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Toxic Contaminants in the Chesapeake Bay and its Watershed: Extent and Severity of Occurrence and Potential Biological Effects

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EXECUTIVE SUMMARY

Purpose of the Report

For many years, scientists and resource managers have recognized that exposure to toxic contaminants can result in adverse effects on biological resources within the Chesapeake Bay and its watershed. In 2010, the Chesapeake Bay Program (CBP), a Federal-jurisdictional partnership, reported that 72 percent of the Bay's tidal-water segments are fully or partially impaired as a result of the presence of toxic contaminants. In some areas of the Bay watershed, fish-consumption advisories have been established as a result of concentrations of toxic contaminants. In recognition of these issues, the CBP developed the Toxics 2000 Strategy, in which commitments were made to prevent and reduce inputs of chemical contaminant and to eliminate toxic impacts on living resources that inhabit the Bay and its tributaries. Since 2000, new concerns, such as intersex conditions in fish, have arisen. Although the causes are undetermined, there is increasing evidence that contaminant exposures may play a role. In 2010, the President's Chesapeake Bay Executive Order (EO 13508) Strategy directed Federal agencies to prepare a report summarizing information on the extent and severity of occurrence of toxic contamination in the Bay and its watershed. Findings in this report will be used by the CBP partnership to consider whether to adopt new goals for reducing inputs of toxic contaminants entering the Bay. This report also identifies research and monitoring gaps that could be considered to improve the understanding of the extent and severity occurrence of toxic contaminants in the Chesapeake Bay and its watershed.

Approach

The findings in this report are based on a review of integrated water-quality assessment reports from the jurisdictions in the Bay watershed (Delaware, Maryland, New York, Pennsylvania, Virginia, West Virginia, and Washington, D.C.), Federal and State reports, and articles in scientific journals. The authors focused on summarizing results of studies conducted mostly since 2000 and, in particular, the 2010 jurisdictional water-quality assessment reports were used to define the extent and severity of occurrence of the following contaminant groups:

- **Polychlorinated Biphenyls (PCBs)**
- **Dioxins and Furans**
- **Polycyclic Aromatic Hydrocarbons (PAHs)**
- **Petroleum Hydrocarbons**
- **Pesticides**
- **Pharmaceuticals**
- **Household and Personal Care Products**
- **Polybrominated Diphenyl Ethers (PBDEs)**
- **Biogenic Hormones**
- **Metals and Metalloids**

The approach used to characterize the extent and severity of occurrence of contaminant groups is described in detail in Chapter 1 of this report. Extent is characterized as "widespread", "localized", or "uncertain" depending on the amount of information acquired from readily available reports and peer-reviewed literature and whether the contaminant has been detected throughout the watershed or only in a limited number of subwatersheds. Severity, as defined in this report, is based entirely on the jurisdictions' impairment determinations as identified in the integrated assessment reports. Contaminants that have caused impairments in many locations are considered to have widespread severity, contaminants associated with impairments in few locations are classified as having localized severity, and other contaminants or contaminant groups are identified as having uncertain severity. Where possible and appropriate, additional information such as peer-reviewed literature is included to provide perspective on potential severity, including evidence of adverse sublethal effects at environmentally relevant concentrations.

Conclusions about the Extent and Severity of Occurrence of Contaminant Groups

Overall conclusions about the extent of occurrence of contaminant groups examined in this report are --

- **Widespread extent:** For polychlorinated biphenyls (PCBs), polycyclic aromatic hydrocarbons (PAHs), herbicides (primarily atrazine, simazine, metolachlor, and their degradation products), and mercury, available information indicates widespread extent of occurrence throughout the Bay watershed.
- **Localized extent:** For dioxins/furans, petroleum hydrocarbons, some chlorinated insecticides (aldrin, chlordane, dieldrin, DDT/DDE, heptachlor epoxide, mirex), and some metals (aluminum, chromium, iron, lead, manganese, zinc), available information indicates localized extent of occurrence.
- **Uncertain extent:** For pharmaceuticals, household and personal-care products, polybrominated diphenyl ether (PBDE) flame retardants, some pesticides, and biogenic hormones, available information is insufficient to determine extent of contamination. However, the widespread distribution of known sources of these contaminants (e.g., wastewater effluents, agricultural runoff, etc.) in the watershed and the summarized occurrence data indicate that some contaminants from each of these groups may have the potential to be found in many locations throughout the Bay watershed.

Overall conclusions about the severity of contaminant groups examined in this report are --

- **Widespread severity:** For PCBs and mercury, impairments have been identified in many locations in the watershed, largely in response to concentrations in sediments and in fish tissues that frequently result in the need for fish-consumption advisories.
- **Localized severity:** For dioxins/furans, PAHs, petroleum hydrocarbons, some chlorinated pesticides (aldrin, chlordane, dieldrin, DDT/DDE, heptachlor epoxide, mirex), and some metals (aluminum, chromium, iron, lead, manganese, zinc), the report identifies localized severity on the basis of impairments in a limited number of areas in the Bay watershed.

- **Uncertain severity:** For atrazine, some pharmaceuticals, some household and personal-care products, some PBDEs, and biogenic hormones, severity as defined in this report could not be assessed. However, recent peer-reviewed research has documented sublethal effects for some compounds at environmentally relevant concentrations, raising concerns about the potential for adverse ecological effects.

Smallmouth bass with skin lesion.

Biological Effects of Toxic Contaminants on Fish and Wildlife

Additional supporting information on the toxic effects of contaminants on fish and wildlife is summarized to inform the discussion of severity. This information provides insights that can be used in assessing the cumulative and interacting effects of toxic chemicals as well as other stressors on fish and wildlife.

The following indicators of compromised fish health have been observed within populations in the Chesapeake Bay watershed: increased incidence of infectious disease and parasite infestations contributing to increased mortality in several species of fish; feminization (intersex, plasma vitellogenin) of largemouth and smallmouth bass and other signs of endocrine disruption; reduced reproductive success and recruitment of yellow perch in tributaries in certain highly urbanized drainage basins; and tumors in bottom-dwelling fish. The evidence for associations between exposure to toxic contaminants and these indicators of compromised fish health is discussed.

Indications of responses to contaminant exposure have also been found among wildlife in the Chesapeake Bay watershed, primarily wild birds. In a few locations, eggshell thinning associated with *p,p′*-DDE is apparent, and reproduction may be impaired. In some cases, organochlorine pesticides are found in eggs of predatory birds at concentrations associated with embryo lethality. Several studies are cited in which PCB concentrations in addled bald eagle eggs may have been high enough to contribute to the failure to hatch. Detectable concentrations of PBDEs have been found in eggs of predatory birds that approach the lowest-observed-adverse-effect level for pipping and hatching success.

Research and Monitoring Gaps

Monitoring gaps were identified for the following contaminant groups: dioxins and furans, petroleum hydrocarbons, some pesticides currently in use (e.g., insecticides and fungicides), pharmaceuticals, household and personal-care products, flame retardants, and biogenic hormones. Biological monitoring at many levels of biological organization (molecular to population) along with systematic evaluation of water contaminants and other stressors would allow for more effective documentation of the extent and severity of occurrence of toxic contaminants in the watershed.

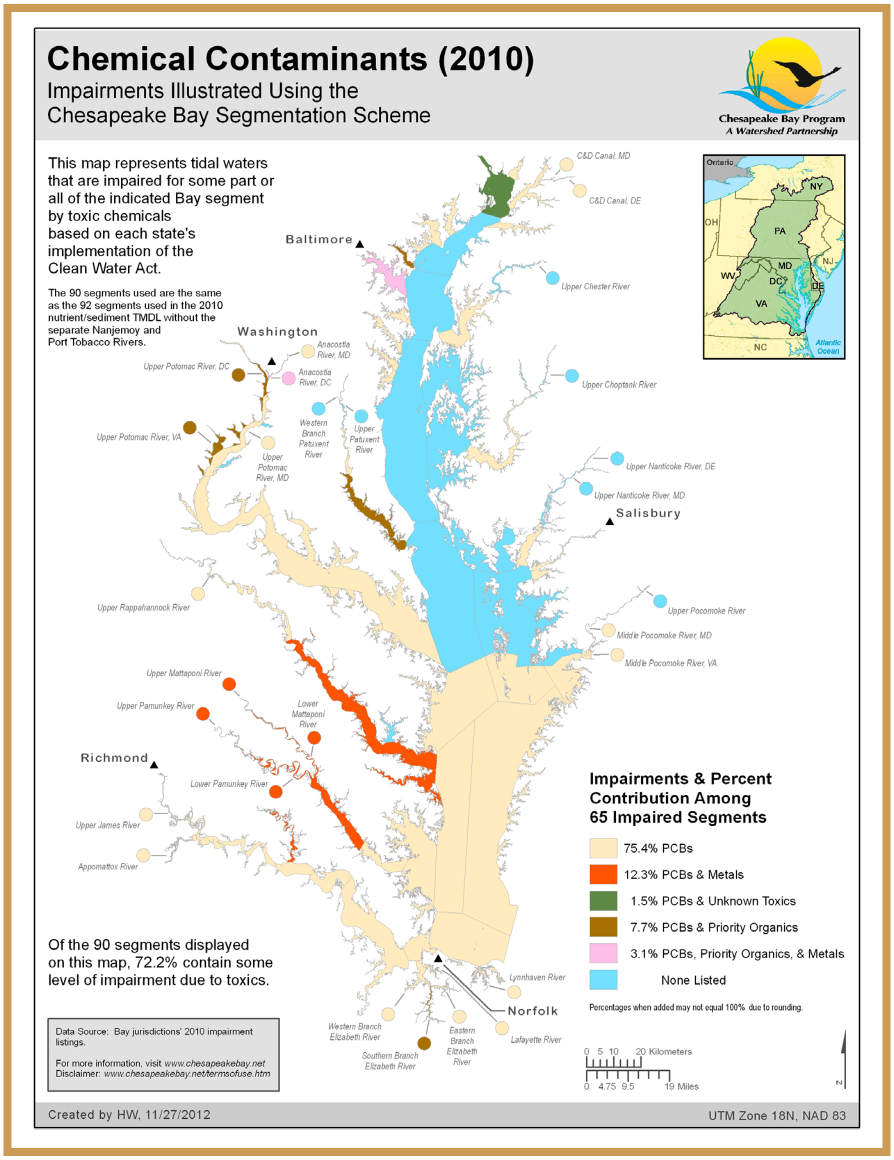
Research gaps that limit understanding of the relations between sources of these contaminants, their pathways to the environment, and exposures to receptor organisms are identified. Research that accounts for the complexities of the effects of contaminant mixtures and multiple stressors, sublethal effects, nonlinear dose-response curves, and the role of contaminant exposure in immune response and subsequent pathogenic disease would help to define relations between contaminant exposures and potential effects in fish and wildlife.

1.0 INTRODUCTION & BACKGROUND

For many years, scientists and resource managers have recognized that exposure to toxic contaminants can result in adverse effects on biological resources within the Chesapeake Bay and its watershed. Some contaminant effects, such as those from chlorinated pesticides, are well documented and have been addressed through various approaches to minimize the occurrence of targeted contaminants. Other contaminants with known effects, such as polychlorinated biphenyls (PCBs), continue to enter the Bay's ecological system. The potential for a range of land use activities such as human and animal waste management to provide sources of contaminants, such as pharmaceuticals, household and personal care products, and biogenic hormones has been documented; however, complete depictions of their occurrence, pathways to the environment, relative source contributions, and severity of effects from these environmental contaminants are the subjects of active research. The presence of toxic contaminants in the Chesapeake Bay has led to:

Source: Jane Thomas/IAN Image Library

- the Chesapeake Bay Program (CBP) adopting the Toxics 2000 strategy (USEPA 2000a), which made commitments to prevent and reduce chemical contaminant inputs and eliminate toxic impacts on living resources that inhabit the Bay and its tributaries,
- impairment of the quality of living-resource conditions to the extent that 72 % of the Bay and its tidal river segments (2010) are fully or partially impaired as a result of toxic contaminants (Figure 1),
- fish consumption advisories as a result of concentrations of certain toxic contaminants in fish in the Bay and its watershed,
- research indicating that conventional toxicological benchmarking approaches may not adequately represent the potential for contaminants to do ecological harm,
- realization that contaminants in the environment occur in mixtures that reflect complex combinations of land uses and contaminant sources and,
- the President's Executive Order strategy for protecting and restoring the watershed calling for new reduction goals and strategies for toxic contaminants.



Chemical Contaminants (2010)

Impairments Illustrated Using the
Chesapeake Bay Segmentation Scheme

This map represents tidal waters that are impaired for some part or all of the indicated Bay segment by toxic chemicals based on each state's implementation of the Clean Water Act.

The 90 segments used are the same as the 92 segments used in the 2010 nutrient/sediment TMDL without the separate Nanjemoy and Port Tobacco Rivers.

Impairments & Percent Contribution Among 65 Impaired Segments

- 🟨 75.4% PCBs
- 🟥 12.3% PCBs & Metals
- 🟩 1.5% PCBs & Unknown Toxics
- 🟫 7.7% PCBs & Priority Organics
- 🟪 3.1% PCBs, Priority Organics, & Metals
- 🟦 None Listed

Percentages when added may not equal 100% due to rounding.

Of the 90 segments displayed on this map, 72.2% contain some level of impairment due to toxics.

Data Source: Bay jurisdictions' 2010 impairment listings.

For more information, visit *www.chesapeakebay.net*
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Created by HW, 11/27/2012

UTM Zone 18N, NAD 83

Figure 1 – Tidal segments with full or partial impairments due to toxic contaminants

The President's Chesapeake Bay Protection and Restoration Executive Order (EO 13508) Strategy (May 12, 2010) directed Federal agencies to prepare a report summarizing information on the extent and severity of toxic contaminants in the Bay and its watershed. The findings in the report will be used by the CBP in 2013 to consider whether to establish new goals for reducing the input of toxic contaminants and, if established, to develop strategies by 2015 to carry out the goals.

Progress on Previous Agreements (*Toxics 2000*)

During December 2000, the CBP Executive Council adopted "Toxics 2000 Strategy: *A Chesapeake Bay Watershed Strategy for Chemical Contaminant Reduction, Prevention, and Assessment"* (USEPA 2000a). The agreement made substantial commitments to:

- prevent and reduce chemical contaminant inputs and eliminate toxic impacts on living resources that inhabit the Bay and rivers
- eliminate all chemical contaminant-related fish consumption bans and advisories
- clean up contaminants in the sediment in the three most urbanized areas referred to as "Regions of Concern" (i.e., Baltimore Harbor, Anacostia River, Elizabeth River)
- sustain progress in the face of increasing population and expanded development within the watershed.

Since 2000, the reduction of nutrient and sediment inputs has been the main emphasis of CBP activities. Progress has been made by Federal and State agencies as well as non-government organizations (NGOs) that are completing ongoing work to control chemical contaminants. Federal agencies such as the United States Environmental Protection Agency (USEPA) have continued to oversee and conduct numerous contaminated site cleanups that have improved conditions in the Bay and in the watershed. The jurisdictions have continued to enforce permit conditions including industrial wastewater permits. The jurisdictions have also continued to monitor fish tissue and other environmental media to fulfill their data needs for determining fish consumption advisories and impairment listings. Federal and jurisdiction agencies charged with implementing and enforcing the hazardous material and waste statutes that control the release of toxic contaminants have continued to fulfill their obligations. Federal agencies with science-based missions such as U.S. Geological Survey (USGS), National Oceanographic and Atmospheric Administration (NOAA), U.S. Fish and Wildlife Service (USFWS) and USEPA monitor the presence of chemical contaminants and assess possible ecological effects. Many of the results of Federal monitoring efforts are discussed in this report.

Progress has been made in at least two of the three previously designated Regions of Concern, the Elizabeth River and Anacostia River, due in part to the leadership provided by the Elizabeth River Project and Anacostia Watershed Restoration Partnership. For example in the Elizabeth River watershed, contaminated soil at a former naval shipyard was removed and the site was replanted to create a wetland. Multiple industrial sites are being cleaned up in the Elizabeth

River to reduce bottom sediment contaminated with PAHs and other pollutants. In the Anacostia watershed, stormwater retrofit projects have been completed to allow for improved treatment of stormwater originating from hundreds of acres in the river's watershed. The Anacostia is benefiting from a Total Maximum Daily Load (TMDL) that targets trash, which will reduce inputs of contaminants associated with household products and other industrial sources of waste. The Anacostia Watershed Restoration Plan (Anacostia Watershed Restoration Partnership 2010) is being implemented through multijurisdictional cooperation and includes projects that will reduce inputs of toxic contaminants to the river. During 2012, the USEPA Chesapeake Bay Program Office focused one million dollars of grant funds toward the Anacostia watershed. Both the Anacostia watershed and Baltimore Harbor were chosen for USEPA's Urban Waters Initiative, which is working to align Federal programs and investments and build local capacity for improving ecological conditions in these watersheds.

In 2006, the CBP completed an analysis of information that led to prioritization of organic pollutants for use in developing management strategies for reducing pollutant inputs. Although several of the contaminant groups that were identified as high priority in 2006 are also identified in this report, the 2006 prioritization was not substantially referred to in this report because the project team believed more current information was available. Strategies for reduction of those high priority pollutants were in development when the CBP organizational decision was made in 2007, to disband the former CBP Toxics Subcommittee to allow for greater focus on development of the nutrient and sediment TMDL. Prior to 2007, the efforts of the Toxics Subcommittee focused on further characterizing the condition of the Bay with regard to ecological impacts from toxic contaminants. The contaminants characterization data generated during that time are referred to in this report.

It is beyond the scope of this report to make a quantitative assessment of the progress made on the original commitments in the Toxics 2000 strategy. Since the strategy was written, the conditions that existed remain. According to the environmental indicator maintained by the CBP (see Figure 1), which measures the number of tidal segments with a partial or full jurisdiction-listed impairment due to toxic contaminants in 2010, a similar extent of impairment exists in the Bay compared with the previous version, based on 2008 jurisdiction impairment listings. Research has augmented our understanding of sublethal effects of contaminant mixtures and new issues, such as intersex characteristics in fish in the Bay watershed, have arisen. The focus of this report, therefore, is to summarize the current conditions of extent and severity of effects from toxic contaminants. The report findings will be used to assist the CBP in considering goals and strategies to reduce risk to the Bay's biological resources.

Report Purpose and Scope

This report summarizes readily available and acquired information about the extent of occurrence and severity of effects for the following groups of toxic contaminants in Chesapeake Bay and its watershed (Table 1).

Table 1 List of Contaminant Groups

1. Polychlorinated Biphenyls (PCBs)	*6. Pharmaceuticals*
2. Dioxins and Furans	*7. Household and Personal Care Products*
3. Polycyclic Aromatic Hydrocarbons (PAHs)	*8. Polybrominated Diphenyl Ether (PBDE) Flame Retardants*
4. Petroleum Hydrocarbons	*9. Biogenic Hormones*
5. Pesticides	*10. Metals and Metalloids*

The report also provides considerations for developing reduction goals if established, and identifies research and monitoring that could be conducted to better define the extent and severity of groups of contaminants. The report focuses on the severity of adverse effects of toxic contaminants on natural resources in the Bay and its watershed. It does not address potential effects on human health except in recognizing fish impairments and the status of fish consumption advisories established by the jurisdictions in the watershed. The extent of occurrence of toxic contaminants is defined for major groups of contaminants, such as PCBs and pesticides, which are known to occur in the Bay and its watershed. Information from the jurisdictions' integrated water quality assessment reports is a key resource in helping to define the extent and severity of toxic contaminants. Additional information from previously published Federal and academic studies was examined. For some groups of contaminants, such as PCBs, polycyclic aromatic hydrocarbons (PAHs), and some pesticides and metals, available information was used to characterize the contaminant as widespread, localized, or uncertain. For other groups of contaminants, including pharmaceuticals, personal care products, flame retardants, and hormones, data were limited; therefore, conclusions about extent of occurrence are constrained and less certain.

The findings will be used by the CBP to consider whether to establish new or updated goals for reduction of toxic contaminants. The primary audience is the decision makers in the CBP who are working to manage fisheries, habitat, water quality, and healthy watersheds. Several CBP Goal Implementation Teams (GITs) will be informed by this report on the effects of toxic contaminants. The Fisheries GIT may use the findings to better understand the health of fisheries in the Bay and its watershed. The Habitat GIT may use the findings to understand effects on wildlife (especially waterfowl) that use coastal wetlands and submerged aquatic vegetation. Because the Water Quality GIT is working to make waters both fishable and swimmable, the information developed in this report may help to develop nutrient and sediment reduction efforts that are designed with consideration of potential toxic contaminant effects. The Healthy Watersheds GIT may work to prevent impacts of toxic contaminants on healthy watersheds. The Water Quality GIT will coordinate with the other GITs and will use the information to work with CBP leadership groups such as the Management Board and Principal's Staff Committee to consider whether to establish new goals to reduce toxic contaminants (during 2013) and, if established, to develop more detailed strategies to carry out the goals (by 2015).

Assessment Approach

Studies published from 2000 onward were used as the source of current environmental data. The primary sources of information were studies prepared by scientists from State and Federal agencies, colleges and universities, consulting firms, and NGOs. It was beyond the scope of this report to assemble a toxic contaminant data base for the Chesapeake Bay watershed and statistically analyze and interpret that data. Trend analysis was also beyond the scope of this report.

Chapter 2 of the report is a detailed technical assessment of the extent and severity of toxic contaminant concentrations and effects for ten contaminant groups (Table 1). These contaminant groups include both chemical classes (e.g., PCBs and PAHs), products with similar use (e.g., pharmaceuticals), and biological products (biogenic hormones). The contaminant groups were selected as representative of major categories of contaminants in terms of potential natural and anthropogenic sources and pathways to the environment, mobility in the environment and potential for widespread extent, and known or suspected adverse ecological impact. Thus, the selected contaminant groups do not include all potential toxic contaminants that could be found in the watershed. For example, volatile organic compounds (VOCs) were not assessed as a specific group of compounds but many individual VOC compounds were included within the representative contaminant groups. These determinations were based on best professional judgment of the writing team and include chemicals that have been studied for decades as well as contaminants of emerging concern. Within each section of Chapter 2, representative and readily available concentrations of the contaminant groups in various media (usually surface water, sediment, and tissue from fish, birds, and mammals) are summarized.

A consistent approach (Figure 2) is used to evaluate the extent and severity of toxic chemical effects in the Bay watershed for each of the ten contaminant groups. Representative and readily available information (i.e. jurisdiction assessment reports, summaries of databases, published reports and journal articles) was acquired and considered for each contaminant group. Geographic extent was categorized as "widespread" if detectable concentrations were widely distributed across the Chesapeake Bay watershed and "localized" if only in specific areas. This is based on representative environmental media including water, sediment, and biota tissue as acquired. In cases where occurrence data were not available, other factors known or suspected to control the geographic extent of a contaminant or contaminant group, such as sources, land use, and pathways to the environment are considered and discussed.

The assessment of severity was based primarily on the monitoring reports that states are required to prepare under the Clean Water Act. Section 305(b) requires each State (and the District of Columbia) to monitor, assess and report on the quality of its waters in terms of designated uses. These uses include supporting aquatic life, fish consumption, recreation, and shellfish harvesting. Monitoring and assessment data are evaluated with respect to these designated uses. If the state agency interprets the concentration data as exceeding the state water quality standards (e.g., due to water column concentrations of specific contaminants or fish tissue concentrations that limit human consumption), the state identifies the water body as impaired for a particular designated use. Section 303(d) of the Clean Water Act requires each State to compile a list (Impaired Waters List) identifying those waters not meeting water quality standards. Impaired waters are defined as any water bodies that are not supporting one or more designated uses. Every two years, each State either prepares a report for each section of the Clean Water Act or an Integrated Water Quality Report that covers both sections. These reports describe the status of all assessed waters, list impaired waters and the causes of impairment, and provide the status of actions being taken to restore impaired waters.

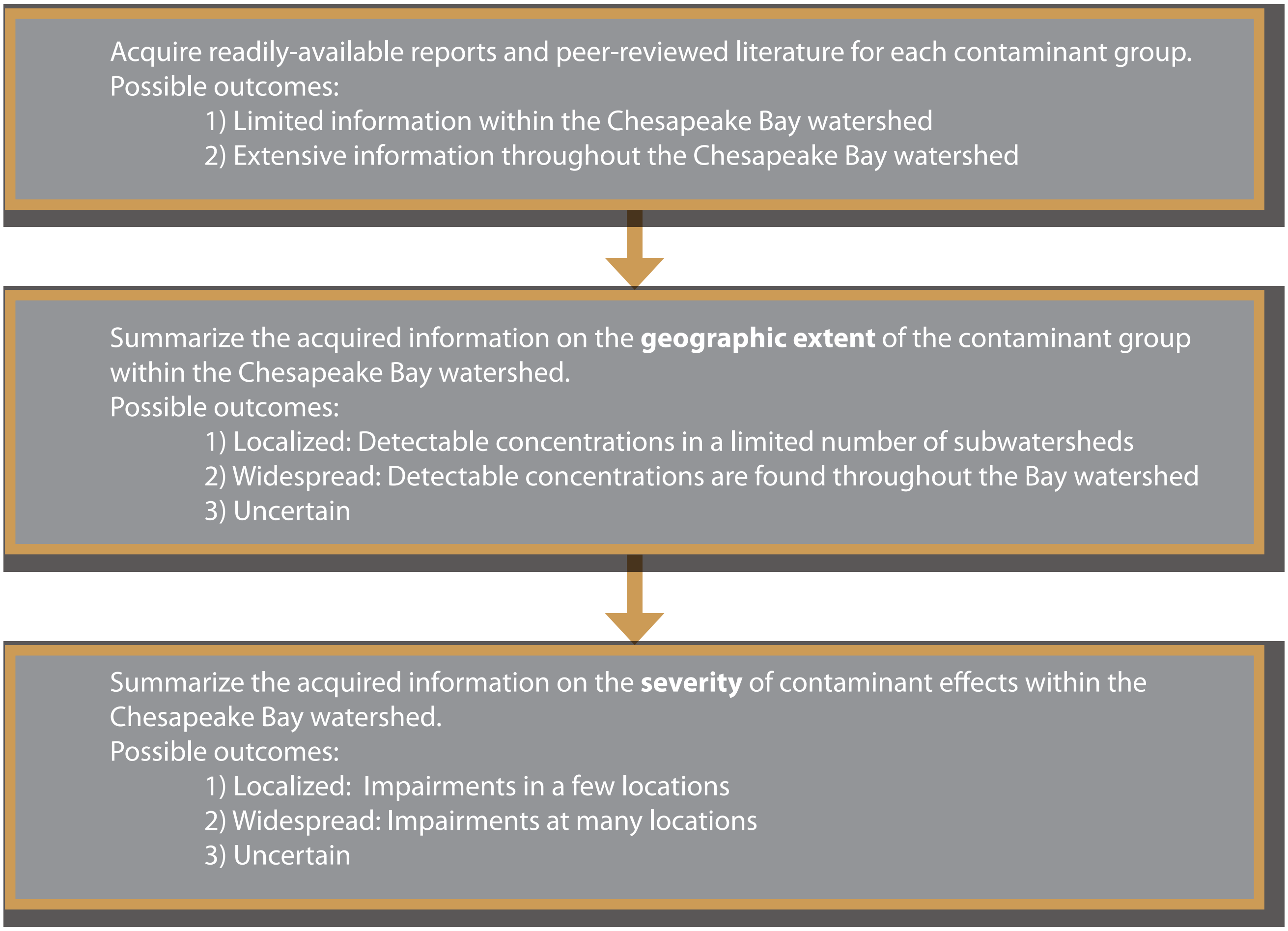
Causes of impairment include chemical contaminants (such as PCBs and metals), physical conditions (such as elevated temperature, excessive siltation, or alterations of habitat), and biological contaminants (such as bacteria).

