987 to 2003. When compared to the established baseline reference concentrations, some sites in Maine had contaminant levels above the Maine coastal norm. Most, however, did not. Mussels with elevated levels of toxic chemicals in their tissues were generally in the most heavily developed ports and harbors or were in the mouths of major industrial rivers, as seen in the overview of lead concentrations in sites sampled since 1987. Figure 5-2 provides an overview of the results of lead sampling sites in the Bay. Note that the elevated and highly elevated levels of lead are seen in areas with high levels of human activity.

CBEP sampling in 1996 and 1998 indicated elevated toxic chemicals at the following sites:

- Lead levels were elevated in Back Cove mussels while dioxins and furans were elevated in Freeport, New Meadows, Jewell Island, Back Cove, and the Harraseeket River; total PCBs were elevated in samples from Back Cove, Quahog Bay, and somewhat elevated in samples from Falmouth.
- Arsenic was elevated at Falmouth and Jewell Island,

For samples collected by CBEP and Maine DEP from 2001 to 2003, Table 5-1 indicates sites where metals were elevated above the State normal baseline. For other toxic chemicals, areas where elevated levels were detected are summarized as follows:

- PAHs were at baseline levels at all sites except the inner Fore River where they were highly elevated.
- PCBs and pesticides were at baseline or below at all other sites except the inner Fore River site, where PCBs were approaching elevated.

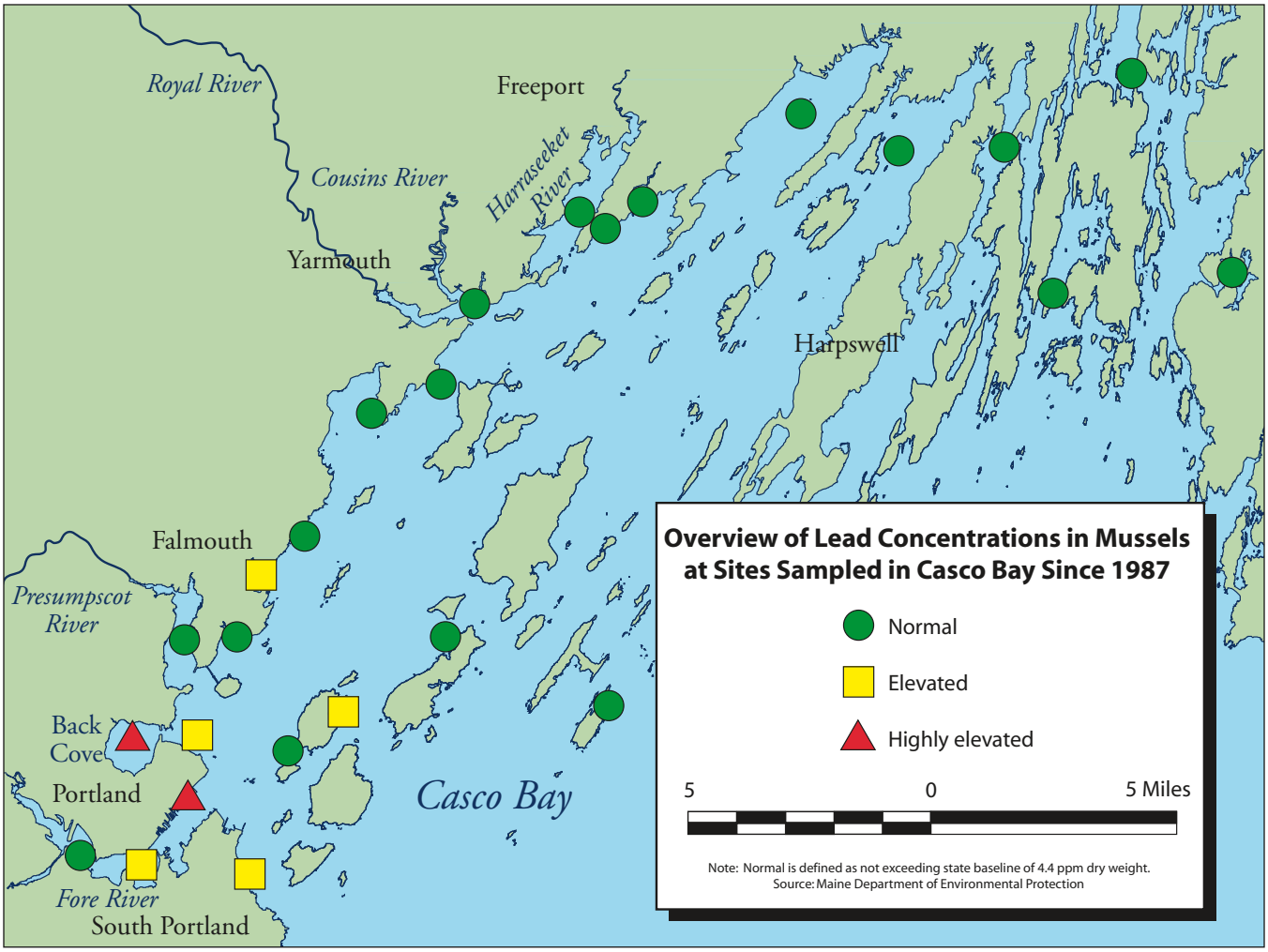


Figure 5-2. *Long-term monitoring of mussels in Casco Bay indicates that elevated levels of metals (such as lead) tend to be found in areas where human activity is concentrated.*

Changes in Toxics Concentrations Over Time

Sampling at the same locations several years apart allows us to look at the way concentrations of contaminants are changing over time. Six of the sites noted in Table 5-1 were also sampled for metals in 1988. The 1988 data was the result of a single sample while the 2001 and 2002 results are based on four replicate samples. Note that aluminum was not included in the 1988 analysis. Along with iron, aluminum is used to indicate the extent to which mussels are ingesting suspended sediments and is reported as elevated in the table to give an indication of the amount of sediment in the gut of the mussel.

Table 5-1: Maine Department of Environmental Protection

Metals Elevated Above Maine Normal Baseline Values Found in Mussels from Sampling Sites in Casco Bay 2001-2003

	Al	Cd	Cr	Cu	Ni	Pb	Zn	Ag	Hg
Great Diamond Island (Cocktail Cove)	X					X		X	
Long Island					X				
Mare Brook	X								
Inner Fore River						X	X		X
Maquoit Bay	X								
East End Beach						X	X		
Spring Point						X	X		
Mill Creek						X			
Outer Fore River						X			

Source: Maine Department of Environmental Protection

The six sites sampled in both 1988 and 2001/2002 are Great Diamond Island, Long Island, the Inner Fore River, East End Beach, Spring Point and Mill Creek. Each of these sites is characterized below, including changes in land use that may have altered the concentration or availability of toxic chemicals to resident blue mussel populations.

- **Great Diamond Island, Cocktail Cove** is a protected cove, heavily used by recreational boaters in the summer. The shoreline is ledge with gravel beaches. Increased development has changed land use dramatically since 1988. A restaurant and marina and new seasonal and year-round homes have increased boat traffic and nonpoint source runoff. In 1988, while levels of metals were within normal concentrations, the lead level was approaching the human health action level (Maine DEP 2005). A repeat sampling in 2001 indicated that lead concentrations were now twice as high. Arsenic and silver were also elevated, but were not sampled in 1988.

- **The Long Island** fuel terminal area (the shore adjacent to the former Navy fuel depot) at Ponce Landing has sand and gravel beaches with rocky outcrops. The area is now residential. In 1988, cadmium and zinc were elevated above normal and lead was elevated above the health screening level. The presence of toxics is likely related to the past history of the site (Maine DEP 2005). In 2001, metals were all in the normal range with the exception of nickel, which was elevated.

- **The Inner Fore River,** upstream of the I-95 Bridge is a soft mud-bottom area that receives freshwater inflows from the Stroudwater River. There is moderate commercial and residential development nearby. Historically, the water quality has been compromised by industry upstream. In 1988, metals were in the normal range with the exception of zinc, a common constituent of road runoff which derives from tire material. Both the Portland Jetport and the Maine Mall are nearby sources of polluted runoff (Maine DEP 2005). In 2002, the concentration of lead was twice as high, zinc had decreased but was still at the high end of the Maine coastal normal baseline, and mercury was in a range similar to the 1988 value.

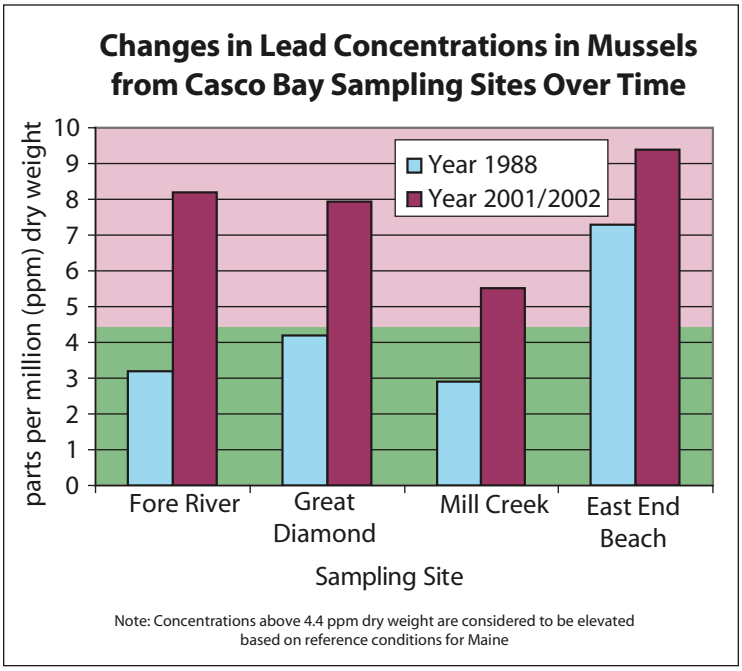
- Off of **East End Beach**, the sediment is composed of fine and coarse rubble, including fill from the old city dump. Water quality at the beach may be affected by urban runoff, leachate from the dump and possibly pollutants carried downstream by the Presumpscot River. The area is densely residential, with a municipal sewage treatment plant nearby. In 1988, both lead and zinc were elevated (Maine DEP 2005). In 2001, lead and zinc were still elevated, with slight increases in concentration. Cadmium increased to 2.66 ppm in 2001, elevated based on reference conditions for Maine (the coastal baseline norm is 2.56 ppm).

- **Spring Poin**t (South Portland) area has a narrow intertidal shoreline which is rocky and drops off steeply to deepwater. The adjacent area is residential with nearby industrial development. There are no direct discharges, but the area is likely impacted by urban runoff and pollutants from the inner harbor carried by the outgoing tide. In 1988, lead was elevated (Maine DEP 2005). In 2001, the lead level was still elevated but the concentration had declined since 1988, as it has in most areas of the Bay.

- **Mill Creek** (Mussel Cove) is an estuary comprised of intertidal mud flats. The Cove has a drainage areas of 5.4 square miles. Over the past 25 years, development along Route 1, including two shopping centers, has greatly increased the amount of impervious surface and stormwater runoff to the Cove. In 1988, metal concentrations were within normal baseline conditions, including a lead concentration of 2.90 ppm (parts per million) in the single replicate sample (Maine DEP 2005). In the 2001 sampling, lead concentrations increased to an average of 5.51 ppm in the four replicates, exceeding the level considered high for Maine.

The increases in lead levels that were seen at four of the sites (Inner Fore River, Great Diamond Island, Mill Creek and East End Beach in Portland) are all likely related to increased development and impervious surface (see Figure 5-3).

Figure 5-3



Marine and Estuarine Areas of Concern in Maine for Toxic Contamination.[1]

Location	Area
Cape Rosier	80 acres
Boothbay Harbor	410 acres
Fore River (Casco Bay)	1,230 acres
Back Cove (Casco Bay)	460 acres
Presumpscot River Estuary (Casco Bay)	620 acres
Piscataqua River Estuary	2,560

Based on sediment and mussel tissue analyses, Maine DEP has identified six areas of concern for toxics along Maine's coast, which are listed above. Three of these areas are in Casco Bay. As noted earlier, the most impacted areas tend to be in heavily developed ports and harbors or in the mouths of major industrial rivers (Maine DEP 2004).

[1]Acreage based on professional judgement.

Comparing Levels of Toxics in Casco Bay and Gulf of Maine Mussels

Gulfwatch is a joint United States/Canadian blue mussel monitoring program sponsored by the Gulf of Maine Council on the Marine Environment. The program is intended to help identify temporal and spatial trends in ecosystem exposure and exposure variability in the Gulf. Since 1993, Gulfwatch scientists have regularly sampled mussels along the coast of the Gulf of Maine. Five "benchmark" sites have been sampled every year. A total of 27 other sites have been sampled every 3 years on a rotating basis. There are also 6 multi-year sites that have been sampled every six years. Mussel tissues have been tested for 9 trace metals,16 pesticides, 24 PCB congeners (varying configurations of chemical structure) and 24 PAHs. Of the 38 sites that have been regularly monitored, three are in Casco Bay: these are located in Portland Harbor, the Presumpscot River estuary and the Royal River estuary.

Data from the first nine years (1993-2001) of Gulfwatch sampling have been analyzed and interpreted (GOMC 2006).

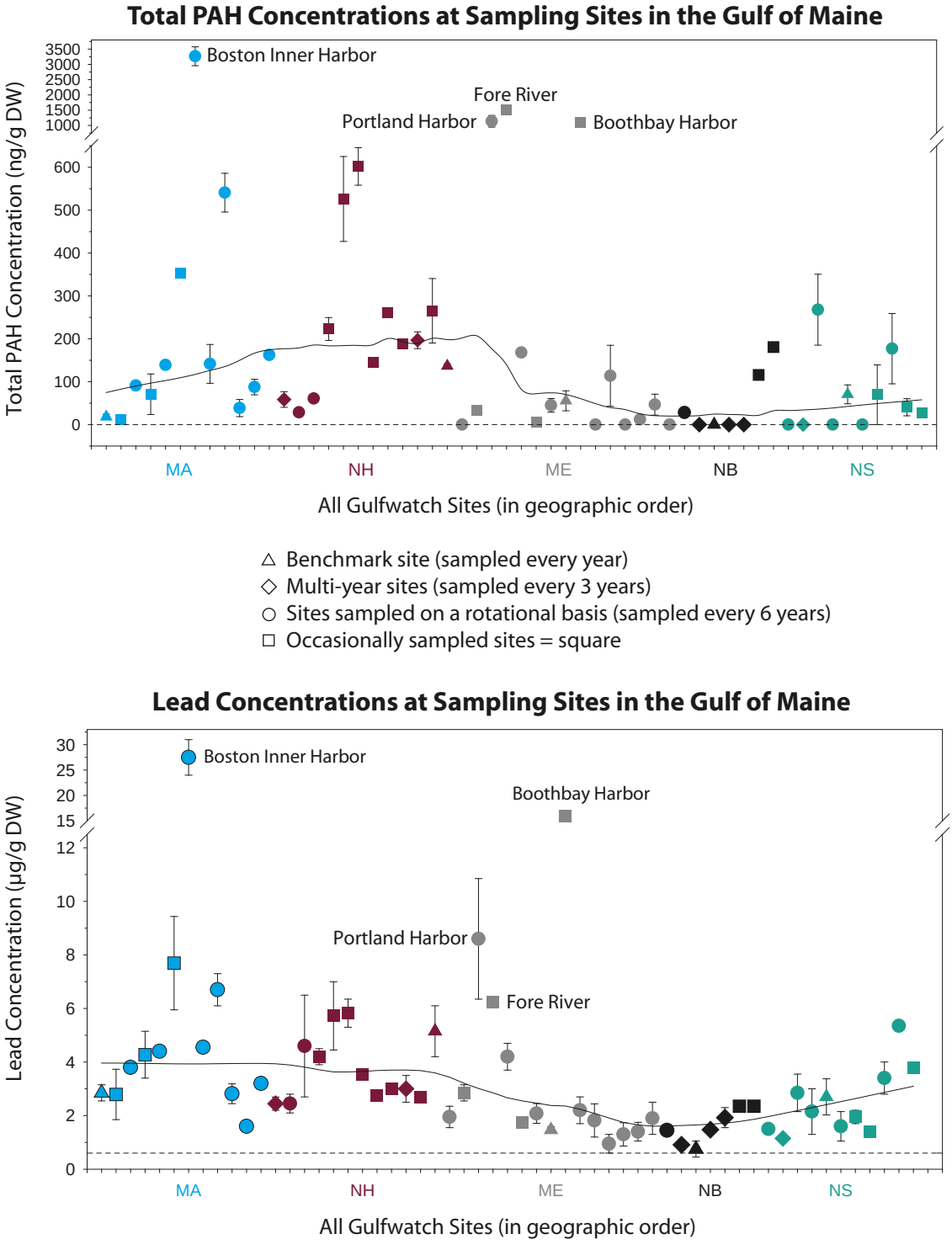
Statisitically significant spatial trends included:

- Decreasing trends south to north for silver, chromium, copper, lead and zinc
- Decreasing trends south to north for total DDT and DDT metabolites p,p-DDE and p,p-DDD, and for total PCBs

Statistically significant temporal trends :

- Silver, chromium, iron, lead, zinc, p,p-DDE and total DDT declined at some of the benchmark sites.
- Total DDT increased in Sandwich, MA.

Figure 5-4. *Nine-year (1993-2001) median and median absolute deviations for the sum of total PAH concentrations in nanograms/gram dry weight (ng/g DW) and lead concentrations in micrograms per gram dry weight (µg/g DW) in mussel tissues at all Gulfwatch sites, in geographic order (south to north along the x axis from Massachusetts to Nova Scotia). Note the elevated levels of PAHs in Portland Harbor and the Fore River. The dashed line indicates the detection limit of the analytical method. The solid line is the Lowess smoother, a statistical smoothing function for scatter plots that results in a locally weighted regression line (GOMC 2006).*



Gulfwatch sites in Casco Bay with elevated concentrations of toxic chemicals

- Lead was detected at concentrations exceeding the federal action level (level at which a consumption advisory may be appropriate) of 11.5 parts per million at the Portland Harbor site in several samples (see Figure 5-4). There were increasing concentrations of lead at the Casco Bay sites, including Portland Harbor. This data is consistent with the results of the Maine DEP and CBEP monitoring studies (see Figures 5-2 and 5-3).
- The sites in Maine with the highest mussel tissue PAH concentrations were in Casco Bay. Mussel tissues from the Fore River site and the Portland Harbor site had 1500 and 1100 nanograms/gram (ng/g) dry weight respectively. The PAH median value for Maine as a whole was 45 ng/g (see Figure 5-4). CBEP and DEP sampling in 2001 to 2003 also showed highly elevated levels of PAHs in the Fore River.

Summary/Conclusions

Most areas in Maine that are away from human activity, past and present, contain background/baseline concentrations of toxic metals and organic chemicals. Based on the blue mussel as an indicator, elevated levels of toxic contaminants in Maine tend to be present in areas with an industrial history (*e.g.*, past manufacturing), in harbors, commercial ports, the mouths of river watersheds and in locations adjacent to population centers. This is also confirmed by regional mussel sampling conducted by the Gulfwatch Program. The geographic distribution of sediment contamination in the Bay (see Chapter 4) is generally confirmed by the analysis of mussel tissue by the Maine DEP, CBEP and Gulfwatch monitoring programs.