This report used the latest available (2010) water quality reports prepared by the jurisdictions in the Bay watershed (Delaware, Maryland, New York, Pennsylvania, Virginia, West Virginia, and Washington, D.C.) as the primary sources of information for describing the extent of impaired waters within the Bay watershed. The agencies include: Delaware Department of Natural Resources (DNREC), Maryland Department of the Environment (MDE), New York State Department of Environmental Conservation (NYSDEC), Pennsylvania Department of Environmental Protection (PADEP), Virginia Department of Environmental Quality (VADEQ), West Virginia Department of Environmental Protection (WVDEP), District of Columbia Department of the Environment (DDOE) The report focuses on impairments due to chemical contamination that limit aquatic life support and/or limit fish consumption. Within each State, only those sub-watersheds that are part of the Chesapeake Bay watershed were evaluated. If impairments are identified at many locations in the watershed, severity is classified as "widespread"; if impairments are identified at few locations, severity is classified as "localized" (Figure 2). The uncertainties associated with these categories are discussed below.

A second closely related major source of information on severity of a contaminant group was the jurisdictions' listings of fish tissue advisories, which overlaps considerably with the waters listed as impaired for fish consumption. The advisories were screened against the Chesapeake Bay watershed boundaries. Although this report is largely focused on ecological rather than human health effects of contaminants, restrictions on fish consumption are included because they represent a lost or restricted use of the water resource.

A third major source of information for describing severity was the sediment chemistry, benthic macroinvertebrate community, and sediment toxicity studies conducted at various locations within the watershed. The synoptic collection of these three types of data (using the same sampling locations and timing) is termed the "sediment triad approach" (e.g., Chapman 1990) and is a useful method for characterizing the quality of freshwater, estuarine, and marine habitats. A Bay-wide study of 210 sediment sampling locations was conducted by the National Oceanic and Atmospheric Administration (NOAA) in 1998, 1999, and 2001 and reported by Hartwell and Hameedi (2007). Smaller scale sediment triad studies within the Bay watershed were prepared by Pinkney et al. (2005) and Fulton et al. (2007).

Figure 2. Approach for assessing the extent and severity of effects for each contaminant group within the Chesapeake Bay watershed.



Acquire readily-available reports and peer-reviewed literature for each contaminant group.
Possible outcomes:
1) Limited information within the Chesapeake Bay watershed
2) Extensive information throughout the Chesapeake Bay watershed

Summarize the acquired information on the **geographic extent** of the contaminant group within the Chesapeake Bay watershed.
Possible outcomes:
1) Localized: Detectable concentrations in a limited number of subwatersheds
2) Widespread: Detectable concentrations are found throughout the Bay watershed
3) Uncertain

Summarize the acquired information on the **severity** of contaminant effects within the Chesapeake Bay watershed.
Possible outcomes:
1) Localized: Impairments in a few locations
2) Widespread: Impairments at many locations
3) Uncertain

Additional reports on Bay tributaries or watersheds aimed at specific environmental questions or problems were summarized. Examples include a series of studies of fish kills within the Potomac watershed (e.g., Blazer et al. 2007, 2010; Ciparis et al. 2012); fish tumor surveys (e.g., Pinkney et al. 2009; Vogelbein and Unger 2006); and assessments of the status of the Anacostia River (e.g., Anacostia Watershed Toxics Alliance 2009, Velinsky et al. 2011; McGee et al. 2009).

Benchmarks, defined as standards or points of reference used for comparisons or assessments, were identified and used to provide context for the concentration data. For many contaminant groups, however, no benchmarks are available. Study authors, including state agencies, frequently compare water column concentrations with state water quality standards (Appendix A, Table A-1). Since the Bay states do not have sediment quality standards, the study authors and state agencies compared sediment concentrations with guidance values they identified (Appendix A, Table A-2). The most commonly used guidelines for evaluating the severity of sediment contamination in estuarine and marine sediment are provided by the effects range-low (ERL) and effects range- median (ERM) developed by NOAA (Long et al. 1995). The ERM is the 50th percentile concentration of a data set reporting adverse effects to a variety of benthic invertebrates from estuarine and marine environments; adverse effects occur more frequently than not above this concentration. The ERL is a lower threshold concentration (10th percentile value from the same data set), below which toxic impacts are unlikely to occur; concentrations between the ERL and ERM may occasionally result in adverse effect (Long et al. 1995). For freshwater sediments, consensus-based guidance values were derived as threshold effects concentrations (TECs) and the probable effects concentration (PECs) (MacDonald et al. 2000) and are functionally similar to the ERL and ERM. TECs identify contaminant concentrations below which harmful effects on sediment dwelling organisms are not expected. PECs are contaminant concentrations above which harmful effects on sediment-dwelling organisms are expected to occur frequently.

All four sediment guidance values are empirically based and are used in this report as screening values and not as risk thresholds. Other more theoretically based guidance values include the equilibrium partitioning approach and evaluation of the relationship between simultaneously extracted metal concentrations (SEM) and acid volatile sulfide (AVS) concentrations. In the reports summarized, the authors compared sediment contaminant concentrations with ERL, ERM, TEC, and PEC values. In the current report, the higher benchmarks (ERM and PEC), which are more frequently associated with adverse effects on the benthic community than the lower benchmarks (ERL and TEC), are emphasized. The authors relied on the guidance values used in the cited studies because they did not have the resources to reevaluate the data and reinterpret the results using alternate approaches. It is recognized that these thresholds are only based on direct effects on benthic organisms and do not address effects on fish (e.g., development of liver pathology such as tumors) or concerns about bioaccumulation. There are also limitations inherent in relying on these empirically-based guidance values, especially for mixtures of PAHs where the narcotic mode of action provides a theoretical basis for applying the equilibrium partitioning approach to estimate benchmarks (DiToro and McGrath 2000).

For tissue data (largely for fish, birds, and mammals), the residue concentrations were compared against critical residue thresholds (those associated with toxic effects) but again, many of the contaminants assessed have limited, or no, threshold values. Many thresholds have been compiled and described in three books (Beyer et al. 1996, Hoffman et al. 2003, and Beyer and Meador 2011), which were used as the primary source of this information in the report. Food chain modeling to assess risks to birds and mammals was beyond the scope of the report.

Chapter 3, *Responses of Fish to Cumulative and Interacting Stressors*, summarizes studies that indicate the adverse effects of the exposure of fish to complex mixtures of both traditional contaminants, contaminants of emerging concern, and multiple stressors (both biological and chemical). Chapter 4, *Contaminant Exposure and Responses in Wildlife*, is a synopsis of the wildlife information spread among the sections on individual contaminant groups in Chapter 2, and is largely focused on effects on birds and mammals. A major source of the wildlife data was the Contaminant Exposure and Effects—Terrestrial Vertebrates (CEE-TV) database (www.pwrc.usgs.gov/contaminants-online) containing over 20,000 geo-referenced data records for marine and estuarine habitats across the nation (Rattner et al. 2005). Chapter 5 presents the summary and conclusions of the report.

There are uncertainties involved in summarizing and categorizing the extent and severity of toxic chemical effects for any contaminant or contaminant group. All determinations and the use of the words "limited", "extensive", "localized", and "widespread" reflect the lack of precise decision boundaries; however, the professional judgment used to support each of these determinations is supplied. Lack of sufficient geographic coverage of monitoring data across the watershed often prevents conclusions on widespread versus localized or uncertain extent of occurrence. In some cases, determining that there is widespread as opposed to localized severity was readily apparent where there are impairments identified at many locations in the watershed. It was beyond the scope of this report to make spatially explicit delineations (such as percentage of stream miles impaired) to establish rules for distinguishing localized versus widespread severity. Additional uncertainty in this report is inherent in its reliance on the jurisdictions' water quality reports to describe the severity of contaminant impacts. The jurisdictions have different methods of summarizing their monitoring data and comparing with standards, which may vary, to determine whether a water body is impaired. For many of the chemicals of emerging concern, there are no existing benchmarks or standards, and, therefore, no opportunity for the jurisdictions to identify impairments.

There may be substantial differences in detection limits used in different studies. In some cases, detection limits for certain analytes were above concentrations associated with adverse effects or even above state water quality standards. These and other factors can lead to additional uncertainty regarding the geographic extent and/or severity of a contaminant group. Consequently in some cases, a determination of the extent or severity was unsupported and a determination of "uncertain" was made (Figure 2). An uncertain determination does not imply presence or absence of potential extent or severity; where possible, relevant exposure and toxicity information was summarized to inform readers on the current status of these contaminant groups.

Finally, recent literature on sublethal effects of contaminants within multiple stressor environments has drawn attention to the potential inadequacies of conventional contaminant-by-contaminant benchmarking approaches (Feingold et al. 2010; Burton et al. 2012). Full consideration of this rapidly emerging aspect of environmental toxicology was not possible for determinations of severity as defined in this report. In some cases, the authors have provided additional context for considerations of severity of contaminants and contaminant groups by citing recent relevant peer-reviewed literature on the potential ecological effects of these contaminants. Representative toxicity studies conducted at environmentally relevant concentrations (i.e. concentrations known to occur in the watershed) were summarized. However, frequently there is insufficient monitoring data to determine the duration of exposure of sensitive organisms or vulnerable life stages, and the likelihood of adverse effects in the field.

The extent and severity of occurrence for the 10 groups of contaminants listed in Table 1 are presented in the following subsections. There is a subsection for each group, which includes an abstract to summarize the main points and conclusions.

2.1 Polychlorinated Biphenyls

Abstract

PCBs are a group of synthetic organochlorine chemicals widely used as dielectric and coolant fluids in transformers and capacitors. In 1977, the U.S. banned the production of PCBs out of concern for their persistence in the environment as well as evidence indicating that PCBs were bioaccumulative and has the potential to cause toxic impacts. Though PCBs are no longer produced, there are continued authorized uses of PCB-containing materials which pose the potential for environmental release. The inadvertent production of PCBs in certain manufacturing processes represents an additional contemporary source. Impairments resulting from PCB contamination are widespread. The Commonwealth of Virginia documents water column impairments in the Potomac and Shenandoah Rivers impacting nine river miles and one square mile of estuarine waters. All of the Bay jurisdictions have waterbodies identified as impaired for human consumption of fish tissue. The District of Columbia and the State of New York have general fish consumption advisories out of concern for contamination of fish tissue from PCBs and other chemical contaminants. The State of New York includes a fish tissue impairment for Koppers Pond, located in the Chemung River basin. Delaware identifies a fish tissue impairment for the Chesapeake and Delaware Canal (C&D Canal). Maryland lists 30 assessment segments as impaired for fish tissue consumption. In Pennsylvania, the Susquehanna River is impaired for 208 river miles, extending from the New York/Pennsylvania state line to the city of Sunbury, PA. Virginia lists five Bay tributaries as impaired, impacting 456 river miles and 2,011 square miles of estuarine waters. In West Virginia, two waterbodies are impaired by PCB contamination in fish tissue: the South Branch of the Potomac River and the Shenandoah River. Unlike concentrations of chlorinated pesticides, concentrations of PCBs in tissues of many species of Chesapeake Bay wildlife have

not declined since the final USEPA rule restricting the manufacture, processing, and distribution of these compounds became effective in 1979. The available data indicates that the extent of PCB contamination within the Chesapeake Bay watershed is widespread, being detected in tissue and sediment in most sampling locations.

Background

PCBs are a group of synthetic organic chemicals with high thermal stability, making them important in applications such as dielectric fluids in transformers and capacitors, heat transfer fluids, and lubricants. In addition, PCBs were used in plasticizers (e.g., carbonless paper), inks, adhesives, sealants and caulk. There are no natural sources of PCBs to the environment. PCBs typically exist as mixtures of chlorinated biphenyl compounds with varying degrees of chlorination. A total of 209 possible compounds, known as congeners, result from the variation of chlorination (1 – 10 chlorine atoms) around the biphenyl rings (Agency for Toxic Substances and Disease Registry (ATSDR) 2000).

PCBs are relatively insoluble in water with solubility decreasing with increasing chlorination. These hydrophobic compounds dissolve readily in nonpolar organic solvents and in biological lipids. Due to different degrees of chlorination, the physical and chemical properties vary among the congeners (ATSDR 2000).

PCBs have not been produced in the U.S. since August of 1977 due to evidence that this group of compounds was persistent and bioaccumulative in the environment and had the potential to cause toxic effects. Aside from the historical contributions of PCBs to the environment, PCBs continue to be released to the environment, however, through leaks or fires in PCB-containing equipment, accidental spills, illegal or improper disposal, burning of PCB-containing oils in incinerators, and leaks from hazardous waste sites (Total Maximum Daily Load reports: MDE 2009a, 2009b, 2009c, 2011a, 2011b, USEPA and VADEQ 2001, Haywood and Buchanan 2007). Point source discharges are regulated; however, discharges of PCBs may continue as a result of historical contamination or inadvertent production (Oregon Department of Environmental Quality (ODEQ) 2012, Du et al. 2008, Hu and Hornbuckle 2010). Specific processes implicated in inadvertent production have been identified as those that involve chlorinated solvents, paints, printing inks, agricultural chemicals, plastics and detergent bars (ODEQ 2012).

In addition, there are continued authorized uses of PCBs including in sealed systems such as transformers and some heat transfer systems (ODEQ 2012). ODEQ (2012) provides additional information on potential sources in waste materials and recycling operations.

In the aqueous environment, the higher molecular weight PCBs (i.e., more chlorinated) are typically sorbed to suspended solids and sediment whereas the lower molecular weight PCBs tend to volatilize to the atmosphere. Once in the environment, PCBs cycle among environmental media (air, water, soil/sediment, biota). Volatilized PCBs are redeposited to land and water through precipitation events (ATSDR 2000).

PCBs in the water column can be removed through volatilization at the air-water interface, through sorption to sediments and suspended solids, and by uptake in aquatic organisms (ATSDR 2000). Uptake in aquatic organisms can occur through bioconcentration and/or bioaccumulation. In bioconcentration, uptake occurs directly from the water column whereas bioaccumulation occurs through the combined uptake of food, water and sediment. Concentrations of PCBs increase through the higher trophic levels. As a result of the lipophilicity of these compounds, they tend to accumulate within the tissues of the organisms (ATSDR 2000).

PCBs are listed as probable human carcinogens by the USEPA and the International Agency for Research on Cancer (ATSDR 2000). Studies of workers reported that PCBs were associated with cancer of the liver and biliary tract. ATSDR (2000) summarized studies that reported that women who consumed high amounts of PCB-contaminated fish gave birth to babies with lower birth weights. ATSDR noted that infants of these women had abnormal responses to behavior tests and that the infants' problems with motor skills and short-term memory persisted. Therefore, prenatal exposure and exposure of children through breast milk is a concern, and fish consumption advisories for PCBs are therefore more restrictive for children and women of child-bearing age than for the general population.

The data for this chapter were derived primarily from the state water quality assessment reports (DDOE 2010, MDE 2010, NYSDEC 2007, PADEP 2010, VADEQ 2010, WVDEP 2010) which documents a water body's attainment of its designated use. Water bodies that fail to meet the water quality standards or criteria applicable for the state's designated use are categorized as "impaired". These numerical thresholds may differ from state to state. Comparisons between the state standards will not be made; however, impairment identifications are noted. For informational purposes, current jurisdiction water quality standards for aquatic life protection and screening values for sediment quality are located in the appendices.

Though the integrated assessment reports do not provide quantitative data on PCB concentrations, such data were available from the TMDL reports prepared for several impaired waterways in the Chesapeake Bay watershed. Under the Clean Water Act, states must develop TMDL reports for each impaired water body, identifying the probable sources of impairment and the required load reductions from each source category necessary to comply with the standard. Several TMDL reports in the Chesapeake Bay watershed have been completed for fish tissue impairments and provide a source for quantitative data. NOAA provided an additional source of quantitative data in its sediment survey of the Chesapeake Bay conducted during 1998 to 2001, characterizing PCB concentrations across a large portion of the watershed (Hartwell and Hameedi 2007).

Measuring low concentrations of PCBs is subject to analytical challenges. Though not yet promulgated in the Federal Register for Clean Water Act programs, USEPA Method 1668, a low-level PCB method, is being used to support many TMDL studies. Though there is on-going debate and concern about the method's reliability at concentrations near its reported detection limit and its sensitivity to false positives, USEPA indicated that its use in state TMDL programs has been successful (USEPA 2012b).

Water

There are two categories of water quality standards applicable to PCBs: standards developed for the protection of aquatic life and standards developed for the protection of human health from the consumption of fish. Documented water column exceedances of the state water quality standards are uncommon. This could be attributable, in part, to the limitations of the analytical methods most commonly used for the routine assessment of state waters. Routine methods typically quantify a small subset of the total 209 PCB congeners and have analytical detection limits several orders of magnitude greater than the state standards (USEPA 2012a).

The Commonwealth of Virginia documents water column impairments in the Potomac and Shenandoah river basins. Approximately 9 river miles are impaired and 1 square mile of estuary is impaired as a result of PCB contamination (Virginia Department of Environmental Quality (VADEQ) 2010).

The multi-jurisdictional Potomac/Anacostia Rivers TMDL report identified a range of water column values for total PCBs from below detection to 0.34 µg/L (congener specific detection limits 2 - 8 pg/L) (Haywood and Buchanan 2007). Completed Maryland TMDL studies reported water column values for total PCBs ranging from 0.00009 to 0.03071 µg/L (MDE 2011a, MDE 2009a, MDE 2009b, MDE 2009c, MDE 2009d). Virginia and West Virginia collaborated for the Shenandoah River TMDL and measured water column values for total PCBs ranging from 0.0000077 to 0.0000791 µg/L (USEPA 2001). Pennsylvania's Susquehanna River TMDL report indicated a water column value of 0.0276 µg/L for total PCBs (PADEP 1999). Water quality standards for total PCBs in water for these states range from 0.014 to 0.030 µg/L (freshwater and saltwater chronic values, respectively).

Sediment

Maryland documents impairments based on exceedances of screening values in sediment (Bear Creek, Curtis Bay, and Baltimore Harbor, MDE 2010). The other states in the Chesapeake Bay watershed did not document impairments for PCBs in sediment. In Virginia, 208 stations were monitored for PCBs in sediment in conjunction with the Commonwealth's freshwater probabilistic monitoring program. PCBs were detected in all samples but were below the PEC screening value of 676 ppb (VADEQ 2010). Virginia conducted additional sediment monitoring as part of a toxicological characterization effort (Roberts et al 2002, 2003, 2004). There were very few instances in which PCB congeners were detected in the sediment. In the Mattaponi and Pamunkey Rivers, the concentrations were "well below the concentrations expected to produce adverse impacts" (Roberts et al. 2004). In the Mattaponi, the results ranged from < 6 – 125 ppb dw and in the Pamunkey, the results ranged from < 5 – 70 ppb. Historical data from the Nanticoke River in Delaware indicate that PCB concentrations "were not detected at levels expected to pose a significant risk to aquatic life or human health" (DNREC 1997). More recent sediment core data were collected to support a maintenance dredging project in the Nanticoke (EA 2006). The resulting data showed that concentrations in the sediment ranged from approximately 29 – 44.5 ppb.

NOAA completed a survey of toxic contaminants in sediments Bay-wide from 1998 to 2001, including PCB analysis for a list of approximately 22 congeners. Sediment concentrations from samples in the Bay tributaries tended to be higher than those collected from the embayments and mainstem (Hartwell and Hameedi 2007) and ranged from below detection to 122 ppb.

None of the sediment concentrations of total PCBs exceed the ERM level of 180 ppb, however, several sites did document levels above the ERL of 22.7 ppb (Susquehanna Flats, Bay Bridge, and the Elizabeth River). The sediment values for the Chesapeake Bay embayments were all below the ERL.

In 1994, NOAA released a report documenting sediment contamination in the Chesapeake and Delaware Bays (NOAA 1994). At that time, sediment concentrations of total PCBs around Fort McHenry (Baltimore, MD) ranked among the highest total PCB sediment concentrations documented at NOAA's National Status and Trends (NS&T) sites around the country (above the 89th percentile). The measured concentration of total PCBs at this site was 679 ppb, above the ERM of 180 ppb. The more recent NOAA report (Hartwell and Hameedi 2007) did not include data from the Fort McHenry area.

The Potomac/Anacostia Rivers TMDL identified a range of sediment values of total PCBs from nondetect – 1,550 ppb dw (Haywood and Buchanan 2007). Completed Maryland TMDL studies reported sediment values ranging from 1.4 – 59.14 ppb dw (MDE 2011a, MDE 2009a, MDE 2009b, MDE 2009c, MDE 2009d). Virginia and West Virginia collaborated for the Shenandoah River TMDL study and measured sediment values for total PCBs ranging from 0.31 – 100 ppb (USEPA 2001).

PCBs in sediments at concentrations above certain thresholds pose risks to aquatic life through several pathways. First, sediment-bound PCBs serve as a source for bioaccumulation in prey that ultimately results in fish contamination. This topic is covered extensively by Haywood and Buchanan (2007) in the TMDL document for the tidal Potomac River. They calculated bioaccumulation factor (BAF)-based target sediment concentrations of 2.8 to 12.0 ppb. These would translate to fish tissue concentrations at or below the impairment thresholds of the District of Columbia, Maryland, and Virginia. A high percentage of the sediments monitored in the tidal Potomac have total PCB concentrations above these targets, hence the requirement for a 96% reduction in PCB loading in order to achieve the TMDL (Haywood and Buchanan 2007).

Eggs, larvae, and juveniles of Bay fish species are exposed to PCB-contaminated sediments and it is likely that these life stages are more sensitive than adults (Eisler and Belisle 1996). In addition, maternal transfer of PCBs occurs during oogenesis (Fisk and Johnston 1998). Calculations of sediment thresholds for toxic effects in fish have been conducted for juvenile salmonids by Meador et al. (2002) who evaluated 15 studies that reported total PCB tissue concentrations and toxic effects. Using literature-based biota sediment accumulation factors (BSAFs) and lipid calculations, they suggested a sediment effect threshold concentration ranging from 75 to 600 ppb dw. The range depended on the BSAF and percent total organic carbon in the sediments. These sediment thresholds for the protection of salmonids are higher than the TMDL targets of Haywood and Buchanan (2007).

Fish and Shellfish

Fish tissue advisories and impairments

The state water quality standards regulating fish tissue concentrations are designed to protect human health by minimizing dietary exposure to PCBs through fish consumption. These concentrations can be elevated to unacceptable levels as a result of interactions with sediment, the water column and through trophic transfer. Though high resolution data may not often be available for sediment and water in waterways that have fish tissue impairments, the bioaccumulation of PCBs in excess of state standards or screening values may indicate that total PCB concentrations in sediment and water are contributing to the impairment.

All of the Bay jurisdictions have water bodies listed with fish consumption advisories due to PCB fish tissue concentrations in excess of a state standard or health department threshold. Most advisories limit exposure in terms of meals per week or month and there is variation in the formulas used to calculate the restrictions.

The District of Columbia issued fishing advisories for all its waters in order to minimize the risk of human exposure to elevated levels of PCBs and other chemicals in fish tissue (DDOE 2010).

PCB contamination is the principal toxicological driver for the fish tissue impairment identified in Delaware's Chesapeake and Delaware Canal (C&D Canal) (DNREC 2010 and R. Greene, DNREC, personal communication). The advisory is based on data generated for a 1999 report. Delaware is updating its toxics data for the C&D Canal in 2013 (R. Greene, DNREC, personal communication).

The State of Maryland lists more than 30 segments in the Bay watershed for fish tissue impairment. In the Severn River mesohaline segment, the State's assessment report indicates that fish tissue concentrations may be low enough to meet the standard; however, additional data are needed for confirmation (MDE 2010). Completed Maryland TMDLs document a range of fish tissue values of 22.1 to 608.9 ppb (MDE 2009a-d, MDE 2011a).

The State of New York has a general fish consumption advisory for all state waters in order to minimize the risk of human exposure to elevated levels of PCBs and other chemicals in fish tissue. The New York Department of Health (NYDH) advises the public to consume no more than four one-half pound meals a month of fish, with no more than one meal per week (NYDH 2012). In the Chemung River basin, one impoundment is identified as impaired for PCBs in fish tissue (Koppers Pond) (NYSDEC 2007).

Within the Chesapeake Bay watershed of Pennsylvania, the Susquehanna River has a PCB fish consumption advisory for 208 miles from the New York/Pennsylvania state line to the city of Sunbury. A TMDL for a portion of the upper Susquehanna River basin has been completed and identifies an average fish tissue value of 860 ppb. The upper limit for unrestricted fish consumption is 50 ppb (PADEP 1999). PADEP is actively monitoring the PCB levels in fish in the lower section from Sunbury to the Bay (2006, 2008, 2011, and 2012) and none of the results have resulted in a fish consumption advisory for PCBs.

In Virginia, all five Bay tributaries have PCB fish tissue impairments with a total of 456 river miles and 2,011 square estuarine miles impacted. The James River has the highest number of impaired river miles (245 miles), whereas the mainstem Chesapeake Bay and its small coastal basins account for 79% of the impaired estuary footprint (VADEQ 2010). PCB TMDLs for the Potomac and Shenandoah Rivers have been completed (Haywood and Buchanan 2007; USEPA and VADEQ 2001).

West Virginia identified the South Branch of the Potomac River as impaired in the 2010 report. In the Shenandoah Jefferson watershed, the Shenandoah is identified as impaired but with a completed TMDL (WVDEP 2010). As indicated in the above discussion, the TMDL for the Shenandoah was completed in collaboration with the Commonwealth of Virginia and the USEPA. The report did not identify any major West Virginia sources of the contamination but identified an industrial point source and a landfill as the major potential sources of PCB contamination to the Shenandoah River (USEPA and VADEQ 2001). In its January 2012 fish consumption advisory, the West Virginia Department of Health and Human Resources (WVDHHR 2012b) indicated that PCB levels in the Shenandoah may be declining. The average fish tissue concentration (skin-off) was 250 ppb ww with a range from non-detect to 2,100 ppb (detection limit = 10 ppb).

All of the fish collected for skin-on analyses were below the WVDHHR screening value of 50 ppb ww (WVDHHR 2012a). The most recent Shenandoah data collected by the Commonwealth of Virginia (2005), however, indicates a continued need for fish consumption advisories because fish tissue concentrations continue to be elevated above the levels of concern (VADEQ 2012a).

Within the entire Bay watershed, the Anacostia and Potomac Rivers in Washington, DC are the areas of greatest concern for PCB fish tissue contamination. The DDOE (http://ddoe.dc.gov/service/fishing-district) currently advises the public not to consume any catfish, carp, or eels from waters of the District of Columbia due to PCBs and other chemicals. The most recent sampling in the Potomac and Anacostia Rivers within the District was conducted in 2007 (Pinkney 2009). Pinkney (2009) found that the highest total PCB concentrations were in American eel (*Anguilla rostrata*), where the median concentration was 2.18 ppm ww, over 100 times the USEPA (2000b) screening value of 0.020 ppm. One eel sample contained 4.00 ppm. Median concentrations in carp (*Cyprinus carpio*), channel catfish (*Ictalurus punctatus*), and blue catfish (*I. furcatus*) were all close to 0.80 ppm.

Pinkney (2009) compared PCB fish tissue concentrations in 2007 with those measured in 2000 (Pinkney et al. 2001a), using similar methods and fish with similar lengths. Median concentrations of PCBs in American eel, carp, and largemouth bass increased in both the Potomac and Anacostia Rivers whereas median PCB concentrations in channel catfish decreased in both rivers. Median PCB concentrations in sunfish decreased slightly. PCB concentrations were generally higher in the Anacostia vs. Potomac fish, but fish from both rivers were well above the screening limit thresholds.

Ecological concerns

Wenning et al. (2011) and Monosson (1999) reviewed literature on toxicological effects of PCBs on fish. Reported effects include mortality, impaired growth and reproduction, disruption of the endocrine and immune systems, biochemical changes, behavioral alteration, and mutagenicity. Iwanowicz et al. (2009a) documented adverse effects on the brown bullhead immune response, disease resistance and endocrine physiology following intraperitoneal exposure to 5 and 0.5 mg/kg of the PCB mixture Aroclor 1248. Similarly, a significant negative correlation has been documented between PCB body burden and the immune response and endocrine physiology in wild-caught brown bullheads and largemouth bass (Iwanowicz et al. 2012). Barron et al. (2000) documented an increased prevalence of hepatic tumors and preneoplastic liver lesions in walleye from the PCB-contaminated Green Bay area of Lake Michigan relative to a reference area. They stated that while these results did not show causation, they are consistent with studies that indicate that PCBs are liver tumor promoters in fish (Weisburger and Williams 1991). Wenning et al. (2011) concluded that data were inadequate to establish no-observable-effect concentrations (NOECs) based on PCB tissue residues.

Meador et al. (2002), in their review of salmonid toxicity data, suggested a total PCB tissue residue threshold of 2,400 ppb in lipid, which corresponds to 140 ppb ww tissue (D. MacDonald, MacDonald Environmental Services Ltd, personal communication). TAMS Consultants Inc. and Menzie-Cura Associates Inc. (2000) addressed the toxicological effects associated with PCB residues as part of the Hudson River Ecological Risk Assessment. Based on their literature review, they recommended a no-observable-adverse-effects level (NOAEL) of 1.9 ppm whole body weight and a lowest observable adverse effect level of 9.3 ppm body weight for total PCB concentrations. These NOAELs and LOAELs were applied to species that are resident to the Hudson River, most of which are also Chesapeake Bay species. Using a whole body to fillet ratio of 1.7 from Amrhein et al. (1999), these are converted to 1.1 ppm fillet (NOAEL) and 5.5 ppm fillet (LOAEL). Others have reported whole body: fillet ratios ranging from 1.7 to 3.1 (D. MacDonald, MacDonald Environmental Services Ltd, personal communication), which would lower the estimated fillet concentration thresholds.

Based on TAMS Consultants Inc. and Menzie-Cura Associates Inc. (2000) and Meador et al. (2002), it is reasonable to suggest that total PCB fillet concentrations above about 1.0 ppm may be associated with adverse biological effects in Bay watershed species. Such concentrations have been reported in bottom-dwelling fish in urban areas such as the District of Columbia (Pinkney 2009; Velinsky et al. 2011) and near USEPA National Priority List sites where PCBs are a contaminant of concern, such as the Marine Corps Base Quantico (Pinkney and McGowan 2006).

Since 1986, NOAA's Mussel Watch program has monitored PCBs in shellfish within the Chesapeake Bay and its tributaries at five locations in Maryland and five locations in Virginia, with data summarized in Kimbrough et al. (2008). In Maryland, 2004-2005 data for oysters (*Crassostrea virginica*) at the five locations were included in the report with concentrations of 21, 23, 60, 64, and 79 ppb. In Virginia, 2004-2005 total PCB concentrations at the five locations were 21, 21, 50, 57, and 157 ppb. Kimbrough et al. (2008) characterized concentrations as low, medium, or high as part of a nationwide comparison. The three highest Maryland concentrations, and the 50 and

57 ppb in Virginia, were characterized as medium concentrations. The 157 ppb concentration in Virginia was characterized as high.

Wildlife

No recent (post 2000) reports of PCB residues or effects in mammals were identified. In general, birds are more tolerant to acute exposure to PCBs compounds than mammals, but a range of effects (e.g., enzyme induction, altered growth and reproduction, chick edema disease, immune dysfunction and endocrine disruption) have been linked to exposure (Rice et al. 2003).

Threshold effect concentrations are based on residues in eggs or from blood samples and vary according to species. For total PCBs, estimated egg residue thresholds for impaired hatching or fledging success is 35,000 ppb ww in raptors and 23,000 to 142,000 ppb in terns, and thresholds for impaired 3- to 5- year productivity in raptors is 25,000 ppb (reviewed in Harris and Elliott 2011). The nestling eagle blood threshold for reproductive success is 189 ppb (Elliott and Harris 2002; Henny and Elliott 2007).

In the past 20 years, total PCBs concentrations have only been reported for a few bald eagle eggs. A single addled egg collected at Aberdeen Proving Ground in Harford County, Maryland in 2008 contained 33,690 ppb ww (Mojica and Watts 2008), and two addled eggs collected from the Naval Support Facility Indian Head in Charles County Maryland in 2008 and 2009 contained 18,400 and 18,300 ppb (Mojica and Watts 2011). Threshold effects for reduced productivity in bald eagle (*Haliaeetus leucocephalus*) have been estimated to be about 25,000 ppb ww of egg (Elliott and Harris 2002; Henny and Elliott 2007; Harris and Elliott 2011). Concentrations of total PCBs from blood samples of 58 nestling eagles from these same sites ranged from 7 to 106 ppb ww (Mojica and Watts, 2008, 2011), and are below the toxicity threshold for impaired reproduction of 189 ppb (Elliott and Harris 2002; Henny and Elliott 2007).

In a large-scale osprey study conducted in 2000 and 2001, total PCB concentrations in eggs collected from Baltimore Harbor and the Patapsco River, and the Anacostia and middle Potomac Rivers averaged 7,250 and 9,280 ppb ww, respectively (Rattner et al. 2004). The upper extreme value was 19,300 ppb from an egg collected near the Naval Research Laboratory on the middle Potomac, and was actually similar to the greatest historical values reported in osprey eggs from the Chesapeake (Wiemeyer et al. 1988). Osprey eggs from the Elizabeth River, the location of the largest naval port in the world, contained the lowest total PCB value, averaging 3,600 ppb. Total

PCB concentrations in eggs from the South, West and Rhode Rivers reference area averaged 4,600 ppb, and ranged up to 12,400 ppb. Concentrations of 15 arylhydrocarbon (*Ah*) receptor-active PCB congeners (but not dioxins or dibenzofurans) were also quantified in these eggs. Concentrations of the toxicologically most potent coplanar and semi-coplanar congeners (i.e., congeners 77, 81, 105, 126 and 169) did not differ much between study sites. Dioxin toxic equivalents (TEQs) of 15 *Ah* receptor-active congeners did not differ among sites in this Chesapeake Bay study, with average site values ranging from 0.0545 to 0.218 ppb TEQ ww. Concentrations of total PCBs and dioxin TEQs in these osprey eggs were slightly above the no-observed-effect-level (0.136 ppb TEQs ww) for hatching success (Woodford et al. 1998; Harris and Elliott 2011).

Total PCBs were quantified in 22 addled peregrine falcon eggs collected between 1993 and 2002 from locations in the Chesapeake Bay, and ranged from 3,460 to 12,500 ppb (Potter et al. 2009). It is difficult to assess the importance of these residues because PCB toxicity thresholds have not been rigorously developed for peregrine falcons.

Findings of high concentrations of PCB congeners and toxic equivalents, as well as cytochrome P450 induction in Baltimore Harbor black crown night herons (Rattner et al. 1997), was the impetus for testing the hypothesis that PCBs might be leading to the declining size of the Baltimore Harbor heron colony (Rattner et al. 2001). Although total PCBs, 12 *Ah* receptor-active PCB congeners and dioxin toxic equivalents were up to 35 times greater in sample eggs from Baltimore Harbor compared to those from the reference area in the southern Chesapeake (Holland Island), overall nest success (0.74) and productivity (2.05 young/hen) were adequate to maintain a stable population. Furthermore, no significant relation was found between hatching, fledging and overall reproductive success and concentrations of PCBs and toxic equivalents. It was concluded that contaminants were not having a dramatic effect on reproduction in the Baltimore Harbor heronry.

In a preliminary study examining potential endocrine disruptive effects of PCBs, common tern eggs collected in 1994 from South Sand Point, off of Barren Island, contained relatively low concentrations of Aroclor 1260 concentrations ranging from 440 to 1,500 ppb ww (J.B. French, USGS, unpublished data). In testing this hypothesis, eggs were subsequently collected from Bodkin Island (Chesapeake Bay) which served as a comparative reference site for the more contaminated samples from Ram Island in Buzzards Bay, Massachusetts. Total PCBs concentrations (<10,000 ppb lipid weight) were much lower at Bodkin Island compared to Ram Island, but no evidence was obtained to suggest that PCBs were evoking toxic effects in embryos (French et al. 2001). In 2010, common tern eggs were collected from Poplar Island for a flame retardant toxicity study, and six that were chemically analyzed were found to have very low total PCB concentrations (range: 310 to 440 ppb ww) and well below suspected adverse effect thresholds for reproductive success (Harris and Elliot 2011).

In portions of the Chesapeake, including USEPA-designated regions of concern (Baltimore Harbor, Elizabeth River, and Anacostia River) there is clearly evidence that total PCBs and *Ah*-receptor active PCB congeners induce cytochrome P450-associated monooxygenases, and may evoke adverse effects on some sensitive species of birds (Rattner et al. 1997; Rattner and McGowan 2007). In a few circumstances, PCBs concentrations in eggs are great enough to contribute to the failure of eggs to hatch (Mojica and Watts 2008, 2011).

Conclusions

Existing data documenting the concentrations of PCBs in fish tissue in the Chesapeake Bay watershed are adequate to characterize the extent of PCB contamination in the watershed as widespread in sediment and fish tissue. Unlike concentrations of chlorinated pesticides, concentrations of PCBs in tissues of many species of Chesapeake Bay wildlife have not declined since the final USEPA rule restricting the manufacture, processing, and distribution of these compounds became effective in 1979. The environmental persistence of PCBs is an impediment to quick remediation of the impairments as legacy deposits remain a primary source of PCBs. However, continued authorized uses and the inadvertent production of PCBs remains a potential source of environmental exposure. Due to the widespread presence of fish consumption advisories within the Chesapeake Bay watershed, it is clear that PCB contamination is negatively impacting the use of watershed resources.

2.2 Dioxins and Furans

Abstract

Dioxins and furans is the abbreviated name for a family of toxic substances that share a similar chemical structure, containing one to eight chlorine atoms attached to the carbon atoms of the parent chemical (dibenzodioxin and dibenzofuran). Dioxins and furans have no known technical use and are not intentionally produced. They are primarily released to the environment during combustion of fossil fuels (coal, oil, and natural gas) and wood, and during incineration processes (municipal and medical solid waste and hazardous waste incineration) and have been shown to be present as contaminants in certain chlorinated pesticides (e.g., 2,4 -D). The few data available were collected from areas associated with a known potential for dioxin contamination. There are three sites within the Bay watershed with documented dioxin contamination in sediment: the Atlantic Wood superfund site located in the Elizabeth River, and two areas within Maryland's North Branch of the Potomac River (maximum values of 2.674 ppb in the Elizabeth River and 0.2966 ppb in the North Branch of the Potomac). The latter two sites appear to be associated with historical dioxin contamination associated with a papermill in Luke, Maryland. Though data on dioxin contamination in the watershed are limited, available information indicates that the extent of dioxin contamination within the Chesapeake Bay watershed is localized. Impairments as a result of elevated levels of dioxin in crab and fish tissue have been identified in two areas of the watershed: the Elizabeth River and the C&D Canal. Whereas the Elizabeth River fish consumption advisory only limits the consumption of blue crab hepatopancreas, the C&D canal fish consumption advisory impacts the consumption of finfish. These localized fish consumption advisories impact the use of the Bay resources identified in the advisories. The severity of effects from dioxin is identified as localized.

Background

Dioxins and furans is the abbreviated name for a family of persistent, bioaccumulative and toxic substances that all share a similar chemical structure. These chemicals contain one to eight chlorine atoms attached to the carbon atoms of the parent chemical (dibenzodioxin and dibenzofuran). The chlorinated dibenzo-p-dioxins (CDDs) include 75 individual compounds and the chlorinated dibenzofurans (CDFs) include 135 compounds. These individual compounds are referred to as congeners. The most widely studied of these compounds, 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), is one of the most toxic to mammals and has received the most attention. Often referred to as "dioxin", 2,3,7,8-TCDD serves as a reference compound for this class of compounds. The chemicals with properties similar to 2,3,7,8-TCDD are called "dioxin-like" compounds. Only seven of the 75 congeners of CDD and 10 of the 135 congeners of CDFs are thought to exhibit "dioxin-like" toxicity (ATSDR 1994, 1998).

Dioxins and furans have no known technical use and are not intentionally produced. They arise through inadvertent production in processes involving chlorine in uncontrolled reactions. Air emissions of dioxins and furans result from the combustion and incineration of fossil fuels, municipal and medical solid waste, and hazardous waste (ATSDR 1994, 1998). Additional sources include the burning of many materials that may contain chlorine, such as plastics, wood treated with pentachlorophenol (PCP), pesticide-treated wastes and PCB-containing materials. They are also created in the pulp and paper industry from a process that bleaches the wood pulp (ATSDR 1998). Pesticides like 2,4-D have been shown to be contaminated with dioxins. Though manufacturing has been optimized to reduce the formation of impurities, the use of these pesticides still represents a potential source of dioxins (USEPA 2005). Other pesticides like triclosan, when in water, can also degrade to different forms of dioxin naturally in sunlight (Latch 2000). USEPA (2001a) breaks down the sources into five types: combustion and incineration; metals smelting; refining and processing; chemical manufacturing and processing; reservoir sources, and biological and photochemical processes. Within the last few decades, regulations on incineration processes have resulted in a reduction of dioxin air emissions (USEPA 2001a).

Dioxin exposure in waterways can be widespread, resulting from the dispersion of soil particles in erosion and run-off, volatilization from land and water, and resuspension of sediment particles. There is some uncertainty as to whether the atmospheric deposition represents contemporary inputs of dioxins or whether this deposition component is resulting from transfers among the environmental media (USEPA 2001a).

Dioxins and furans are found in the environment together with other structurally related chlorinated chemicals. Therefore, the Toxicity Equivalent Factor (TEF) methodology was developed to estimate the risk from exposure to these mixtures. Because very limited data on the toxicity of these chemicals exist, TEFs were developed and validated in studies in animals. TEFs are the result of expert scientific judgment using all of the available data and taking into account a variety of uncertainties. The TEF approach compares the relative toxicity of individual congeners to that of 2,3,7,8-TCDD, which is the most extensively studied. 2,3,7,8-TCDD was assigned a TEF of 1.0 and the TEF for the other congeners range from 1.0 to 0.00001. The toxic potency of a mixture of congeners (i.e., the Toxicity Equivalents, or TEQ) is the sum of the products of the TEFs for each congener and its concentration in the mixture (ATSDR 1994, 1998).

Dioxins and furans can cause a number of health effects with complex mixtures identified as "likely human carcinogens" (USEPA 2001a). At ten times greater than background exposure, humans may experience changes in markers of early development and hormone levels. High dose exposures have caused chloracne skin disease. Animal studies show that animals exposed to dioxins and furans experienced changes in their hormone systems, changes in the development of the fetus, decreased ability to reproduce, and suppression of the immune system (USEPA 2012c).

Water and Sediment

No water column data were found in the sources of information reviewed; this is not unexpected because dioxins and furans are hydrophobic and tend to sorb to soil and sediments. Additionally, dioxins may not be commonly assessed in jurisdiction waters. Water quality standards for human health protection exist in each of the jurisdictions and range from $5x10^{-9}$ to $5.1x10^{-7}$ µg/L.

Dioxin/furan contamination was noted at three locations. One is in the Southern Branch of the Elizabeth River in Portsmouth, VA (approximately 12 miles from the Chesapeake Bay), the location of the Atlantic Wood Industries (AWI) Superfund site. One of the compounds used at the AWI facility as a wood preservative, pentachlorophenol, has been reported to contain dioxin and furan impurities. Sediment sampling conducted as part of the Superfund investigation showed that the dioxin in samples included predominantly octachloro dibenzo-p-dioxin (OCDD), with minor amounts of heptachloro dibenzo-p-dioxin (HpCDD). Sampling conducted in shallow sediment in the vicinity of the AWI site had a maximum TEQ of 2.674 ppb. Sediment sampling also showed detections of dibenzofurans, but at lower levels (USEPA 2007a). Remediation of the site includes a plan to dredge contaminated sediments.

Another area with reported dioxin contamination is in the North Branch of the Potomac River. In the late 1980s and early 1990s, dioxin contamination in the North Branch of the Potomac River resulted from the discharge from the Westvaco paper mill in Luke, Maryland. Fish consumption advisories were issued at that time by both Maryland and West Virginia for a portion of the Potomac between Luke Maryland and Paw Paw, West Virginia (Wheeler 1992). Westvaco implemented control measures to reduce dioxin levels in the discharge and fish advisories were discontinued in the early 1990s. Given the hydrophobic nature of dioxins, the sediment may still be contaminated. However, no information was available on any sediment concentrations close to this discharge (Wheeler 1992).

The third area where dioxin contamination has been reported is also in the North Branch Potomac River. In Cumberland, Maryland, sediment sampling was conducted in 2009 as part of the Cumberland Dam Removal Feasibility Study (Princeton Hydro 2010). Sediment samples collected from within the dam and upstream detected dioxin-like compounds with the 2,3,7,8-TCDD concentration in each sample ranging from 0.57 to 0.93 parts per trillion (ppt). The sum of dioxin-like compounds detected in sediment samples ranged from 99.5 to 296.6 ppt. There was no information regarding the source of contamination, however, the report notes that the dam is approximately 28 miles downstream from the Westvaco paper mill in Luke, Maryland. The report recommended collection of additional information; however, no further studies have been conducted since that study was completed (Princeton Hydro 2010).

To put these sediment values in context (maximum values of 2.674 ppb in the Elizabeth River and 0.2966 ppb in the Potomac), the USEPA (1993) developed guidelines for evaluating potential risk to fish species. Low level risk is estimated at 0.060 ppb. Low level risk was based on a no effects threshold for reproductive endpoints. At 0.100 ppb, there is a high risk to sensitive species. These high risk concentrations are "derived from TCDD doses expected to cause 50 to 100% mortality in embryos and young of sensitive species".

Fish and Shellfish

In their water quality assessment reports, the states use fish advisories as the basis for listing streams as impaired. The fish advisories are coordinated with the respective states' health departments. Only two states, Virginia and Delaware, listed streams in the Chesapeake Bay watershed, as impaired by dioxins. The listings are reported as *"Dioxin"*, or *"Dioxin, including 2,3,7,8- TCDD".* Specifically, Delaware listed the Chesapeake and Delaware Canal (C & D Canal), from the Maryland line to Delaware River (15 miles). This listing was based on an advisory for no consumption of all finfish due to dioxin, among other contaminants. The principal toxicological driver for this listing is to limit human health exposure to fish tissue contaminated with PCBs (R. Greene, DNREC, personal communication). No source for the dioxin is specified for this segment. VADEQ listed the Elizabeth River Southern Branch and its tidal tributaries (an area of 23 river miles) on the basis of a fish advisory issued for no consumption of blue crab hepatopancreas. The edible tissue, however, is identified as safe for consumption. As stated above, dioxin contamination was reported in the Southern Branch of the Elizabeth River as a result of contamination from the AWI Superfund Site. As part of the Superfund investigation, shellfish sampling was conducted as follows (results in parenthesis are maximum detections):

- crab meat and whole crab (crab meat and hepatopancreas) collected adjacent to the AWI site (total dioxin TEQ 0.00026 ppb and 0.012 ppb, respectively);
- oyster meat from oysters collected adjacent to the AWI site (0.0014 ppb);
- crab meat and whole crab (crab meat and hepatopancreas) from near Scuffletown Creek (0.000023 ppb and 0.0042 ppb, respectively) (USEPA 2007a).

As indicated above, two streams in the Bay were listed as impaired by dioxins, one in the Southern Branch of the Elizabeth River and one in the C & D Canal based on fish advisories. The fish advisories were issued due to the exceedance of each of the states' benchmarks, the purpose of which is to minimize human health exposure through the consumption of fish tissue. The states' schedule for issuing TMDL plans for these impairments is from 5 to 10 years. In the near-term, however, Superfund remediation is expected to address contamination in sediments near the AWI Superfund site in the Southern Branch of the Elizabeth River (USEPA 2007a). USEPA (1993) identified 50 ppt fish tissue concentration as a low risk threshold for effects in fish. For pisciverous mammals and avians, the low risk thresholds for fish tissue concentrations are 0.7 ppt and 6 ppt, respectively. High risk levels for effects in sensitive species were identified as 80 ppt for fish, 7 ppt for pisciverous mammals, and 60 ppt for pisciverous fish.

Wildlife

Apparently, only one study has examined the concentrations of dioxins and dibenzofurans in Chesapeake Bay wildlife. Addled and post-term peregrine falcon eggs were sampled for dioxin (2,3,7,8-TCDD: ranging from not detected to 97 ppt ww, not adjusted for moisture loss) and dibenzofurans (2,3,7,8-TCDF: ranging from not detected to 146 ppt ww, not adjusted for moisture loss) (USFWS et al. 2004). These data, in combination with coplanar PCB congener levels, were subsequently concentration-corrected for moisture loss to estimate their fresh weight values (Clark et al. 2009). These concentration values were used to estimate dioxin toxic equivalents. Geometric means of the peregrine egg samples from middle Chesapeake Bay (n=9) and upper Chesapeake Bay (n=2) were at or below the NOAEL of 0.23 ppb TEQ ww derived for the American kestrel (*Falco sparverius*) (USEPA 2003a).

Conclusions

Existing data documenting the environmental occurrence of dioxins and furans in the Chesapeake Bay watershed are limited though sediment and fish tissue contamination has been identified near areas associated with historical contamination. Specifically, sediment contamination has been identified at the Atlantic Wood Superfund site located in the Elizabeth River and at two sites in the North Branch of the Potomac River downstream of a Luke, Maryland papermill. Dioxin (as measured by 2,3,7,8 TCDD) has been found to be elevated in fish tissue resulting in impairment listings in the Elizabeth River and the C&D Canal. An advisory restricting the consumption of blue crab hepatopancreas was issued for the segment of the Elizabeth River associated with the Superfund site (no consumption allowed). The C&D Canal also has a limit on the quantity of finfish that can be consumed, though the principal driver for this advisory is to limit human exposure to PCB contamination. These listings have resulted in localized extent and severity of contamination.

2.3 Polycyclic Aromatic Hydrocarbons

Abstract

Polycyclic aromatic hydrocarbons (PAHs) are a class of hundreds of chemicals composed solely of hydrogen and carbon in structures containing two or more benzene rings. In environmental surveys, scientists often refer to 16 USEPA priority PAHs (acenaphthene, acenaphthylene, anthracene, benz(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(ghi)perylene, benzo(k)fluoranthene, chrysene, dibenz(a,h)anthracene, fluoranthene, fluorene, indeno(1,2,3-c,d)pyrene, naphthalene, phenanthrene, and pyrene). Other researchers examine as many as 34 compounds including these 16 and alkylated PAHs. PAHs are either petrogenic (derived from petroleum and coal) or pyrogenic (derived from the combustion of fossil fuels or wood products). PAHs are detected more often in sediments than in other media. Whereas PAHs can bioaccumulate in invertebrates such as mussels and clams, they are rapidly metabolized in fish and, therefore, are less frequently measured in fish-tissue monitoring programs than PCBs and metals. PAHs are widely detected in sediments in the Chesapeake Bay watershed at varying concentrations, with the highest reported in or near Baltimore Harbor, Anacostia River, and Elizabeth River. In the Elizabeth River, sediment remediation projects, with the aim of reducing contaminant concentrations (including PAHs) and associated biological effects, have been completed or are in progress. Liver tumors in bottom-dwelling fish from the Anacostia and Elizabeth Rivers have been statistically associated with exposure to PAH-contaminated sediments and are used to monitor conditions. Three jurisdictions (Maryland, Virginia, and DC) list impairments. The severity of effects due to PAHs is identified as localized.

Background

Polycyclic aromatic hydrocarbons (PAHs) are a class of hundreds of chemicals, composed solely of hydrogen and carbon in structures containing two or more benzene rings. In environmental surveys, scientists often refer to 16 USEPA priority PAHs (acenaphthene, acenaphthylene, anthracene, benz(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(ghi)perylene, benzo(k)fluoranthene, chrysene, dibenz(a,h)anthracene, fluoranthene, fluorene, indeno(1,2,3-c,d)pyrene, naphthalene, phenanthrene, and pyrene) (USEPA method 610) Other researchers examine as many as 46 separate PAH compounds and groupings including these 16 compounds and additional alkylated PAHs (Hartwell and Hameedi 2007). PAHs in water will readily adsorb to sediments (USEPA 2008b; Van Metre et al. 2006). They may persist in the environment, and some have half-lives of up to 5 years (Greenfield et al. 2004; Oros et al. 2007). There are two types of PAHs: petrogenic PAHs are found naturally in petroleum and coal, whereas pyrogenic PAHs are formed during the burning of gasoline, coal tar, aluminum, fuel oil, and other fossil or modern biomasses. Petrogenic PAHs tend to have lower molecular weights (typically < 4 carbon rings), whereas pyrogenic PAHs tend to have higher molecular weights (≥4 carbon rings). PAHs with higher molecular weights tend to have lower solubility (ATSDR 1995, NIH 2005). With their decreased solubility, the higher molecular weight compounds tend to be found in sediment, whereas the lighter compounds may be found dissolved in water. Studies indicate that the load of PAHs in Bay tributaries may have a larger proportion of pyrogenic (e.g., combustion byproducts) compounds than the load in the mainstem (Hartwell and Hameedi 2007).

Human health effects of PAHs were summarized in the Agency for Toxic Substances and Disease Registry (ATSDR 1995) toxicological profile. Many PAHs are classified as probable human carcinogens by the USEPA and the International Agency for Research on Cancer.

Water

In Maryland, two segments of the Patuxent River are listed as impaired by PAHs resulting from an oil spill in 2000. The April 7, 2000, oil spill resulted from a break in a Pepco pipeline; two segments, Craney Creek and Buena Vista, have yet to meet the Phase I or Phase II cleanup status. PAH contamination as a result of this oil spill resulted in impairment of 0.33 estuarine acres (MDE 2010).

In the District of Columbia, there are impairment listings for PAHs in more than 12 stream segments as part of the District's organics TMDL (DDOE 2010). A study titled "Sediment contamination studies of the Potomac and Anacostia Rivers around the District of Columbia" was completed by the Interstate Commission on the Potomac River Basin in 1992. The study included the tidal basin. Results of this study showed higher levels of PAHs at sampled outfalls and storm sewers to the tidal basin than in basin sediments. Results did not indicate a specific outfall as the source. The study indicated that the primary source of these hydrocarbons likely was much more diffused and probably related to vehicular traffic.

There are no reported impairments resulting from PAH contamination in water in the other states within the Bay watershed (DNREC 2010, WVDEP 2010, PADEP 2010, NYSDEC 2010). In a USGS study of organic wastewater compounds in Pennsylvania stream water samples collected from 2007 to 2009, PAHs were occasionally detected in concentrations ranging from 4 to 42 µg/L (Reif et al. 2012). In Virginia, impairments are based on fish tissue (VADEQ and Virginia Department Conservation and Recreation (VADCR) 2010).

Sediment

Sediment triad studies typically use synoptically collected samples that are analyzed for sediment chemistry and benthic macroinvertebrate community structure, and tested for toxicity as an approach for evaluating habitat quality (Chapman 1990). The CBP funded two sediment triad studies, Pinkney et al. (2005) and Fulton et al. (2007), to focus on Bay tributaries identified by USEPA (1999) as having insufficient data to characterize the extent and magnitude of sediment contamination. Additional triad studies to characterize areas with data gaps were funded by VADEQ and conducted by Roberts et al. (2002, 2003, and 2004). Only the sediment PAH data from those studies are reported here.

Pinkney et al. (2005) collected samples from tidal sections of the Bohemia, Elk, Northeast, and Severn Rivers in Maryland. Total PAH concentrations ranged from 0.717 to 16.9 ppm, substantially less than the ERM of 44.792 ppm.