The concentration of toxic chemicals found in blue mussel tissues is one of the fourteen indicators used by the CBEP to evaluate the environmental health of Casco Bay in *State of the Bay* (CBEP 2005). Continued mussel monitoring in the Bay will be useful to establish temporal and spatial trends in ecosystem contaminant levels and to assess potential health risks to human seafood consumers. The health implications of elevated levels of contaminants in blue mussels from Casco Bay are discussed in Chapter 8.

References

Casco Bay Estuary Partnership. 2005. *State of the Bay.*

Gulf of Maine Council. 2004. *Gulfwatch Contaminants Monitoring Program: Mussels as Bioindicators*. (http://www.gulfofmaine.org/gulfwatch/mussels.asp) (July 17, 2006).

Gulf of Maine Council. 2006. *The Nine-Year Gulfwatch Program 1993-2001: A Review of the Results and Program Design*. Authors: L. White, S. Jones, P. Wells, G. Brun, G. Harding, P. Hennigar, C. Krahforst, D. Page, J. Schwartz, S. Shaw, D. Taylor, P. Trowbridge.

Maine Department of Environmental Protection. 2004. *Integrated Water Quality Monitoring and Assessment Report ("305b")*. (http://www.maine.gov/dep/blwq/docmonitoring/305b/index.htm#2004) (July 17, 2006)

Maine Department of Environmental Protections. 2005. *A Decade of Toxics Monitoring*. John Sowles. (http://www.maine.gov/dep/blwq/docmonitoring/toxics/index.htm) (July 17, 2006).

National Oceanic and Atmospheric Administration (NOAA) 1998 (on-line) *"Chemical Contaminants in Oysters and Mussels"* by Tom O'Conner. (NOAA's State of the Coast Report. Silver Spring, MD) (http://www.oceanservice.noaa.gov/websites/retiredsites/sotc_pdf/CCOM.PDF) (January 17, 2007).

What are the impacts of mercury on wildlife?

6

USF&WS

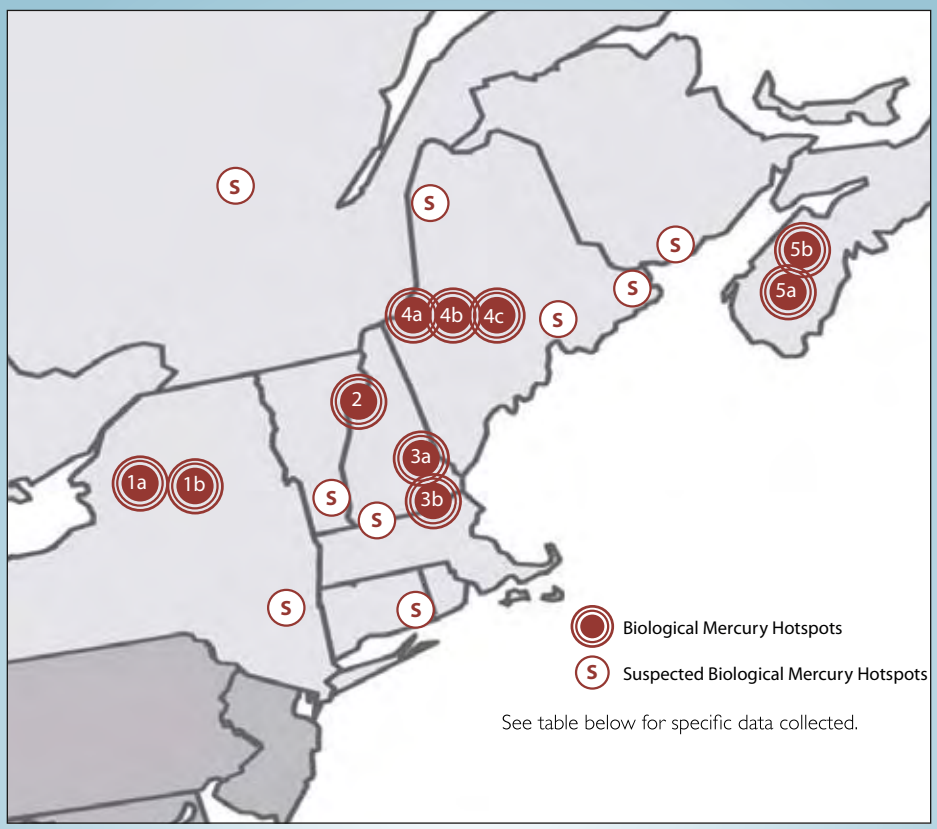
Background

Maine DEP has noted that mercury levels in the state's fish, loons and eagles are among the highest in North America (Maine DEP 2005a). Much of the research supporting this statement resulted from a call for studies on the regional bioavailability of mercury in freshwater and marine ecosystems, a need identified at the 1998 conference of the New England Governors and Eastern Canadian Premiers (NESCAUM 1998). This chapter briefly summarizes recent studies on the impacts of mercury on wildlife in Maine and, in particular, in Casco Bay and its watershed. Chapter 8 addresses the human health implications of consuming fish and shellfish with elevated mercury.

The heavy metal mercury can enter the environment through industrial processes, such as chlorine manufacturing (Evers 2005), and through combustion of coal, oil, wood, natural gas and mercury-containing trash. Over the past century, anthropogenic inputs of mercury into the environment have significantly increased (Evers *et al.* 2004). Once in the environment, elemental mercury can be transformed by bacteria into a highly toxic organic compound (methyl mercury) which is readily absorbed into living tissues. Mercury is poorly excreted, leading to bioaccumulation and biomagnification up the food chain (see Chapter 1). Exposure to methyl mercury can result in serious damage to the nervous system and kidneys of fish, birds, and mammals. Mercury can also affect the reproductive system, including reduced fertility and reduced survival of young. It has been shown that it can induce genetic mutations and interfere with embryonic development (Chan *et al.* 2003).

Figure 6-1. *Evers et al. (2007) used a new method to identify biological mercury hotspots, based on the mercury concentrations in yellow perch and Common Loons. A mercury hotspot of human health concern occurs where there are 10 or more independent sites with yellow perch concentrations above 0.3 ppm within grids that average 890 square miles in size. A biological mercury hotspot of ecological concern occurs where 25 percent or more of the Common Loons sampled in a grid containing at least 14 samples have mercury blood levels above 3.0 ppm.*

Biological Mercury Hotspots



Mercury Levels in Biological Hotspots

Biological Hotspot	State/Province	Yellow Perch		Common Loon		
		Average (ppm)	Range (ppm)	Average (ppm)	Range (ppm)	% of loons > adverse effect level
1a. Adirondack Mountains—west	NY	0.73	0.57-0.96	1.5	1.1-2.1	0%
1b. Adirondack Mountains—central	NY	0.54	0.39-0.80	2.0	0.3-4.1	25%
2. Upper Connecticut River	NH, VT	0.35	0.14-0.58	1.1	0.1-2.9	0%
3a. Merrimack River—middle	NH	0.78	0.05-5.03	2.6	0.7-7.1	28%
3b. Merrimack River—lower	NH, MA	0.65	0.23-3.81	no data		
4a. Upper Androscoggin River	NH, ME	0.44	0.21-1.25	1.9	0.15-5.5	14%
4b. Upper Kennebec River—west	ME	0.40	0.24-0.52	3.1	0.6-14.2	43%
4c. Upper Kennebec River—east	ME	0.38	0.14-0.72	2.2	0.6-4.1	26%
5a. Kejimkujik National Park	NS	0.50	0.14-0.85	5.5	2.9-7.8	93%
5b. Central, Nova Scotia	NS	0.58	0.14-3.79	no data		

Source: Driscoll *et al.* 2007. Courtesy of the Hubbard Brook Research Foundation.

Mercury in the Northeast

During the period from 2001 through 2005, the BioDiversity Research Institute in Gorham, Maine worked together with a group of researchers in the northeast, including the New England states and Canadian provinces, US Fish and Wildlife Service, the Canadian Wildlife Service, and Environment Canada, to compile a comprehensive database on mercury sources and impacts. The database focused primarily on northeastern freshwater environments. The results of the analysis demonstrated that "mercury levels are high and pervasive in northeastern North America." The mercury comes from both atmospheric deposition (with highest levels of mercury in precipitation associated with regional transport from the west and southwest) and from local point sources (Evers and Clair 2005).

An examination of large-scale spatial distribution patterns of mercury in surface waters from Massachusetts to Newfoundland indicated that there were areas of elevated total mercury near the urban regions of Boston and Portland. However, the highest total mercury and methyl mercury were found in flat, wet areas (wetlands) located far from point sources (Dennis *et al*. 2005), likely delivered via atmospheric deposition. Research undertaken by CBEP indicated that atmospheric deposition is the dominant source of mercury to Casco Bay and its watershed (Ryan *et al.* 2003) (see Chapter 2).

Mercury has been found in the northeast in the tissues of aquatic wildlife from crayfish and salamanders to fish, birds, mink, river otters (Evers 2005) and seals (Shaw 2002) (see Chapter 7). Recent studies suggest that even terrestrial insect-eating birds, such as Bicknell's Thrush, a mountain-dwelling woodland songbird, show elevated body burdens of mercury, indicating that methyl mercury can be produced in terrestrial ecosystems in Maine as well (Rimmer *et al*. 2005).

The term "hotspots" is used to describe areas where mercury deposition is unusually high or where the levels of mercury in wildlife are especially elevated in two or more species (biological hotspots). They occur where conditions are especially conducive to methyl mercury production or where there are local emissions sources. Evers *et al*. (2007) identified at least three biological mercury hotspots in Maine, none of which is in the Casco Bay watershed (see Figure 6-1). Additional data are being collected to confirm the number and location of hotspots in Maine.

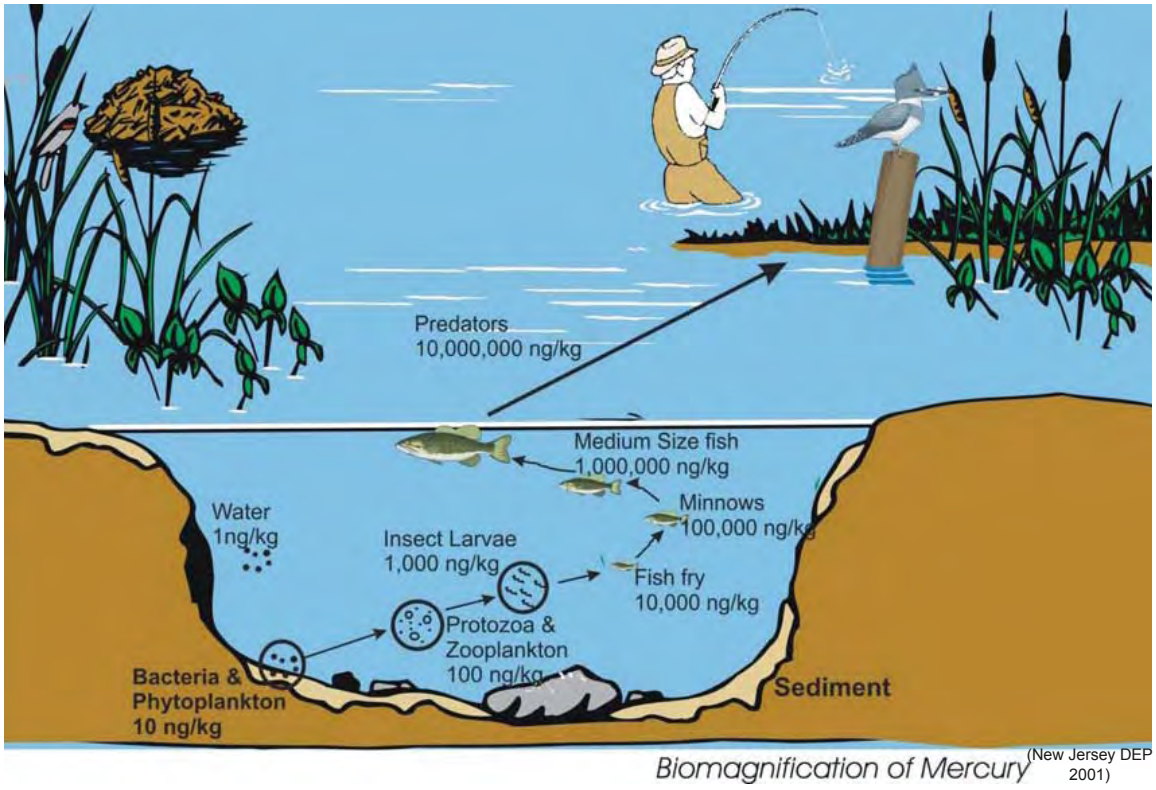


Figure 6-2, Biomagnification of Mercury: *The concentration of a pollutant can increase from one link in a food chain to the next highest trophic level through the process of biomagnification. This diagram illustrates a typical pathway for biomagnification of mercury.*

Maine DEP

This image of a gutted striped bass filled with juvenile alewives from a recent meal illustrates one reason why biomagnification occurs—higher level predators consume large numbers of prey in order to meet their energy requirements.

Maine DEP

Barry Mower from Maine DEP collects a fish blood sample for the Maine SWAT monitoring program.

Mercury in Fish

Through the process of biomagnification, the tissues of predatory freshwater fish near the top of the food chain may contain levels of methyl mercury that are 100,000 to 1,000,000 times higher than the concentration in the water (Maine DEP 2002; Mower 2006) (see Figure 6-2). In 1993, Maine DEP began studying the levels of toxic contaminants, including mercury, in the tissues of fish in lakes and ponds. Of 1800 potential candidate water bodies in Maine, 150 lakes from all over Maine, including some in the Casco Bay watershed, were chosen for study using a statistical design program based on probability sampling. Top predators and omnivorous fish species were collected from each lake. The results of the study indicated that mercury levels in composite tissue samples exceeded the Maine Department of Health and Human Services Level of Concern for human consumption (0.43 ppm or higher) in fish from 65% of the lakes sampled (Maine DEP 2005b). In the Casco Bay watershed, for example, chain pickerel from Forest Lake in Windham had mercury levels of 0.80 to 1.22 ppm in composite tissue samples (DiFranco *et al.* 1995).

These results led the State to issue a mercury health advisory for consumption of fish from Maine lakes and ponds in May 1994. Subsequent freshwater fish sampling through the Maine DEP's Surface Water Ambient Toxic Monitoring Program (SWAT) supports continuation of the health advisory. For example, Maine DEP SWAT sampling conducted in Pleasant Lake in 1998-99 showed mercury levels in fish ranging from a mean of 0.89 ppm in tissues of smallmouth bass and 0.83 ppm in tissues of white perch (Maine DEP 1999). Mercury concentrations in fish from Maine rivers also are elevated and warrant consumption advisories (Maine CDC 2006). The new fish tissue Action Level of 0.2 ppm (wet weight) is also Maine's ambient water quality criterion for human health for mercury. Fish consumption advisories in Maine are discussed further in Chapter 8.

Regional monitoring studies show that there is considerable variation in methyl mercury body burdens among species and types of fresh water bodies across the northeast. For example, bass species, pike, lake trout, white perch and walleye had the highest mercury concentrations of the fish species sampled in the northeast. Surface water characteristics that lead to elevated body burdens in fish include high acidity, presence of wetlands along the shore, low nutrient levels, and a complex food web (Kamman *et al.* 2005, Evers 2005).

Throughout the Gulf of Maine, elevated body burdens of mercury have also been found in saltwater fish, including swordfish, shark and tuna. Consumption advisories have been issued for these species by the Maine Center for Disease Control and Prevention. While humans may be protected from the health impacts of mercury-laden fresh and saltwater fish by public advisories, top-level predators dependent on fish as their main source of food are potentially at risk. The following sections examine the impact of trophic level transfer of mercury from fish to fish-eating birds. Chapter 7 addresses the impacts of mercury and other toxic chemicals on seals in Maine and Casco Bay.

Mercury in Fish-Eating Birds

Predatory birds whose diet is high in fish are at risk of both sub-lethal and lethal effects of mercury poisoning. Over their lifetime, predatory birds can accumulate a substantial body burden of mercury through biomagnification. The impacts of mercury on birds can be manifested in individuals as well as in entire populations through changes in behavior, reproduction and body chemistry (Evers 2005). It is difficult to assess these impacts and risks to fish-eating wildlife because the bioavailability of mercury to fish varies geographically, is influenced by the age and species of fish consumed, and because bird species often feed from multiple aquatic habitats. With such broad ecological variation, it is necessary to sample multiple target species in a variety of habitats that can represent the broader biological community. These selected species serve as indicator organisms or "biosentinels" (Lane *et al*. 2004). Belted Kingfishers, Bald Eagles, and Common Loons are examples of fish-eating indicator species.

Mercury in Belted Kingfishers

Belted Kingfishers (*Ceryle alcyon*) are found throughout Maine in areas where fish are available as food, including small streams, large rivers, ponds, lakes and estuaries. They feed on a variety of fish species ranging from 4 to 14 centimeters in length (as well as crayfish, insects and small amphibians) (Davis 1982). Because the bird is common and widely distributed, it was assessed as a potential methyl mercury biosentinel species in a study sponsored by the Maine DEP's SWAT monitoring program. The 68 nest sampling sites included the Androscoggin and Kennebec River watersheds as well as Flagstaff Lake/Dead River Reservoir, Merrymeeting Bay (estuarine habitat), and Casco Bay (Lane *et al.* 2004). The 4 Kingfisher nests sampled in Casco Bay were located at Winslow Park, Freeport.

During the four-year study, blood and feathers were collected for analysis from adults and young. Prey fish were also sampled. The results indicated that, compared to birds from Michigan, Vermont and Massachusetts, Maine's Belted Kingfishers had higher blood levels of methyl mercury. This is likely due to distribution patterns of mercury as it is transported by the atmosphere from the west to the east. The lowest levels of blood mercury were found in the marine birds (Casco Bay). Samples from Casco Bay, Merrymeeting Bay and the rivers fell below 1 ppm, a value considered to be below a No Observed Adverse Effect Level (NOAEL) critical concentration (US EPA 1997). Birds from the lake/reservoir habitat had much higher levels, with several exceeding the 1 ppm, a level at which there can be reproductive impairments (Lane *et al*. 2004). The study suggests that Kingfishers eating a diet of marine fish have lower exposure to methyl mercury than estuarine, river and especially lake/reservoir birds.