Fulton et al. (2007) collected samples from a total of 60 stations in five areas of the Bay for which data previously had been insufficient: the Chester River, Nanticoke River, Pocomoke River, Lower Mobjack Bay (Poquosin and Back Rivers), and South and Rhode Rivers. Among these locations, the total PAH concentrations were highest at stations in the upper section of the South River. The average total PAH concentration in the South River ranged from 2.723 ppm in the lower section to 4.704 ppm (just above the ERL of 4.022 ppm) in the upper section. Average total PAH concentrations in the other tributaries did not exceed the ERL values: Chester River, 0.784 ppm; Nanticoke River, 1.036 ppm; Pocomoke River, 0.890 ppm; and Rhode River, 0.777 ppm. The average total PAH concentrations in Lower Mobjack Bay ranged from 0.167 ppm in the Back River to 0.309 ppm in the Poquosin River.

Roberts et al. (2002) studied the tidal freshwater areas in the James and Appomattox Rivers in 2001. In the nine sediment samples analyzed, total PAH concentrations ranged from 0.955 to

5.785 ppm, with the highest six samples exceeding the TEC of 1.610 ppm and none exceeding the PEC of 22.800 ppm. In 2002, Roberts et al. (2003) collected 12 samples from Lower Mobjack Bay portion of the Chesapeake which included the area south of the York River and included the Poquoson and Back Rivers. PAHs were only detected in five samples; the maximum total PAH concentration was 0.184 ppm, far below the ERL of 4.022 ppm. In 2003, Roberts et al. (2004) collected a total of 14 sediment samples from the Mattaponi and Pamunkey Rivers. PAHs were detected in only sample, with a total PAH concentration of 0.040 ppm, far below the TEC.

Hartwell and Hameedi (2007) reported the results of the NOAA sediment triad study of the Chesapeake Bay conducted at 210 sites in 1998, 1999, and 2001. They found that total PAH concentrations varied from just over 4 ppb to 22.6 ppm. They stated that concentrations at only one mainstem and six tributary stations exceeded the ERL, and no concentrations exceeded the ERM. They stated that the highest concentrations in tributaries were in Baltimore Harbor, the James and Elizabeth Rivers, and the mouth of the Patuxent River.

The objective of the Hartwell and Hameedi (2007) study was to conduct a broad-scale survey of the Bay; therefore, it did not focus on highly contaminated areas. Thus, in the three Regions of Concern (Baltimore Harbor, Anacostia River, and the Elizabeth River), only eleven samples were collected: two from Baltimore Harbor and nine from the Elizabeth River. Velinsky and Ashley (2001) analyzed surface sediment collected at 114 locations in the Anacostia River, including the Washington Channel, for contaminants in 2000. Total PAH concentrations averaged 22.6 ppm, just below the 22.8-ppm freshwater PEC (MacDonald et al. 2000a). The maximum concentration was 90.4 ppm. Samples from 52 of the 114 locations had concentrations above the PEC. Therefore, based on a comparison of concentrations of total PAHs with the PEC, concentrations of total PAHs appear to be frequently detected at concentrations associated with adverse effects on the benthic community. However, in a sediment triad study of 20 locations with total PAHs ranging from 1.005 to 57.907 ppm, McGee et al. (2009) reported only sublethal toxicity at the station with the highest total PAH concentration and no evidence of sediment toxicity at the other 19 stations.

The most extensive survey of Baltimore Harbor sediment chemistry and toxicity was conducted in 1996 (Ashley and Baker 1999). Total PAH concentrations ranging from 90 ppb to 46.2 ppm (which exceeds the ERM) were reported for 80 sampling sites, with several of the highest in Bear Creek near the Sparrows Point industrial facility. More recently, EA Engineering, Science and Technology, Inc. (EA 2009, 2011) collected sediment, water, and tissue PAH concentration data in the Coke

Point Offshore Area near Sparrows Point. The mean total PAH concentrations in the 18 sediment samples collected in the Coke Point Offshore Area was 569 ppm (range: 5.97 to 7,354 ppm) which greatly exceeds the ERM of 44.792 ppm and the average total PAH concentration in Baltimore Harbor channels of 3.24 ppm (EA 2009). In a risk assessment, EA (2011) concluded that there were ecological risks to aquatic organisms from exposure to PAHs in the Coke Point Offshore Area based on exceedance of the probable effect level (PEL) screening benchmarks of 1.442 ppm for low molecular weight PAHs and 6.676 ppm for high molecular weight PAHs (Buchmann 2008; MacDonald et al. 1996). A PEL is defined as an estimate of the concentration above which adverse effects on the benthic community frequently occur.

The Elizabeth River Project (2008; a grass-roots citizens' organization dedicated to the cleanup of the Elizabeth River) reported that concentrations of PAHs in some areas of the Elizabeth River were as much as 1,000 times the average concentration in the Chesapeake Bay. Concentrations of individual and total PAHs at 16 locations in the Elizabeth River sampled in 2007 ranged from 0.736 to 383.2 ppm (Vogelbein et al. 2008). The highest concentration was found in the South Branch near the Atlantic Wood Superfund site. This site, operated from 1926 to 1992 as a wood-treating facility, released creosote, PCP, metals, and dioxins into the Elizabeth River. Hartwell and Hameedi (2007) reported lower total PAH concentrations at the nine sites sampled in the Elizabeth River in 2001, ranging from 1.678 to 24.617 ppm but their survey did not attempt to sample highly contaminated locations.

Fish and Shellfish

Fish tissue advisories and impairments

In Virginia, fish tissue impairments are listed for 1 square mile of estuary (Pohick Creek, due to benzo[k]fluoranthene), 74 acres of lakes, and 7 river miles (Gold Mine Creek in the James River due to benzo(a)pyrene, benzo[b]fluoranthene, and/or benzo[k]fluoranthene) (VADEQ and VADCR 2010). VADEQ lists PAH tissue data for fish and invertebrates within the Chesapeake Bay watershed for 2000, 2001, 2003, 2005, and 2008 on its Web site (http://www.deq.state.va.us/Programs/Water/WaterQualityInformationTMDLs/WaterQualityMonitoring/FishTissueMonitoring/FishTissueResults.aspx). VADEQ uses a screening value of 15 ppb (as a PEC (potency equivalent concentration)) for total PAHs based on the sum of the following seven carcinogenic compounds, weighted according to the following relative potency factors: benzo(a)pyrene (potency =1.0), benzo(a)anthracene (0.145), benzo(b)fluoranthene (0.167), benzo(k)fluoranthene (0.020), chrysene (0.0044), dibenzo(a,h)anthracene (1.11), and indeno(1,2,3-c,d)pyrene (0.055).

In all the sampling years, no concentration exceeded the PEC. The highest PEC concentration in fish sampled in these years was 5.521 ppb in a 2005 composite sample of mummichogs (*Fundulus heteroclitus*) collected from St. Julian Creek in the Elizabeth River watershed.

Fish tissue samples were analyzed for PAH concentrations as part of monitoring conducted by Washington, DC to update fish tissue advisories (Pinkney et al. 2001, Pinkney 2009). In 2007, samples of the following species were collected from the Potomac and Anacostia Rivers: sunfish, largemouth bass, carp, blue catfish, channel catfish, and American eel (Pinkney 2009). PAH concentrations were compared with the USEPA (2000) screening value of 0.00547 ppm, which is based on the cancer slope factor for benzo(a)pyrene, the most potent PAH carcinogen. Concentrations were also evaluated with respect to Toxic Equivalent Factors (TEFs) by using the values of Nisbet and LaGoy (1992), which relate the toxicities of various PAHs to that of benzo(a)pyrene. For the TEF approach, the screening values developed by the DNREC and the Delaware Department of Health and Social Services (DHSS) were used (DNREC-DHSS 2005).

In general, the two- and three-ring PAHs such as naphthalene and anthracene were detected at higher concentrations than the five- and six-ring compounds such as benzo(a)pyrene (Pinkney 2009). Concentrations of PAHs in all samples except one sunfish sample exceeded the screening value of 0.00547 ppm. The highest total PAH concentrations by far were detected in carp and ranged from 0.0706 ppm in the lower Potomac River sample to 0.384 ppm in the upper Potomac River sample. The median concentration (0.184 ppm) was about 34 times the screening level. Median total PAH concentrations in the other species ranged from 0.0079 ppm in the single smallmouth bass sample (from the lower Potomac River) to 0.0814 ppm in American eel. By using the TEF approach, however, no concentrations in any of the samples for any species exceeded the DNREC-DHSS (2005) screening value of 7 ppb. Median TEF concentrations ranged from 0.002 ppb in smallmouth bass to 0.129 ppb in carp. The highest TEF, 0.217 ppb, was measured in a carp sample from the upper Potomac River.

The District of Columbia's advisory is not linked to a specific chemical but states that the advisory is due to presence of "PCBs and other chemicals" (http://green.dc.gov/service/fishing-district). In view of the uncertainty regarding the risk from PAHs in fish tissue, it is reasonable to attribute most of the risk from fish consumption in District of Columbia waters to PCBs and chlorinated pesticides rather than PAHs (based on data in Pinkney 2009).

Ecological concerns

Logan (2007) reviewed the ecotoxicology of PAHs on fish. Although PAHs can be detected in fish muscle tissue, these compounds tend to be metabolized by vertebrates and eliminated through the liver. Elimination results in detoxification but also the generation of genotoxic metabolites (French et al. 1996). Therefore the most common biomarkers used to monitor PAH exposure in fish are bile PAH-like metabolites (Leadley et al. 1999) and DNA adducts (Reichert et al. 1998).

Chronic exposure of fish embryos to PAHs may result in death, deformities, or decreased growth. Narcosis is believed to be an important mechanism of toxicity and is attributable to low molecular weight volatile PAHs (French-McCay 2002). There is also concern about sediment toxicity to bottom-dwelling organisms, which can be associated with phototoxicity (Barron 2007). A diminished benthic biomass could affect the survival, growth, and reproduction of their fish predators.

There are documented cases of liver tumors in bottom-dwelling fish that have been causally linked with exposure to PAH-contaminated sediments (Myers et al. 2003; Baumann and Harshbarger 1998). In the Chesapeake Bay watershed, the clearest linkages between liver tumors in fish and PAH exposure are in the Anacostia (Pinkney et al. 2009) and Elizabeth Rivers (Vogelbein and Unger 2006). There are also genetic changes associated with long-term exposure to PAHs. In a laboratory study with Elizabeth River sediments, Ownby et al. (2002) documented an increased tolerance to the acute toxic effects of creosote. The authors found that tolerance was hereditable, indicating that it resulted from natural selection.

Pinkney et al. (2001b, 2004) used biomarkers of exposure and response, tumor data, and sediment data to evaluate the association between PAHs and liver tumors in brown bullheads. In both studies, the concentrations of biliary PAH-like metabolites were examined as an indicator of PAH exposure. Pinkney et al. (2004) also measured concentrations of polycyclic aromatic compound (PAC)-DNA adducts, which are bulky molecules attached to the DNA that serve as an indicator of response to PAHs. A specific pattern (diagonal radioactive zone (DRZ)) in the radiographic determination of these adducts is indicative of PAC adducts. Using logistic regression, the authors reported a statistical association between liver tumors and biliary PAH-like metabolites. They also reported equally high concentrations of PAC-DNA adducts in 1- to 2-year-old fish, which already had a 10 to 17% liver tumor prevalence, as in the 3-year-old and older fish, which had a 50 to 68% tumor prevalence. Therefore, the younger age classes were likely to have a high prevalence as they reach age 3 or greater. The finding of high concentrations of PAC-DNA adducts with a strong DRZ signal, elevated bile PAH-like metabolites, and elevated sediment PAHs (15–31 ppm total PAHs within 1 kilometer of the fish collection sites) provide strong evidence linking the liver tumors with exposure to PAH-contaminated sediments.

Monitoring has been conducted over the past 25 years to evaluate the status of sediment contamination in the Elizabeth River using mummichog liver pathology. Vogelbein et al. (1990) reported total PAH concentrations as high as 2,200 ppm in sediments from a creosote-contaminated site. They reported that 35% of mummichogs collected near this site exhibited hepatocellular neoplasms and 73.3% of them had foci of cellular alteration, which are hypothesized to be pre-neoplastic lesions. They also noted a variety of other pathologies, including elevated prevalence of exocrine pancreatic (Vogelbein and Fournie 1994; Fournie and Vogelbein 1994) and vascular neoplasms (W. Vogelbein, VIMS, personal communication). In contrast, mummichogs from two relatively uncontaminated study sites exhibited no proliferative liver lesions or other pathologies.

Vogelbein et al. (1990) demonstrated a strong positive association between sediment PAH concentrations derived from creosote spills and development of proliferative liver lesions. As a result, the VADEQ and the Elizabeth River Project adopted the mummichog as a sentinel of chemical contamination. Since the late 1990s, this fish has been used in a long-term field monitoring program in the Elizabeth River (Elizabeth River Project 2008). Results of these studies indicate that liver histopathology in mummichogs is an effective bioindicator of sediment chemical contamination (Vogelbein et al. 1997, 1999, 2008; Vogelbein and Zwerner 1999; Vogelbein and Unger 2003). The greatest liver disease prevalence was found in mummichogs from the most heavily contaminated sites and the lowest disease prevalence occurred in fish from the least contaminated sites.

Currently, mummichog liver histopathology and sediment PAH measurements are being used in association with ongoing sediment remediation efforts in the Elizabeth River, such as the Money Point Project started in 2009. Remediation efforts included nearshore sediment dredging and sand capping, and construction of a new onshore salt marsh. The constructed inshore salt marsh appears to have created a refuge for aquatic life from the sediment chemical contaminants found just offshore. Remediation reduced the bioavailability of sediment PAHs, which has already caused a decrease in liver lesion prevalence at the site (Vogelbein and Unger 2011).

Since 1986, NOAA's Mussel Watch program has monitored PAHs in shellfish within the Chesapeake Bay and its tributaries at five locations in Maryland and five locations in Virginia with data summarized in Kimbrough et al. (2008). In Maryland, 2004-2005 data for oysters at three of the five locations were included in the report with concentrations of 266, 306, and 481 ppb. In Virginia, 2004-2005 total PAH concentrations at the five locations were 200, 207, 232, 307, and 1583 ppb. Kimbrough et al. (2008) characterized concentrations as low, medium, or high as part of a nationwide comparison. The 1583 ppb concentration was characterized as a medium concentration.

Wildlife

From 1986 to 1988, 35 muskrats (*Ondatra zibethicus*) were trapped on the Elizabeth and Nanesmond Rivers in Virginia. Twenty-two of 35 carcasses had detectable concentrations of PAHs (naphthalene, phenanthrene, anthracene, fluorene, pyrene, and chrysene), and the muskrat with the greatest concentration (phenanthrene) contained 0.15 ppm dry weight (Halbrook et al. 1993). To the best of the authors' knowledge, PAH concentrations have not been reported in other wildlife species in the Chesapeake Bay.

Conclusions

PAHs are widely detected in sediments in the Chesapeake Bay watershed at varying concentrations, with the highest reported in or near Baltimore Harbor, Anacostia River, and Elizabeth River. Concentrations of PAHs have also been detected in the water column and in fish tissues, although, in general, fish tend to metabolize rather than accumulate these compounds. There are localized impairments in Virginia (fish tissue), Maryland (water), and DC (water). Concentrations of PAHs in sediments of the Elizabeth River and the Anacostia River have been linked with a high prevalence of liver tumors in bottom dwelling fish. Fish lesion prevalence in the Elizabeth River has been used as a tool to monitor the success of sediment remediation projects.

2.4 Petroleum Hydrocarbons

Abstract

Petroleum hydrocarbons are a mixture of several hundred chemicals originating from crude oil. The chemical components of this group of compounds vary considerably in chemical properties, affecting their bioavailability, fate and effects. The compounds vary with regard to solubility in water, with the lower molecular weight aromatic hydrocarbons (benzene, toluene, ethylbenzene, xylene (BTEX)) exhibiting a relatively high degree of solubility relative to the higher molecular weight compounds. For petroleum compounds, states typically evaluate compliance with a narrative standard (i.e., no visible sheen) or with a numeric standard, such as that for oil and grease compounds (which include petroleum and non-petroleum-based oils). Data on reported spills and/or observable sheens are readily available, though concentration-based data are limited. The data that are available indicate that petroleum contamination within the Chesapeake Bay watershed is predominantly localized to areas with heavy boating or shipping activity. Two of the partner jurisdictions have documented water column impairments for the surrogate parameter, oil and grease. In Washingrton D.C., there are approximately 1.7 miles of impairment in the Anacostia River and Hickey Run. Pennsylvania documents a total of five miles of river impairments; three miles of the Conococheague River are impaired. These impairments indicate localized severity of effects.

Background

Petroleum hydrocarbons are a mixture of several hundred chemicals originating from crude oil. The chemical components of this group of compounds vary considerably in chemical properties (i.e., some are characterized by relatively high vapor pressures, whereas others exhibit low vapor pressures). Compounds with high vapor pressures, such as those commonly found in gasoline, volatilize readily, whereas those with low vapor pressures tend to remain in liquid form. The individual compounds vary with regard to solubility in water, with the lower molecular weight aromatic hydrocarbons (BTEX) exhibiting a relatively high degree of solubility relative to the higher molecular weight compounds. The individual petroleum compounds have widely ranging organic carbon-water partitioning coefficients, indicating that some compounds partition preferably to solids and sediment that may be present in the water column, whereas others remain in water. Those compounds remaining in the aqueous fraction are highly mobile and can be transported considerable distances from the point of entry into the environment. Of the BTEX compounds, benzene is classified as a mobile compound whereas toluene, ethylbenzene, and xylene are classified as intermediately mobile. Upon exposure of these compounds to the environment, microbial populations capable of utilizing petroleum hydrocarbons as a carbon source begin to biodegrade them, provided that nutrients are also readily available (Horel et al. 2012). Horel et al. (2012) determined that addition of inorganic nutrients to the sandy beaches in the northern Gulf of Mexico both stimulated and enhanced the biodegradation of these compounds following the Deepwater Horizon accident in 2010.

Petroleum hydrocarbon contamination can arise from a number of sources, given the common use of petroleum-derived products (e.g., gasoline, kerosene, asphalt). Combustion and accidental releases of these products into the environment are common and are associated with the use of any tool, vehicle, or equipment that requires their use (DDOE 2003). An analytical method exists to estimate quantities of total petroleum hydrocarbons when assessing environmental prevalence; however, the more commonly used methods to assess risk to aquatic life and human health involve the measurement of surrogates such as the BTEX compounds or oil and grease.

Chesapeake Bay jurisdictions assessing their waters for aquatic life protection commonly refer to either a narrative standard ("no visible sheen") or a numeric standard for oil and grease promulgated by the USEPA in 1986. A water standard for total petroleum hydrocarbons has not been developed; however, this measurement of oil and grease includes plant-based oils as well as those derived from petroleum products. The measurement of oil and grease, like that of total petroleum hydrocarbons, captures a wide array of organic compounds, each with distinct physical, chemical, and toxicological properties (USEPA 1986).

The information on impairments was derived primarily from the jurisdiction water quality reports (DDOE 2010, PADEP 2010). Quantitative data were extracted from a completed TMDL report (DDOE 2003).

Water

Only two jurisdictions cite oil and grease impairments within the Chesapeake Bay watershed: DC and Pennsylvania (DDOE 2010; PADEP 2010). The Distict of Columbia identifies approximately 1.7 miles as impaired (Anacostia River and Hickey Run); all but 0.6 miles have a completed TMDL. In Pennsylvania, approximately five river miles in the watershed are identified as impaired for this contaminant, with three miles in the Conococheague River. These impairments will not impact the Bay and will be remediated as per state regulatory requirements.

Water-column data for oil and grease concentrations were generated in support of the Anacostia/Hickey Run TMDL study. These concentrations ranged from less than detection (less than 5 mg/L) to 116 mg/L. Of the 37 samples collected during this effort, concentrations in five were at or above the water-quality standard of 10 mg/L. However, although the most recently approved integrated assessment report from the DDOE (2010) indicated the presence of a visible sheen in Hickey Run, it noted that the suspected sources are currently being addressed.

Petroleum is released to the environment through spills both small and large, including chronic releases of crankcase oil from vehicles and discharges from boat engines. The National Response Center (NRC) staffed by U.S. Coast Guard personnel is to be contacted upon discharge of a "harmful quantity" of oil, defined as "any quantity that violates state water quality standards, causes a film or sheen on the water's surface, or leaves sludge or emulsion beneath the surface" (USEPA 2006). Minor spills occur frequently within the Bay watershed and are reported to the NRC. For the 5-year period from 2007 to 2011, USFWS personnel received 2,734 spill-response notifications from the NRC for the Chesapeake Bay region. Most spills were very small and none involved wildlife. Though the majority of these spills could not be attributed to a known source, many were associated with boating or shipping activity. Virginia had the most reports. The largest number of oil and hazardous chemical spill incidents were reported in the Elizabeth River area (S. Lingenfelser, USFWS, personal communication).

Because of the wide variety of compounds present in the general category of "petroleums," completing a risk assessment for the group as a whole is challenging. USEPA (1976) promulgated a water-quality criterion for oil and grease to protect aquatic life from the harmful effects of petroleum and nonpetroleum oils. The potential effects that were identified include sublethal effects on cellular activity as well as physiological processes affecting feeding and reproduction. These sublethal effects resulting from chronic exposure could occur when petroleum concentrations reach levels of 10 µg/L. In many cases, the most toxic and long-lived components of petroleum in the water column are PAHs. The toxicology of PAHs is complex and is discussed in more detail in a previous section of this report.

Both petroleum and non-petroleum oils can cause deleterious physical effects on aquatic organisms by coating gill surfaces; by increasing biochemical oxygen demand in the water body, potentially leading to fish kills; and by combining with surface debris to form tarballs that settle out of the water column and may smother benthic organisms. Though the physical effects of non-petroleum-based oils are the same as those of petroleum-based compounds, vegetable-based oils are non-toxic (USEPA 1976).

Sediment and Fish

Sediment and fish tissue residue data for petroleums were not available. See the section of this report on PAHs for more information.

Wildlife

Since the early 1990s, two moderate-sized oil spills have resulted in wildlife kills in the Chesapeake Bay. On March 28, 1993, a 36-inch high-pressure pipeline running from the Gulf of Mexico to Maine ruptured and released more than 400,000 gallons of No. 2 heating oil into Sugarland Run Creek and the Potomac River, affecting a 9-mile stretch. Twenty-three oiled birds, including wood ducks (*Aix sponsa*), Canada geese (*Branta canadensis*), mallards (*Anas platyrhynchos*), and a kingfisher (*Megaceryle alcyon*), were observed, 18 of which succumbed despite rehabilitation efforts (Research Planning, Inc. 1993). Undoubtedly, more birds were affected by this spill. Another oil spill on April 7, 2000, involved a pipeline rupture that released 126,000 gallons of No. 2 and No. 6 fuel oil at the Potomac Electric Power Company Chalk Point Facility near Aquasco, Maryland. The spill spread to Swanson Creek, a tributary of the Patuxent River; 55 dead birds (principally waterfowl, but also osprey, herons, gulls, and terns) were found, and 109 birds were collected for rehabilitation (12 of these died) (Cardona et al. 2001). This spill occurred near osprey nests and, although 10 adult ospreys were observed to be oiled, there was no evidence of effects on reproductive success (Michel et al. 2001). In addition, some visible evidence of oiling from this spill was found on 10 snapping turtles (*Chelydra serpentine*), 7 diamondback terrapins (*Malaclemys terrapin*), 1 Eastern box turtle (*Terrapene carolina*), 18 common mud turtles (*Kinsternon subrubrum*), 2 Eastern painted turtles (*Chrysemys picta picta*), 1 garter snake (*Thamnophis sirtalis*), and 8 Northern water snakes (*Nerodia sipedon*) (Tri-state Bird Rescue and Research Inc., historical records, Newark DE).

Conclusions

Existing data on the occurrence of petroleum spills or observable sheens are readily available through the NRC. However, concentration-based data are much more limited and typically present in the form of the surrogate parameter "oil and grease". Review of the NRC data indicates that the extent of petroleum contamination within the Chesapeake Bay watershed is predominantly localized to areas with heavy boating or shipping activity. Two areas of the watershed (i.e., District of Columbia and PA) have documented water column impairments for oil and grease, indicating localized severity for this contaminant group.

2.5 Pesticides

Abstract

The USEPA defines the term "pesticide" to include many kinds of ingredients in products, such as insect repellants, weed killers, disinfectants, and swimming pool chemicals, which are designed to prevent, destroy, repel, or reduce pests. Unlike most of the other toxic contaminant groups (other than some PAHs) discussed in this report, pesticides are intentionally applied to land and water surfaces throughout the Chesapeake Bay watershed. Occurrence data on some pesticides in the watershed are available and therefore do not limit general interpretations about their extent and severity. For example, the extent of occurrence of herbicides, and specifically atrazine and its degradates, is identified as widespread. Insecticides such as aldrin, chlordane, dieldrin, DDE/DDT, heptachlor epoxide, mirex, and their degradates occur in localized areas. These pesticides persist for long periods of time in areas of usage. Data and research gaps exist for other pesticides including some current-use insecticides and fungicides, and consequently the extent and severity remains uncertain and cannot be evaluated at this time. The severity of some pesticide effects as determined by conventional benchmarking approaches has been documented in individual integrated state assessment reports by the number of river miles that are considered to be impaired (i.e. aldrin, chlordane, dieldrin, DDE/DDT, heptachlor epoxide, and mirex). For other pesticides, data and understandings of the potential environmental behavior and effects are not as complete for the low level concentrations typically detected in the environment. In addition, recent laboratory and field research indicate some pesticides can cause adverse sublethal effects on receptor organisms at environmentally relevant concentrations. Although there are data gaps for some pesticides in the watershed, there are a large number of potential sources (e.g. agricultural, residential, etc.). When considered with the emerging literature on the sublethal effects of some pesticides (e.g. atrazine) and their degradates, there is potential for adverse ecological effects in the Chesapeake Bay watershed.

Background

Generally chemical or biological agents, pesticides are used for a range of governmental, residential, industrial, and agricultural applications. This report summarizes chemical pesticides only. Some pesticides target only one or a few species, whereas others are broad-spectrum and target all similar species. Pesticides can be classified or grouped by (1) similarities in the target organism(s) (e.g., herbicide, insecticide, fungicide, etc.); (2) similarities in the molecular structure (e.g., organophosphates, organochlorines, carbamates, etc.); or (3) the mode of action (e.g., acetylcholinesterase inhibition, inhibition of sterol biosynthesis, growth regulation, etc.).

Application rates and procedures vary greatly depending on the targeted organism(s), mode of action, chemistry, and other factors such as whether the pesticide is no longer in use. For example, some pesticides are applied before plants emerge, whereas others are applied after they emerge. Some are applied directly to plant or animal tissue, whereas others are applied to the soil or other physical components of habitat (e.g., open-water surfaces). Application procedures as well as delivery modes such as irrigation waters, aerial spraying, and tractor-applied dictate to a large extent how and where the active ingredients will be mobilized, deposited, and/or transformed. Changes in amounts of pesticides manufactured and used over time also influence their environmental occurrence and distribution. These pathways then determine the extent to which receptors (included target and nontarget organisms) are exposed. In order to facilitate application and uptake, various adjuvants are commonly used in pesticide mixtures. Consideration of pesticide adjuvants, although they are potentially important toxic chemicals, is beyond the scope of this chapter, which focuses on active pesticide ingredients and their degradation products. In addition, the USEPA definition of pesticide includes disinfectants. This broadens the scope of pesticides as a group of toxic contaminants and includes potential sources such as disinfectants and disinfection by-products in wastewater effluents and other environmental pathways. Those pesticides were beyond the scope of this section but are discussed elsewhere in the report (e.g. Household and Personal Care Products).

Agricultural uses account for about 75% of all pesticides, but at least one pesticide is stored in 85% of U.S. households, and one to five pesticides are stored in 63% (Delaplane, 1996), and pesticides are also used to control forest pests and for other purposes as well. Kiely et al. (2004) estimate a per capita home, garden, and personal-care pesticide loading of 0.42 pounds per year in the Chesapeake Bay watershed, which results in a loading to the watershed of about 6.5 million pounds annually, not including agricultural uses. Hively et al. (2011) showed that the fate of herbicides (atrazine and metolachlor) is related to landscape features and characteristics in 15 subwatersheds in the Choptank River system. Although agricultural and residential uses account for much of the pesticide use in the watershed, other land activities associated with golf courses, mosquito control, gypsy-moth control, control of invasive or non-native species, and weed control on rights-of-way are also potential sources of these contaminants. Therefore, virtually all land uses in the Chesapeake Bay watershed are potential sources of pesticides, and many provide direct pathways to the environment.

In 2009, a study (Maryland Pesticide Network 2009) of pesticides in the Chesapeake Bay watershed was published with input from a range of stakeholders and technical experts, including representatives of Federal and State agencies, and academia. The report noted that, although more data are needed, some pesticides have potential adverse effects on the watershed. Many previous studies have prioritized pesticides on the basis of combinations of occurrence (extent) and toxicity (severity) as potential environmental contaminants. For example, in 2006 the CBP Toxics Subcommittee assessed organic chemicals, including a range of pesticides previously documented to occur in the Bay, to occur in its tidal tributaries, or to have been used in the watershed with the potential to reach the Bay. The report further prioritized 35 contaminants, including 10 organochlorine and 2 organophosphate pesticides, for additional study on the basis of the following criteria: (1) the chemical appeared on lists of priority chemicals submitted by the Chesapeake Bay Program Toxics Subcommittee, (2) loading estimates for the chemical were available in the 1999 Chesapeake Bay Toxics Loading and Release Inventory (CBP 1999), and (3) detections of the chemical were reported in the 1999 Toxics Characterization Report (USEPA 1999). These results represent only one attempt to prioritize pesticides in the watershed, with the acknowledgment that other prioritization schemes may yield different results. That prioritization is another indication that multiple pesticides warrant attention as potential environmental contaminants in Chesapeake Bay watershed.

There are many point and nonpoint sources of the pesticides that enter the Chesapeake Bay watershed. As previously mentioned, these chemicals are deliberately applied to the land and water surfaces. In addition to intentional applications, spills and other sources and indirect pathways to the environment, such as wastewater effluents (solid and/or liquid), landfill leachates, spray drift, runoff to surface water, and leaching to groundwater, provide inputs. These sources are all closely related to the usage and use patterns of pesticides. A multitude of pesticides, each with its own "footprint" in terms of geographic extent and location, is used in the Chesapeake Bay watershed. Pesticide usage changes over time as new products are registered, old ones are phased out, and pest-control needs and strategies evolve. There are many reasons for phasing out or modifying older pesticides in favor of new ones: loss of effectiveness due to increased resistance in target organisms; technological advances that enable the same level of effectiveness to be achieved with smaller quantities of pesticides (e.g., increases in potency of active ingredients); and changes associated with re-registration processes. For example, Hartwell (2011) notes that the total mass of pesticides being applied to the Chesapeake Bay watershed declined during the period 1985 to 2004; however, because the potency of these chemicals increased during the same period, the "toxic units" (a measure of the toxicity of the active ingredients to a range of animal and plant species) remained approximately static or increased depending on the bioassay test organism(s) used. As a result of increases in usage and new analytical methods synthetic pyrethroids, are detected in increasing frequency in both surface water/suspended sediment and stream bed sediment (Kuivila et al. 2012). In addition, recent agricultural uses of pesticides have begun to shift toward the use of transgenic crops. Two of the most common examples include those that are resistant to targeted pesticides (e.g., Roundup Ready® soybeans) and/or are capable of creating pesticides through otherwise normal metabolic activities (e.g., "Bt corn"). In the Chesapeake Bay watershed Roundup Ready® soybeans are planted more extensively than corn with atrazine and metolachlor being used as pre-emergent herbicides on corn. As a result of transgenic crops, pesticide usage and geographic patterns are changing. For example, glyphosate herbicide usage increases where Roundup Ready® soybeans are grown, whereas usage of other herbicides (e.g., metolachlor) decreases. Toxins produced in situ by transgenic crops such as Bt corn are released to the environment in unquantified amounts, but recent studies have documented their occurrence and potential effects on aquatic invertebrates (Rosi-Marshall et al. 2007).

The fate and transport properties of pesticide formulations in the environment vary with active ingredient, adjuvant characteristics and purposes, application procedures, seasonality, and other factors (Barbash and Resek 1996). Generally, pesticides are biologically active agents capable of persisting long enough to be effective in environmental applications. Therefore, the half-life of all currently registered active pesticide ingredients ranges from hours to days to weeks to months depending on the nature of the molecule. Some pesticides are water soluble and can be expected to be mobile in aquatic environments, whereas others are hydrophobic and are likely to sorb to solids. Foster et al. (2000) documented that measured concentrations of organophosphorus pesticides in tributaries to the Chesapeake Bay varied mainly in response to the timing of their application, whereas the particle-reactive organochlorine insecticides were more directly correlated with stream discharge. Finally, although not a focus of the summaries in this report, atmospheric transport of pesticides has been shown by a number of studies across the globe to be an important ecological pathway for exposure (Majewski and Capel 1995). Kuang et al. (2003) detected metolachlor, atrazine, simazine, endosulfan, and chlorpyrifos in air and rain, with maximum concentrations during the time when crops were planted; they suggested that an atmospheric source from outside the watershed was likely contributing to some of the high values. Summarizing the environmental behavior of all pesticides is beyond the scope of this report; however, information on half-life/persistence and the potential for the active ingredient to enter and move in aquatic environments are part of the registration process considerations required by USEPA. Many documented cases show long-term persistence of pesticides and/or their degradates (e.g., DDT/DDE; ATSDR 2002) as well as mobility in the environment (Barbash and Resek 1996). In addition, the environmental behaviors exhibited by the environmental transformation products of pesticides can be very different from those of the parent active ingredient (Barbash and Resek 1996).

Pesticide occurrence in ambient water resources, as residues on produce and other food products, and in our drinking waters has been documented by a number of nationally relevant studies. The USGS National Water-Quality Assessment (NAWQA) program has monitored and assessed the extent of pesticide occurrence in the Nation's water resources since the early 1990s. These assessments have targeted a range of insecticides, herbicides, and some fungicides, and have shown close relations between pesticide applications and occurrence in nearby water resources throughout a range of hydrogeologic environments (Gilliom et al. 2006) Other factors such as the physiochemical characteristics of the pesticide molecules, the nature and properties of associated soils, and other hydrogeologic controls have been shown to be related to pesticide occurrence in water resources. Gilliom et al. (2006) note that the major findings of the NAWQA program with respect to pesticides include: (1) pesticides are frequently present in streams and groundwater; (2) pesticides are seldom present at concentrations that exceed human-health benchmarks; however, current benchmarks have been, and will be continuously, updated and revised as understandings of pesticide modes of actions in nontarget organisms, mixture effects, and other nonconventional toxicological effects are advanced; and (3) pesticides occur in many streams at concentrations that may have effects on aquatic life or fish-eating wildlife. The U.S. Department of Agriculture (USDA) publishes annual summaries of the Pesticide Data Program that include occurrence data for pesticide residues on a range of food commodities and drinking-water sources in several states across the Nation, including some samples collected in Maryland, New York, Pennsylvania, and Virginia (USDA 2009). Although individual State data were not summarized for this report, nationally representative findings include: (1) 17% of food samples analyzed contained residue of only one pesticide; (2) 40% of food samples analyzed contained more than one pesticide; (3) 29 different pesticide residues (including metabolites) representing 19 parent compound pesticides were detected in the 278 groundwater samples collected from farm wells, schools, and domestic wells; (4) 53 different pesticide residues (including metabolites) representing 42 pesticides were detected in the 306 finished municipal drinking-water samples analyzed; and (5) 49 different pesticide residues (including metabolites) representing 38 pesticides were detected in 306 samples of untreated (at intake locations) municipal drinking water. The detections of some pesticides cited above in sources of drinking water indicate the likelihood of at least one other potential exposure route that is unrelated to the workplace or application of pesticides. The presence of these contaminants in human tissue and drinking water does not necessarily indicate adverse health effects; however, in referring to low-level environmental exposures of pesticide residues from food sources (another exposure pathway for humans and animals), the National Institutes of Health notes that "scientists do not yet have a clear understanding of the health effects of these pesticide residues". However, Birnbaum (2012) cited a growing body of research that links pesticide exposures to both acute and long-term health impacts including certain cancers, neurological, developmental, psychiatric, learning, behavioral and respiratory impacts, birth defects and immune system disorders.

As mentioned above, pesticides are a unique group of toxic contaminants because they are applied directly to land surfaces in a variety of land-use settings across the watershed. As a result of advances in modern detection technologies such as mass spectroscopy and gas or liquid chromatography and the range in their hydrophilic and lipophilic properties, pesticide residues (including parent and degradate compounds) are likely to be found in detectable concentrations in various environmental matrices including water, sediment, air, and fish tissue. Because the USEPA pesticide registration process requires testing and data, many sources of information about individual pesticide toxicities and potential behavior in the environment are available, particularly for the concentrations likely to be found as a result of pesticide applications in the field. This and other information sources (e.g., the Safe Drinking Water Act) have produced benchmarks to interpret environmental pesticide data. For example, the USEPA Office of Pesticides Program Aquatic Life benchmark for atrazine is 65 μg/L; however, Tillitt et al. (2010) showed that chronic exposures to concentrations as low as 0.5 μg/L will cause fathead minnows to spawn less frequently and to produce fewer eggs than controls. Recently, the usefulness of conventional "benchmarks," which have been, and continue to be, developed on a contaminant-by-contaminant basis for a limited range of toxicological endpoints, has been questioned (Feingold et al. 2010). In other recent research on the potential for pesticide usage to do environmental harm, the adjuvants have been implicated in potentially increasing the toxicity of pesticide mixtures in the environment (Brausch et al. 2007; Katagi 2008).

Impairments cited in the following paragraph are focused on pesticides that are no longer in use in the watershed. In Virginia, along the Potomac and Shenandoah Rivers, 7 river miles are impaired as a result of the insecticides heptachlor epoxide (five impaired segments) and chlordane in the James River Basin. Impairments are listed from chlordane (3 impaired miles) and from other insecticides (aldrin and DDE/DDT), totaling 9 impaired river miles; mirex, also an insecticide, impairs 5 river miles in the James River and 49 miles in the Rappahannock River (VADEQ and VADCR 2010). Two river segments in Maryland, the Back River and the Baltimore Harbor, and one impoundment, Lake Roland, are listed as impaired waters for chlordane. One location in the northwest portion of the Anacostia River in Maryland is listed as impaired by heptachlor epoxide contamination (MDE 2010). In the Potomac and Anacostia watersheds in the District of Columbia, chlordane, DDE, DDD, DDT, dieldrin, and heptachlor epoxide all contribute to impairments (DDOE 2010). Pesticides are also responsible for the impairment of 17 river miles in the Susquehanna River Basin of Pennsylvania (PADEP 2010). New York and West Virginia do not report any pesticide contamination entering the Chesapeake Bay from their portions of the watershed (NYSDEC 2010, WVDEP 2010).

Water

Many sources of data and information on pesticide extent in waters of the United States at local, regional, and national levels are available. Among the Federally-funded national programs that address pesticide occurrence in the environment, the U.S. Geological Survey's NAWQA Program (Ator et al. 2005; Ator 2008; Ator and Denver 2012; DeBrewer et al. 2007, 2008; Denver et al. 2004, 2010; Domagalski et al. 2008; Phillips et al. 2007) has found:

- Pesticides are widely detectable in non-tidal streams and groundwater of the Bay watershed in areas of use.
- Pesticides are detectable year-round in many streams. Concentrations rarely exceed 1µg/L but can increase for example during, or shortly after, application if runoff transports contaminants to receiving water bodies.
- Concentrations of degradates of metolachlor, alachlor, and acetochlor often exceed those of parent compounds in groundwater and streams, in some cases by orders of magnitude.
- Many pesticides (such as atrazine, metolachlor, and degradates) occur widely in groundwater, and groundwater serves as an important pathway for transport from the land surface to streams.

» The extent of pesticide occurrence varies in time as new pesticide products come into use and others are no longer used. For example, concentrations of diazinon decreased significantly in Accotink Creek (a small stream in suburban Distict of Columbia) following usage restrictions in the early 2000s. The introduction of genetically modified crops and their associated pesticides into the Chesapeake Bay watershed has also changed pesticide usage over time.

Water samples collected in Chesapeake Bay tidal waters during spring 2000 and late summer 2004 were analyzed for a range of pesticides (McConnell et al. 2007). Of all pesticide groups analyzed for, agricultural herbicides were detected most frequently. Among the herbicides analyzed for, the parent compounds atrazine, simazine, and metolachlor were detected at maximum concentrations of 1.29, 0.49, and 0.61 µg/L, respectively. The highest concentrations of these compounds were observed in the Nanticoke, Pocomoke, and Chester Rivers on the Delmarva Peninsula, reflecting the intensity of agricultural production in the watersheds. Degradates of these parent compounds were also frequently detected, with the metolachlor degradation product, metolachlor ethane sulfonic acid (MESA), found in greater concentrations (2.9 µg/L) than the parent compound in almost all of the tidal regions assessed. Atrazine was detected in 100% of the samples collected in 2004. McConnell et al. (2007) noted that herbicides and associated products are present within both agricultural and nonagricultural tidal areas of the Chesapeake Bay throughout the year, even though they are used primarily in the spring. In a more detailed study of pesticides in upland areas of a Delmarva Peninsula agricultural watershed, outlets of 15 non-tidal sub-watersheds of the Choptank River were monitored approximately monthly under baseflow conditions from 2005 to 2007 (Hively et al. 2011). This work was part of a national experiment called the USDA Conservation Effectiveness Assessment Project (CEAP). Across all stations, the maximum atrazine, metolachlor and MESA concentrations were 7.6, 4.4, and 8.6 µg/L respectively with the greatest concentrations occurring between approximately April 1 and June 1 corresponding to corn planting activities. At most sites, only a moderate increase in metolachlor occurred after soybean planting in June, indicating that most soybeans planted were "Roundup-ready®" and did not include the use of metolachlor as a pre-emergent herbicide. Mean concentrations for the entire sampling period were 0.19, 0.29, and 4.2 µg/L for atrazine, metolachlor and MESA, respectively.

Recognizing the need to develop estuarine criteria for low levels of pesticides in the Chesapeake Bay, Hall et al. (1997) studied the chronic toxicity of atrazine to Sago pondweed (*Potamogeton* *pectinatus*) under varying conditions of salinity. This study showed that toxicity as well as bioavailability was influenced by salinity and highlights the typically subtle and complicating factors involved in determining conventional "benchmarks". Since then, many other studies have concluded that conventional benchmarking approaches to decision making are inadequate for a variety of reasons. For example, Fatima et al. (2007) exposed goldfish to environmentally relevant mixtures of herbicides (atrazine, simazine, diuron, and isoproturon) and observed biomarkers indicative of immune suppression. They concluded that these common environmental exposures cause immune suppression in goldfish, thereby representing an endpoint and exposure scenario not quantified in conventional benchmarking studies.

Whitall et al. (2010) analyzed water samples collected from seven sites in the Choptank River estuary on the Delmarva Peninsula during base flow and documented the seasonal signal in herbicide (atrazine, simazine, and metolachlor) concentrations known to dominate pesticide occurrence in streams throughout the Nation. Observed concentrations of individual herbicides did not approach established levels of concern (10-20 µg/L) for aquatic organisms (USEPA 2006b) and were below the USEPA drinking-water standard for atrazine (3 µg/L). Total triazine concentrations (atrazine, simazine, CIAT, and CEAT), however, exceeded 3 µg/L in eight samples, highlighting the importance of assessing the effects of mixtures. Additionally, the degradation products (CIAT, CEAT, MESA, and MOA) were associated with groundwater pathways and were frequently found in higher concentrations than the parent compounds (Whitall et al. 2010). Hladik et al. (2005) also documented that degradates of triazine herbicides were found in higher concentrations than parent products in the upper Chesapeake Bay, and noted the importance of assessing degradate compounds in ecological studies.

Hall et al. (2009) studied the potential effects of irgarol (an algicide used as an anti-fouling agent in paints for boat hulls) and its major metabolite on phytoplankton in the Port Annapolis marina and Severn River system, Maryland. These sites were chosen to represent a range in use of irgarol including a high-use area (marina) and a background site (Severn River near its confluence with the Chesapeake Bay). In the study, a probabilistic approach was used to determine ecological risk by comparing exposure data with toxicity endpoints (NOEC). Field data could not confirm the microcosm NOEC in the high-exposure area (marina).

As noted above, there are many local environmental studies that assessed pesticide occurrence in the Chesapeake Bay watershed. Recently, passive-sampler technologies have been used to detect a broader range of pesticides in the Potomac River Basin (Alvarez et al. 2009). In general, pesticide occurrence data from several more recent studies including Ator and Denver (2012), Chambers and Leiker (2006), McConnell et al. (2007), Battaglin et al. (2009), Loper et al. (2007), and Hively et al. (2011) reflect changes in pesticide trends (e.g., increases in glyphosate use) as well as changes in sampling and analytical methods to include a wider range of degradate compounds.

Kingsbury et al. (2008) collected raw and treated drinking water from nine community water systems ranging in size from a system serving about 3,000 people to one that serves about 2 million people. The study included one site on the Potomac River where simazine, atrazine, DEA, and metolachlor were consistently detected throughout the year (i.e., 100% detection frequency). Other pesticides frequently detected include 2,4-D (58%), 2-hydroxyatrazine (92%), acetochlor (17%), benomyl (8%), carbaryl (33%), diazinon (33%), deisopropylatrazine (8%), 3,4-dichloroanaline (17%), diuron (25%), fipronil (17%), prometon (92%), MCPA (33%), and tryclopyr (17%).

Sediment

The NOAA Contaminated Sediment report (Hartwell and Hameedi 2007) identifies organophosphate-type pesticides as being among the most common of all synthetic organic chemicals present in tidal sediment, with the largest concentrations reported for the Choptank and Nanticoke River watersheds in Maryland during spring runoff. Elevated levels of insecticides including chlordanes, heptachlors, nonachlors, aldrin, dieldrin, endrin, and endosulfan were found in sediments in the Elizabeth River in Virginia, and trace amounts of insecticides were found throughout the other tidal portions of the Bay. Hexachlorocyclohexane (HCH) concentrations were elevated in the Patuxent, Potomac, and Eastern Shore tributaries of Maryland. DDT was found throughout the Bay but was concentrated mainly in the upper Bay, and exceeded the ERM only in the Elizabeth River. Other pesticides detected were mirex, chlorpyrifos, butyltins, tributyltin (TBT), and chlorinated benzenes (Hartwell and Hameedi 2007).

Organochlorine pesticides that are no longer in use in the watershed can be extremely persistent in soils and runoff events may move these soils into waterways where they can be accumulated in sediments and in wildlife. Areas that have historically been used for agriculture and residential areas that received high concentration applications of chlordane for termite control may be important sources of these compounds. In a recent study by Goel et al. (2010) concentrations of these "legacy" compounds were measured in air and rain from three different locations on the

Delmarva Peninsula over a four-year period. Overall, concentrations of these compounds were found to be slowly decreasing over time reflecting a gradual decrease in soil concentrations. However, results indicate significant local sources of the insecticide dieldrin and the DDT degradation product DDE in soils of the Delmarva Peninsula. Little is known regarding the location and magnitude of contamination from these historical sites which are likely distributed throughout the Chesapeake Bay watershed.

Fish

Analyses of fish tissue for pesticide residues have been conducted for a variety of monitoring and research objectives throughout the watershed and it is beyond the scope of this report to summarize all these results. For illustrative purposes however it is instructive to highlight some selected results in order to provide further perspective on the extent and(or) severity of some pesticides in the watershed. For example, surveys in the District of Columbia (Pinkney et al. 2001a; Pinkney 2009) have shown elevated levels of chlordane in the tissue of certain fish species across many portions of the Anacostia and Potomac Rivers. Blazer et al. (2010) analyzed organ-specific fish tissue from fish in the Potomac River watershed; the following pesticides were measured in at least one tissue analyzed: chlorpyrifos, the phenyl pyrazole pesticides (fipronil, finpronilsulfide, and desulfynylfipronil), hexachlorobenzene, pentachloronitrobenzene, pentachloroanisole, oxychlordane, trans-chlordane, cis-chlordane, trans-nonachlor, cis-nonachlor, dieldrin, and DDx (DDE, DDD, DDT). One or more organochlorine pesticides were found in all tissues of both sexes. In addition, the ovary from a female smallmouth bass collected in the South Branch Potomac River contained 30.5 ppb total organochlorine pesticides, with 13.6 ppb DDx; skin from the same individual contained 6.4 ppb total organochlorine, with 2.6 ppb from DDx. Pesticide compounds detected in individual organ tissue varied in presence and magnitude; muscle tissue generally contained the lowest concentrations.

Ripley et al. (2008) discuss possible links between atrazine exposures and immune suppression in fish of the Potomac River system. A recent summary article by Rohr and McCoy (2011) showed potential behavioral and immune suppression effects when fish are exposed to environmentally relevant concentrations of pesticides and pharmaceuticals. In more recent work within the Chesapeake Bay watershed, Kolpin et al. (2013) have shown statistically significant correlations between atrazine concentrations in the water column above smallmouth bass nesting sites in the Potomac River and intersex conditions in male smallmouth bass collected at those sites (see Chapter 3.0).

Wildlife

Rohr et al. (2008) exposed green frog (*Rana clamitans)* tadpoles to environmentally relevant concentrations of one of four pesticides (atrazine, glyphosate, carbaryl, or malathion) for one week, then half were exposed to a trematode parasite, (*Echinostoma trivolvis*), for 24 hours, and then all were returned to the same pesticide exposure for an additional week. The authors reported that exposure to each of the pesticides increased the percentage of encysted cercariae compared with tadpoles exposed to a solvent control. They stated that the increased susceptibility may be a result of immunosuppression. Additional studies have shown sublethal and other effects on receptor organisms exposed to low levels of pesticides. For example, Hayes et al. (2003) showed that atrazine can induce hermaphrodism in leopard frogs (*Rana pipiens*), and Blakley et al. (1999) showed immunotoxicological effects of pesticides to a variety of organisms.

Organochlorine Pesticides

Toxicological effects of organochlorine pesticides and metabolites upon Chesapeake waterbirds occurred principally through impairment of reproduction rather than through altered survival of adults (Heinz and Wiemeyer 1991; Blus 1996). As described in various reviews (Ohlendorf and Fleming 1988; Heinz and Wiemeyer 1991; Rattner and McGowan 2007), concentrations of the DDT metabolite *p,p'*-DDE in osprey (*Pandion haliaetus*) and bald eagle (*Haliaeetus leucocephalus*) eggs in the Chesapeake were among the highest in the nation and appear to have been the principal cause of the population declines that were observed in these species from the 1950's through 1970's in this region (Wiemeyer et al. 1984, 1988, 1993). Many other organochlorine pesticides and metabolites were also detected in eggs and young during this era (e.g., dieldrin, endrin, heptachlor epoxide, chlordane, oxychlordane, mirex, toxaphene). Since the phase out of some organochlorine pesticides in the early 1970's, Chesapeake Bay osprey and bald eagle populations gradually began to rebound and currently populations are the greatest since they began to be systematically monitored (Watts et al. 2004, 2007).

Since 2000, data are available for only a few addled eggs of bald eagles from the Chesapeake. A single addled egg collected at Aberdeen Proving Ground in Harford County Maryland in 2008 contained *p,p'*-DDE at a concentration of 9.46 ppm wet weight, and chlordane constituents and metabolites (e.g., oxychlordane, *cis*- and *trans*-nonachlor, *cis*- and trans-chlordane) totaling 3.34 ppm (Mojica and Watts 2008). Notably, *p,p'*-DDE exceeded 5.5 ppm, and threshold concentrations associated with embryo lethality (Elliott and Harris 2002; Henny and Elliott 2007). Two addled eggs collected from the Indian Head Naval Support Facility in Charles County Maryland in 2008 and 2009 contained *p,p'*-DDE at concentrations of 3.77 and 5.61 ppm, chlordane constituents and metabolites at concentrations of 2.12 and 4.34 ppm (Mojica and Watts 2011). Organochlorine pesticides and metabolites in blood of eagle nestlings were also quantified in 58 blood samples of nestling eagles from these same sites (Mojica and Watts 2008, 2011). The sum of DDT and metabolites (< 0.1 ppm wet weight) and the sum of chlordane constituents (< 0.02 ppm) were below the thresholds for toxicity (Elliott and Harris 2002; Henny and Elliott 2007).

In 2000 and 2001, a large-scale study was undertaken in which osprey eggs were collected from nests in these regions of concern (Baltimore Harbor, Anacostia River, Elizabeth River) and nearby tributaries, and from a reference area (South, West and Rhode Rivers near Annapolis) (Rattner et al. 2004). Of 27 organochlorine pesticides and metabolites quantified, 17 or more were detected in half of the sample eggs. Concentrations of *p,p'*-DDE ranged from 0.26-1.92 ppm wet weight in the regions of concerns compared to 0.24-0.98 ppm in the reference area. Over half of the eggs sampled from the Anacostia and middle Potomac Rivers had *p,p'*-DDE concentrations within the 95% confidence interval (1.2 to 3.0 ppm) associated with 10% percent eggshell thinning in osprey eggs, but no samples exceeded the value (>2.6 ppm) associated with population instability (production of 1 fledgling per active nest) (Wiemeyer et al. 1988). The concentration of *p,p'*-DDD ranged up to 0.24 ppm, while other compounds (e.g., dieldrin, endrin, heptachlor epoxide, α- and γ-chlordane, oxychlordane, *cis*- and *trans*-nonachlor, mirex, HCB) were well-below 0.1 ppm, and seemingly below reproductive toxic thresholds associated with in bird eggs. Chlordecone (also known as Kepone®) an organochlorine insecticide had been found at concentrations of up to 5.0 ppm wet weight in osprey eggs collected from Chesapeake tributaries in Virginia in 1977 (Wiemeyer et al. 1988), but was not detected in a subset of osprey eggs from the Elizabeth River in 2001 (Rattner et al. 2004).

Between 1993 and 1999, post-term and addled peregrine falcon (*Falco peregrinus*) eggs were collected from the Chesapeake region (e.g., Chincoteague and Martin National Wildlife Refuges, Clay, Cobb, Fisherman, Metompkin, South Marsh, and Wallops Islands, Baltimore, Wachapreague, and Fort Eustis) as part of a mid-Atlantic states monitoring effort (Clark et al. 2009).

Concentrations of *p,p'*-DDE ranged from 0.94 to 5.05 ppm wet weight. Other organochlorine pesticides in these egg samples were generally low (e.g., β-hexachlorocyclohexane <0.01 ppm, oxychlordane <0.5 ppm, *cis*-nonachlor <0.25 ppm, *trans*-nonachlor <0.3 ppm, HCB <0.02 ppm, hepatochlor epoxide <0.3 ppm, and *o,p'*-DDD, *p,p'*-DDD, *o,p'*-DDT, and *p,p'*-DDT <0.02 ppm), with the possible exception of dieldrin (range 0.05 to 1.31 ppm). Despite the presence of these organochlorine pesticides, 11 of 14 clutches that were followed in detail were successful in producing young. In another study, 22 peregrine eggs that failed to hatch in 2001 and 2002 from the Chesapeake region had *p,p'*-DDE concentrations ranging from 1.38 to 12.4 ppm wet weight (Potter et al. 2009). Notably, concentrations in two of the eggs exceeded 10 ppm, and thus approached the threshold for 20% eggshell thinning and impaired reproduction.

A study assessing reproduction and contaminant exposure of the Baltimore Harbor black-crowned night heron (*Nycticorax nictocorax*) colony was undertaken in 1998 (Rattner et al. 2001). Based on analyses of a large number of sample eggs, concentrations of *p,p'*-DDE (0.023-1.292 ppm wet weight) and dieldrin (not detected to 0.262 ppm) were clearly lower than observed in 1991, and levels of other organochlorine pesticides were also quite modest. Pesticides in night-heron eggs concurrently collected from Holland Island, a remote reference colony in the southern Chesapeake, were but a fraction of those observed in Baltimore Harbor. There was neither eggshell thinning nor impaired reproduction in night–herons that could be associated with DDT exposure in this study.