C. Schlawe

Belted Kingfisher

Mercury in Bald Eagles

In 2001-2006, researchers studied fresh-water based populations of Maine bald eagles (*Haliaeetus leucocephalus*) to determine if exposure to dietary mercury may be slowing the recovery of Maine's eagle population. Researchers visited nests to collect nestling blood samples, which reflect recent dietary uptake, and shed adult feathers, which reflect mercury bioaccumulated over time. Sampling sites were distributed throughout Maine in lake and river habitats, including a site on Little Sebago Lake in the Casco Bay watershed. Preliminary results from sampling over 300 nestlings from over 200 nests during 2001-2006 suggested a statistically significant negative correlation between nestling blood mercury exposure and productivity, and no relationship between adult feather mercury and productivity. Researchers found that eagle mercury exposure patterns on Maine's landscape were often consistent

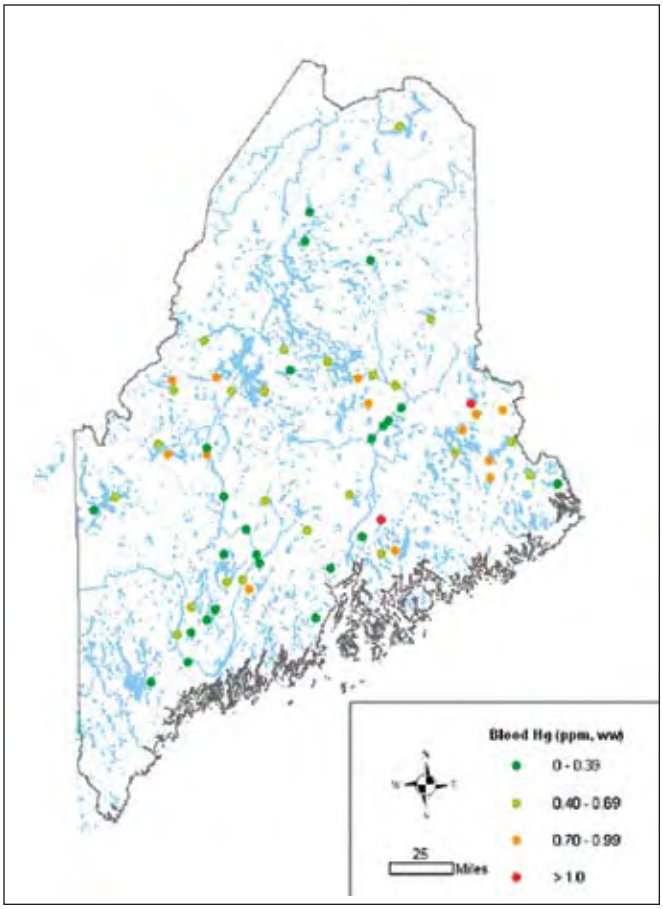


***Figure 6-3**: Mercury exposure indicated by eagle nestling blood samples 2001-2006, (DeSorbo* et al. *2006)*

Christian Niven

Researcher Chris DeSorbo climbs to an eagle nest to collect nestling blood and adult feather samples.

Chris DeSorbo

Young eagles are exposed to elevated levels of methyl mercury through their fish diet in lake and river ecosystems in Maine. Results from studies of 4.5 to 8 week old nestlings indicates that there is a statistically significant negative correlation between nestling blood mercury exposure and productivity (DeSorbo and Evers 2005)

with those observed in other wildlife and fish, and this information is being used to identify regions of specific mercury concern (DeSorbo *et al.* 2006, DeSorbo and Evers 2005) (see Figure 6-1). Comparisons to eagle mercury levels documented in 1991-1992 (Welch 1994) indicated that dietary mercury exposure may be similar on lakes, and potentially higher in rivers, in comparison to 14 years ago. Researchers will also analyze PCBs and DDE (a metabolite of DDT) in nestling eagle blood samples in order to determine how these chemicals relate to productivity (DeSorbo *et al*. 2006).

In all the eagle tissues analyzed, samples from Maine lakes displayed significantly higher mercury levels than samples from Maine rivers. Mercury exposure in Maine eagle tissues are elevated in comparison to most populations in the U.S., and most comparable to populations associated with significant point source pollution problems (*i.e.,* dredging, mercury mines). While nestling eagle blood mercury levels from the Casco Bay watershed indicated low mercury exposure in the local foodweb (see figure 6-3), feather samples from territorial adult eagles indicated significantly elevated exposure and bioaccumulation to levels of concern (DeSorbo *et al*. 2006, DeSorbo and Evers 2005).

Mercury in Common Loons

The Common Loon *(Gavia immer)* is a long-lived bird that is found throughout New England. It has emerged as a suitable biosentinel species, serving as an indicator of aquatic methyl mercury pollution. Studies of the New England breeding loon populations conducted from 1994-2003 show that the birds are at a high level of risk to mercury contamination. In Maine, 22% of the breeding population is considered to be at risk (Evers *et al.* 2004). During the study period, 324 abandoned eggs and blood and feathers from 408 adults and 142 juvenile Common Loons were collected from Maine lakes. In addition, a focused study was conducted in the Rangely Lakes area. Loon blood mercury levels from Forest Lake in Windham were quite high, perhaps because of the lake's proximity to the Portland municipal incinerator (Evers 2006). The results of the loon studies were used to relate mercury to behavioral and reproductive impacts. The studies confirmed that mated pairs whose blood levels exceeded the Low Observed Adverse Effect Level (LOAEL) produced 40% fewer fledged young than birds with mercury blood levels below the NOAEL (Evers *et al*. 2004).

The physiological impacts of increasing levels of mercury in the blood of loons were observed using two indicators: increasing corticosterone hormone stress levels (which can lead to suppression of the immune system) and asymmetry of flight feathers (which may be related to disruption of embryonic development and overall decline in reproductive fitness). Behavioral changes were also observed with increasing methyl mercury exposure. High risk adults left eggs abandoned and showed reduced hunting and foraging. All of these impacts challenge the birds' ability to maintain their population successfully.

Mercury in Insect-Eating Birds: The Saltmarsh Sharp-tailed Sparrow

A study conducted in 2004-2005 suggests that a small estuarine bird, the Saltmarsh Sharp-Tailed Sparrow, may be a good indicator of methyl mercury availability to insect-eating birds in Maine and New England. Sparrow blood was collected at Scarborough Marsh State Wildlife Management area, Libby River and Nonesuch River estuaries in Scarborough, and five estuaries in the Rachel Carson National Wildlife Refuge. In addition, samples were collected at sites in Massachusetts, Rhode Island, and Connecticut. The sparrows had elevated blood mercury levels at all the sampling sites. While the blood mercury concentrations were highest in the Parker River National Wildlife Refuge in Massachusetts) and Ninigret Marsh National Wildlife Refuge in Rhode Island, the 62 birds sampled in Maine had blood concentrations ranging between 0.23 and 0.84 parts per million. For several sites in Maine, the mercury concentration exceeded levels considered to impact the health of insect-eating songbirds (Lane and Evers 2005, 2006). This study suggests that invertebrates (such as insects) in the food chain in freshwater and estuarine wetlands are an important part of the mercury bioaccumulation problem that is just now being discovered (Evers 2006).

Oksana Lane

Researcher collects a blood sample from a Saltmarsh Sharp-tailed Sparrow using a capillary tube.

Management Tools to Protect Freshwater Wildlife from Mercury

In an effort to provide a wildlife management tool applicable throughout Maine, researchers have been using a modeling approach to develop a Maine-based wildlife criterion value (WCV). Current models of loon populations in Maine suggest that breeding population sinks exist (*i.e.* areas where loons attempt to nest but are unsuccessful because of mercury concentrations in the environment). A WCV is under development that would indicate the maximum allowable total mercury concentration in fresh water that is protective of loons at the population level. WCV levels are also in development for mink and river otters, animals that are also highly susceptible to elevated levels of methyl mercury due to their fish-heavy diet and rapid metabolism (Evers *et al.* 2004, US EPA 1997, Yates *et al*. 2004).

Summary/Conclusions

Mercury levels are elevated in many Maine species, including freshwater fish species, some marine fish species, fish-eating birds and mammals, and even in insect-eating birds. Elevated blood levels and health impacts from exposure to methyl mercury have been observed in populations of bald eagles and loons from Casco Bay. The widespread bioaccumulation of mercury in fish tissues has led to fish consumption advisories for human consumers throughout Maine (see Chapter 8). Protection of Maine's animal species from the impacts of mercury will require an ongoing commitment to dramatically reduce inputs of mercury into the environment. See Chapter 9 for a discussion of state and federal efforts to reduce the loading of mercury to our Bay ecosystem.

Ron Singer

A wildlife criterion value (the maxiumum total allowable total mercury concentration in fresh water protective at the population level) is under development for river otters, such as the animal shown above, as well as mink and loons.

References

Chan, H.M, Scheuhammer, A.M., Ferran, A., Loupelle, C., Holloway, J., and S.Weech. 2003. "Impacts of Mercury on Freshwater Fish-Eating Wildlife and Humans: Human and Ecological Risk Assessment" *Hum. Ecol. Risk Assess*. 9 (4) 867-883.

Davis, W.J. 1982. Territory size in *Megaceryle alcyon* along a stream habitat. *Auk.* 99. pp. 353-362.

Dennis, I.F., T.A. Clair, C.T. Driscoll, N. Kamman, A. Chalmers, J. Shanley, S.A. Norton, and S. Kahl. 2005. "Distribution patterns of mercury in lakes and rivers of northeastern North America." *Ecotoxicology*. 14. 113-125.

DeSorbo, C.R. and D.C. Evers. 2005. *Evaluating exposure of Maine's Bald Eagle population to Mercury: assessing impacts on productivity and spatial exposure patterns*. Report to Maine Department of Environmental Protection, no. BRI 2005-08. BioDiversity Research Institute, Gorham, Maine. 27 pp.

DeSorbo, C.R., C.S. Todd, D.C. Evers, S.E. Mierzykowski, W. Hanson, W.W. Bowerman, C. Romanek, and R. Taylor. 2006. *Evaluating exposure patterns and impacts of methylmercury on freshwater-feeding Bald Eagles in Maine, USA*. Poster presented at Mercury 2006: Conference on Mercury as a Global Pollutant, Madison, Wisconsin. August 6-11, 2006.

DiFranco, J., L. Bacon, B. Mower, and D. Courtemanch. 1995. *Fish Tissue Contamination in Maine Lakes: Data Report.* Maine Department of Environmental Protection. Funded through USEPA Regional Environmental Monitoring and Assessment Program (REMAP).

Driscoll, C.T., D. Evers, K.F. Lambert, N. Kamman, T. Holsen, Y-J Han, C. Chen, W. Goodale, T. Butler, T. Clair, and R. Munson. *Mercury Matters: Linking Mercury Science with Public Policy in the Northeastern United States*. Hubbard Brook Research Foundation. 2007. Science Links Publication. 1 (3).

Eisler, R. 1987. Mercury hazards to fish, wildlife and invertebrates: a synoptic review. *U.S. Fish and Wildlife Service. Biological Report*. 85.

Evers, D. C., O. P. Lane, L. Savoy, and W. Goodale. 2004. *Assessing the impacts of methylmercury on piscivorous wildlife using a wildlife criterion value based on the Common Loon, 1998-2003*. Report to Maine Department of Environmental Protection no. BRI 2004-05. BioDiversity Research Institute, Gorham, Maine.

Evers, D. C. 2005. Mercury Connections: *The extent and effects of mercury pollution in northeastern North America*. Biodiversity Institute. Gorham. Maine. 28 pp.

Evers, D.C. and T.A. Clair. 2005. Mercury in northeastern North America: a synthesis of existing databases. *Ecotoxicology.* 14 (1,2) 7-14.

Evers, D. 2006. BioDiversity Research Institute. Personal Communcation.

Evers, D.C., Y-J Han, C.T. Driscoll, N.C. Kamman, M.W. Goodale, K.F. Lambert, T.M.Holsen, C.Y. Chen, T.A. Clair, and T. Butler. 2007. Biological Mercury Hotspots in the Northeastern U.S. and Southeastern Canada. *BioScience*. 57 (1).

Kamman, N.C., N.M. Burgess, C.T. Driscoll, H.A. Simonin, W. Goodale, J. Linehan, R. Estabrook, M. Hutcheson, A. Major, A.M. Scheuhammer, and D.A. Scruton. 2005. Mercury in freshwater fish of northeast North America—A geographic perspective based on fish tissue monitoring databases. *Ecotoxicology*. 14 (1,2) 163-180.

Lane, O., D. Evers, D. Albano, T. Haines and R. Taylor. *Belted Kingfishers as indicators of methyl mercury availability in aquatic systems (1997-2003)*. 2004. Submitted to Maine Department of Environmental Protection Surface Water Ambient Toxic Monitoring Program. BioDiversity Research Institute. Gorham, Maine.

Lane, O. and D. Evers. 2005. *Developing a geographic exposure profile of methylmercury availability in salt marshes of New England*. Report BRI 2005-04. BioDiversity Reserch Institute. Gorham, Maine.

Lane, O. and D. Evers. 2006. *Methylmercury availability in New England estuaries as indicated by Saltmarsh-Sharp-Tail Sparrow, 2004–2005*. Report BRI 2006-01. BioDiversity Research Institute. Gorham, Maine.

Maine Center for Disease Control. 2006. *Freshwater Fish Safe Eating Guidelines*. (http://www.maine.gov/dhhs/eohp/fish/2KFCA.htm) (May 16, 2006).

Maine Department of Environmental Protection. 1999. *SWAT Monitoring Program Report. Part 2. Lakes*. (http://mainegov-images.informe.org/dep/blwq/docmonitoring/swat/99lakes.pdf) (August 8, 2006)

Maine Department of Environmental Protection. 2002. *Mercury in Maine. A Status Report.* Prepared for the Joint Standing Committee of the Maine Legislature Having Jurisdiction Over Natural Resources.

Maine Department of Environmental Protection. 2005a. *Mercury: A Significant Environmental Problem*. (http://www.maine.gov/dep/mercury/index.htm) (May 16, 2006).

Maine Department of Environmental Protection. 2005b. *Fish Tissue Contamination in the State of Maine*. (http://www.maine.gov/dep/blwq/hg_pres.htm) (April 7, 2006).

Mower, B. 2006. Maine Department of Environmental Protection. Personal Communication.

NESCAUM (Northeast States for Coordinated Air Use Management). 1998. *Northeast States and Eastern Canadian Provinces mercury study.* NESCAUM/NEWMOA/ NEWPCC/EMAN. Boston, MA.

New Jersey Department of Environmental Protection. 2001. *New Jersey Mercury Task Force. Vol. 1. Executive Summary and Recommendations*. (http://www.state.nj.us/dep/dsr/nj-mercury-volume1.PDF) (December 5, 2006).

Rimmer, C.C., K.P. Mcfarland, D.C. Evers, E.K. Miller, Y. Aubrey, D. Busby, and R.J. Taylor. 2005. Mercury concentrations in Bicknell's Thrush and other insectivorous passerines in montane forests of northeastern North America. *Ecotoxicology.* 14 (1,2) 223-240.

Ryan, P.A., H.R. Hafner, and S.G. Brown. 2003. *Deposition of Air Pollutants to Casco Bay.* Casco Bay Estuary Partnership.

Shaw, S.D. 2002. *An investigation of persistent organic pollutants and heavy metals I tissues of harbor seals* (Phoca vitulina concolor) *and gray seals* (Halichoerus grypus) *in the Gulf of Maine.* Final Report to the Maine Department of Environmental Protection, Augusta, ME, 16 pp.

United States Environmental Protection Agency. 1997. *Mercury Study Report to Congress. Volume VI: An ecological assessment of anthropogenic mercury emissions in the United States*. 452/R-97-008.

Welch, L. 1994. *Contaminant burdens and reproductive rates of bald eagles breeding in Maine.* M.S. Thesis, University of Maine, Orono, Maine, USA. 86pp.

Yates, D., D.C. Evers, and L. Savoy. 2004. *Developing a mercury exposure profile for mink and river otter in Maine.* Report BRI 2004-09 submitted to Maine Department of Environmental Protection and Maine Inland Fisheries and Wildlife. Biodiversity Research Institute, Gorham, Maine.

7 How are seals, as top predators, impacted by toxic contaminants in Casco Bay and the Gulf of Maine?

Harbor seal (Phoca vitulina concolor), *the most abundant marine mammal in the Gulf of Maine and mid-Atlantic region.*

Peter Ralston

Introduction

Harbor seals *(Phoca vitulina)* are widely distributed in the temperate near-shore waters of the northern hemisphere and are important indicators of coastal contamination because they occupy a high trophic level, are long-lived (35-40 years), and accumulate high concentrations of persistent organic pollutants (POPs) and mercury through the food chain. Lipophilic (fat soluble) POPs including PCBs, dioxins, and DDT build up in fatty tissues such as blubber and have been shown to cause immune- and endocrine-disrupting effects in seals and other marine wildlife (De Swart *et al.* 1994, De Guise *et al.* 2001). Evidence amassed over three decades suggests that these compounds have caused reproductive impairment, hormone abnormalities, and population declines in seals inhabiting industrialized regions of Europe, North America, and Asia. It is widely believed that immunotoxic chemicals such as PCBs and dioxins have played a role in the recurring distemper virus outbreaks and mass mortalities reported among seals since the 1980s, by altering the animals' normal immune resistance to disease (Dietz *et al*. 1989, Van Loveren *et al.* 2000, Harding *et al.* 2002). Unlike POPs, mercury preferentially accumulates in muscle and liver tissue, and at high levels, may place young seals at risk for liver damage and immune and neurotoxic effects following exposure *in utero* and through nursing (AMAP 1998, Shaw 2002).

Seals as Sentinels

Dr. Susan Shaw and co-workers at the Marine Environmental Research Institute (MERI), Center for Marine Studies, in Blue Hill, Maine, have been studying the impacts of environmental pollutants on seals in the Gulf of Maine and along the mid-Atlantic coast since 2001 as part of the *Seals as Sentinels* project. This project has generated the first extensive data reported in 25 years on levels and effects of toxic contaminants in northwestern Atlantic harbor seals *(Phoca vitulina concolor)*.

At present, there are an estimated 99,340 harbor seals inhabiting the northwestern Atlantic coast extending from the Gulf of Maine southward to the coast of New Jersey (Gilbert *et al*. 2005). Considered relatively non-migratory, harbor seals feed on a variety of fish including hake, herring, alewife, haddock, redfish, and winter flounder in coastal and estuarine environments and are exposed to contaminated habitats and prey across their range. In the southerly portion of the range, coastal urban development has resulted in some of the densest concentrations of human populations in North America, and environmental pollution has been a concern at least since the 1950s. Similar to European seals, the harbor seal population has experienced a series of mass mortalities since the 1980s (Geraci *et al*. 1982, Duignan *et al.* 1995). The most recent event occurred in 2004 among harbor seals in southern Maine where approximately 300 animals, primarily pups, were found dead on beaches in and around Saco Bay. The possible role of immunotoxic chemicals (*e.g.,* PCBs, dioxins) in these outbreaks is not clear.