Two studies examined organochlorine pesticides in common tern (*Sterna hirundo*) eggs. In 1997, 10 three-egg clutches were collected from Bodkin Island, and organochlorine pesticide concentrations (e.g., DDT and metabolites, chlordane and metabolites, dieldrin, endrin, heptachlor epoxide and mirex) were below the detection limit of 0.061 ppm dry weight (French et al. 2001). In a 2010 collection on Poplar Island, eggs of 6 terns contained low levels (<0.08 ppm wet weight) of *p,p'*-DDE, *cis*- and *trans*-nonachlor, mirex and hexachlorobenzene (Rattner et al. 2010).

Organophosphorus and Carbamate Pesticides

Use of organophosphorus and carbamate pesticide in the United States expanded greatly following the ban of many organochlorine pesticides in the 1970's. These compounds or their activated metabolites inhibit the enzyme acetylcholinesterase, thereby disrupting neurotransmitter processes in the central nervous system and normal neural functioning of the sensory, integrative, and neuromuscular systems (Ballantyne and Marrs 1992). Definitive evidence of poisoning in wildlife usually entails detection of the parent compound in the digestive tract and substantial inhibition of brain acetylcholinesterase activity (Hill 2003).

For the period of 1988 through 2011, the Contaminant Exposure and Effects-Terrestrial Vertebrates database (Rattner et al. 2005; Rattner and McGowan 2007) contained over 130 records documenting at least 50 incidents of unintentional bird poisonings in the Chesapeake Bay region that involved anticholinesterase pesticides, and most of these incidents occurred in agricultural settings. Diagnoses are derived from inhibition of brain cholinesterase activity, circumstantial evidence of use in nearby fields, and in a few instances, detection of parent compound in the gastrointestinal tract. These compounds do not accumulate appreciably in tissue of higher vertebrates, so tissue residue data are not available. These die-offs involved six orders of birds, most frequently passerines, birds of prey, and waterfowl. These incidents were principally caused by carbofuran exposure, and affected many different species of birds, including at least one bald eagle (Stinson et al. 1994). Use of granular formulations of this carbamate was restricted by the USEPA in 1994 due to bird die-offs. The remaining incidents involved chlorpyrifos, diazinon, fenthion, phorate, parathion and the carbamate oxamyl.

With the exception of aforementioned pesticides, there appear to be no residue data for the vast majority of some other compounds (e.g. fungicides, some insecticides, rodenticides, etc.) in free-ranging Chesapeake Bay wildlife (amphibians, reptiles, birds and mammals). This apparent data gap reflects the absence of routine monitoring for exposure to newer chemical groups of compounds, their shorter environmental half-lives, rapid metabolism, lower potential to bioaccumulate, paucity of analytical laboratories that routinely quantify newer compounds, and the implementation metrics that associate both exposure and declines in health and condition of wildlife.

It would appear that the frequency of outright mortality of wildlife from pesticide exposure in the Chesapeake region has declined considerably since the organochlorine pesticide era. There are probably still anticholinesterase poisoning incidents, but their frequency and magnitude do not seem to be exerting population level effects. Of the newer groups of pesticides, the ecological consequences of chronic low level exposure and potential adverse effects are inadequately known.

Conclusions

Because of the diverse uses, modes of action, and chemical characteristics of pesticides, the available data on environmental occurrence and related toxicology range from minimal to extensive. In addition, some pesticides are included in current monitoring programs, whereas many others are not. On the basis of data collected within the Chesapeake Bay watershed, herbicides as a group are known or are suspected to occur throughout the watershed. The likelihood that current and future land uses will include regular and widespread applications of herbicides in agricultural, as well as other areas where lawns and other plantings are maintained increases their potential to be widely distributed in the environment. Specifically, atrazine and its degradates are widely distributed throughout the agricultural parts of the watershed, and recent studies have shown the potential for atrazine to cause adverse effects at environmentally relevant concentrations. Insecticides and fungicides, though potentially important environmental contaminants as a result of their potential effects on non-targeted insects, fungi, and other organisms (e.g., broad-spectrum insecticides, systemic fungicides), have not been monitored as broadly or as consistently as herbicides; however, some of the more recent studies indicate the potential for a range of effects related to insecticides and fungicides at environmentally relevant concentrations. Therefore the occurrence of herbicides, and specifically atrazine and its degradates, is identified as widespread. Insecticides such as aldrin, chlordane, dieldrin, DDE/DDT, heptachlor epoxide, mirex, and their degradates occur in localized areas at levels concentrations high enough to indicate that adverse effects are possible. Some legacy pesticides persist for long periods of time locally as well as regionally in areas of current and past usage. Data and research gaps exist for many pesticides including some current-use and some legacy pesticides, and consequently the extent and severity remains uncertain and cannot be evaluated at this time. For example, the potential sublethal effects of low concentrations of many pesticides (and degradates) and their mixtures (including adjuvants, etc.) in the environment is poorly understood.

2.6 Pharmaceuticals

Abstract

Pharmaceuticals are chemicals used in the diagnosis, mitigation, treatment, cure, and prevention of disease in humans and other animals. There is potential widespread extent of occurrence of some pharmaceutical compounds in the environment due to the abundant human (e.g., municipal wastewater-treatment plant discharges) and animal waste (e.g., livestock feeding and related activities) sources in the watershed and their pathways to the environment. The few occurrence studies in the watershed as well as many studies elsewhere in the United States and across the globe have documented pharmaceuticals as environmental contaminants. In addition, although effects on receptor organisms are still poorly understood, several studies have shown biological uptake in a wide variety of exposed aquatic and other species, as well as a range of

sublethal effects of some compounds at environmentally relevant concentrations (e.g. synthetic hormones, some antidepressants, diclofenac, ciprofloxacin and sulfamethoxazole).
Although there is evidence these compounds are of potential concern as toxic contaminants in the Bay watershed, major data gaps and lack of understanding of effects limit assessment. For these reasons, extent and severity of pharmaceuticals are identified as uncertain.

Background

Pharmaceutical compounds have been considered potential environmental contaminants since the 1960's (Richardson and Bowron, 1985) and their occurrence in rivers, groundwater, and untreated drinking water sources of the United States has been documented for many years (Kolpin et al. 2002; Focazio et al. 2008). Although broader definitions of pharmaceuticals include herbal preparations, nutraceuticals, cosmeceuticals, and other remedies, this section focuses on the conventional prescription and nonprescription pharmaceuticals as noted in the Food, Drug and Cosmetic Act (FD&C Act, sec. 201(g)(1)). Excipients, which are additives in pharmaceuticals that give a drug its form, control its release into the bloodstream, facilitate its transport to targeted sites in the body, preserve its shelf life, and enhance its taste, are beyond the scope of this report but could be important compounds for future research on environmental contaminants generally. This report focuses only on the active pharmaceutical ingredients. In addition, biological drugs or so-called "biopharmaceuticals" such as vaccines that are typically administered by injection in the doctor's office are not considered here. The chemically derived prescription and nonprescription drugs covered in this report range from popular over-the-counter analgesics to rigorously controlled chemotherapy or other drugs.

Pharmaceuticals are also commonly used to treat domestic pets as well as livestock. Livestock usages include growth promotion, estrus modulation, and various purposes that are not common goals of human pharmaceutical usage. Many human-use pharmaceuticals are also used as veterinary pharmaceuticals, though many veterinary pharmaceuticals are restricted to animal use only. In addition, many over-the-counter pharmaceuticals are used for domestic pets and livestock. Veterinary drugs are administered for a variety of reasons, including nervous system (anesthetic), digestive tract and metabolism (antacid), antiparasitic (anthelmintic, antibacterial, antimicrobial, antiseptic, astringent, anti-infective), respiratory (antihistamine), steroidal and nonsteroidal anti-inflammatory, respiratory (bronchodilator), genitourinary (diuretic, emetic), estrus synchronization (sex hormones), nervous system (sedative, tranquilizer), and growth promotant purposes.

According to 1995 Toxics Release Inventory (TRI) data, facilities released (discharged to the air, water, or land without treatment) and transferred (shipped off-site) a total of 177 million pounds of contaminants, made up of 104 different chemicals (USEPA 1997). This amount represents about 3 percent of the 5.7 billion pounds of TRI chemicals released and transferred by all manufacturers in that year. In comparison, the chemical industry as a whole produced 1.7 billion pounds that year, accounting for about 30 percent of all releases and transfers. Of the pharmaceutical industry's TRI releases, 57% go to the atmosphere, 25% go to underground injection, 17% go to surface waters, and 1% go to the land. The reported chemical releases from pharmaceutical manufacturing facilities are not commonly active pharmaceutical ingredients (APIs), but rather volatile organic compounds (VOCs) (e.g., solvents) and other chemicals used in the manufacturing process itself. It is not possible at this time to summarize the API releases. USEPA defines high-production-volume chemicals as those produced or imported at quantities exceeding 1 million pounds per year; therefore, some pharmaceuticals and classes of pharmaceuticals are considered high-production-volume chemicals whereas many are not. In addition effluents associated with pharmaceutical manufacturing plants have been shown to be important localized sources of active ingredients in receiving streams (Phillips et al. 2010). Consequently national estimates of production volume may not be an adequate indicator of the potential for a pharmaceutical to affect ecological receptors.

Approximately half of the U.S. population takes at least one prescription drug during any given month, with concurrent use of several drugs being common in the older populations (Gu et al. 2010). Therefore, as more than 17 million people live in the Chesapeake Bay watershed, about 8.5 million prescription drugs are consumed in any given month, or about 102 million per year. Nonprescription drug use is more difficult to track for many types of drugs (e.g., analgesics, antihistamines, etc.). It is beyond the scope of this report to assess and summarize the mass of pharmaceuticals manufactured and released in the Chesapeake Bay watershed; however, it is clear that they are manufactured in large quantities throughout the United States, are consumed within the Bay watershed by the majority of the population and, therefore, are being released to the environment in human and animal wastes and waste-management activities in many locations throughout the watershed.

As a group, pharmaceuticals are complex molecules with various acidic and basic functionalities; they exhibit a range of neutral, cationic, anionic, or zwitterionic behaviors in the environment, are of high molecular weight (commonly ranging from 300 to 1,000 daltons), and are biotransformed in the body, commonly resulting in changes in their physical and chemical properties upon excretion (Kummerer 2004). For example, Sarmah et al. (2006) listed selected antibiotics and showed the range of physical and chemical properties that exists for this one group of pharmaceuticals. Once in the environment, APIs can further undergo a range of potential transformations including photolysis, bacterially remediated degradation, hydrolysis, and dilution. Generally, APIs are biologically active and are mobile because they have high water solubilities in relation to molecular weight (Kummerer 2004).

Pharmaceuticals are associated with point as well as nonpoint sources and pathways to the environment. In sewered areas, pharmaceuticals are transported from human excretion or disposal in residential settings to municipal wastewater-treatment plants (WWTPs). In this way, WWTPs represent centralized locations where various residential, hospital, manufacturing, and other potential pharmaceutical wastes are accumulated before being discharged as a point source of mixtures of APIs and their metabolites to receiving water bodies. Most households in urban areas of the U.S. are connected to municipal sewers, which often include hospitals and can include pharmaceutical manufacturing facilities within the same sewersheds as well. Although WWTPs are not designed to remove APIs or their metabolites, studies have shown that existing treatment processes can decrease the concentrations of some parent compounds, in some cases to below detection (Phillips et al. 2012; Drewes 2007) before being discharged. Barber et al. (2012) documented a decrease in estrogenicity of wastewater effluents and an associated decrease in intersex conditions of exposed fish due to an upgrade in treatment. Other APIs, however, are discharged relatively unchanged because of their ability to resist the type of treatment typically found in municipal WWTPs. Other studies (Benotti and Brownawell 2007, Stackelberg et al. 2007) have shown that drinking-water treatment is effective in decreasing concentrations of parent pharmaceutical compounds in many cases; in some limited cases, however, the pharmaceutical compounds are recalcitrant. Considering the amount of pharmaceuticals manufactured and prescribed as well as factors controlling the removal or dilution of pharmaceuticals as they are transported from wastewater treatment plants to receiving streams, Anderson et al. (2004) developed an empirical model to determine the potential environmental concentrations of targeted pharmaceuticals released to aquatic environments via wastewater treatment plants throughout the United States. Schwab et al. (2005) used the model to predict concentrations of 26 human-use pharmaceuticals in U.S. surface waters and concluded that "no appreciable human health risk exists." Recently the model was revised to include predictions of pharmaceutical concentrations in biosolids generated in wastewater treatment plants (Cunningham et al. 2012).

Other sources of human-waste-related APIs include point-source leachate from onsite sewage disposal such as septic tanks (Conn et al. 2006). Phillips et al. (2010), in an analysis of effluent from WWTPs in New York State (outside the Chesapeake Bay watershed) with a focus on sewersheds that contain pharmaceutical manufacturing facilities, conclude that manufacturing practices can result in pharmaceuticals concentrations from 10 to 1,000 times higher than those typically found in WWTP effluents. Point sources of wastewater effluent are the primary source pathways of human-use pharmaceuticals to the environment in the urbanized areas of Chesapeake Bay watershed, as they are elsewhere in the United States. Therefore, although some generalizations can be made regarding the expected environmental occurrence and persistence of some individual or classes of pharmaceuticals, accurate understanding of the ultimate fate of these compounds and their severity in the environment requires site- and compound-specific assessments.

Pharmaceuticals are also associated with nonpoint sources and pathways to the environment where agricultural land uses, particularly animal feeding operations, are present (Sarmah et al. 2006). Sources of APIs include feed or drinking-water additives, direct injections, implants, drenches, and pastes. The use and length of treatment and whether the drug is delivered to an individual animal or to a herd or flock determine, in part, how a specific drug is ultimately reaches the environment (Sarmah et al. 2006). Potential pathways to the environment reflect overall manure management practices and include composting and/or application of biosolids/manures on crop or other lands, deposition by animals in liquid or solid wastes excreted directly into surface-water bodies, and releases of liquid animal wastes from lagoons or other holding tanks, as well as the local hydrologic condition. Therefore, the potential agricultural sources and the pathways depend, in large part, on how animals and their wastes are managed. In a recent summary of the literature on animal manures, the USEPA (2000c) showed that the amount of manure per acre of farmland in states within the Chesapeake Bay watershed is among the highest in the Nation.

As a group, pharmaceuticals represent a wide array of potentially biologically active compounds with a variety of potential mechanisms of action. Additionally, although these compounds are designed for specific pharmacological purposes, they also can exhibit a range of unintended effects on target as well as nontarget species at therapeutic doses. The toxicity of therapeutic doses of pharmaceuticals is well understood because these chemicals are intentionally ingested and because the FDA registration process requires it (U.S. Department of Health and

Human Services 2007). The effects of pharmaceuticals on aquatic or terrestrial organisms at environmentally relevant concentrations are less well understood. Recent research is focused on the potential effects on organisms that are exposed to low concentrations of pharmaceuticals in the environment. Currently, no aquatic-life or related water-quality or sediment-related benchmarks exist for pharmaceuticals; however, several studies have documented a range of lethal and sublethal effects on receptor organisms in laboratory, mesocosm, and field settings. Swan et al. (2006) was one of the first studies to link environmental exposures of a pharmaceutical to an adverse ecological effect. They found that vultures (*Gyps* spp) in South Asia feeding on cattle carcasses with high levels of diclofenac (a veterinary pharmaceutical) died of renal failure in numbers that threatened their extinction. Since then an increasing number of studies, many focused on aquatic organisms, have been undertaken. In addition, there continue to be more efforts to demonstrate and quantify biological uptake of environmental pharmaceuticals in various species and environmental source pathways. For example, Kinney et al. (2012) documented bioaccumulation of wastewater indicator compounds (commonly indicative of sources of environmental pharmaceuticals) in earthworms exposed to biosolids. Other studies, such as Ramirez et al. (2009), Bringolf et al. (2010), and Schultz et al. (2011), are providing baseline data and information on biological uptake and effects of targeted pharmaceuticals by various species living in municipal wastewater-affected streams. In related work, Li et al. (2012) documented a range of antibiotics including quinolones, macrolides, and sulfonamides in mollusks collected from the Bohai Sea of China. Kidd et al. (2007) documented population-level declines in native fathead minnows in a Canadian lake exposed to environmentally relevant concentrations of ethinyl estradiol (synthetic estrogen). Other pharmaceuticals such as antidepressants have been shown to affect the behavior of fish living downstream of wastewater treatment plants (Schultz et al. 2011). Thus, an increasing number of studies are showing effects of pharmaceuticals on various organisms and ecosystems at environmentally relevant concentrations.

In this report, "pharmaceutical" (or synthetic) hormones are considered separately from naturally occurring (or biogenic) hormones (see the "Biogenic Hormones" section of this report). Although their modes of action are similar, their potencies, sources, and other factors are different. For simplicity, therefore, these two major groups of hormones have been separated. A third group with similar hormone-related modes of action includes other chemicals that have been shown to interfere with normal endocrine functioning through antagonistic or synergistic effects on hormone receptors. These other known, or suspected, "endocrine-disrupting" chemicals, which include some pesticides, surfactants (e.g., alkylphenol ethoxylates), ingredients in plastics (e.g., bisphenol A), and flame-retardant mixtures (e.g., polybrominated diphenyl ethers), are discussed in other sections of this report.

Although representative studies conducted in the Chesapeake Bay watershed are highlighted in this section, several studies conducted outside the watershed are included for illustrative purposes. A short list of representative laboratory, mesocosm, and field studies that have linked various categories of environmental exposures to three major classes of pharmaceuticals (synthetic hormones, antidepressants, and antibiotics) to a range of adverse effects on biological receptors is included. This list is meant to illustrate important effects of these pharmaceuticals on fish and wildlife health only and does not represent a complete literature review.

Water

Human-health or ecologically based water-quality standards are not common for pharmaceuticals and they are not regulated water-quality constituents, consequently, they are not commonly monitored in the environment. However, a few research-based datasets for the Chesapeake Bay watershed and a few for the Nation as a whole are available. State integrated assessments did not include pharmaceuticals, but the few studies that assessed pharmaceuticals as environmental contaminants in the Bay watershed are summarized below. Several studies that provide a national perspective are summarized as well.

Glassmeyer et al. (2008) reviewed and summarized the available literature on the occurrence of pharmaceuticals and personal-care products in the environment worldwide and showed that most of the current papers originated in the United States. Although the individual studies summarized in the report included a variety of compounds with different analytical methods, detection limits, and research objectives, it is clear that some groups of pharmaceuticals tend to be detected more frequently than others in the aquatic environments sampled (including groundwater, surface water, WWTP influents, and WWTP effluents, as well as biosolids and streambed sediments). Sixty-seven compounds were found in wastewater influents, 105 were detected in wastewater effluents, and 22 were present in biosolids. One-hundred twenty-four compounds were detected in surface waters, 35 were detected in groundwater, and 4 were found in bed sediments. Of the 126 compounds measured in at least one matrix, 18, including analgesics, antibiotics, antihyperlipidemics, an antiepileptic, and other compounds such as galaxolide and tonalide, which are used as fragrances in personal-care products, were detected in more than 10 different studies. In another national study by the USGS (Focazio et al. 2008), 25 groundwater and 49 surface-water sites that are sources of drinking water were sampled for the occurrence of 100 targeted chemicals including pharmaceuticals, personal-care products, and other organic wastewater compounds. Forty percent of the 36 pharmaceuticals analyzed were detected at least once, and carbamazepine was the most commonly detected pharmaceutical in both surface water and groundwater.

Phelan and Miller (2010) collected samples from 23 stream sites in Rock Creek National Park (Washington, DC) selected on the basis of warm water thermal infra-red images. The samples were analyzed for a suite of nine human-use pharmaceuticals. Butalbital and oxycodone were detected but concentrations could not be quantified as a result of analytical uncertainties. Although the sources of the warm water were not specifically linked to pharmaceutical detections, they nevertheless indicate the presence of leaking municipal wastewater pipes or other anthropogenic influences on the water.

Loper et al. (2007) present environmental and quality-control data from analyses of water samples from 11 streams and 6 wells in south-central Pennsylvania for 46 pharmaceuticals (31 of which were human and veterinary antibiotics). Five of the streams received municipal wastewater and six received runoff from agricultural areas dominated by animal-feeding operations. For all 11 streams, samples were collected at locations upstream and downstream from the municipal effluents or animal-feeding operations. All six wells were in agricultural settings. For the stream samples, 24 pharmaceuticals (the authors of this report included caffeine and its metabolite as pharmaceutical compounds) were detected at least once. Of the pharmaceuticals detected, 11 were antibiotics. Seventy-eight percent of all detections were in samples collected downstream from municipal-wastewater effluents. The maximum concentrations of compounds other than caffeine in the wastewater effluents include azithromycin (1.65 µg/L; antibiotic), sulfamethoxazole (1.34 µg/L; antibiotic), carbamazepine (0.516 µg/L; antiepileptic), ofloxacin (0.329 µg/L; antibiotic), ibuprofen (0.277 µg/L; nonsteroidal anti-inflammatory), and trimethoprim (0.256 µg/L; antibiotic). Concentrations and detections were much lower in streams receiving runoff from animal-feeding operations than in those receiving municipal-wastewater effluents during baseflow. The pharmaceuticals detected were acetaminophen, carbamazepine, diphenhydramine, oxytetracycline, sulfadimethoxine, sulfamethoxazole, and tylosin. The maximum concentration for all pharmaceuticals was 0.157 µg/L. In samples from wells used to supply livestock, 3 of the 24 pharmaceutical compounds were detected—diphenhydramine, tylosin, and sulfamethoxazole.

There were five detections in all the well samples. The maximum concentration for cotinine detected in well water was 0.024 µg/L (estimated).

Alvarez et al. (2008) used passive samplers to assess the occurrence of a limited number of pharmaceuticals and other wastewater indicator compounds at nine locations in the Shenandoah River Basin and two in the James River Basin, Virginia. Detections included codeine (a narcotic analgesic) and carbamazepine (an anticonvulsant drug), each of which was detected at several sites. Trimethoprim, an antibi¬otic commonly prescribed in tandem with sulfamethoxazole, was detected at 8 of 10 sites; however, sulfamethoxazole was detected only at 1 of these sites. The antidepressant venlafaxine, currently the 13th most prescribed drug in the United States was detected at several sites.

Arikan et al. (2008) collected water samples from 22 stream sites in the Choptank River, Maryland, including 7 sites on the mainstem and 15 on tributaries during baseflow. Samples were collected in all four seasons (April, June, September, and December) in a reconnaissance effort to assess the occurrence of key tetracycline and sulfur classes of antibiotic compounds. Of the sulfonamide class of antibiotics, sulfamethoxazole (19% detection) and sulfadimethoxine (12% detection) were detected at the mainstem stations at maximum concentrations of 0.002 and 0.003 µg/L, respectively. The most frequently detected compounds at the tributary stations were sulfamethoxazole (5% detection) and sulfadimethoxine (14% detection), with maximum concentrations of 0.007 and 0.009 µg/L, respectively. Of the tetracycline class of antibiotics, chlortetracycline (19% detection) and oxytetracycline (15% detection) were the most frequently detected of the tetracycline group of antibiotics at the mainstem stations, with maximum concentrations of 0.034 and 0.047 µg/L, respectively. Tetracycline and doxycycline were detected only once (4% detection) at the mainstem stations, at concentrations of 0.005 and 0.020 µg/L, respectively. Chlortetracycline (21% detection) and oxytetracycline (18% detection) were the most frequently detected tetracycline antibiotics at the tributary stations, with maximum concentrations of 0.180 and 0.084 µg/L, respectively. Tetracycline and doxycycline were detected at 5% of the tributary stations, with maximum concentrations of 0.003 and 0.146 µg/L, respectively. In this limited dataset, seasonal trends were not clear; however, the authors report more samples with detections for antibiotics in December (chlortetracycline was detected in 14 of 19 samples collected in December) than in the other seasons. The highest concentration for all sulfonamide antibiotics was 0.694 µg/L for sulfamerazine in a sample collected in April.

Barnes et al. (2008) reported datasets used in a national reconnaissance of pharmaceuticals and other organic wastewater compounds in untreated sources of drinking water in 25 states throughout the United States, including three states in the Chesapeake Bay watershed (six wells in Pennsylvania, three wells in West Virginia, and four surface-water intakes in Virginia). Results indicated that sulfamethoxazole (antibiotic) was detected in a Pennsylvania well and none of the compounds analyzed for were detected in the West Virginia wells. Sarafloxacin (antibiotic) and carbamazepine (antiepileptic) were each detected once in samples from Virginia surface-water sites.

The USGS is conducting ongoing research throughout the Chesapeake Bay watershed to determine whether fish health is related to chemical exposure. Initial research included the sampling of water and bed sediment at seven active smallmouth bass nesting sites during spawning (Kolpin et al. 2013). During this study, 14 prescription and nonprescription pharmaceuticals (including caffeine and its metabolite as well as cotinine, a metabolite of nicotine) were detected in the water samples collected. The two most frequently detected compounds were caffeine (86%, stimulant) and iso-chlorotetracycline (71%, antibiotic degradate). Other pharmaceuticals detected include acetaminophen (29%, analgesic), azithromycin (14%, antibiotic), carbamazepine (29%, antiepileptic), diphenhydramine (14%, antihistamine), epi-iso-chlorotetracycline (15%, antibiotic degradate), epi-tetracycline (29%, antibiotic degradate), oxytetracycline (43%, antibiotic degradate), sulfamethazine (29%, antibiotic), sulfamethoxazole (29%, antibiotic), sulfathiazole (14%, antibiotic), tetracycline (57%, antibiotic), and tylosin (14%, antibiotic).

Sediment

Very few sediment analyses have been published for samples collected in the Chesapeake Bay watershed; however, Glassmeyer et al. (2008), in a national summary of pharmaceutical and other organic wastewater data, note that whereas the concentrations of pharmaceuticals in liquid effluents typically are in the range of low parts per billion to high parts per trillion (low micrograms per liter (µg/L) to high nanograms per liter (ng/L)), they are found in biosolids at parts per million (milligrams per kilogram (mg/kg)) concentrations. These values reflect, in part, the affinity of many of these compounds to attach to solid particles and the manner in which solid wastes from municipal wastewater-treatment plants and other sources are managed.

Arikan et al. (2008) analyzed bed sediment from four sites on the Choptank River. Chlortetracycline (100% detection) and sulfamethoxazole (75% detection) were the most frequently detected of the antibiotics. The maximum concentrations were 10.0 and 0.15 ppb dry weight, respectively. Sulfamethazine was detected at one site at a concentration of 0.82 ppb dry weight. Chlortetracycline and sulfamethoxazole were also the most frequently detected antibiotics in water samples at these sites. Oxytetracycline, the second most frequently detected compound in water samples from these sites, was not detected in bed-sediment samples. Kolpin et al. (2013) also analyzed bed sediment for a limited number of compounds.

Fish and Wildlife

No fish tissue analyses for pharmaceuticals from the Bay watershed were identified. However, several studies conducted outside the watershed have shown that pharmaceuticals such as antidepressants can be detected in the brain tissue of fish (Schultz et al. 2011) where the fish have been exposed to wastewater effluents in aquatic environments.

Pharmaceutical (Synthetic) Hormones

Much of the environmental research regarding pharmaceutical hormones has focused on the synthetic sex hormones such as ethinyl estradiol, the active ingredient in human birth control pills. Leet et al. (2011) summarized the state of the science on the role of exposure to estrogens and androgens in sexual differentiation during early life stages of fish. Other hormonally active pharmaceuticals such as those used for thyroid-related therapies, corticoid-related therapies, and others are beginning to receive more attention as potential environmental contaminants; however environmentally relevant studies of these other pharmaceutical hormone groups are not common (Kugathas and Sumpter 2011).

Some of the earliest work linking reproductive endocrine disruption effects and potential environmental exposures to pharmaceuticals (ethinyl estradiol and diethylstilbestrol) was demonstrated in Japanese quail (*Coturnix japonica*) by Halldin et al. (1999). The study showed embryonic exposure to these synthetic estrogens can affect sexual differentiation and cause reproductive impairment. Research on synthetic and other exogenous hormones has improved the understanding of the effects of hormones in the environment regardless of whether the hormones are derived from natural sources or are synthetic. Additional research has been performed on other chemicals that are known to interfere with normal hormone-receptor functioning in vertebrates. For example, the effect of embryonic diethylstilbesterol (DES, a synthetic estrogen) exposure demonstrated that estrogenic chemicals affect the differentiation of estrogen target organs (Colborn et al. 1993). Data on androgen- and thyroid-active compounds reveal similar deleterious potential for long-term effects from these compounds through different mechanisms of action (Custer et al. 1999; Chen et al. 2008; Zoeller 2008).

Other research has shown that in some cases exposure to estrogens at environmentally relevant concentrations can result in immunosuppressive effects (Milla et al. 2011). For example, Roberston et al. (2010) have shown that expression of a protein critical to immune suppression (hepcidin) can be disrupted in fish exposed to exogenous estrogens at environmentally relevant concentrations (also see the "Biogenic Hormones" section of this report). Cubero-Leon et al. (2010) have documented that natural (estradiol) as well as synthetic (ethinyl estradiol) estrogen can disrupt serotonin receptor functioning as well as mRNA expression levels of cyclooxygenase (an enzyme critical to production of prostaglandins) in the marine bivalve *Mytilus edulus*. More recently, Kidd et al. (2007) dosed a Canadian lake with environmentally relevant concentrations (i.e., concentrations detected by other studies in environmental waters) of synthetic estrogen (17α-ethinyl estradiol) over a 3-year period. Population-level effects including intersex and severe reproductive failure were observed in fathead minnows (*Pimephales promelas*) from the dosed lake but not in those from two control lakes. Populations returned to normal when the contaminant dosing was discontinued. Other studies have shown that total estrogenicity of common mixtures of contaminants (composed of natural, pharmaceutical, and other hormonally active compounds) such as those commonly found in municipal wastewater effluents can cause intersex in exposed fish (Vajda et al. 2011). Critically, once the wastewater-treatment plant was updated with treatment technologies capable of removing these estrogenic compounds, the intersex conditions in fish were no longer evident (Barber et al. 2012).

Pharmaceutical Antidepressants

Schultz et al. (2011) exposed fathead minnows to environmentally relevant concentrations of antidepressants (bupropion, fluoxetine, sertraline, and venlafaxine) and observed anatomical and physiological effects, including the presence of intersex biomarkers such as elevated plasma vitellogenin in male fish as well as severe effects on male secondary sexual characteristics. Some exposures to venlafaxine (305 ng/L) and sertraline (5.2 ng/L) resulted in mortality. Painter et al. (2009) showed that predator avoidance behaviors were adversely affected in larval fathead minnows exposed to these antidepressants at environmentally relevant concentrations. Avoidance success decreased significantly in individuals exposed to these pharmaceuticals, thus potentially compromising survival and reproductive fitness.

Pharmaceutical Antibiotics

It is well known that antibiotic therapies can create antibiotic resistance in clinical settings as a result of repeated exposures of bacteria to individual antibiotics. The potential for antibiotics that have been released to the environment to enhance resistance patterns in receptor microbial communities is not well understood; however, recent research has focused attention on this topic. For example, D'Costa et al. (2011) discuss potential effects on resistance patterns of indigenous soil microbial communities exposed to antibiotics, and Duriez and Topp (2007) explore various manure management techniques at swine farms and their influence on antibiotic resistance patterns in the manure. Haack et al. (2012) showed that microbial community composition and structure can be affected by environmentally relevant exposures to an antibiotic (sulfamethoxazole). These investigators also noted that ecological function could be affected at concentrations two to three orders of magnitude smaller than those used in clinical applications and could promote antibiotic resistance through the selection of naturally resistant bacteria. In a related experiment, Underwood et al. (2011) showed that sulfamethoxazole exposures at environmentally relevant concentrations can affect the denitrifying activity of indigenous soil microbial populations in an aquifer that previously received wastewater effluents. LaPara et al. (2011) have identified tertiary treated municipal wastewater effluents to be significant point sources of antibiotic resistance genes to receiving surface waters in Minnesota. In another example of pharmaceutical antibiotic effects in the environment, Ebert et al. (2011) researched potential risks to photoautotrophic aquatic organisms (an alga, a cyanobacterium, and two macrophytes) resulting from exposure of fluroquinolone antibiotics (ciprofloxacin, enrofloxacin). The study identified risks (growth inhibition effects) to two of the test species at environmentally relevant concentrations of ciprofloxacin (risks were not identified at these concentrations for enrofloxacin).

Conclusions

Existing data documenting the environmental occurrence of pharmaceuticals in the Chesapeake Bay watershed are limited so the extent and severity is identified as uncertain. However the limited data indicate a potentially wide extent of some pharmaceuticals for several reasons: 1) the sources of pharmaceuticals, which include human and animal waste management operations such as municipal wastewater treatment effluents and animal agriculture, are widely distributed throughout the watershed; 2) the few studies that have been completed in the watershed have detected some pharmaceutical and related indicator chemicals in some streams, groundwater, and streambed-sediment; and 3) other studies conducted across the United States and elsewhere consistently point to a wide extent of pharmaceuticals wherever source pathways to aquatic environments exist. The severity of pharmaceuticals as environmental contaminants is the subject of active research and very few studies have linked pharmaceutical exposures to individual or population-level mortality. However several studies in the U.S. and elsewhere have linked some of these chemicals to adverse sublethal effects such as predator avoidance behavior and incidence of intersex in a range of exposed organisms at environmentally relevant concentrations. In summary, sources of pharmaceuticals are fairly extensive in the watershed and studies on effects are increasing throughout the U.S. and therefore pharmaceuticals are important toxic contaminants of concern. However, data gaps and lack of complete understanding of potential adverse impacts continue to limit broader conclusions regarding extent and severity throughout the Chesapeake Bay watershed.

2.7 Household and Personal Care Products

Abstract

Household and personal-care products (HPCPs) represent a wide range of organic chemicals that are used frequently in residences, workplaces, and other locations. Used in a variety of applications such as cosmetics, detergents, soaps, pest control, and food additives, these products are often formulated as a mixture of chemicals (including inorganic and organic active and inert ingredients). These contaminants can enter the environment in a number of ways but the predominant pathways are related to management and disposal of residential and human wastes such as landfills, on-site septic and municipal wastewater treatment plants. Findings included in the few local and regional occurrence datasets in the Chesapeake Bay watershed tend to mirror national studies of these compounds as potential environmental contaminants. For example, the compounds often detected in the environment include N,N-diethyltoluamide (insect repellant), surfactants (i.e. alkyl phenols), triclosan (an antimicrobial), and musks (tonalide, galaxolide). The environmental health implications of the presence of these contaminants in the environment are poorly understood; however, it is clear that: 1) these compounds often occur in complex mixtures; 2) the concentrations detected in environmental settings tend to be low as compared to the few existing health-based benchmarks; 3) some studies indicate that targeted compounds can produce estrogenic and other sublethal effects in exposed organisms (e.g. some surfactants, fragrances or musks, triclosan and triclocarban); and 4) due to the fact that sources of HPCPs are widely distributed throughout the built environment, it is reasonable to conclude that these compounds have the potential to occur extensively in receiving waters throughout the Chesapeake Bay watershed. Because of limited monitoring data and incomplete understandings of sublethal and other effects, extent and severity of HPCPs are identified as uncertain.

Background

HPCPs include a wide range of inorganic as well as organic chemicals that are used frequently in residences, workplaces, and other locations. These products, such as cosmetics, detergents, soaps, and food additives, are typically formulated as a mixture of ingredients. For example, some soaps and toothpastes include antimicrobial compounds in their formulation (e.g., triclosan and triclocarban); some cosmetics include compounds commonly known as musks that provide fragrance (galaxolide and tonalide); detergents and cleaning products consist of several ingredients, including surfactants (nonylphenol ethoxylates, alkylphenol ethoxylates, and octylphenol ethoxylates); and other organic compounds (e.g., menthol) are used to enhance flavors in a range of products.

Chemical constituents in HPCPs can enter the environment through a variety of pathways. Solid forms of these products are commonly placed in trash receptacles destined for landfills, whereas liquid forms are flushed in toilets or poured into sink drains. Therefore, most frequent source pathways to the environment are associated directly or indirectly with human-waste management activities such as municipal wastewater-treatment facility effluent discharges, septic tanks, and landfill leachates (Glassmeyer et al. 2008; Barnes et al. 2008). Chemical constituents associated with HPCPs can have a variety of sources and therefore are not always associated with household wastes (Phillips et al. 2012).

Water and Sediment

These chemical constituents associated with HPCPs could derive from a variety of waste sources. Therefore, the analytes listed in this section are referred to as "indicators" of HPCPs when detected in the environment. Water quality standards generally do not exist for all chemicals that are indicators of HPCPs; therefore, these analytes are not commonly monitored in the Chesapeake Bay watershed. Several studies conducted throughout the United States and elsewhere, however, have provided monitoring data and associated ecological impacts (Vajda et al. 2011) and perspective on the few local data that are available. Most of the environmental data for these chemicals are for concentrations in water and some sediment samples. Tissue analyses are not currently available.

Kolpin et al. (2002) were among the first to document the widespread occurrence of chemical indicators of HPCPs in streams in the United States. Glassmeyer et al. (2008) reviewed and summarized the available literature on the occurrence of pharmaceuticals and chemical indicators of HPCPs in the environment worldwide and showed that 124 different compounds were detected in surface waters, 35 were detected in groundwater, and 4 were detected in bed sediments. Of the 126 compounds measured in at least one matrix, 18, including galaxolide and tonalide (which are used as fragrances in personal care products), were detected in more than 10 different studies.

Halden and Paull (2005) documented widespread occurrence of triclosan and triclocarban (antimicrobials) in water resources of the greater Baltimore area. Results of this study were compared to other locations throughout the United States and the authors concluded that these contaminants are widely distributed and frequently occurring. Elevated concentrations were not entirely due to incomplete removal of contaminants during wastewater treatment as maximum concentrations (triclocarban, 6.75 µg/L; and triclosan, 1.6 µg/L) in the Baltimore area were due to spills and leakage.

Phelan and Miller (2010) reported results from base-flow, stormwater, and bed-sediment synoptic sampling of 23 sites in Rock Creek Park, Washington, DC. Of the indicators of HPCPs analyzed for, N,N-diethyltoluamide (insect repellant applied topically to clothing and skin), organophosphate, flame retardants, and galaxolide (musk) were detected at more than half the sites. Other indicators of HPCPs detected in water samples included alky phenol surfactants, triclosan (antimicrobial additive in soaps), and tonalide (musk). Compounds detected in bed-sediment samples include N,N-diethyltoluamide, galaxolide, and surfactants.

Alvarez et al. (2008) used passive samplers to assess the occurrence of a limited number of pharmaceuticals and other wastewater indicator compounds at nine locations in the Shenandoah River Basin and two in the James River Basin, Virginia. The objective of this study was to test the general hypothesis that declining fish health is linked to stream chemistry, but not to determine possible sources or source pathways. Detections of indicators of HPCPs included celestolide, galaxolide, phantolide, tonalide, and traseolide (musks used in a variety of cosmetics and other personal care products), N,N-diethyltoluamide, as well as several phosphate-based flame retardants used in a variety of consumer products such as clothes and furniture.

Barnes et al. (2008) reported datasets used in a national reconnaissance of pharmaceuticals and other organic wastewater compounds in untreated sources of drinking water in 25 states throughout the United States. Sites in the Chesapeake Bay watershed were located in Virginia, Maryland, and Pennsylvania. Surfactants that are common in several types of detergents and cleaning products were detected at several sites. Other indicators of HPCPs detected included triclosan, menthol, and N,N- diethyltoluamide.

Reif et al. (2012) conducted reconnaissance sampling from 2006 to 2009 in selected locations across Pennsylvania to identify contaminants of emerging concern in (1) groundwater from wells used to supply livestock, (2) streamwater upstream and downstream from animal feeding operations, (3) streamwater upstream from and streamwater and streambed sediment downstream from municipal wastewater effluent discharges, (4) streamwater from sites within 5 miles of drinking-water intakes, and (5) streamwater and streambed sediment where fish health assessments were conducted. Analytes included a range of indicators of HPCPs.

Kolpin et al. (2013) analyzed discrete (grab samples) and integrated (passive samplers) water samples as well as bed sediment from smallmouth bass nesting sites in the Potomac River Basin. Chemical indicators of HPCPs detected in water samples include a range of surfactants, camphor, galaxolide, N,N-diethyltoluamide, tonalide, and triclosan.

Fish and Wildlife

Residues of HPCPs have yet to be reported in tissues of wildlife (amphibians, reptiles, birds, and mammals) in Chesapeake Bay. Ramirez et al. (2009) conducted a survey of five wastewater-effluent-dominated streams across the United States and analyzed fish tissue for a range of pharmaceuticals and HPCPs. Galaxolide and tonalide (musks) and triclosan were among the compounds detected.

Perfluorinated compounds are surface protectors and surfactants that are distributed worldwide. Perfluorooctanoic acid, an active ingredient of Scotch Guard®, was voluntarily removed from the marketplace in 2000 because it was increasingly being detected in the environment, wildlife, and people. Several perfluorinated compounds were detected in osprey eggs collected in the Chesapeake Bay watershed in 2000 and 2001, with upper extreme concentrations of 428 ppb wet weight for perfluorooctanesulfonate, 671 ppb for perfluorordecanesulfonate, 13.7 ppb for perfluorodecanoic acid, and 27.1 ppb for perfluoroundecanoic acid (Rattner et al. 2004). Recent controlled exposure studies and risk assessments in Northern bobwhite (*Colinus virginianus*) and mallards concluded that environmental exposure to perfluorooctanesulfonate does not pose a significant risk to avian populations (Newsted et al. 2005).

A series of alkylphenol and ethoxylate surfactants were also analyzed in 15 osprey eggs collected from the Chesapeake Bay watershed in 2000 and 2001. Nonylphenol was detected in all egg samples, with a maximum concentration of 16.7 ppb wet weight. Nonylphenol ethoxylates, octylphenol, and octylphenol ethoxylates were generally not detected, with the exception of octylphenol ethoxylate in 1 of 15 eggs at a concentration of 14.6 ppb.

Lozano et al. (2012) documented seasonal variations in concentrations of alkylphenol and alkylphenol ethoxylate surfactants in tissue samples collected from largemouth bass living downstream from municipal wastewater-treatment plants in Chicago, IL. In comparison, the tissue concentrations of other persistent organic compounds such as PCBs, polybrominated diphenyl ethers (PBDEs), and some pesticides such as DDT, did not vary over time. Although these analyses were not done in the Chesapeake Bay watershed, the contaminants associated with wastewater (e.g., surfactants) are similar.

The toxicological effects of HPCPs in the environment are an area of active research. Some compounds, such as the antimicrobials triclosan and triclocarban, have been shown to have sublethal effects at environmentally relevant concentrations ranging from stimulation of embryo production in freshwater snails (*Potamopyrgus antipodaru*) (Guidice and Young 2009) to disruption of thyroid homeostasis (Dann and Hontela 2010). Dann and Hontela (2010) also point out that mammalian toxicity studies of triclosan have shown that it is not acutely toxic, carcinogenic, mutagenic, or a developmental toxicant; however, they also note that some aquatic species such as algae, invertebrates, and some fish are sensitive to triclosan. Bedoux et al. (2012) reviewed the literature on triclosan toxicity and environmental occurrence and showed that benchmarks such as EC50 generally were much lower (µg/L or lower) in plants than in animals, with toxicity to animals well above most environmentally relevant concentrations. On the other hand, some indigenous soil bacteria and aquatic algal species did exhibit sublethal and other effects (e.g., inhibition of soil microbial respiration) at environmentally relevant concentrations. Evidence of the biological effects of HPCPs in the environment can be found for other contaminants as well. For example, surfactants have been identified as contributing to the estrogenicity of wastewater effluent and endocrine disruption in Boulder, Colorado (Vajda et al. 2011) and Lozano et al. (2012) showed that APE surfactants can elevate plasma vitellogenin in male largemouth bass living in streams receiving wastewater effluents. Breitholtz et al (2003) concluded that synthetic musks are not likely to adversely impact copepods at environmentally relevant concentrations.

However, Carlsson and Norrgren (2004) studied the toxicity of synthetic musks to early life stages of zebrafish (*Danio rerio*) and concluded that some musks can have adverse impacts on wild fish at environmentally relevant concentrations.

Conclusions

HPCPs represent a group of contaminants with many potential uses and a diverse array of chemicals. Many studies throughout the world have shown these contaminants to be associated with a wide range of sources such as municipal wastewater effluents, on-site septic tanks, and other waste management operations (e.g. landfills). Therefore, although the existing occurrence data are limited in the watershed, the sources such as wastewater effluents are widespread indicating a potential for some of the commonly used chemicals within this group to occur in many locations throughout the watershed. Significant gaps exist in understanding the range of potential adverse ecological effects of HPCPs, their degradates and mixtures, at environmentally relevant concentrations thereby limiting broader conclusions about their severity in the Chesapeake Bay watershed.

2.8 Polybrominated Diphenyl Ether Flame Retardants

Abstract

Polybrominated diphenyl ethers (PBDEs) have been used as flame retardants in a range of consumer products since the 1970s. As a contaminant group, PBDEs include more than 200 potential congeners, making this one of the most extensive groups in this report in terms of total number of individual chemicals. Because PBDE congeners are highly hydrophobic, they are more likely to be found in sediments and tissue than in the water column. PBDEs are known to bioaccumulate; therefore, environmental monitoring for PBDEs has focused on animal tissue (e.g., fish, shellfish, and a range of terrestrial animals) in the Chesapeake Bay watershed and around the world, including remote regions of the Arctic. Because of their documented persistence and tendency to bioaccumulate, along with concerns about potential toxicity, certain penta- and deca-BDE formulations were phased out in 2004 and deca-BDE formulations will be phased out by the end of 2013. Also known to exist in coastal marine sediments globally, PBDEs are well documented in terms of their extent elsewhere. and therefore have the potential to be widespread in Chesapeake Bay. On the other hand, the potential severity of toxicological and ecological effects of PBDEs as environmental contaminants are less well understood. Currently there are no fish consumption advisories for PBDEs in the Chesapeake Bay watershed; however, adverse sublethal effects at environmentally relevant concentrations have been documented

in a limited number of test organisms. Because of limited monitoring data and incomplete understandings of sublethal and other effects, extent and severity of PBDEs are identified as uncertain.

Background

Polybrominated diphenyl ether (PBDE) flame retardants have been commonly used as fire retardants in textiles, electronics, polymers, and other materials since the 1970s. Although the deca-BDE mixture has been produced in the greatest volumes, the demand for penta-BDE in North America has exceeded that in other markets by an order of magnitude (Hale et al. 2006). Pathways for PBDEs to enter the environment are poorly understood and quantified. Because PBDEs are chemically incorporated into consumer products, the most likely environmental pathways include air emissions from manufacturing and product usage. Hale et al. (2002) suggest that these compounds enter the environment as polyurethane foams, disintegrate, and release congeners in the resulting fragments.

Because PBDEs are hydrophobic and lipophilic, the majority of environmental occurrence studies for PBDEs are focused on animal tissue and soils or bed sediment. Hale et al. (2002) found elevated concentrations of PBDEs in soils, bed sediment, and bluegill tissues downwind and downstream from a polyurethane foam manufacturing plant in the Roanoke River watershed (outside the Chesapeake Bay watershed). Due to their persistence, bioaccumulation and potential toxicity, USEPA and manufacturers reached agreement to phase out the use of certain penta- and octa-BDE formulations by 2004, and more recently agreed to phase out the use of the deca-BDE formulation by the end of 2013 (USEPA 2012d).

Water and Sediment

Because PBDEs have low solubility and there are no regulatory standards for PBDEs, no water-column data for these compounds were identified in the literature or found in any of the data searched for this report.

Kimbrough et al. (2009) measured concentrations of PBDEs (mono- through hepta- congeners) in paired sediment and oyster tissues (see below) collected at the NOAA nationwide Mussel Watch stations from 2004 to 2007. The Chesapeake Bay was placed in the lowest of three clusters (low, medium, and high) for paired sediment and tissue concentration data. In the six Maryland stations in the Bay, four had no detectable PBDE concentrations in sediments; the other two were designated as medium concentrations of 0.1 and 0.3 ppb dry weight. In the four Virginia samples, two were non-detects one had 0.1 ppb, and one had 0.4 ppb (both designated as medium concentrations).

Fish and Shellfish

According to the ATSDR (2004), the main source of human exposure to PBDEs may be through foods with high fat content, such as fatty fish. Some lower brominated PBDEs have been detected in air samples, indicating that people can also be exposed by inhalation. The ATSDR (2004) public health statement notes that rats and mice exhibited thyroid effects after consuming food with moderate amounts of lower brominated PBDEs for short periods. Rats and mice that ate smaller amounts over longer time periods (weeks, months) had liver and thyroid effects. The statement speculates that the thyroid effects are specific to the test animals and may not be relevant for humans. Understanding of the carcinogenicity of PBDEs is incomplete although USEPA lists decaBDE as a possible human carcinogen. Birth defects have not currently been linked to PBDE exposures but research is on reproductive effects is ongoing.

There are no fish tissue advisories for PBDEs in the Chesapeake Bay watershed. PBDE concentrations were included as part of monitoring conducted to update fish tissue advisories for District of Columbia (Pinkney 2009). In 2007, samples of the following species were collected from the Potomac and Anacostia Rivers: sunfish, largemouth bass, carp, blue catfish, channel catfish, and American eel. Total PBDE concentrations were compared with the Virginia Department of Health (VDH) Guidance Value of 5 ppm (R. Tripathi, VDH, personal communication) because no USEPA fish-tissue screening value exists for PBDEs. The maximum concentration was 0.136 ppm in a channel catfish sample. No concentration in any of the other samples exceeded 0.1 ppm.

Klosterhaus and Baker (2010) examined the bioavailability of decabromdiphenyl ether (BDE 209) to the marine polychaete worm, *Nereis virens*. Bioaccumulation of this compound was minimal but uptake of two degradation products (BDE 207 and 208) did occur. Klosterhaus et al. (2011) reported much higher bioaccumulation of penta-BDE, with biota-sediment accumulation factors as high as 0.6 compared with of 0.0003 for BDE 209. This is consistent with studies showing tissue residues of tetra- and penta-BDEs in wildlife.

In their review of the literature on the toxicity of PBDEs, Wenning et al. (2011) stated that PBDEs are expected to be toxic to aquatic organism but dose-response relations have not been developed. Recent studies of single PBDE compounds in the laboratory demonstrate a range of sublethal effects. For example, Kuiper et al. (2008) exposed zebrafish (*Danio rerio*) to a purified pentabromodiphenylether (congener BDE-71) in water in a 30-day exposure that focused on egg production and fertilization. Eggs were hatched and exposure continued until 45 days post-hatch. The authors also measured whole-body BDE-71 concentrations in adults and juveniles. They found that, although larval survival was significantly reduced, the adult tissue concentration associated with the exposure (126 ppm wet weight) was more than 55 times the highest reported environmental concentrations in wild fish (burbot, *Lota lota* in a Norwegian lake at 2.27 ppm wet weight). In contrast, Chou et al. (2010) reported adverse effects on swimming speed and level of activity in juvenile zebrafish exposed to the pentabromodiphenylether BDE-47 from day 21 through day 54 post-hatch. They stated that these effects occurred at concentrations approaching those reported in environmental samples of wild fish.

As stated above, Kimbrough et al. (2009) measured concentrations of PBDEs (mono- through hepta- congeners) in paired sediment and oyster tissues collected at NOAA's nationwide Mussel Watch stations from 2004 to 2007. The six Maryland stations were designated as having medium concentrations in oyster tissue (7.1–24.1 ppb total PBDEs). All five Virginia sites had medium concentrations ranging from 8.5 to 14.8 ppb. Kimbrough et al. (2009) concluded that the toxicity and ecosystem effects of PBDEs on marine biota have not been well studied.

Wildlife

In osprey eggs collected in 2000 and 2001, BDE congeners 47, 99, 100, 153, and 154 were detected in all samples from Chesapeake Bay urbanized areas and the South, West, and Rhode Rivers reference sites near Annapolis, Maryland (Rattner et al. 2004). Total PBDE concentrations ranged up to 928 ppb wet weight (some of the greatest concentrations reported in bird eggs to date), and approach the lowest-adverse-effects-level of 1,800 ppb eggs for pipping and hatching success derived in American kestrels (McKernan et al. 2009). Tetra- and penta-BDE congeners typically dominate eggs of piscivorous birds. In a collection of addled peregrine falcon eggs from 13 nests in the Chesapeake Bay watershed (Potter et al. 2009), total PBDE concentrations ranged from 32.8 to 354 ppb wet weight. The deca-BDE congener 209 was detected at concentrations ranging up to 48.2 ppb, and levels of this congener were found to be positively correlated with human population density in proximity to the nests (Potter et al. 2009). Polybrominated biphenyl congener 153 was also quantified in many of the peregrine eggs, ranging from 5.88 to 51.8 ppb wet weight, although toxicity thresholds in avian eggs are unknown. In 2010, six common tern eggs collected from Poplar Island were analyzed for PBDEs. These eggs contained low levels of BDE congeners (total PBDE ranged from 10.2 to 51.0 ppb wet weight), making them ideal for their intended use in a controlled exposure penta-BDE egg injection (Rattner et al. 2011).

Congeners of PBDEs detected in penta- and octa-BDE formulations bioaccumulate and biomagnify in food chains (de Wit 2002), and detailed investigations in captive and free-ranging birds have documented developmental, endocrine, immunologic, and reproductive effects at environmentally relevant concentrations (Chen and Hale 2010).

Conclusions

Limited data suggests that PBDEs occur in the tissue of aquatic and terrestrial organisms and in sediment; however, the amount of data is not sufficient to determine extent of occurrence in the Bay. It is notable that recent research on the adverse effects of PBDEs is focusing on specific congeners and results are varied. Research on sublethal effects would inform broader conclusions as to their severity as potential environmental contaminants in the Chesapeake Bay watershed. Because of limited monitoring data and incomplete understandings of sublethal and other effects, extent and severity of PBDEs are identified as uncertain.

2.9 Biogenic Hormones

Abstract

By definition, biogenic hormones are created, biologically processed, and excreted by humans and other organisms. Therefore, there is potential widespread extent of occurrence of some biogenic hormones in the environment. Recent monitoring studies have focused on natural and synthetic estrogens as well as several other chemicals that are known or suspected to interfere with normal estrogen receptor functioning in a range of organisms. Monitoring results have shown that municipal wastewater effluents as well as animal agricultural activities can be major pathways for biogenic hormones to reach the environment. Other studies have shown that exposures to these environmental hormones cause intersex and other abnormalities such as immune suppression in fish and other species. Although these compounds do not have immediate lethal effects at environmentally relevant concentrations, they may have more subtle effects on developmental and maturational processes and may diminish individual fitness and reproductive life span. These key sublethal effects are typically difficult to detect in wild populations and present an insidious challenge to research on the health of fish and wildlife. Currently (2012), the weight of evidence indicates that environmental exposures to biogenic

hormones can cause adverse effects to some receptor organisms. The occurrence data on biogenic hormones in water and sediment is presently limited to a few focused studies and is identified as uncertain. Conclusions about the severity of biogenic hormones in the Chesapeake Bay watershed are limited by the available data and research. Because of limited monitoring data and incomplete understandings of sublethal and other effects, extent and severity of biogenic hormones are identified as uncertain.