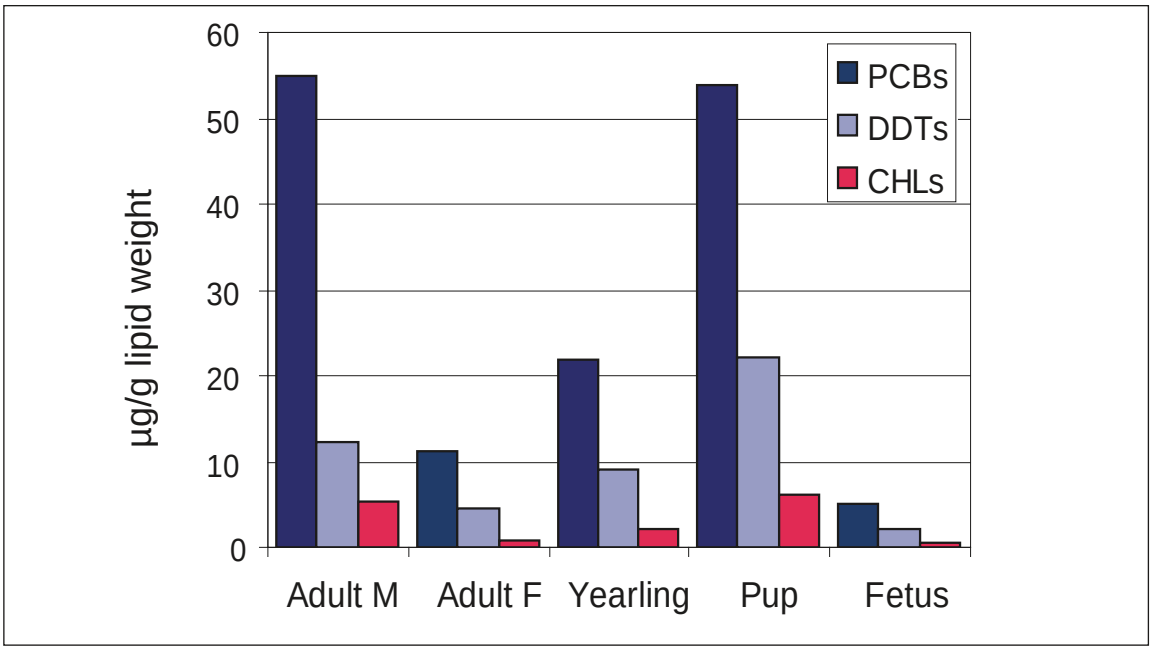


Figure 7-1: *Major POPS (PCBs, DDT, CHLs) (µg/g, lipid wt) in harbor seal blubber by age class*

Contaminant Levels in Seals

A total of 34 stranded harbor seals *(Phoca vitulina concolor)* and 3 gray seals *(Halichoerus gryphus)*, primarily pups and yearlings, were collected by MERI at locations along the coast from Mount Desert Island, Maine to Long Island, New York between 1991 and 2001-2002. Seal blubber, liver, and kidney samples were analyzed for a wide range of organic contaminants and metals. PCBs, DDT, and chlordane-related compounds (CHLs), chemicals which were banned in the U.S. in the late 1970s, were the predominant organic compounds found in harbor seal tissues, reflecting the extreme persistence of these substances in the marine food chain. The highest concentrations were found in the adult male harbor seals and pups, with mean PCBs of 55 and 54 µg/g, lipid weight (lw), respectively, followed by the yearlings, adult females, and fetuses (see Figure 7-1). Gray seals tend to have lower levels of POPs than harbor seals (PCBs 18-27 µg/g, lw) which is likely due to their pelagic migratory patterns and feeding habits.

The accumulation pattern in the harbor seals reflects an age-dependent increase in adult males, whereas females lower their levels by transferring a proportion of their body burdens to pups (Addison and Brodie 1977). In phocid (true or earless) seals, lipophilic POPs are transferred from maternal lipid stores to some extent during gestation but particularly during lactation, so that the body burdens of PCBs and DDTs are often higher in pups at weaning than in their mothers. Although MERI did not examine mother-pup pairs, levels in pups were five times higher than those in adult females. Compared with the fetuses, pups had PCB burdens an order of magnitude higher, reflecting the greater importance of breast milk as an exposure route.


Cynthia Stroud

Harbor seal mother making first contact with newborn pup, mid-coast Maine.

Lactational transfer may also pose an increased toxic risk to pups compared with that of adult exposure through feeding. As the lactating seal does not feed, the bulk of her circulatory lipids are derived from the blubber layer rather than from lipid sources in her diet (Addison and Brodie 1987). During the fasting period, as the mother loses weight, the nursing pup may be exposed to the more toxic PCBs mobilized from the mother's fat stores, as compared with lower chlorinated, relatively less toxic PCBs which, if the mother were feeding, would be obtained from fish.

Persistent Organic Pollutants

Figure 7-2a shows mean concentrations of PCBs and DDTs in blubber of harbor seals (all ages) from different regions of the northwestern Atlantic. The animals from southern Maine were harbor seal pups and yearlings collected in Cape Elizabeth (Casco Bay), Saco, Wells and Kennebunk. Across the range, PCB concentrations in these seals exceed the estimated threshold value of ~17µg PCB/g, lw (ppm) in blubber for adverse effects including effects on immune and endocrine functions in the species (Kannan *et al*. 2000). Region-wide, the highest concentrations were found in the seals from Narragansett Bay/Long Island Sound, although this distribution undoubtedly reflects the large effect of age class on body burdens.

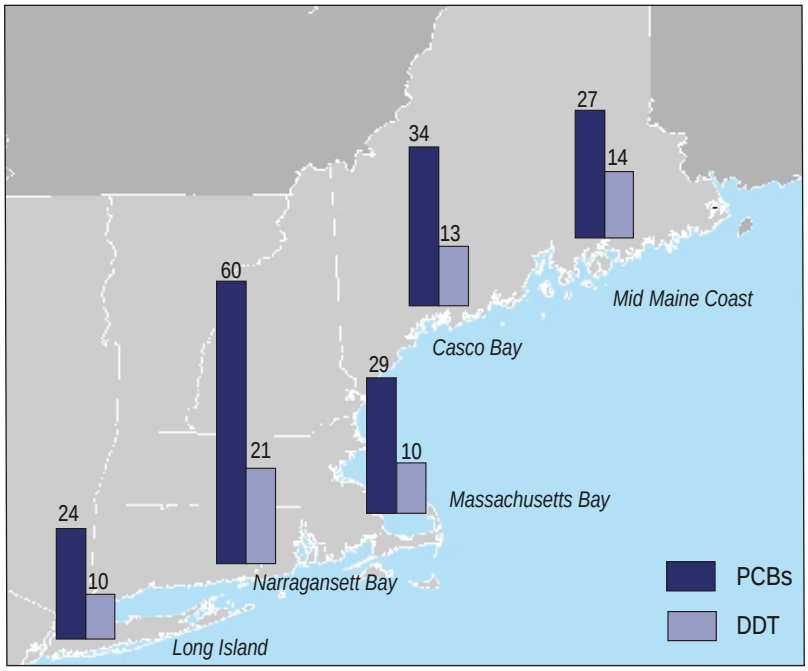


Figure 7-2a: *PCB and DDT concentrations (µg/g, lipid wt) in blubber of NW Atlantic harbor seals*

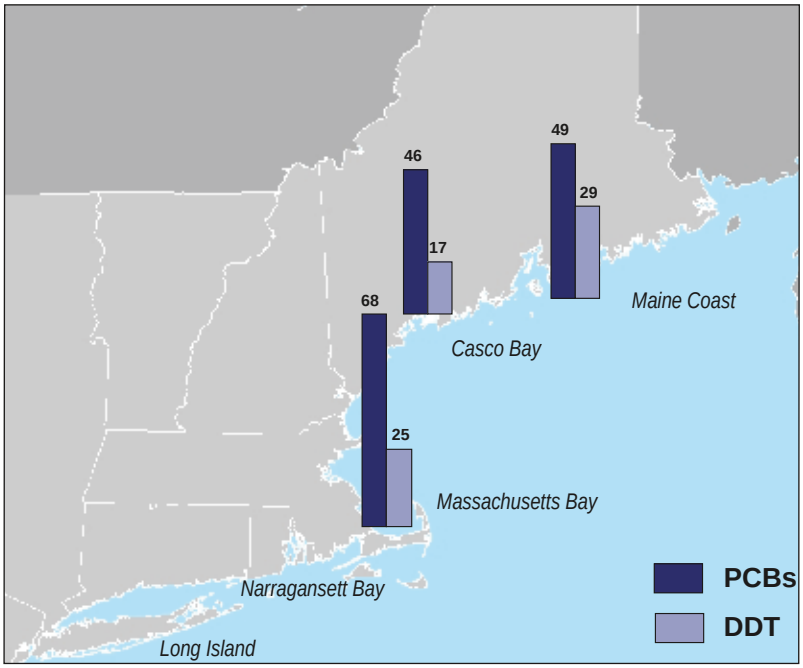


Figure 7-2b: *PCB and DDT concentrations (µg/g, lipid wt) in blubber of harbor seal pups*

Figure 7-2b shows the higher PCB and DDT levels in harbor seal pups from three regions—mid-coast Maine, southern Maine, and Massachusetts Bay. The highest PCB concentrations (mean 67.5 µg/g, lw) were found in pups from Massachusetts Bay, whereas levels were slightly lower (mean 46 and 49 µg/g, lw) in pups from Casco Bay/southern Maine and eastern Maine, respectively. In the four pups from Casco Bay/southern Maine, PCB concentrations were highly variable, ranging from 11 to 110 µg/g, lw.

As is clear from Figures 7-2a and 7-2b, harbor seals from the northwestern Atlantic have elevated tissue burdens of toxic organic contaminants that place them at risk for adverse health effects (Shaw *et al*. 2005). This is especially true for the seal pups, which may be vulnerable to health impacts when concentrations are an order of magnitude lower (Shaw *et al*. 1999). In fact, the levels of PCBs found in these pups were 18 times higher than the concentrations (~3 µg/g, lw) associated with altered immune and endocrine function biomarkers (indicators) in stranded, rehabilitated harbor seal pups from the California coast (Shaw *et al*. 1999).

One of these markers is the lymphocyte proliferation assay. Lymphocytes are a type of white blood cell—T and B cells—involved in immune response to foreign substances. The assay measures the ability of the circulating lymphocytes to respond to foreign substances *in vitro* (*i.e,* in cell culture). This assay is an important indicator of contaminant-induced alterations in nonspecific immune function. A lowered proliferative response is indicative of an animal's reduced ability to resist infection by viruses and other pathogens, while an enhanced response may reflect autoimmune disease or cancer. Recently, Levin *et al.* (2005) reported enhanced lymphocyte proliferative responses in free-ranging harbor seals from British

Columbia with mean PCB concentrations in blubber as low as 2.5 g/g, lw. This is consistent with earlier findings by Shaw *et al.* (2003) of PCB-dioxin-related immune enhancement in free-ranging adult harbor seals from the Gulf of Maine.

Thyroid hormone levels and retinols (vitamin A) in plasma are important biomarkers of contaminant-induced endocrine disruption. Adequate levels of thyroid hormones and vitamin A are critical to normal growth and development, including development of the immune system, the reproductive system, and the brain. Exposure to PCBs and related POPs can reduce hormone and retinol levels in animals and humans by various mechanisms such as competitive binding to receptors on carrier proteins.

MERI

Week-old harbor seal pup rescued at Blue Hill Falls, mid-coast Maine.

Mercury and Other Metals

Concentrations of mercury found in liver of the adult harbor seals are shown in Figure 7-3 (Shaw, unpublished data). Seal liver and kidney samples were also tested for arsenic, cadmium, chromium, lead, silver, selenium, copper and zinc, but these metals were not detected at levels of concern. While hepatic (liver) mercury levels in the younger seals were relatively low, concentrations in adult seals (mean 64.8 µg/g, wet weight) exceed the threshold level of 60 µg/g, ww, for liver damage in mammals (AMAP 1998). Elevated mercury levels are known to be common in livers of marine mammals, and seals have evolved biochemical mechanisms involving selenium to detoxify (demethylate) and store mercury in the form of less toxic (divalent) mercury-selenide complexes (Wagemann *et al.* 2000). However, the ability to detoxify and store mercury may not be present in newborn and young seals following exposure to the mother's burden *in utero* and while nursing, thus, these young and developing seals may be at risk for mercury-related neurotoxicity and other effects.

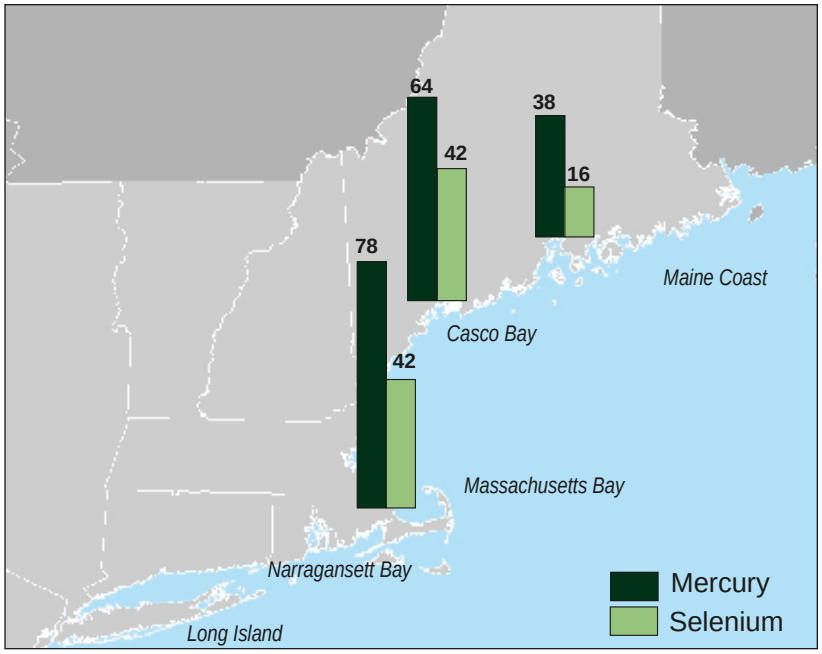


Figure 7-3: *Mercury and Selenium concentrations (µg/g, wet wt) in liver of adult harbor seals*

Temporal Trends

DDT and, to a lesser extent, PCB burdens in northwestern Atlantic harbor seals have declined from the very high levels reported in the 1970s (Gaskin *et al.* 1973, Shaw *et al.* 2005). Between 1971 and 2001, DDT levels in harbor seal blubber (all ages) decreased by ~82% while PCB levels decreased by ~66%. In the adult males and pups, a smaller decline of ~45% in PCB levels was observed over this thirty year period. This is consistent with trends in other industrialized areas where a more rapid decline of DDT was observed after these compounds were banned (Kennish 1992), while PCBs are still being released from stockpiled residues (Tanabe 1988). In seals from the highly polluted Baltic Sea, DDT levels have decreased by 72-85% since the 1970s, while PCB levels showed only

a minor decrease of 25% in females and no decrease in males (Nyman *et al.* 2002). A similar trend was observed in most Arctic marine mammal populations (AMAP 2000).

To examine changes over the past decade, MERI compared contaminant levels in blubber of yearling harbor seals collected in 1991 and 2001-2002 (Shaw *et al.* 2005). Due to the small sample size (n=3) of the 1991 samples, no conclusions could be drawn, but the data show only small decreases in absolute concentrations of the major contaminant groups, PCBs, DDTs, and CHLs, in seal blubber over this ten-year period, suggesting an equilibrium in environmental cycling of these POPs in the northwestern Atlantic.

Global Comparisons

The levels of PCBs and DDTs found in northwestern Atlantic harbor seals are at the upper middle of the contamination spectrum on a global scale (see Figure 7-4). PCB concentrations in the adult males and pups (55 and 54 µg/g, lw) are approaching the high levels reported in stranded seals from the polluted Baltic Sea, Wadden Sea, western Mediterranean, and Caspian Sea (Luckas *et al*. 1990, Borrell *et al*. 1997, Kajiwara *et al*. 2002), and are slightly higher than levels reported in blubber of harbor seals from the the coasts of Denmark (Storr-Hansen and Spiid 1993), eastern England (Law *et al.* 1989) and northern Ireland found during the 1988 morbillivirus epizootic (an epidemic among animals) (Mitchell and Kennedy 1992). Compared with Pacific coast seals, PCB concentrations in the harbor seal pups, including the four pups from Casco Bay/southern Maine, are three-fold higher than those reported in stranded harbor seal pups from southern Puget Sound, Washington, an area considered relatively polluted (Shaw 1998, Hong *et al.* 1996), and an order of magnitude higher than the levels reported in stranded harbor seal pups from the California coast (Shaw *et al.* 1999).

DDT concentrations in harbor seal pups sampled by MERI were similar to those of Baltic seals and western Mediterranean monk seals *(Monachus monachus)* (Luckas *et al.* 1990; Borrell *et al.* 1997), reflecting the widespread production and application of DDT in these areas. However, DDT levels in the pups were an order of magnitude lower than the extremely high concentrations found in Caspian seals *(Phoca caspica)* (Kajiwara *et al*. 2002), reflecting recent uses of this pesticide in the former USSR (Federov 1999).

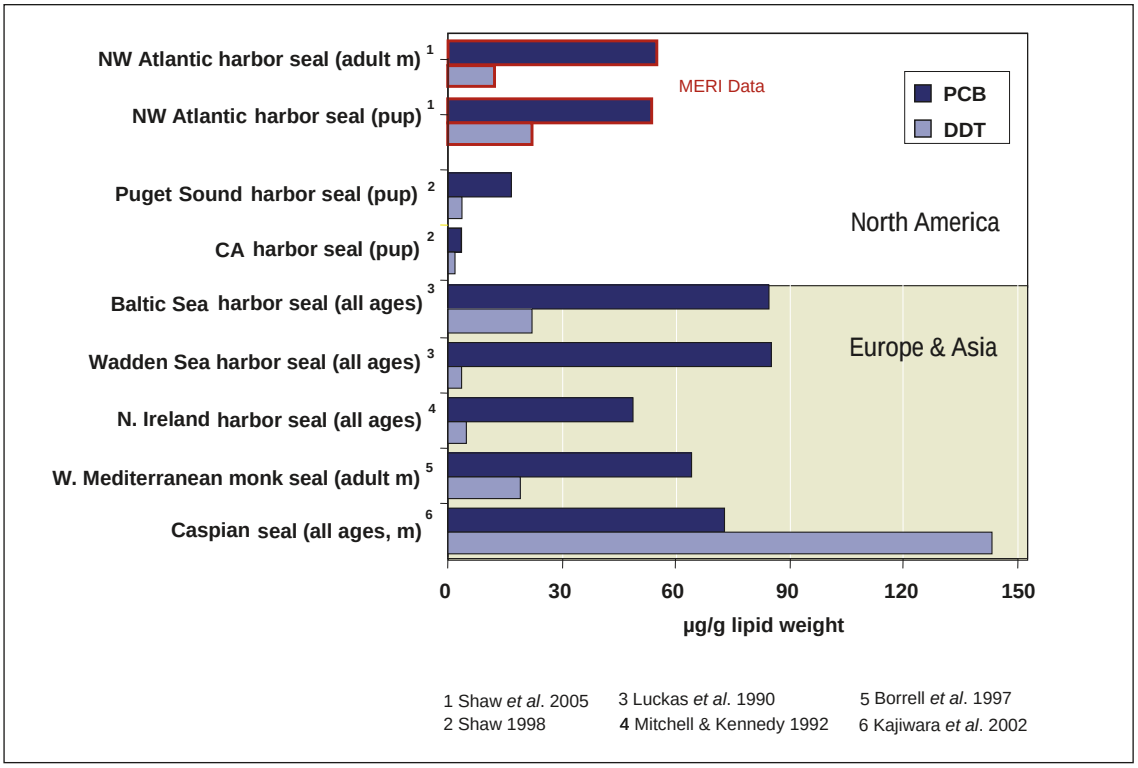


Figure 7-4: *PCB and DDT concentrations (µg/g, lipid wt) in pinnipeds from different regions*

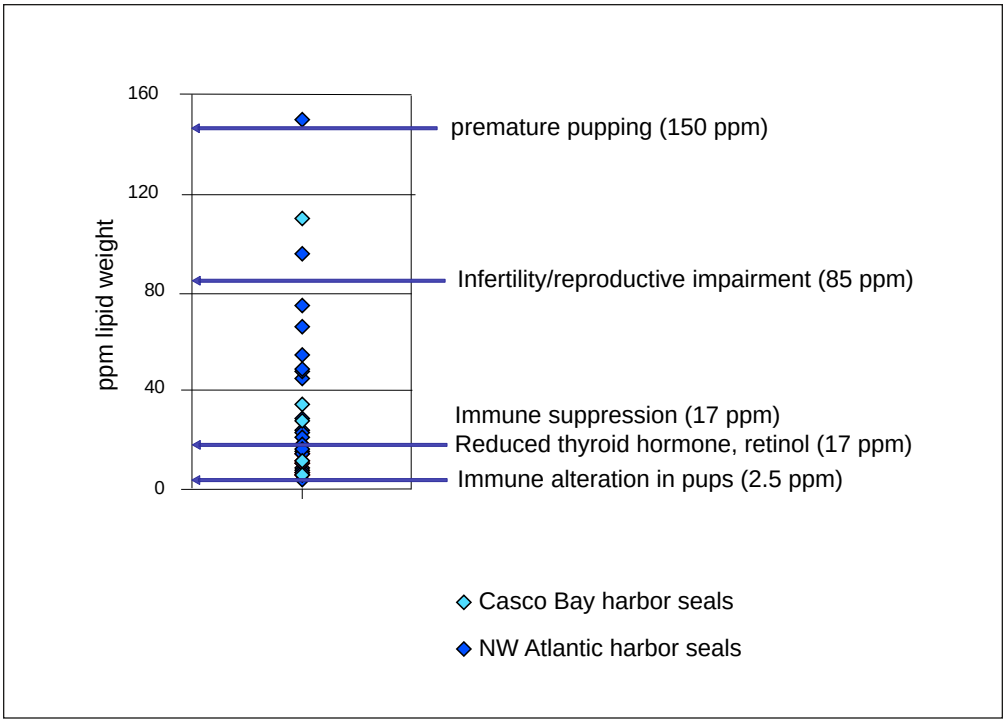


Figure 7-5: *PCB concentrations (ppm, lipid wt) in NW Atlantic harbor seals and estimated threshold levels of effects in pinnipeds*

Mercury levels in liver of the adult harbor seals (males and females) were similar to those reported in adult seals from other polluted areas including grey and ringed seals (*Phoca hispida*) from the Baltic and harp seals (*Phagophilus groenlandicus*) from the Greenland Sea (Nyman *et al.* 2002; Fant *et al.* 2001; Brunborg *et al.* 2005). Their mercury levels were two-fold higher than the levels found in Greenland hooded seals *(Crystophora cristata)* (Brunborg *et al.* 2005) and an order of magnitude higher that those of ringed seals from Svalbard, Norway, (Fant *et al.* 2001). Much higher concentrations (mean 134-4250 µg/g, ww) were reported in livers of dolphins from the Mediterranean Sea (Frodello *et al*. 2000).

Cynthia Stroud

Harbor seal with three-week old nursing pup

Toxic Impacts: Conclusions

As shown in Figure 7-5, PCB burdens in harbor seals from the northwestern Atlantic exceed the estimated threshold level of 17µg PCB/g, lw in blubber for adverse effects on immune function (Kannan *et al*. 2000), and fall within the estimated threshold level of 25-77 µg PCB/g, lw for reproductive effects in marine mammals (AMAP, 2000). PCB burdens in the pups, including those from Casco Bay/southern Maine, are an order of magnitude higher than the concentrations associated with reduced immune responses and hormone levels in stranded harbor seal pups from California (Shaw *et al.* 1999) and with altered immune responses in free-ranging pups from British Columbia (Levin *et al.* 2005). Moreover, in a previous study MERI reported significant correlations between dioxin-like compounds in plasma and altered immune responses in free-ranging adult harbor seals from the Gulf of Maine (Shaw *et al.* 2003).

Mercury concentrations found in liver of adult harbor seals exceed the estimated threshold level of 60 µg/g lw for liver damage in mammals (AMAP 1998), suggesting that harbor seal pups may be exposed to harmful levels of mercury during gestation and lactation. These observations, together with reports of at least two, and possibly three large-scale outbreaks of viral disease among these seals since the 1980s, suggest that the population is currently at risk for contaminant-related health effects. Although the present study was limited by a small sample size distributed over a large geographic area, the toxic impacts of the current POP and mercury body burdens in these seals would be expected to be considerable, particularly among the pups, leading to developmental deficits and compromised immune resilience, which in turn, may place them at risk for future disease outbreaks.

The data generated by the *Seals As Sentinels* project are the first extensive, region-wide data in 25 years on levels and effects of toxic contaminants in harbor seals from the northwestern Atlantic. While levels of the legacy POPs (PCBs, DDT) are slowly declining in marine biota, blubber concentrations in northwestern Atlantic harbor seals declined only slightly over the ten-year period 1991-2001, suggesting that these compounds are at equilibrium in the marine ecosystem. Moreover, thousands of new chemicals are being released every year, and we have recently documented the presence of the widely used flame retardants polybrominated diphenyl ethers (PBDEs), perfluorooctanesulfonate (PFOS), and related perfluorinated chemicals at relatively high concentrations in harbor seal tissues (Shaw *et al*. 2006a,b). These compounds of emerging concern are now being studied for their capacity to biomagnify and provoke effects in marine mammals and humans.

In view of the past vulnerability of northwestern Atlantic harbor seals to viral outbreaks, there is a clear need for continued research on larger sample sizes to ascertain body burdens and toxic impacts of the complex mixtures of contaminants to which these seals are exposed.

References

Addison, R.F, Brodie, P.F. 1977. Organochlorine residues in maternal blubber, milk, and pup blubber from grey seals *(Halichoerus grypus)* from Sable Island, Nova Scotia. *J. Fish Res. Bd Can.* 34, 937-941.

Addison, R.F., Brodie, P.F. 1987. Transfer of organochlorine residues from blubber through the circulatory system to milk in the lactating grey seal *(Halichoerus grypus)*. *Canadian J.Fish.Aquat Sci.* 44(4), 782-786.

AMAP. 1998. *AMAP Assessment Report: Arctic Pollution Issues. Chapter 7: Heavy metals*. Arctic Monitoring and Assessment Programme (AMAP), Oslo, Norway.

AMAP. 2000. *AMAP Assessment Report: Arctic Pollution Issues*. Arctic Monitoring and Assessment Programme (AMAP), Oslo, Norway.

Borrell, A., Aguilar, A. and Pastor, T. 1997. Organochlorine pollutant levels in Mediterranean monk seals from the western Mediterranean and the Sahara coast. *Marine Pollution Bulletin* 34: 505-510.

Brunborg, L.A., Graff I.E., Froyland, L, Julshamn, K. 2005. Levels of non-essential elements in muscle from harp seal *(Phagophilus groenlandicus)* and hooded seal *(Crystophora cristata)* caught in the Greenland Sea area. *The Science of the Total Environment* 366: 784-798.

De Guise, S., Shaw, S.D., Barclay, J., Brock, J., Brouwer, A., Dewailly, E., Fair, P., Fournier, M., Grandjean, P., Guillette, L., *et al.* 2001. Consensus Statement: Atlantic Coast Contaminants Workshop 2000. *Environmental Health Perspectives* 109:1301-1302.

De Swart, R.L., Ross, P.S., Vedder, L.J., Timmerman, H.H., Heisterkamp, S.H., Van Loveren, H., Vos, J.G., Reijnders, P.J.H., and Osterhaus, A.D.M.E. 1994. Impairment of immune function in harbor seals *(Phoca vitulina)* feeding on fish from polluted waters. *Ambio* 23:155-159.

Dietz, R., Heide-Jorgensen, M.P., and Harkonen, T. 1989. Mass deaths of harbor seals *(Phoca vitulina)* in Europe. *Ambio* 18:258-264.

Duignan, P.J., Saliki, J.T., St. Aubin, D.J., Early, G., Sadove, S., House, J.A., Kovacs, K., Geraci, J.R. 1995. Epizootiology of morbilli virus infection in North American harbor seals *(Phoca vitulina)* and gray seals *(Halichoerus grypus). J. Wildlife Diseases* 31(4), 491-501.

Fant, M.L., Nyman, M., Helle, E., Rudback, E., 2001. Mercury, cadmium, lead and selenium in ringed seals (Phoca hispida) from the Baltic Sea and from Svalbard. *Environ. Pollution* 111, 493–501.

Federov, L.A. 1999. Persistent organic chemicals in the former Soviet Union. *Environ. Pollution* 105:283-287.

Frodello, JP, Romeo, M., Viale, D. 2000. Distribution of mercury in the organs and tissues of five toothed whale species of the Mediterranean. *Environ. Pollution*. 108:447-452.

Gaskin, D. E., Frank, R., Holdrinet, M., Ishida, K., Walton, C. J., and Smith, M. 1973. Mercury, DDT, and PCB in harbour seals *(Phoca vitulina)* from the Bay of Fundy and Gulf of Maine. *J. Fish. Res. Board Can*. 30: 471-475.

Geraci, J.R., St. Aubin, D.J., Barker, I.K., Webster, R.G., Hinshaw, V.S., Bean, W.J., Ruhuke, J.H., Prescott, J.H., Early, G., Baker, A.S., Madoff, S., Schooley, R.T. 1982. Mass mortality of harbor seals: pneumonia associated with influenza A virus. *Science* 215:1129.

Gilbert, J. R., Waring, G.T., Wynne, K.M., and Guldager, N. 2005. Changes in abundance of harbor seals in Maine, 1981-2001. *Marine Mammal Science* 21:519-539.

Harding, K.C., Härkönen, T., Caswell, H. 2002. The 2002 European seal plague: epidemiology and population consequences. *Ecology Letters* 5:727-732.

Kannan, K., Blankenship, A.L., Jones, P.D., and Giesy, J.P. 2000. Toxicity reference values for the toxic effects of polychlorinated biphenyls to aquatic mammals. *Human Ecol. Risk Assess*. 6 (1):181-201.

Kajiwara, N., Niimi, S., Watanabe, M., Ito, Y., Takahashi, S., Tanabe, S., Khuraskin, L., and Miyazaki, N. 2002. Organochlorine and organotin compounds in Caspian seals *(Phoca caspica)* collected during an unusual mortality event in the Caspian Sea in 2000. *Environ. Pollution* 117:391-402.

Kennish, M.J. 1992. Chlorinated hydrocarbons. In: *Ecology of Estuaries: Anthropogenic Effects,* CRC Press, Boca Raton, pp. 183-248.

Law, R. J.; Allchin, C. R. and Harwood, J. 1989. Concentrations of organochlorine compounds in the blubber of seals from eastern and northeastern England (1988). *Marine Pollution Bulletin* 20:110-115.

Levin, M., De Guise, S., and Ross, P.S. 2005. Association between lymphocyte proliferation and polychlorinated biphenyls in free-ranging harbor seal *(Phoca vitulina)* from British Columbia, Canada. *Environ. Toxicol. Chem*. 24:1247-1252.

Luckas, B., Vetter, W., Fisher, P., Heidmann, G., Plötz, J. 1990. Characteristic chlorinated hydrocarbon patterns in the blubber of seals from different marine regions. *Chemosphere*: 21:3-19.

Mitchell, S.H. and Kennedy, S. 1992. Tissue concentrations of organochlorine compounds in common seals from the coast of Northern Ireland. *The Science of the Total Environment* 115:163-177.

Nyman, M., Koistenen, J., Fant, M. L., Vartiainen, T., and Helle, E. 2002. Current levels of DDT, PCB and trace elements in the Baltic ringed seals *(Phoca hispida baltica)* and grey seals *(Halichoerus grypus). Environ. Pollution* 119:399-412.

Shaw, S.D. 1998. Organochlorines and biomarkers of immune and endocrine effects in Pacific harbor seal and northern elephant seal pups. Doctoral Thesis, Columbia University School of Public Health, Division of Environmental Health Sciences, New York.

Shaw, S.D., Brenner, D., Hong, C-S., Bush, B., and Shopp, G.M. 1999. Low-level exposure to PCBs is associated with immune and endocrine disruption in neonatal harbor seals *(Phoca vitulina richardsi)* from the California coast. *Organohalogen Compounds* 42:11-14.

Shaw, S.D. 2002. An Investigation of Persistent Organic Pollutants (POPs) and Heavy Metals in Tissues of Harbor Seals *(Phoca vitulina concolor)* and Gray Seals *(Halichoerus grypus)* in the Gulf of Maine. *Final Report to the Maine Department of Environmental Protection*, Augusta, ME.

Shaw, S.D., Brenner, D., Mahaffey, C.A., De Guise, S., Perkins, C.R., Clark, G.C., Denison, M.S., and Waring, G.T. 2003. Persistent organic pollutants and immune function in US Atlantic coast harbor seals *(Phoca vitulina concolor). Organohalogen Compounds* 62:220-223

Shaw, S.D., Brenner, D., Bourakovsky, A., Mahaffey, C.A., and Perkins, C.R. 2005. Polychlorinated bipenyls and chlorinated pesticides in harbor seals *(Phoca vitulina concolor)* from the northwestern Atlantic coast. *Marine Pollution Bulletin*. 50:1069-1084.

Shaw, S.D., Berger, M.L., Brenner, D., Fang, F., Hong, C-S., Storm, R., and O'Keefe, P. 2006a. Polybrominated diphenyl ethers (PBDEs) in harbor seals (Phoca vitulina concolor) from the northwestern Atlantic. *Organohalogen Compounds* 68:600-603.

Shaw, S.D., Berger, M.L., Brenner, D., and Kannan, K. 2006b. Perfluorooctane sulfonate and related perfluorinated hydrocarbons in harbor seals (Phoca vitulina concolor) from the northwest Atlantic. *Organohalogen Compounds* 68:2042-2046.

Storr-Hansen, E. and Spliid, H. 1993. Distribution patterns of polychlorinated biphenyl congeners in harbor seal *(Phoca vitulina)* tissues: statistical analysis. *Arch. Environ. Contam. Toxicology* 25:328-345.

Tanabe, S. 1988. PCB problems in the future: Foresight from current knowledge. *Environ. Pollution* 50:5-28.

Van Loveren, H., Ross, P. S., Osterhaus, A.D.M.E. and Vos, J.G. 2000. Contaminant-induced immunosuppression and mass mortalities among harbor seals. *Toxicology Letters* 112-113:319-324.

Wagemann R., Trebacz E., Boila G., Lockhart W.L. 2000. Mercury species in the liver of ringed seals. *The Science of the Total Environment* 261(1-3): 21-32.

Are human consumers potentially at risk from toxic chemicals in Casco Bay fish and shellfish?

Background

Through the process of bioaccumulation (see Chapter 1) toxic metals and organic chemicals present in the sediments and water column can concentrate in the tissues of aquatic organisms. Predators at the top of the food chain, including large, long-lived fish and humans who consume them, are especially at risk of exposure to elevated levels of toxic contaminants. The widespread atmospheric deposition of mercury (see Chapters 2 and 6) has led to bioaccumulation of mercury in the tissues of predatory fish throughout the continental United States. In addition, toxic organic chemicals such as dioxins and PCBs have bioaccumulated in the tissues of some fish.

As of 2004, fish consumption advisories (see box on following page) for freshwater fish had been issued in every state but Alaska and Wyoming, representing 35% of the lake acreage and 24% of the total river miles in the US, plus all of the Great Lakes and their connecting waters. In addition, almost 65% of the US coastline was under advisory for consumption of certain fish (US EPA 2005). This widespread and ongoing problem impacts fish consumers in Casco Bay and across the State of Maine.

Timothy Knepp

Consumption advisories and consumer guidance have been issued by the Maine Center for Disease Control (Maine CDC) for striped bass (shown above), bluefish, and shark as well as saltwater tilefish, swordfish, king mackerel, halibut, tuna and all fish caught in Maine fresh waters.

Action Levels and Fish Consumption Advisories

State health agencies, including the Maine Center for Disease Control and Prevention, use action levels as a guide to determine whether they should issue a fish consumption advisory warning consumers to limit meals of fish from certain waters (Maine CDC 2001). Action levels are defined as concentrations of a contaminant in fish or shellfish tissue below which there should be negligible risk of deleterious health effects, at a consumption rate of one meal per week (US EPA 1993,1997). An action level takes into account exposure level for a human population, including sensitive subpopulations such as pregnant women and children, body weight, and fish consumption rate. For example, for carcinogens (cancer-causing agents), action levels are based on the assumption that consumption of edible fish tissue at a rate of one 8-ounce meal per week over a 70-year lifetime would result in a 1 in 100,000 incremental lifetime cancer risk (Maine CDC 2001). The tables below provides Maine action levels for PCBs, dioxins, PAHs, pesticides, and metals for both cancer risk and non-cancer risk.

Examples of Maine Fish Tissue Action Levels for Fish Filet (wet weight)

Table 8-1

Organic Chemicals	Non-Cancer Action Level ppb (parts per billion)	Cancer Action Level ppb
PCBs	43	11
Dioxin	0.0019	0.0015
PAHs	**ppb**	**ppb**
Benzo(a)pyrene		3.0
Acenaphthene	130	
Anthracene	648	
Fluoranthene	86	
Fluorene	86	
Biphenyl	108	
Naphthalene	43	
Pyrene	65	
Chlorinated Pesticides	**ppb**	**ppb**
DDT	1080	64
Dieldrin	108	1.4

Source: Maine CDC 2001

Table 8-2

Metals	Non-Cancer Action Level ppm (parts per million)	Cancer Action Level ppm
Arsenic (inorganic)	0.6	0.014
Cadmium	2.2	
Chromium VI	11	7
Lead	*	*
Manganese	302	
Methylmercury - fetal	0.2	
Methylmercury -adult	0.65	
Nickel	43	
Selenium	11	
Silver	11	
Tributyl tin (oxide)	0.6	
Vanadium	6	
Zinc	648	

*The need for advisories based on lead is determined using US EPA's biokinetic model to estimate typical lead exposure given the species and population of interest (Frohmberg 2006)

Consuming Fish from Maine Waters

Elevated levels of mercury in all fresh waters in Maine, including those in the Casco Bay watershed, have resulted in elevated levels of mercury in the tissues of resident fish populations (Maine DEP 2004). Freshwater fish species from some rivers and ponds in Maine also have elevated levels of PCBs, dioxins, and DDT. Both PCBs and dioxins have the potential to cause cancer in humans. Tissue concentrations of PCBs and dioxins that exceed the State action level have been found in some saltwater fish species as well, including striped bass and bluefish. Dioxin has also been found at elevated levels in lobster tomalley, the organ that serves as the lobster's hepatopancreas (pancreas and liver). Monitoring results have led the State of Maine to issue fish consumption advisories since 1994 and to provide consumers with safe-eating guidelines. All advisories are currently undergoing review by the State (Frohmberg 2007). The results of State fish tissue studies and the ongoing Maine DEP Surface Water Ambient Toxic Monitoring Program (SWAT) are also discussed in Chapter 6.

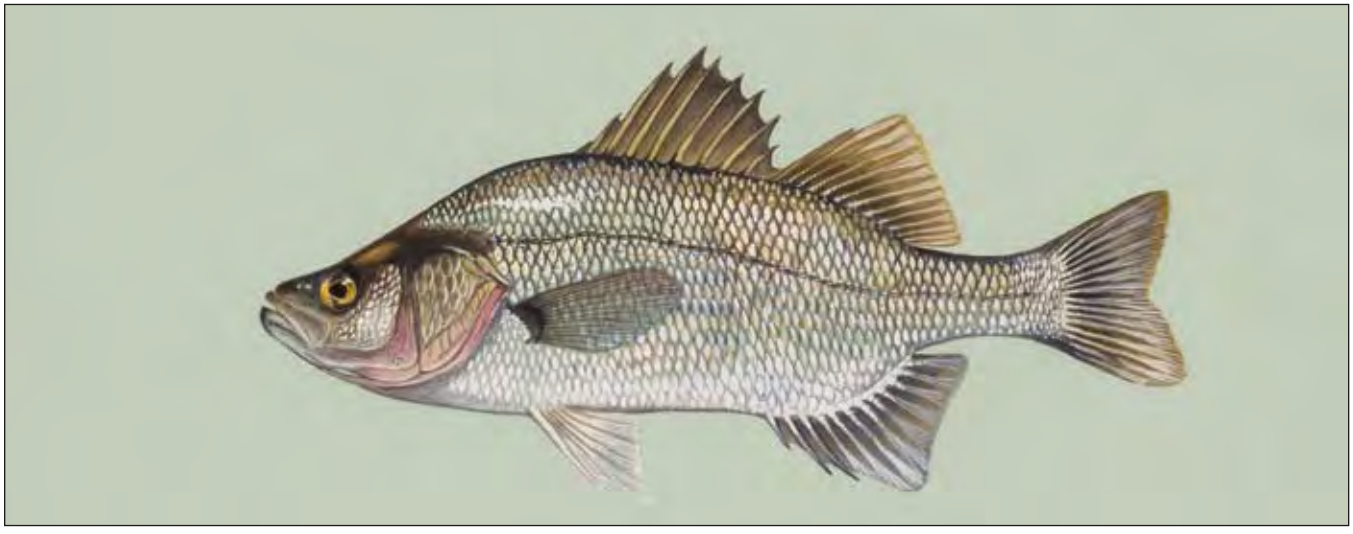
State Guidance on Eating Freshwater Fish from the Casco Bay Watershed

Because mercury has the potential to harm forming or growing brain tissue, unborn babies, infants and young children are at greatest risk of harm from exposure to small amounts of mercury. In higher dosages, older children and adults can also experience neurological damage (Maine CDC 2006a). State consumer guidance on fish consumption for adults and children over 8 is based on an 8-ounce meal (an upper estimate of fish consumption). Four ounces is the amount promoted by dietary organizations and the Maine Family Fish Guide as an appropriate serving size (Maine CDC 2006b, Frohmberg 2007).

- **Pregnant and nursing women, women who may get pregnant, and children under 8:** Consumers in this high risk category are advised not to eat any freshwater fish from Maine's inland waters. The only exceptions are freshwater smelt, brook trout and landlocked salmon, for which the guidance suggests a limit of 1 meal per week.

- **Adults and children older than 8:** Consumers are advised to eat no more than 2 meals per week of freshwater fish from Maine's inland waters, with a limit of 1 meal per week for freshwater smelt, brook trout and landlocked salmon.

  Additional State fish consumption limits are suggested for fresh waters where fish have elevated levels of PCBs, dioxins or DDT. Fortunately, none of these waters is in the Casco Bay watershed (Maine CDC 2006a). The Maine DEP SWAT program continues to monitor fish in the Casco Bay watershed, including recent dioxin and coplanar PCB sampling in fish from the Presumpscot River at Westbrook and Windham. The sampling was funded with assistance from the CBEP.



Duane Raver

Consumption advisories and consumer guidance have been issued by the Maine CDC for all fish caught in Maine fresh waters, including white perch, pictured above.

State Guidance on Eating Saltwater Fish

Consumption advisories for saltwater species cover all marine waters in Maine, including Casco Bay.

- **Pregnant and nursing women, women who may get pregnant, and children under 8**: These high risk consumers should not eat the following fish due to elevated mercury levels:

  Swordfish
  Shark
  King Mackerel
  Tilefish

  The following marine fish should be limited to one meal per week:

  Tuna steak
  Canned white tuna
  Halibut steak

  These consumers should limit meals of other ocean fish and shellfish (including canned light tuna), to 2 per week (Maine CDC 2006c). The exceptions are bluefish and striped bass which have elevated levels of PCBs and should be limited to 2 meals per month by all consumers (Maine CDC 2006c).

Timothy Knepp

Saltwater fish that are low in mercury include fresh and canned salmon (Atlantic salmon is shown above), sardines and herring, smelt, Atlantic mackerel, mussels, scallops and clams, flounder and sole, shrimp, haddock, hake, Pollock, cod, and lobster (Maine CDC 2006b).

The current advice for striped bass and bluefish is under review by the Maine CDC. New advice will be released by spring of 2007 (Frohmberg 2007). A review of the data can be found at http://www.maine.gov/dhhs/eohp/fish/PCBSTBhome.htm (Maine CDC 2006d).

- **Adults and Children Older Than 8:** The State guidance advises no more than 2 meals per month of:

  Swordfish
  Shark
  King Mackerel
  Tilefish
  Bluefish
  Striped Bass (Maine CDC 2006c)

Maine DEP

Consumers are advised to avoid eating the greenish tomalley (shown above) due to elevated levels of dioxin.

State Guidance on Eating Lobster Tomalley

While lobster meat is low in mercury and other toxics and safe to eat, the lobster tomalley (the soft, green substance found in the lobster's body cavity) has been shown to contain elevated levels of dioxin. The tomalley functions as the lobster's liver and pancreas, concentrating lipophilic (fat-soluble) organic contaminants. The State guidance advises that no one consume lobster tomalley (Maine CDC 2006c).

Casco Bay Mussel Toxics Study

Chapter 5 describes the Maine DEP's mussel sampling program, which uses the shellfish *Mytilus edulis* as an indicator organism to assess the health of the Casco Bay ecosystem. In the decade from 1987 to 1997, the DEP mussel sampling program found that only the metals mercury and lead exceeded State action levels for toxics at a few stations in Maine. Since 1996, CBEP has been supplementing the DEP blue mussel monitoring program by periodically collecting samples at additional sites in Casco Bay. Selection of sites for testing takes into consideration the results of CBEP sediment contamination studies (see Chapter 4), the intensity of local land use, and past history of pollution, focusing on areas where the mussels might have maximum exposure to elevated concentrations of toxics.

To assess the potential human health impacts from mussel contamination, the results of the 1996 and 1998 CBEP sampling were submitted to the Maine CDC, Environmental and Occupational Health Program, previously called the Maine Bureau of Health, Environmental Toxicology Program. The samples were collected at eight sites, which were selected for the following reasons:

- Back Cove in Portland was selected because of its historically elevated levels of PAHs and metals in the sediments, the result of CSO overflows into the Cove
- Harraseeket River in Freeport was selected because of the recent huge increases in impervious surface in the watershed and the heavy vehicle traffic.
- Quahog Bay in Harpswell was selected because the sediments have high levels of cadmium.
- Falmouth was selected due to its close proximity to boating and boat yards.
- Middle Bay was selected because of potential PAH and other chemical contamination from the Naval Airbase.
- The Wolfe's Neck site in Freeport was selected because there is an air deposition monitoring site there.
- Jewell Island in Outer Casco Bay and the Basin in the New Meadows River were selected as potential reference sites, because they had no known local sources of toxics.

The results of the data analysis are presented below:

- Levels of lead in samples from Back Cove were slightly elevated above the action level for this neurotoxin. Since lead is a serious concern for young children, regular consumption of mussels from Back Cove could pose a risk. Back Cove is currently closed for all shellfishing.
- Total PCB levels were elevated in mussels from Back Cove and Quahog Bay and somewhat elevated in mussels from Falmouth.
- For the PAH compounds evaluated for their potential to cause cancer, levels indicated an incremental cancer risk for frequent consumers of mussels of less than 2 in 100,000. Of the compounds evaluated for non-carcinogenic effects, none approached levels of concern.
- Arsenic was elevated above the action level at Falmouth and Jewell Island. The report noted that most of the arsenic found in seafood tends to be in a relatively non-toxic form (Maine CDC, 1999).

Because mussels are widely harvested in Casco Bay, the risk associated with human consumption is of great interest to the Maine CDC. There is, however, no licensing program for recreational mussel harvesting in the Bay and no data available on the frequency of harvesting or the quantities of wild mussels consumed in a typical meal (Maine DHHS, 1999). In 2002, field studies conducted by CBEP determined that recreational harvesting is taking place in the Bay in a few mussel beds where elevated levels of pollutants have been observed. Further studies would be needed to determine whether local harvesters and their families are consuming enough mussel meals from polluted beds to pose a public health risk (CBEP 2002).

Maine DEP also periodically samples soft-shell clams *(Mya arenaria)* for toxic chemicals and makes the data available to the Maine CDC for risk analysis. No advisories for clam consumption have been issued by the Maine CDC.

Summary/Conclusions

While many species of saltwater fish remain safe for all consumers to eat, inputs of mercury and organic chemicals generated by human activities have resulted in fish consumption advisories in Maine and all across the United States. The good news is that the levels of mercury, PCBs and dioxins (as well as many pesticides) entering the aquatic environment across the country have greatly declined over the past two decades. Chapter 9 discusses some of the ways federal, state and local governments and citizens are helping to reduce the loading of toxic chemicals to our environment.

Robert W. Hines

References

Casco Bay Estuary Partnership. 2002. *Human Exposure to Toxic Chemicals through Subsistence Shellfishing in Casco Bay.*

Frohmberg, E. 2006, 2007. Maine Center for Disease Control. Personal Communication.

Maine Center for Disease Control and Prevention. 2001. *Bureau of Health Fish Tissue Action Levels*. Updated 2/20/01. (http://maine.gov/dhhs/eohp/fish/documents/Action%20Levels%20Writeup.pdf) (February 5, 2007).

Maine Center for Disease Control and Prevention. 2006a. *Freshwater Fish Safe Eating Guidelines*. (http://www.maine.gov/dhhs/eohp/fish/2KFCA.htm) (November 8, 2006).

Maine Center for Disease Control and Prevention. 2006b. *Maine Family Fish Guide: Advice from the Maine Center for Disease Control and Prevention* (http://www.maine.gov/dhhs/eohp/fish/documents/MeFFGuide.pdf) (February 5, 2007).

Maine Center for Disease Control and Prevention. 2006c. *Saltwater Fish Safe Eating Guidelines* (http://www.maine.gov/dhhs/eohp/fish/saltwater.htm) (November 8, 2006).

Maine Center for Disease Control and Prevention. 2006d. *Home Page for Evaluation of Atlantic Coastal Advisory for Striped Bass and Bluefish Based on PCBs*. (http://www.maine.gov/dhhs/eohp/fish/PCBSTBhome.htm) (January 3, 2007).

Maine Department of Environmental Protection. 2004. *2004 Integrated Water Quality Monitoring and Assessment Report* (http://www.maine.gov/dep/blwq/docmonitoring/305b/index.htm#2004) (May 16, 2006).

Maine Department of Health and Human Services.1999. *Interdepartmental Memorandum,* Oct. 27, 1999 from Andrew Smith, SM,ScD, Bureau of Health, Environmental Toxicology Program to Lee Doggett, Maine Department of Environmental Protection.

United States Environmental Protection Agency. 1993. *Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories: Volume 1. Fish Sampling and Analysis.* EPA 823-R-93-002.

United States Environmental Protection Agency. 1997. *Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories: Volume 2. Risk Assessment and Fish Consumption Limits.* EPA 823-B-97-009.

United States Environmental Protection Agency. 2005. 2004 National Listing of Fish Advisories: Fact Sheet. (http://epa.gov/waterscience/fish/advisories/fs2004.html) (August 2, 2006).

9 Overview and Next Steps: What are CBEP and our partners doing to reduce the loading of toxics to the Bay and its watershed?

The voluntary Clean Marinas and Boatyards program is reducing toxics by promoting best management practices at participating facilities.

Summary of Report Findings

Toxic chemicals generated through human activities have entered and continue to enter Casco Bay and its watershed via multiple routes including outfall pipes, industrial smokestacks, internal combustion engines, stormwater runoff, oil spills, and atmospheric deposition. While most of these sources are local, atmospheric deposition is contributing toxic chemicals, including mercury and PAHs, both from local and distant sources through the movement of polluted air masses. As a result of past and ongoing activities both here in Maine and in other parts of the United States, toxic chemicals are found throughout Casco Bay and its watershed. Both heavy metals and organic contaminants have accumulated in the sediments of the Bay and, in many cases, in the tissues of aquatic organisms.

The levels of toxic chemicals found in the waters and sediments of the Bay are below the levels that would cause negative biological effects throughout most of Casco Bay. The exceptions are the elevated levels of PAHs found in the sediments in some inner parts of the Bay, and the levels of PCBs and some metals, including mercury, in the sediments of the Fore River.

While low levels of toxic contaminants are found in most parts of the Bay, these chemicals are becoming concentrated in the tissues of organisms, including predatory aquatic organisms, through the processes of biomagnification and bioaccumulation. Blue mussels, which serve as an indicator organism for Maine DEP,

Dave Menke

Bald eagles, like these chicks, are exposed to mercury through their fish diet.

CBEP and the Gulf of Maine Council on the Marine Environment, show elevated levels of metals including lead and the organic pollutants PCBs and PAHs at some sites in the Bay with an industrial history in harbors, commercial ports, at the mouths of river watersheds and in locations adjacent to population centers.

Elevated body burdens of mercury have been found in predatory species from insect-eating birds to fish-eating birds and mammals. Studies have shown that the mercury levels in Maine's fish, loons and eagles are among the highest in the country and that the productivity of Maine's loons and eagles is being impacted by mercury. As top-level predators, seals are especially vulnerable to bioaccumulation. Research indicates that seals from southern Maine have elevated body burdens of mercury and the organic chemicals PCBs and DDE (a metabolite of DDT). Casco Bay seals, especially the pups, are likely at risk of health impacts to their livers and to their neurological and endocrine systems.

As consumers of fish, humans are also at the top of the food chain and are potentially at risk of health impacts from bioaccumulated toxic chemicals in fish. Levels of mercury and some organic chemicals found in freshwater and certain marine fish have led the State to issue fish consumption advisories and guidelines on safe fish and lobster consumption practices. These are especially important for the most vulnerable consumers, including pregnant women and children.

Despite the clear evidence that toxic chemicals are found throughout Casco Bay and its watershed, impacting both the ecosystem and our ability to safely eat certain fish, there is some good news. The levels of mercury, PCBs, dioxins, and many pesticides entering the environment have declined greatly over the past two decades (US EPA 2005). *State of the Bay* (CBEP 2005) reported that levels of most heavy metals, pesticides, tributyltin, PCBs and low molecular weight PAHs decreased in the sediments of the Bay between 1991/1994 and 2000/2001.

CBEP's state, federal and local partners are using a variety of regulatory and non-regulatory tools to reduce the overall loading of toxics to the Bay and its watershed. A summary of state and federal programs follows.

Oksana Lane

Insect-eating Saltmarsh Sharp-Tailed Sparrows from southern Maine have elevated body burdens of mercury.

Cynthia Stroud

Harbor seals hauled out on pupping ledges. Seals in Maine, including Casco Bay, are likely at risk for contaminant-related health effects from mercury and persistent organic pollutants.

Federal and State Programs that Reduce Toxics Loading

US EPA New England and Maine DEP are helping to reduce toxics loading through enforcement of environmental laws and regulations. Inspections of facilities that produce pollutants, reporting, and sampling and monitoring programs are tools that help to determine compliance. Violations can result in civil or criminal penalties. Through compliance assistance programs, US EPA helps business and industry to understand and meet regulatory requirements (US EPA 2006a). State and US EPA pollution prevention (P2) programs provide guidance, tools, and resources to promote pollutant elimination/reduction through more efficient use of materials, energy, water, and land (US EPA 2006b, Maine DEP 2005a). Some of the programs that regulate toxics are included below.

Water Enforcement Programs:

- US EPA is nationally responsible for compliance monitoring under the **Clean Water Act (CWA)**, first passed in 1972. CWA enforcement programs include the **National Pollutant Discharge Elimination System (NPDES)**, which regulates point-source discharges to the waters of the United States, and, recently began regulating stormwater discharges as well. The State is delegated by US EPA to oversee the NPDES program in Maine. Through Maine DEP, permits are issued to facilities that discharge to the surface waters of the State. Compliance monitoring is used to ensure that State water quality standards are not violated.

- Other CWA programs include the **Pretreatment Program**, which regulates discharges to publicly-owned treatment works; the **Combined Sewer Overflow (CSO) Control Policy**; and the water discharge aspect of the **Pulp and Paper "Cluster Rule"** (US EPA 2006a). The CSO Control Policy has resulted in the elimination of twenty combined sewer overflows in the Casco Bay Watershed (see *State of the Bay,* CBEP 2005a). The federal **Pulp and Paper "Cluster Rule"** is significantly reducing the amount of pollutants in wastewater from mills, mandating a 95% reduction in dioxin and furan (US EPA 1997). Maine also has a strict **dioxin wastewater discharge law** and has developed an inventory of dioxin discharges to the State's water (see Maine DEP's dioxin website http://www.maine.gov/dep/dioxin/)

- In September of 2005, the Maine Board of Environmental protection voted to adopt new State rules (the **Water Toxics Rule**) which contain numeric surface water quality criteria for toxic pollutants for the protection of aquatic life and human health. These revisions were approved by US EPA in July 2006. Chapter 584 also includes testing requirements, data evaluation and impact assessment (Maine DEP 2005b).

- Every two years, Maine DEP reports to the U.S. Congress and the Maine Legislature on the health, current status, and trends of the State's waters. The **Integrated Water Quality Monitoring and Assessment Report** satisfies reporting requirements under the Clean Water Act and Maine statutes. The report includes a list of impaired waters that require development and submission to US EPA of **total maximum daily load (TMDL) assessment reports**. When specific toxics have been identified as pollutants of concern in a particular water, Maine DEP develops TMDLs, or chemical-specific limits for a certain waterbody, for those toxics. If the source of toxics is wastewater discharge, water quality-based effluent limits are then incorporated into the discharge permits. Reduction in toxics from diffuse sources such as stormwater or nonpoint source runoff are achieved by implementing best management practices (BMPs) that are effective in promoting infiltration of stormwater to the groundwater. Low impact development strategies are BMPs that allow runoff from paved surfaces to flow over pervious or vegetated surfaces where they naturally infiltrate the ground or are treated before entering a drainage collection system.

- In 1972, Congress passed the **Marine Protection, Research, and Sanctuaries Act (MPRSA),** or Ocean Dumping Act to control ocean dumping and to protect the marine environment and human health. The MPRSA bans radiological, chemical, and biological warfare agents, high-level radioactive wastes, medical wastes, sewage sludge, and industrial wastes from ocean disposal. Anyone seeking a permit to dump other types of waste must show that the dumping will not pose a danger to human health or the environment, and that there are no better alternatives for reuse or disposal. Most of the material dumped in US ocean waters today is sediment dredged from the bottom of water bodies to maintain the nation's navigation system. US EPA has issued stringent environmental criteria, including bioaccumulation and toxicity testing, for evaluating materials proposed for ocean dumping (US EPA 2006c). If it is determined that dredged material has the potential to cause unacceptable, adverse environmental effects, it may not be disposed of in the ocean.

Air Enforcement Programs:

- Since 1970 when the **Clean Air Act** was first enacted, US EPA and Maine DEP have implemented control programs that have significantly reduced air pollution, including air toxics from mobile sources, stationary and area sources. The State is delegated by US EPA to oversee certain federal regulations such as the New Source Performance Standards and the National Emission Standards for Hazardous Pollutants which control emissions of air toxics and criteria pollutants. The **Clean Air Mercury Rule** (2005) is intended to cap and reduce mercury emissions from coal-fired power plants with the goal of reducing utility emissions of mercury by nearly 70% (US EPA 2005). The **Clean Air Act Amendments** of 1990 directed US EPA to identify sources of dioxin emissions and to implement regulations to reduce dioxin emissions to the environment. As a result of federal, state, and industrial efforts, there has been an overall 90% reduction in emissions of dioxin from industrial sources in the US since 1987 (US EPA 2003). The 1998 **Pulp and Paper "Cluster Rule"** is also significantly reducing toxic air pollutant emissions, including dioxin, from mills.

Hazardous Waste & Toxics Use and Release Programs:

- Maine's **Toxic and Hazardous Waste Reduction Law** encourages Maine businesses to reduce toxics use, toxics release, and hazardous waste generation. Regulated businesses must develop a Pollution Prevention plan, set company-specific reduction goals, report to the Maine DEP biennially on their progress, and pay an annual fee to the Maine DEP's Toxics Program (Maine DEP 2003). The law sets non-binding statewide reduction goals which include a statewide reduction of toxics releases of 60% by 2006.

- The 1986 federal **Emergency Planning and Community Right-to-Know Act (EPCRA)** helps to increase the public's knowledge of the presence of hazardous chemicals and their releases into the environment. The provisions of this regulation include a requirement that facilities quantify and submit releases of toxic chemicals into a national database, the **Toxics Release Inventory** or TRI. The data is a valuable tool for state, federal and local regulatory and emergency planning.

- The **Toxic Substances Control Act (TSCA)** is a federal law that protects the pubic from exposure to toxic substances by regulating the importation, manufacture and distribution of listed toxic chemicals in the US. The TSCA PCB Program prohibits the manufacture of PCBs (production ceased in 1977), controls the phase-out of existing uses, and oversees their safe disposal. TSCA lead regulations focus on protecting the public from lead-based paint hazards.

- The federal **Oil Pollution Act** requires facilities that store large quantities of oil to prepare spill plans and to adopt measures to keep any accidental spills from reaching waterways. See Chapter 3 to learn more about what the State of Maine is doing to reduce the impacts of oil spills, including the *Maine Oil Spill Contingency Plan*.

- **Pesticide Enforcement**—The use of DDT was banned in the US in 1972. US EPA works in partnership with the State of Maine to regulate the use of legal pesticides through inspections and certification training for applicators. Under federal and State regulations, the use of tributyltin, a toxic anti-fouling ingredient added to marine paints, is banned in Maine (except for vessels over 25 meters in length or vessels with an aluminum hull).

- The **Superfund Enforcement Program** implements the federal **Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA)** and the **Resource Conservation and Recovery Act (RCRA)**. Under CERCLA, US EPA responds to releases or threatened releases of hazardous substances and negotiates with the responsible parties to conduct the clean-up. Under RCRA, US EPA works with the State of Maine to regulate businesses that generate, transport, treat and store hazardous wastes. Any release to the environment requires the business to conduct clean-up and monitoring.

Toxics Source and Risk Assessment Programs:

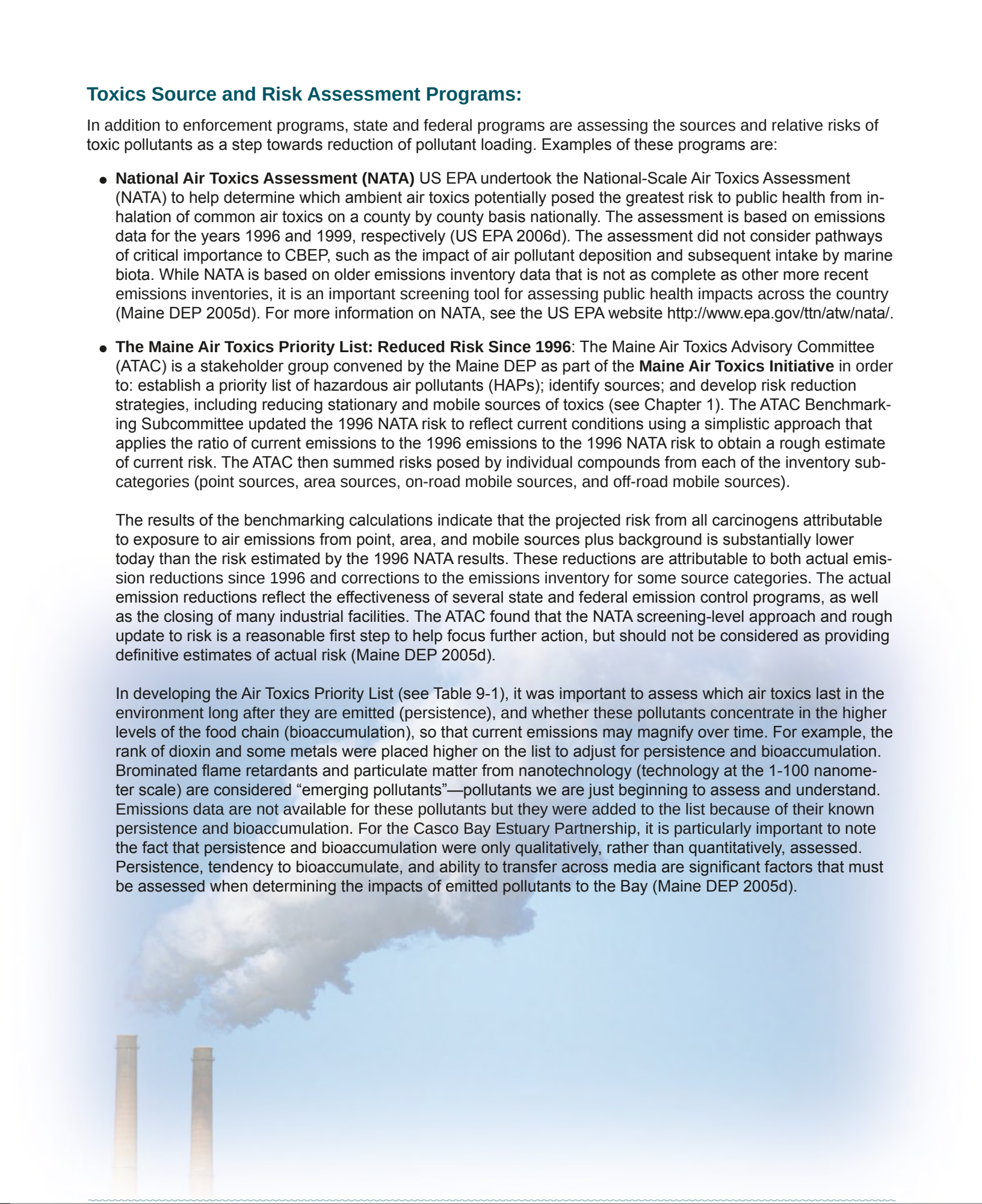
In addition to enforcement programs, state and federal programs are assessing the sources and relative risks of toxic pollutants as a step towards reduction of pollutant loading. Examples of these programs are:

- **National Air Toxics Assessment (NATA)** US EPA undertook the National-Scale Air Toxics Assessment (NATA) to help determine which ambient air toxics potentially posed the greatest risk to public health from inhalation of common air toxics on a county by county basis nationally. The assessment is based on emissions data for the years 1996 and 1999, respectively (US EPA 2006d). The assessment did not consider pathways of critical importance to CBEP, such as the impact of air pollutant deposition and subsequent intake by marine biota. While NATA is based on older emissions inventory data that is not as complete as other more recent emissions inventories, it is an important screening tool for assessing public health impacts across the country (Maine DEP 2005d). For more information on NATA, see the US EPA website http://www.epa.gov/ttn/atw/nata/.

- **The Maine Air Toxics Priority List: Reduced Risk Since 1996**: The Maine Air Toxics Advisory Committee (ATAC) is a stakeholder group convened by the Maine DEP as part of the **Maine Air Toxics Initiative** in order to: establish a priority list of hazardous air pollutants (HAPs); identify sources; and develop risk reduction strategies, including reducing stationary and mobile sources of toxics (see Chapter 1). The ATAC Benchmarking Subcommittee updated the 1996 NATA risk to reflect current conditions using a simplistic approach that applies the ratio of current emissions to the 1996 emissions to the 1996 NATA risk to obtain a rough estimate of current risk. The ATAC then summed risks posed by individual compounds from each of the inventory subcategories (point sources, area sources, on-road mobile sources, and off-road mobile sources).

  The results of the benchmarking calculations indicate that the projected risk from all carcinogens attributable to exposure to air emissions from point, area, and mobile sources plus background is substantially lower today than the risk estimated by the 1996 NATA results. These reductions are attributable to both actual emission reductions since 1996 and corrections to the emissions inventory for some source categories. The actual emission reductions reflect the effectiveness of several state and federal emission control programs, as well as the closing of many industrial facilities. The ATAC found that the NATA screening-level approach and rough update to risk is a reasonable first step to help focus further action, but should not be considered as providing definitive estimates of actual risk (Maine DEP 2005d).

  In developing the Air Toxics Priority List (see Table 9-1), it was important to assess which air toxics last in the environment long after they are emitted (persistence), and whether these pollutants concentrate in the higher levels of the food chain (bioaccumulation), so that current emissions may magnify over time. For example, the rank of dioxin and some metals were placed higher on the list to adjust for persistence and bioaccumulation. Brominated flame retardants and particulate matter from nanotechnology (technology at the 1-100 nanometer scale) are considered “emerging pollutants”—pollutants we are just beginning to assess and understand. Emissions data are not available for these pollutants but they were added to the list because of their known persistence and bioaccumulation. For the Casco Bay Estuary Partnership, it is particularly important to note the fact that persistence and bioaccumulation were only qualitatively, rather than quantitatively, assessed. Persistence, tendency to bioaccumulate, and ability to transfer across media are significant factors that must be assessed when determining the impacts of emitted pollutants to the Bay (Maine DEP 2005d).



The Maine Air Toxics Priority List

The final Air Toxic Priority list is shown below. This list is based on the best information available in 2006, but due to uncertainties in this information, it is only a rough estimate of rank (with acrolein ranked highest), and pollutants will be added and deleted as new information comes to light and emission reductions are implemented. The Air Toxics Advisory Committee finds that every six months, it should re-evaluate whether any previously unknown pollutants should be added to the list (Maine DEP 2005d).

Note that many of the pollutants on the list (like acrolein) are primarily a concern due to human inhalation risks rather than potential impacts to the aquatic environment .

Table 9-1. Final Maine Air Toxics Priority List

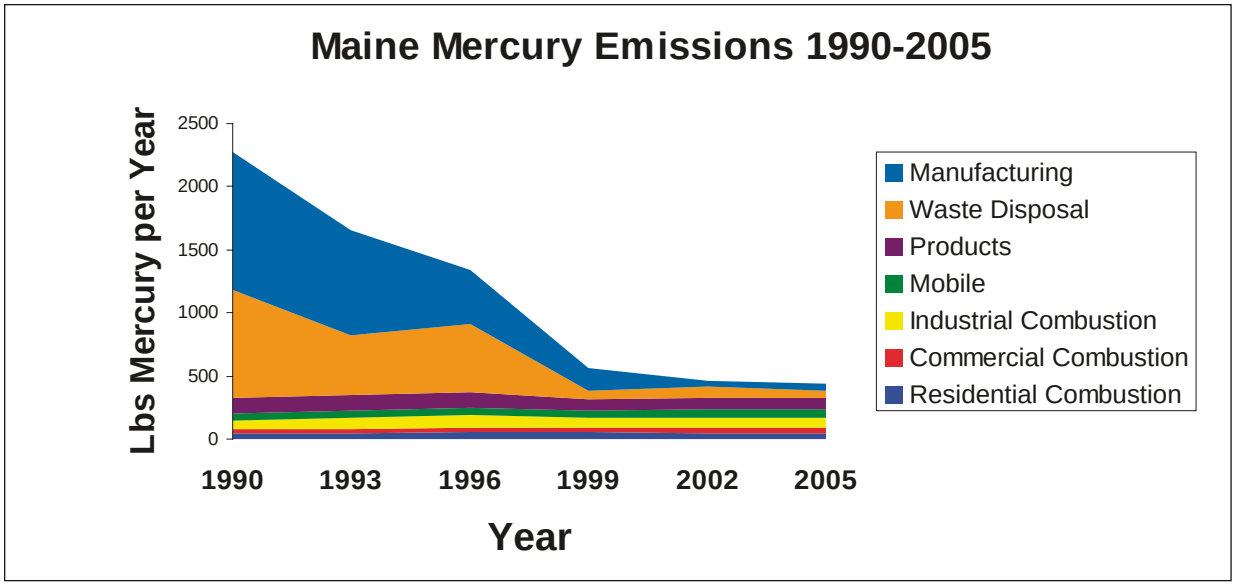
Rank	Pollutant Category
1	Acrolein
2	Polycyclic Organic Matter
3	Manganese
4	Formaldehyde
5	Nickel
6	1,3-Butadiene
7	Diesel PM
8	2,4-Toluene Diisocyanate
9	Sulfuric Acid
10	Benzene
11	Lead
12	Cadmium
13	Dioxins
14	Chromium
15	Arsenic
16	Cyanide & Compounds
17	Mercury
18	Brominated Flame Retardants
19	Particulate Matter from Nano-Technology
20	Acetaldehyde
21	Tetrachloroethylene (Perchloroethylene)
22	Chloroform
23	Carbon Tetrachloride
24	Ethylene Dichloride
25	Ethylene Dibromide
26	Methyl bromide
27	Chlorine
28	Hydrochloric acid
29	Chlorine dioxide

Source: Maine DEP 2005d

Focus on Mercury Reductions in Maine

Historically, mercury has been the compound of greatest concern to the Maine DEP in terms of persistence and bioaccumulation (Maine DEP 2005d). The effects of mercury on wildlife were explored in Chapter 7 and the resulting impacts to human consumers of fresh and saltwater fish were discussed in Chapter 8. Regulatory efforts by the Maine DEP have substantially decreased emissions of mercury in Maine during the past 15 years, as shown in Figure 9-1 below.

Figure 9-1 (Maine DEP 2005d)



There have been many major steps taken in Maine to reduce mercury loading from all sources. These include:

1989 Mercury discharges to State waters prohibited; ambient water quality criteria set.

1994 First in the nation statewide fish consumption advisories issued by Maine.

1998 New England Governors and Eastern Canadian Premiers adopt a landmark goal to "virtually eliminate" releases of mercury from human activities into the environment. Action plan called for elimination of 50% of mercury emissions by 2003.

2000 State statute lowered mercury emissions standards. Also, HoltraChem Manufacturing Company chlor-alkali plant in Orrington, Maine was closed down. It was the last plant in New England that used a mercury-cell process to produce chlorine gas and caustic soda.

2001 Maine bans the sale and distribution of thermostats containing added mercury.

2002 The Natural Resources Council of Maine and US EPA worked with businesses to ensure the removal and storage of 185,000 pounds of surplus mercury from HoltraChem. Maine achieves 65% mercury emissions reductions since 1998. Law passed that requires automobile manufacturers to recover mercury-containing switches from vehicles before they are scrapped.

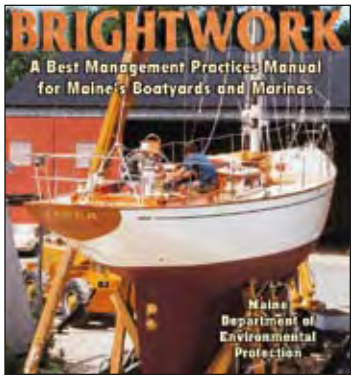
2003 Maine bans the sale of most switches, relays, and measuring devices containing added mercury. After a dialogue begun in its Portland, Maine store, Wild Oats becomes the first national chain to post mercury levels in fish. Law passed that requires dentists to separate mercury from dental wastewater.

2006 Law passed banning the sale of button-cell batteries with added mercury and products containing such batteries after January 30, 2011. Mercury-free alternatives will replace these batteries (NRCM 2006).

How Citizens and Businesses Can Reduce Toxics Loading

The following are a few examples of the many ways that citizens and businesses can act as stewards of the environment by helping to reduce the loading of toxic chemicals to the Bay and its watershed.

Businesses can:



- **Participate in voluntary toxics reduction approaches:** For example, marinas and boatyards across the state are now participating in the Maine Clean Boatyards & Marinas Program first piloted in Casco Bay on a voluntary basis. Guidance for environmentally sound practices can be found in *Brightwork: A Best Management Practices Manual for Maine's Boatyards and Marinas* (2005c). Golf courses can participate in Audubon International's Golf Course Certification Program, which includes management approaches to reduce the use of chemicals. In 2003, CBEP helped to sponsor training in the program for Maine golf course superintendents and continues to work with courses in the watershed toward certification through its Presumpscot Watershed Initiative.

Everyone can:

- **Manage lawns and gardens in a more environmentally sustainable way:** Educational programs such as Maine Board of Pesticide Control's Yardscaping Program (http://www.yardscaping.org/), Friends of Casco Bay's Bayscaping program (http://www.state.me.us/agriculture/pesticides/bayscaper/), and the Maine DEP's Lakesmart program (www.maine.gov/dep/blwq/doclake/lakesmart/index.htm) teach and promote the reduction of toxic chemical use and other environmentally friendly techniques for maintaining an attractive landscape.

- **Minimize impacts from driving**: Combustion engines are a significant source of pollution. Minimize driving by ride-sharing whenever possible. Don't idle the engine unnecessarily. Avoid gasoline spills and the release of fumes from your car, boat, or lawnmower. Make sure that your car's catalytic converter is functioning well. One of its functions is to reduce the release of PAHs and other hydrocarbons and volatile organic compounds produced from unburned fuel. Maximize fuel efficiency by traveling at a medium, steady speed. Trade in old car batteries for recycling when buying a new one. Also, have your oil, brake and transmission fluid changed at a service station that recycles.

- **Conserve electricity:** The generation of electricity by coal-fired power plants is a major source of atmospheric mercury. Tips for saving on electricity can be found at http://www.efficiencymaine.com/energytips.htm.

- **Reduce & properly dispose of household hazardous waste:** Reduce the use of toxic chemicals in your home by replacing them with less toxic substitutes. Dispose of solid and liquid household hazardous wastes properly (*e.g.,* fluorescent tubes and old thermostats containing mercury, house paint, solvents, pesticides, waste oil). To determine when there is household hazardous waste collection day in your area, visit http://www.state.me.us/spo/recycle/hhw/collections.php

- **Use woodstoves and fireplaces sparingly and wisely**: Use dry, well-seasoned wood and keep your chimney clean. Wood burning releases PAHs, acrolein, and other toxic chemicals.

Friends of Casco Bay

Casco Bay Estuary Partnership (CBEP) Efforts to Reduce and Monitor Toxics in the Bay and its Watershed

Beverly Bayley-Smith

Some of the CBEP Board members and staff gather at a Casco Bay marina.

In addition to the many enforcement activities and voluntary programs of our government and citizen partners, CBEP has developed specific actions in the *Casco Bay Plan* that are focused on reducing toxic pollution. CBEP will continue to monitor the levels of toxic chemicals in the sediments of the Bay and in the tissues of mussels, two of our suite of environmental indicators. CBEP is also supporting continued reductions in the number and volume of combined sewer overflow discharges into the Bay. By supporting the ongoing efforts of the 14-community Casco Bay Interlocal Stormwater Working Group (ISWG), CBEP is helping to reduce the loading of toxics via stormwater runoff. One of the 1996 *Casco Bay Plan* action recommendations was to "research the contribution of deposition of pollutants from the air." The results of CBEP's atmospheric deposition studies are described in Chapter 2. In partnership with Maine DEP, CBEP is continuing to assess the contribution and sources of mercury and trace metals to the Bay and its watershed through atmospheric deposition.

Four new recommended actions in the updated 2005 *Casco Bay Plan* (CBEP 2005b) are:

- ***Casco Bay Plan* Toxics Action #1: Support efforts to develop a comprehensive management strategy for dredged material.**
  In order to facilitate sound dredge material disposal as a result of necessary dredging in the Bay, CBEP will support efforts to develop a comprehensive approach to management of dredged materials by providing input to policy dialogues of state and federal governments and by working in partnership with other groups and agencies to provide state-of-the science information and guidance for dredgers on alternatives for dredge disposal.

- ***Casco Bay Plan* Toxics Action #2: Develop Biological Indicators for Marine Waters**
  CBEP will work with Maine DEP and others to develop scientifically sound biological indicators as a foundation for developing marine biological standards to regulate Maine's marine waters. Biological indicators can integrate and reflect multiple water quality impacts to an ecosystem and are already being used by Maine DEP to ensure that the state's freshwaters meet their designated uses.

- ***Casco Bay Plan* Toxics Action #3: Develop Sediment Quality Thresholds for Assessment of Contaminated Sediments**
  CBEP will work with EPA, Maine DEP and others to develop sediment quality thresholds for contaminated sediments in Maine. The sediment thresholds will be used to interpret sediment quality data, to report on contamination levels in the State of the Bay Report, and to help inform other agencies and partners in their development of thresholds as well.

- ***Casco Bay Plan* Toxics Action #4: Research the Feasibility of and Best Approach to Monitoring New Environmental Analytes**
  Tens of thousands of chemicals are now known or suspected to be present in marine and freshwater bodies as a result of their use by humans. These so-called "emerging contaminants" include pharmaceuticals and personal care products, (such as antibiotics, steroids, hormones and other endocrine disruptors), and a variety of chemicals such as caffeine, cholesterol, fire retardant and insect repellents which may have or, in some cases, have been shown to have detrimental effects on aquatic organisms and ecosystems. As a starting point for a monitoring program for emerging contaminants in Casco Bay, CBEP will conduct research on potential methods for monitoring and prioritizing these contaminants.

CBEP is committed to the implementation of these new *Casco Bay Plan* actions. In 2006, CBEP began working on the implementation of Toxics Action #2: Develop Biological Indicators for Maine's Waters. The Tiered Aquatic Life Uses (TALU) framework relates the declining health of an aquatic ecosystem to increasing human disturbance (including toxic pollution) along a gradient and associates tiers along the gradient with designated water body uses. TALU has already been used to develop biological criteria for rivers and streams in Maine. In partnership with Maine DEP, USEPA and other National Estuary Programs, CBEP is helping to develop biological criteria for estuarine waters by exploring application of the TALU approach in Casco Bay.

Summary/Conclusions

Toxic chemicals are found throughout Casco Bay and its watershed: in the air, in the sediments, in the aquatic environment, and in the tissues of many types of wildlife, in some cases at levels that threaten ecosystem health. Through the dedicated efforts of state, federal, and local government, businesses and citizens, the loading of many toxic chemicals, including mercury, PCBs, dioxin, tributyltin, and pesticides, has declined dramatically over time.

Continued decreases in the loading of toxic chemicals will require an ongoing commitment from government agencies and the citizens of the Casco Bay watershed to use all of the available tools, including: regulatory enforcement; monitoring and assessment of sources, risks, and impacts; development of new approaches to reduce the use and release of toxic chemicals; and vigorous environmental stewardship at every level.

References

Casco Bay Estuary Partnership. 1996, updated 2005. *The Casco Bay Plan*. (http://www.cascobay.usm.maine.edu/) (August 21, 2006).

Casco Bay Estuary Partnership. 2005a. *State of the Bay.* (http://www.cascobay.usm.maine.edu/) (August 21, 2006).

Casco Bay Estuary Partnership. 2005b. *Draft Casco Bay Plan Update.* (http://www.cascobay.usm.maine.edu/pdfs/DRAFT_CBP_Update.pdf) (August 24, 2006).

Maine Department of Environmental Protection, 1997. *Marine Oil Spill Contingency Plan*. (http://mainegov-images.informe.org/dep/rwm/publications/pdf/contplan.pdf) (March 8, 2006).

Maine Department of Environmental Protection. 2003. *Toxics and Hazardous Waste Reduction Pollution Prevention (P2) Planning Guidebook.* (http://mainegov-images.informe.org/dep/oia/thwrp/thwrpguide.pdf) (August 21, 2006).

Maine Department of Environmental Protection. 2005a. *Pollution Prevention(P2).* (http://www.maine.gov/dep/p2.htm) (August 21, 2006).

Maine Department of Environmental Protection. 2005b. *The Water Toxics Rule*.(http://www.maine.gov/dep/blwq/docstand/wd/toxics/index.htm) (August 21, 2006).

Maine Department of Environmental Protection. 2005c. *Brightwork: A Best Management Practices Manual for Maine's Boatyards and Marinas.* (http://www.maine.gov/dep/blwq/docwatershed/marina/bmp.htm) (August 22, 2006).

Maine Department of Environmental Protection. 2005d. *Draft Maine Air Toxics Priority List & Basis Statement*. (http://www.maine.gov/dep/air/toxics/mati-docs.htm) (October 7, 2005).

Natural Resources Council of Maine. 2006. *Progress on Mercury Reductions in Maine*. (http://www.maineenvironment.org/mercury_timeline.asp) (August 22, 2006).

United States Environmental Protection Agency. 1992. *What You Can Do to Reduce Air Pollution*. EPA 450-K-92-002.

United States Environmental Protection Agency. 1997. *The Environmental and Health Benefits of the Final Pulp and Paper "Cluster Rule" and the Incentives Program*. (http://www.epa.gov/waterscience/pulppaper/jd/fs2.pdf) (August 21, 2006).

United States Environmental Protection Agency. 2003. *Questions and Answers About Dioxin*. (http://www.epa.gov/ncea/dioxinqa.htm) (August 21, 2006).

United States Environmental Protection Agency. 2005. *2004 National Listing of Fish Advisories.* (http://www.epa.gov/waterscience/fish/advisories/fs2004.html#2004) (August 21, 2006).

United States Environmental Protection Agency. 2006a. *Enforcement in New England*. (http://www.epa.gov/region1/enforcement/) (August 21, 2006).

United States Environmental Protection Agency. 2006b. *Pollution Prevention (P2) Resources* (http://www.epa.gov/region1/assistance/p2/) (August 21, 2006).

United States Environmental Protection Agency. 2006c. *Dredged Material Management.* (http://www.epa.gov/owow/oceans/regulatory/dumpdredged/dredgemgmt.html) (September 14, 2006).

United States Environmental Protection Agency. 2006d. *Technology Transfer Network 1999 National Scale Air Toxics Assessment.* (http://www.epa.gov/ttn/atw/nata1999/) (August 21, 2006).

Glossary

acrolein: A toxic organic chemical found in vapor form that is used in some industrial processes. It can also be formed when organic matter is burned.

action level: The concentration of a contaminant in fish or shellfish below which there should be negligible risk of deleterious health effects, at a consumption rate of one meal per week.

Ag: Silver

Al: Aluminum

As: Arsenic

ambient water quality: The natural concentration of water quality constituents prior to the mixing of either point or nonpoint source load of contaminants.

anthropogenic: This term pertains to the influence of human activities. For example, anthropogenic sources of water quality impacts include septic systems and treatment plant discharges as well as road and agricultural runoff.

ATAC: Maine Air Toxics Advisory Committee

atmospheric deposition: The process by which airborne pollutants fall to the ground in raindrops, in dust, or due to gravity.

background or baseline reference condition: An environmental condition that is relatively free of industrial and anthropogenic influences. Background or baseline reference levels of toxic chemicals are compared to the results of monitoring (for example, blue mussel tissue monitoring) to assess pollution impacts.

BEAM: Maine DEP's Breathing Easier through Monitoring program

benthic: This term refers to the bottom of a body of water. For example, benthic organisms are bottom-dwellers.

bioaccumulation: The sequestering of toxic chemicals in the tissues of an organism at a higher concentration than the source. Bioaccumulation results from contact with contaminated water or sediment or by consuming prey.

bioindicator: Resident organism that serves as an indicator of environmental contamination.

biomagnification: The increasing concentration of toxics in organisms with each step up the food chain from the lowest to the highest links.

biomarker: An indicator that can be used to measure a biological process.

biosentinel: Resident organism that serves as an indicator of environmental contamination.

biota: The animal and plant life of a given region.

BMP: Best Management Practice. A BMP is a method for preventing or reducing the pollution resulting from an activity. The term originated from rules and regulations in Section 208 of the Clean Water Act.

BOD: Biochemical oxygen demand. This is the amount of oxygen used for biochemical oxidation by a unit volume of water at a given temperature and for a given time. BOD is an index of the degree of organic pollution in water.

body burden: The amount of a chemical present in the body of an organism.

BT: Butyltin

butyltins: Toxic organometallic compounds, i.e., molecules in which metal is bonded to a carbon atom in an organic molecule.

carcinogen, carcinogenic: A substance or agent that can cause or aggravate cancer.

CBEP: Casco Bay Estuary Partnership

Cd: Cadmium

CERCLA: The federal Comprehensive Environmental Response, Compensation, and Liability Act; 42 USC §§ 9601 *et seq.* (1980)

CHLs: Chlordane-related compounds. Chlordane is a pesticide banned in the US.

Clean Air Act: Federal legislation that regulates air pollution; 42 USC. §§ 7401 *et seq.*

Combined Sewer Overflow Control Policy: A national framework for control of combined sewer overflows through the National Pollutant Discharge Elimination System permitting program.

congeners: A chemical term for varying configurations in the same chemical family.

Cr: Chromium

CSO: Combined Sewer Overflow. A combined sewer system collects both stormwater runoff and wastewater in the same pipe where they are usually transported to a treatment plant before discharge to a water body. During heavy rainfall events, the volume of water can exceed the capacity of the sewer system or treatment plant, leading to a CSO in which untreated wastewater is discharged directly to a water body.

Cu: Copper

CWA: The federal Clean Water Act, 33 USC §§ 1251 *et seq.*

DBT: Dibutyl tin

DDT: The pesticide *1,1,1-trichloro-2,2-bis(*p-*chlorophenyl)ethane*, also known as *dichloro-diphenyl-trichloroethane* was the first chlorinated hydrocarbon insecticide. US EPA banned sale and use of DDT in the United States in 1972 due to its persistence in the environment and bioaccumulation in the food chain.

dioxins and furans: Toxic organic chemicals that are formed when organic material is burned in the presence of chlorine. Incineration, pulp paper manufacturing, coal-fired utilities, diesel vehicles and metal smelting are all sources.

DW: Dry weight

emerging contaminants: These contaminants include pharmaceuticals and personal care products (such as antibiotics, steroids, hormones and other endocrine disruptors) and a variety of chemicals such as caffeine, cholesterol, fire retardant and insect repellents which may have or, in some cases, have been shown to have, detrimental effects on aquatic organisms and ecosystems.

endocrine disruptor: A chemical that mimics or disrupts the normal activity of hormones.

epizootic: An epidemic among animals

EPCRA: The federal Emergency Planning and Community Right-to-Know Act; 42 USC §§ 11001 *et seq.*

ERL: Effects Range Low (possible biological effects)

ERM: Effects Range Median (probable biological effects)

estuary: A semi-enclosed coastal water body having a free connection to the open sea and within which seawater is measurably diluted with fresh water.

Fe: Iron

FOCB: Friends of Casco Bay

GOMC: Gulf of Maine Council on the Marine Environment

HAPs: Hazardous air pollutants

hydrophobic: A term that refers to chemicals that do not readily dissolve in water.

heavy metals: Dense metallic elements such as lead, mercury, arsenic, cadmium, silver, nickel, selenium, chromium, zinc, and copper.

Hg: Mercury

high molecular weight PAHs: Polycyclic aromatic hydrocarbons with four or more benzene rings. They result from combustion processes.

indicator organisms: Resident organisms that serve as indicators of environmental contamination.

intertidal zone: Areas between high tide and low tide that are alternately exposed to seawater and air.

in utero: Within the uterus.

in vitro: In cell culture.

Lipophilic: Fat soluble.

LOAEL: Lowest Observed Adverse Effect Level is the lowest exposure level of a stressor at which there are statistically or biologically significant increases in frequency or severity of adverse effects between the exposed organisms and organisms that are not exposed to the stressor.

load, loading: The total amount of a material (pollutant) entering a system from one or multiple sources.

low molecular weight PAHs: Polycyclic aromatic hydrocarbons with three or fewer benzene rings. They are typically derived from weathered petroleum and diesel fuel.

lymphocytes: A type of white blood cell –T and B cells- involved in immune response to foreign substances.

lymphocyte proliferative response: An assay that measures the ability of circulating lymphocytes to respond to foreign substances in cell culture.

lw: lipid weight

Maine DEP: Maine Department of Environmental Protection

MATI: Maine Air Toxics Initiative

MDN: Mercury Deposition Network

MERI: Marine Environmental Research Institute

metabolite: A substance that is the product of biological changes to a chemical.

methyl mercury: A highly toxic organometallic compound. It is the form of mercury that is most easily absorbed and bioaccumulated into organisms.

MPRSA: The federal Marine Protection, Research, and Sanctuaries Act. It is also known as the Ocean Dumping Act; 33 USC §§ 1401 *et seq.*

mobile sources: Sources of air pollution from internal combustion engines that propel cars, trucks, trains, buses, airplanes, ATVs, snowmobiles, boats, etc.

µg/g: micrograms per gram

mutagenic: Causing alteration in the DNA (genes or chromosomes) of an organisms.

neurotoxin: A substance that causes damage to the tissues of the nervous system.

NOAA: National Oceanic and Atmospheric Administration

NADP: The federal National Atmospheric Deposition Program

NATA: National Air Toxics Assessment

ng/g: nannograms per gram

Ni: Nickel

NOAEL: No Observed Adverse Effect Level. This is the highest exposure level of a stressor at which there are no statistically or biologically significant increases in the frequency or severity of adverse effects between the exposed organisms and organisms that are not exposed to the stressor.

nonpoint source: An indirect discharge, not from a pipe or other specific source, such as stormwater runoff.

NPDES: The National Pollutant Discharge Elimination System regulates point source and municipal stormwater discharges to the waters of the United States; 33 USC § 1342.

Oil Pollution Act: Federal legislation that requires facilities that store large quantities of oil to prepare spill plans and adopt measures that prevent spills from reaching waterways; 33 USC §§ 2702 *et seq.*

organometallic: Molecules in which a metal is bonded to a carbon atom in an organic molecule.

oriented strand board: An engineered wood product formed by layered flakes of wood, bonded with wax and resin adhesives.

PAHs: Polycyclic aromatic hydrocarbons. These are toxic organic chemicals that come primarily from combustion of fossil fuels and wood, as well as fuel spills.

parapodia: The paired appendages of segmented marine worms.

Pb: Lead

PBDEs: Polybrominated diphenyl ethers. PDBEs are toxic chemicals widely used as flame retardants.

PCBs: Polychlorinated biphenyls. These are persistent, toxic organic chemicals that were formerly used in electric transformers and capacitors for insulating purposes and in gas pipelines as lubricant. Sale and new uses of PCBs were banned by US EPA in 1979.

PCB conformation: The spatial arrangement of atoms and bonds in a PCB molecule.

pelagic: Relating to or living in the open sea (*i.e.,* offshore not coastal).

PFOS: Perfluorooctanesulfonate. This is a highly persistent toxic chemical widely used as a flame retardant.

pg/g: picograms per gram

pinnipeds: Carnivorous, fur-bearing marine mammals with feet modified as flippers.

planar PCBs: The most toxic conformation of PCBs, based on health effects. They are also referred to as "dioxin-like" compounds.

point source: Any confined or discrete conveyance (such as a pipe) from which pollutants are or may be discharged into a watershed.

POPs: Persistent organic pollutants. Examples are PCBs, dioxins, and DDT.

ppb: parts per billion

ppm: parts per million

Pretreatment Program: A federal program that regulates discharges to publicly owned treatment works; 33 USC §§ 1251 *et seq.*

Pulp and Paper "Cluster Rule": Provides federal air and water emissions standards for the pulp and paper industry that reduce toxic pollution releases and virtually eliminates all dioxin discharges into surface waters.

RCRA: The federal Resource Conservation and Recovery Act; 42 USC §§ 321 *et seq.* .

retinol: Vitamin A.

sentinel or indicator organisms: Resident organisms that serve as indicators of environmental contamination.

sink: A place in the environment where a compound or material collects.

stressor: An ecological stressor is something, such as a chemical, that can potentially cause an adverse effect.

SWAT: Maine DEP's Surface Water Ambient Toxics Monitoring program

TALU: Tiered Aquatic Life Uses. This is a framework that relates the declining health of an aquatic ecosystem to increasing human disturbance along a gradient and associates tiers along the gradient with designated water body uses.

TBT: Tributyltin

TMDL: Total Maximum Daily Load. This is a calculation of the maximum amount of a pollutant that a waterbody can receive and still meet water quality standards, and an allocation of that amount to the pollutant's sources.

tomalley: The organ that serves as a lobster's pancreas and liver, a place where contaminants can accumulate.

trophic level: The position of an organism in the food chain.

TSCA: The federal Toxic Substances Control Act; 15 USC §§ 2601 *et seq.*

TSS: Total Suspended Solids. This is a measure of the suspended solids in wastewater, effluent, or water bodies.

USC: United States Code. This is the codification by subject matter of the general and permanent laws of the United States.

US EPA: United States Environmental Protection Agency

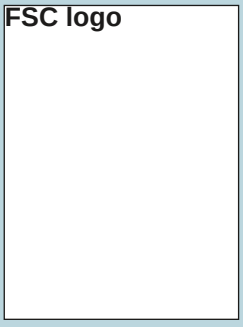
VOCs: Volatile organic compounds. These chemicals produce vapors readily. Gasoline and benzene are examples.

WCV: Wildlife criterion value refers to a derived maximum allowable surface water concentration of a pollutant, such as mercury, that should protect at-risk wildlife at the population level.

wt: weight

ww: wet weight

Zn: Zinc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