Background

The presence of natural hormones in the environment, a result of animal and plant waste excretion and other natural biological processes, can be anthropogenically enhanced by various waste-management practices. As a compound group, natural hormones are not commonly monitored by regulatory agencies as contaminants; however, the combination of potentially large quantities of these compounds reaching the environment as a result of waste-management activities and the fact that they are biologically active at low concentrations has increased research attention on these compounds as potential environmental contaminants.

All multicellular organisms including plants and animals produce hormones. Hormones are required for a variety of biological functions and include, for example, estrogen and androgen, which are critical for normal sexual reproduction in vertebrate animals. Other examples of animal hormones include tyrosine-based hormones associated with thyroid functioning and corticosteroids produced in the adrenal cortex and associated with a range of stress and immune responses and metabolic processes. Phytoestrogens are produced by plants and, although they are weakly estrogenic compared to animal estrogens, they are produced in abundance, and their environmental pathways also are affected by waste-management activities.

This section of the report focuses on naturally produced hormones because of current related research on fish health and intersex in the Chesapeake Bay watershed (Ciparis et al. 2011; Blazer et al. 2012; Kolpin et al. 2013) and elsewhere in the world including studies that have linked estrogenic contaminants and intersex in fish (Vajda et al. 2011). Current research is focused on the naturally occurring steroidal hormones as well as other environmental contaminants that bind to or otherwise affect estrogen and androgen receptors; however, there is a growing list of other chemical compounds (e.g. some personal care products, pharmaceuticals, etc.) that are known or suspected to interact with hormone receptor signaling pathways. These other compounds are not considered in this section of the report, but are discussed in other sections.

Because these compounds are key components of all animal and plant life, their environmental sources are ubiquitous. Some management activities associated with human, animal, and plant wastes and associated biological material can be key point and(or) nonpoint source pathways for hormones in the environment. Municipal wastewater treatment plants are centralized locations where human wastes (and associated hormones) are treated before being discharged to the environment. These treatment systems are not specifically designed to remove hormones and consequently some municipal wastewater effluents are potential point sources of natural hormones to the environment. Consequently successful removal (or biochemical transformation) of parent hormone compounds as they pass through existing wastewater treatment plants can be highly variable (Chimchirian et al. 2007). Effluent from WWTPs can be a significant source of natural estrogens from human excretion to surface waters (Lagana et al. 2004; Muller et al. 2008; Petrovic et al. 2002; Ying et al. 2008; Phillips et al. 2012). Finally, because sewersheds can encompass large land areas and various sources, the treatment plants can become central locations where hormone-laden wastes accumulate and potentially become concentrated before release to the environment.

Other sources include land uses and related operations where animal manure is created and accumulated (e.g. lagoons at livestock feeding operations) and inadvertently (or intentionally) released to the environment as point sources. Rangeland or pasture grazing of livestock can be another source of hormones to the environment especially where livestock have direct access to streams or other water bodies (Kolodziej and Sedlak 2007). Other agricultural activities such as where animal manures are spread on cropland as a source of soil fertility, biosolid uses, and compost operations accumulate animal and plant wastes and eventually release them to the environment in various forms and conditions of decomposition. The latter are important nonpoint sources of hormones to surface and groundwater during runoff and leaching events (Finlay-Moore et al. 2000; Kjaer et al. 2007; Matthiessen et al. 2006; Shore et al. 1995).

Metabolic processes associated with plants and animals produce a range of parent and conjugated forms of hormones. Therefore, hormones excreted from animals can be in the original parent form as well as a range of conjugated forms such as glucoronides and sulfur compounds, which in turn are subject to further transformation processes. Where environmental conditions are amenable, the conjugated forms can be cleaved to the parent compound and other processes can produce the conjugated forms again. Several studies have documented concentrations of parent steroids as well as their glucoronide and sulfate conjugated metabolites in livestock manures and municipal biosolids destined for land applications (Hanselman et al. 2003; Andaluri et al. 2012). The glucoronide metabolites have been shown to deconjugate back to the parent steroid in the environment (Ternes et al. 1999; Panter et al. 1999). In contrast, current evidence on sulfate conjugates indicates they are much more resistant to transformation (Johnson and Williams 2004; D'Ascenzo et al. 2003). Hutchins et al. (2007) analyzed samples from several different lagoons at confined animal-feeding operations (CAFOs) and concluded that estrogen conjugates contribute to the overall estrogen load. Therefore, fate and transport studies of natural plant and animal hormones must account for the parent as well as the conjugated forms, especially when their role as hormonally active environmental contaminants is being investigated.

Hanselman et al. (2003) completed a literature review to assess the current state of science regarding estrogen physicochemical properties, livestock excretion, and the fate of manure-borne estrogens in the environment. Unconjugated steroidal estrogens have low solubility in water (0.8-13.3 (mg/L) and are moderately hydrophobic (log K_{ow} 2.6-4.0). Cattle excrete mostly 17α-estradiol, 17β-estradiol, estrone, and respective sulfated and glucuronidated counterparts, whereas swine and poultry excrete mostly 17β-estradiol, estrone, estriol, and respective sulfated and glucuronidated counterparts.

Khanal et al. (2006) reviewed the literature on the fate, transport, and biodegradation of natural estrogens in the environment. Although the environmental fate of estrogens is not clearly known, Hanselman et al. (2003) summarized the results of laboratory-based studies that found the biological activity of these compounds is greatly reduced or eliminated within several hours to days as a result of degradation and sorption. Bradley et al. (2009) studied the biodegradation of 17β-estradiol, estrone, and testosterone in three streams in the United States that receive wastewater-effluent effluents. The results showed that biodegradation of these hormones in conjunction with sorption to bed sediments can be mechanisms for the attenuation of hormonal endocrine disruptors in effluent-affected streams. Gray and Sedlak (2005) showed that constructed wetlands function to remove or transform estrogenic contaminants. Approximately 36% of the 17β-estradiol was attenuated, with the most likely cause being sorption to hydrophobic surfaces in the wetland coupled with biotransformation. However, the attenuation processes in these studies were still not sufficient to completely mineralize or sequester these compounds. Other studies (Khanal et al. 2006) have demonstrated that estrogens are sufficiently mobile and persistent to affect surface water and groundwater quality and, therefore, aquatic organisms can be exposed to these hormones even in locations that are removed from their sources or pathways to the environment.

Khanal et al. (2006) found that conventional wastewater treatment is efficient in the removal of 17β-estradiol, but estrone removal is relatively poor. The removal occurs mainly through sorption by sludge and subsequent biodegradation. Barber et al. (2012) showed that estrogenic compounds were removed from treated wastewater by upgrading a wastewater-treatment plant from a trickling-filter/solids-contact process to an activated-sludge process. The removal was in large part, the result of increases in hydraulic retention time and solids retention time. Before the upgrade, 17β-estradiol was partially removed by the trickling filter, whereas estrone concentrations actually increased in the effluent, likely as a result of biologically mediated oxidation of 17β-estradiol. Concentrations of estriol, 17β-estradiol, and estrone in treated effluent decreased to below detection levels after the upgrade.

Studies and monitoring data on steroidal hormones in the Chesapeake Bay watershed are limited. A few example studies conducted by Federal agencies and academia are cited below; however, available information is insufficient to warrant individual subsections on water, sediment, and tissue.

Water and Sediment

Many studies have documented the presence of steroidal hormones in aquatic environments (Kolpin et al. 2002). Most of these studies have focused on dissolved constituents in the water column, with fewer studies of those in bed sediment. Kinney et al. (2006) analyzed biosolids produced from wastewater-treatment-plant sources and detected several steroidal hormones including 3-β-coprostanol, cholesterol, β-sitosterol, and stigmastanol at high concentrations relative to the concentrations of other organic compounds detected. These commercially available biosolids are commonly applied to land surfaces as soil amendments and are therefore potential source pathways for steroidal hormones to the environment. Data on phytoestrogen occurrence in aquatic environments is much more limited; however, Kolpin et al. (2010) analyzed water from 15 streams draining agricultural land in Iowa for a range of phytoestrogens. Target compounds that were frequently detected included formononetin (80%), equol (45%), daidzein (32%), biochanin A (23%), and genistein (11%).

Reif et al. (2012) analyzed 270 streamwater samples collected near drinking-water intakes in Pennsylvania for a range of biogenic hormones. Six of the 17 hormones and 2 of the animal sterol compounds were detected at least once. The most frequently detected hormones were estrone (18% of samples; maximum concentration 3.1 ng/L), cis-androsterone (5% of samples; maximum concentration 6.2 ng/L), and 4-androstene-3,17-dione (3% of samples; maximum concentration 1.8 ng/L). All other hormones were detected in fewer than 1 percent of samples. Only 1.5% of all 5,130 analyses resulted in hormone detections. Dorabawila and Gupta (2005) documented the presence of estradiol (17β-estradiol) in water samples from the Wicomico, Manokin, and Pocomoke Rivers with highest concentrations downstream from sewage-treatment plants.

Ciparis et al. (2012) sampled tributaries in the Shenandoah River watershed during three different seasons and analyzed the water by using an estrogen assay. The bioluminescent yeast estrogen screen (BLYES) indicates the presence of compounds that can bind to the estrogen receptor. In this way, BLYES is an indicator of "total estrogenicity" of the water and includes activities associated with all natural and synthetic estrogens or other chemicals that are estrogen agonists or antagonists. Consequently results of these assays cannot entirely be attributed to biogenic hormones; however, for simplicity the total concentration of estrogenic compounds in the water samples is reported relative to 17β-estradiol (E2) as E2 equivalents (E2Eq). Concentrations in 18 samples from 10 sites were >1 ng/L E2Eq, and animal feeding operation (AFO) densities in the watersheds of most of these sites were >1 per 1,000 acres. An E2Eq > 1 ng/L was the predicted no-effect concentration of total estrogens on fish reproduction. This study documented statistically significant (R^2=0.39-0.75) relations between watershed densities of AFOs and in-stream concentrations of E2Eq. The range in estrogenic activity observed during this study was similar to estrogen concentrations and total activity measured in other studies across the United States, including (1) streams with concentrated sources, such as streams draining fields that receive wastes from poultry AFOs; (2) streams adjacent to pastures receiving waste from dairy and beef AFOs; and (3) rangeland streams directly accessed by grazing cattle. A similar range in estrogenic activity has been measured in treated WWTP effluent (Salste et al. 2007) and in rivers receiving effluent from WWTPs (Vermeirssen et al. 2005).

Arikan et al. (2008) analyzed 26 water samples from seven river stations and 56 water samples from 15 subwatershed stations in the Choptank River Basin for several biogenic hormones. Estriol (12 ng/L) and estrone (13 ng/L) were each detected once in subwatersheds, and progesterone was detected in three different subwatersheds (12, 12, and 14 ng/L). The synthetic estrogen 17α-ethinylestradiol was also detected in one subwatershed water sample. Testosterone (16 ng/L) was detected once at a river station. One other hormone (17β-estradiol) was analyzed for but not detected in any water sample.

As part of a national AFO study, the USGS has monitored runoff from streams and ditches draining a pastured cattle operation in the Rappahannock River Basin. Samples were collected during a range of high and low flows by using both grab samples and passive samplers. Preliminary results indicate detections of steroidal hormones in water and bed sediment, including cholesterol, cis-androsterone, coprostanol, estrone, epitestosterone, testosterone, 4-adrostene-3, 17-dione, 17α-estradiol, and 17β-estradiol (D.W. Kolpin, USGS, personal communication).

In a continuing investigation regarding the potential connections between contaminants and fish health and intersex in the Chesapeake Bay watershed, the USGS collected samples of water and bed sediment at six active smallmouth bass nesting stream sites and one control site during spawning (Kolpin et al. 2013). Although hormone analyses in this study have been limited, the steroidal hormones detected in water and sediments include 17α-estradiol, 17β-estradiol, cholesterol, estrone, sitosterol, and stigmastanol. In addition, estrogenicity of sediments collected at nest sites was positively correlated with incidence of fish intersex.

Fish

Although natural endogenous steroidal hormones are present in all animals, hormone concentrations in tissue such as blood plasma are potentially useful indicators or markers of exposures to environmental or exogenous sources of hormones. Iwanowicz et al. (2009b) analyzed blood plasma of smallmouth bass collected from two tributaries in the Chesapeake Bay watershed for an estrogen (17-β estradiol) and an androgen (testosterone). Fish upstream and downstream from municipal wastewater effluents were compared. The general increase in blood plasma hormone concentrations in fish downstream from wastewater sources indicates likely exposures to these or other hormonally active compounds in the wastewater effluents.

Citing weight of evidence for endocrine disruption in fish, the Environment Agency of England and Wales recently focused on risk-management strategies for natural (and all other) estrogenically active effluents that discharge to the aquatic environment (Gross-Sorokin et al. 2006). To date (2012), most studies of environmental hormones have focused on estrogenicity and related effects. Khanal et al. (2006) cited estrogenicity monitoring studies in more than 30 countries and concluded that natural steroidal estrogens such as estrone, 17β-estradiol, estriol, and 17α-estradiol are potent endocrine disrupters found in the environment. Phytoestrogens are structurally similar to 17β-estradiol (estrogen in vertebrate animals) and their interaction with vertebrate estrogen receptors is a topic of active research in human and ecological health sciences. Currently (2012), research on other natural hormones as potential environmental contaminants is limited.

Yonkos et al. (2010) exposed fathead minnows (*Pimephales promelas*), sheeepshead minnows (*Cyprinodon variegatus*), and mummichogs (*Fundulus heteroclitus*) to solutions leached from poultry litter. The minnows exhibited intersex conditions whereas the mummichogs were unresponsive in all trials. Again, the potential mixture of hormonally active chemicals in the litter likely includes biogenic hormones as well as other endocrine disruptors; however, this study did not test for individual chemicals. In a positive controlled exposure experiment, 17β-estradiol (biogenic estrogen) had no effect on the mummichogs but did have effects on the minnows (Yonkos et al. 2010).

Recent research has linked environmental exposures of estrogenic compounds to fish intersex in Boulder Creek, Colorado, and the Potomac River Basin, Virginia (Vajda et al. 2008; Blazer et al. 2011; Kolpin et al. 2013). Because the sources of biogenic hormones in human and animal wastes are consistently associated with other estrogenic chemicals, these intersex studies have not linked specific effects to individual naturally occurring animal or plant hormones. For example, among the targeted chemicals analyzed by Vajda et al. (2008) that are known or suspected endocrine disruptors, 17β-estradiol, estrone, estriol, and 17α-ethynylestradiol as well as estrogenic alkylphenols and bisphenol A were identified and their mixtures were linked to intersex conditions in fish. Vajda et al. (2008) also showed that as separate groups the steroidal estrogens and the nonsteroidal estrogens were both in concentrations high enough to disrupt normal endocrine functioning. However, VanDenBelt et al. (2004) compared the estrogenic potencies of 17β-estradiol, estrone, 17α ethinyl estradiol, and nonylphenol (surfactant) and showed that 17α ethinyl estradiol can be as estrogenic as, or many times more estrogenic than 17β-estradiol (estrogenic potencies are commonly reported relative to 17β-estradiol), but nonylphenol is likely orders of magnitude less potent. As stated above, Kolpin et al. (2013) analyzed bed sediment at smallmouth bass nest sites in the Potomac River watershed and found a positive correlation between total biogenic estrogens (17β-estradiol, estrone, sistosterol, stigmastanol, and progesterone) in the sediments and incidence of intersex in fish using those nests. Therefore, the biogenic estrogens are likely critical components of total estrogenicity in terms of potency and therefore must be considered key environmental contaminants.

An additional effect of hormones is their role as potential promoters of liver tumors in fish. Nunez et al. (1989) first demonstrated that 17β-estradiol promotes hepatocellular carcinoma in fish. Cooke and Hinton (1999) reported greater liver tumor prevalence in females of six fish species and theorized that endogenous estrogens serve as tumor promoters. Although the effect of estrogens on liver tumors in the Chesapeake Bay watershed is unclear, Pinkney et al. (2011) found a similar higher prevalence of liver tumors in females versus males and incorporated sex as a covariate in a logistic regression analysis of prevalence across sampling locations.

Wildlife

A primary mode of exposure to exogenous steroids or environmental chemicals in birds is through maternal deposition (Adkins-Regan et al. 1995; Ottinger et al. 2005), which is significant across a range of endocrine disruptors and contaminants, with the distribution of the toxicant in the egg dependent on the chemical characteristics of that compound (Lin et al. 2004; Ottinger et al. 2005). This potential route of exposure is supported by a study in which Japanese quail hens were given estradiol implants (Adkins-Regan et al. 1995). Assay of plasma samples showed increased circulating estradiol levels, with either daily estradiol injections or with a silastic implant containing crystalline estradiol. Eggs produced by treated females contained significantly greater estradiol concentrations in the yolks than those produced by control females; therefore, these data provide evidence for maternal transfer of steroid hormones to the offspring through the yolk (Adkins-Regan et al. 1995). Similarly, other lipophilic compounds, including the soy phytoestrogens, also transfer from the hen into the egg and. more specifically, the yolk (Lin et al. 2004).

Embryonic exposure to exogenous steroid hormones alters sexual differentiation in birds and results in reproductive impairment in adults (Adkins-Regan et al. 1990, 1995; Halldin et al. 1999; Ottinger and vom Saal 2002; Ottinger et al. 2005). Embryonic exposure to estradiol or androgen greatly affects sexual differentiation, whereas adult exposure is ineffective (Adkins-Regan et al. 1990; Ottinger and Abdelnabi 1997). Similar results have been observed in bobwhite quail and also in mammals, indicating that this effect may be common across species and even phyla (Lien et al. 1987).

Conclusions

Although only few studies have been conducted on the occurrence of biogenic hormones in the watershed, it is clear that these naturally occurring compounds are widespread. Previous studies have shown that wastewater management operations such as municipal sewer systems can result in point sources of biogenic hormones that are reflective of the combined inputs within the sewershed. Other studies have shown that upgrades in treatment plants can lower the estrogenicity concentrations of the effluents. Animal agricultural practices, particularly those associated with manure management and others where livestock have direct access to streams are also known sources of these contaminants. In addition, although these contaminants are naturally occurring they are likely to be elevated in concentrations above background downstream from these pathways to the environment. Hormones are known to be physiologically active at very low levels and have been shown to cause adverse effects on receptor organisms at environmentally relevant concentrations. There are many potential sources and pathways of biogenic hormones to the environment in the Chesapeake Bay watershed therefore they likely are widely distributed in water and sediment. Current research is addressing how these contaminants may be impacting aquatic environments but at this time there is insufficient information to assess the overall severity of biogenic hormones in the watershed.

2.10 Metals and Metalloids

Abstract

Parsing the effects of metals in the environment is a complex process. Metals occur naturally in all media and are typically present in the environment as mixtures. Chemical and biological processes can alter metals valence states and can convert them between organic and inorganic forms. These factors, along with different physical and chemical properties of each metal, makes environmental risk assessment challenging. Although the concentrations of trace metals have declined slowly since the 1980s, elevated concentrations remain in some regions of the Bay. The most prevalent cause of impairment is the presence of mercury in fish tissue at concentrations in excess of State guidelines, affecting more than 600 river miles and approximately 20,000 impoundment acres in the Chesapeake Bay watershed. In the water column, other metals are present at concentrations in excess of State standards for the protection of aquatic life. Specifically, both Maryland and West Virginia have identified impairments for aluminum and iron in water, largely limited to each jurisdiction's portion of the North Branch of the Potomac River. Maryland also included manganese as a cause of impairment in the North Branch of the Potomac

River. In addition, Maryland's Bodkin Creek is identified as impaired for copper. Pennsylvania has completed more than 70 TMDLs for metals impairments, covering several hundred miles within the bay watershed. The great majority of Pennsylvania's metal impairments are for aluminum, iron, and manganese originating from abandoned mine discharges in the coal areas of the upper Susquehanna watershed. Maryland listed additional impairments for chromium, lead and zinc in sediment (Baltimore Harbor, Bear Creek and Curtis Bay Creek). These impairments were minimal in extent; zinc was the most widespread contaminant, impacting approximately seven square miles of estuarine waters. Based on the spatial extent of the listed impairments, the extent and severity of mercury contamination within the Bay watershed is considered to be widespread, whereas contamination with other metals (i.e., aluminum, chromium, iron, lead, manganese and zinc) is more localized.

Background

Metals are ubiquitous in the environment as constituents of both rock and sediment, and concentrations vary across geographic regions. They may be delivered to aquatic environments through natural erosion and weathering but also from anthropogenic sources. When concentrations in aquatic environments exceed natural background levels as a result of human activities, metals are considered to be contaminants. The term "metals" as used in this document refers to true metals, metalloids and organometals.

Risk from metals contamination in aquatic environments is complex because of the unique nature and behavior of elements. For example, because metals occur naturally in all environmental media, they typically are present in the environment in mixtures. Some metals are nutrients that are essential for life and maintaining the health of humans, animals, plants, and microorganisms. Unlike organic contaminants, metals are not degraded or destroyed by chemical or biological processes, but instead those processes can transform metals from one species to another (valence states) and can convert them between organic and inorganic forms. These factors, along with differing characteristics for each metal, create highly variable biological and ecological effects in the environment, making ecological risk assessment uniquely challenging. Extended exploration of these topics is beyond the scope of this report. Moreover, this chapter is not intended to be an exhaustive review of all metal contaminants; its focus is on those metals that have been found within the Chesapeake Bay watershed.

According to Kimbrough et al. (2008), fossil fuel and waste burning, mining and ore processing, chemical production, and agriculture are the sources largely responsible for the elevated metals concentrations observed in coastal waters. Metals are transported to coastal waters primarily from runoff and atmospheric deposition. The relative contribution from each source varies by metal, proximity to sources, and chemical phase (dissolved or particulate-bound).

Mercury, one of the most prevalent of the metals contaminants, is delivered to most aquatic ecosystems by deposition from the atmosphere, primarily through precipitation (USGS 1995). Anthropogenic sources of mercury contamination in the atmosphere include coal combustion, chlorine alkali processing, waste incineration, and metal processing (USGS 1995). Once deposited in aquatic ecosystems, mercury may be transformed to methylmercury; an organic form of mercury that is the most toxic and that readily bioaccumulates and is biomagnified (USGS 2000). Methylation occurs in sediments of freshwaters, estuaries and coastal zones; however the rate of net methylmercury creation from different sediment types is highly variable with higher methylation rates occurring in the organically rich, anoxic sediments common in wetland areas (Luoma and Rainbow 2008). Methylmercury has been identified as potent neurotoxicant (USEPA 2001b). Factors such as chemical form, dose, route of ingestion, exposed organism's species, sex, age, and overall health play a role in determining the level of toxicity and the environmental effects. Acute exposure to mercury most commonly affects the central nervous system and kidneys in fish, birds and mammals (USEPA 2011b).

The presence of ecological and biological effects resulting from metal contamination has been evident since the height of the Industrial Revolution; a substantial amount of literature related to metals in the environment dates as far back as the mid-1800s (Kapustka et al. 2004). Trace metal concentrations in the Chesapeake Bay peaked in the 1970s and have slowly declined since the 1980s; despite this downward trend, concentrations remain elevated in some regions of the Bay (Hartwell and Hameedi 2007).

Occurrence information for this chapter was derived primarily from the 2010 Water Quality Assessment Reports (Clean Water Act 303(d)/305(b) Integrated Reports) of each Bay jurisdiction. This chapter indentifies impairments and occurrences and does not attempt to compare loadings among jurisdictions. In addition to the assessment reports, information was captured from State fish consumption advisories, fish tissue monitoring program data from Maryland and Virginia, various reports from NOAA and other Federal agencies, as well as several studies related to metal contamination throughout the Chesapeake Bay region (DDOE 2010, DNREC 2010, MDE 2010, PADEP 2010, VADEQ 2010, WVDEP 2010, Mason et al. 1999, TetraTech 2006, Hartwell and Hameedi 2007, Fulton et al. 2007).

Because in-depth review of the biological and ecological effects is beyond the scope of this report, a focus has been placed on selected metal contaminants that have occurred within the Chesapeake Bay Watershed. The exclusion of a particular metal in this discussion does not imply insignificance. Although the science surrounding metals and their biological and ecological effects continue to be studied intensively and is evolving rapidly, some aspects still lack sufficient information to support a quantitative assessment (USEPA 2007).

Water Column

Mercury

Mason et al. (1999) characterized mercury concentrations within the Bay (sites located in Baltimore Harbor, Potomac, Patuxent and Anacostia Rivers, and Maryland's Bay waters). Overall mercury concentrations in the Chesapeake Bay were < 3 ng/L except for highly urbanized area sites such as the Baltimore Harbor and the confluence of the Anacostia and Potomac rivers. Total mercury concentrations in the upper Bay and in the subestuaries decreased down-estuary, coinciding with decreases in suspended particulate matter. Increased concentrations of mercury, especially methylmercury, were observed during seasonal bottom water anoxic conditions at the Baltimore Harbor. None of the Chesapeake Bay jurisdictions reported impairments for mercury in the water column.

Other Metals

Maryland, Pennsylvania and West Virginia each identified additional impairments for metals other than mercury. Both Maryland and West Virginia listed waters for aluminum and iron in the Bay watershed. West Virginia completed a TMDL addressing these impairments in the North Branch of the Potomac River Watershed (TetraTech 2006). Maryland identified additional water column impairments for copper and manganese. The copper impairment is isolated to Bodkin Creek in the Patapsco River watershed. The manganese, aluminum and iron impairments were located in the Upper North Branch of the Potomac River with acid mine drainage cited as a primary source (MDE 2010). Pennsylvania has completed more than 70 TMDLs for metals impairments, covering several hundred miles within the Bay watershed. The great majority of Pennsylvania's metal impairments are for aluminum, iron, and manganese originating from abandoned mine discharges in the coal areas of the upper Susquehanna watershed. Pennsylvania has a successful abandoned mine remediation program to address these water quality concerns.

Between 2008 and 2011 the Estuarine Probabilistic Monitoring Program (ProbMon) of the VADEQ collected clean dissolved trace metals samples from near surface waters at 182 estuarine sites in Virginia. Of these, 130 sites were within tidal portions of the Chesapeake Bay watershed. No exceedances of chronic saltwater standards were observed for any of the toxic metals evaluated (As, Cd, Cu, Pb, Hg, Ni, Se, Zn). Vanadium was added to the analyses beginning in 2010, and no exceedances of USEPA's recommended saltwater criterion for vanadium (50 µg/L) were observed among the 58 samples evaluated from the Chesapeake watershed (D. Smith, VADEQ, personal communication).

Sediment

Mercury

None of the Bay jurisdictions documented sediment impairments for mercury. Concentrations of mercury in sediment in the Baltimore Harbor region have been found to exceed 1 ppb with methylmercury concentrations approaching 0.010 ppb (Mason et al. 1999).

Other metals

Maryland identified sediment impairments specifically attributable to metals concentrations (chromium, lead and zinc). These metals impairments were localized to the Baltimore Harbor area, Bear Creek and Curtis Bay Creek. Zinc was the most widespread of the metals contaminants, affecting an area of approximately seven square estuarine miles. Sediment concentrations in excess of the State standards for the identified metal compounds have the potential to adversely affect aquatic life (MDE 2010).

Virginia conducted additional sediment monitoring as part of a toxicological characterization effort (Roberts et al. 2002, 2003, 2004). Sediment metals concentrations were below the ER-M with a few exceptions: in the Mattaponi, manganese exceeded the ER-M with a value of 3.38×10^6 ppb (Roberts et al. 2004). Various other metals were detected; however, no other metals exceeded the respective ER-M. In each study, the calculated SEM/AVS ratios (simultaneously extracted metals/acid volatile sulfides) indicated that the sediments may have additional metals binding capacity (Roberts et al. 2002, 2003, 2004).

Additional work on sediment in the Nanticoke identified detectable concentrations of all seven metals tested (arsenic, cadmium, chromium, copper, lead, mercury and zinc). Zinc concentrations were the highest, with values ranging from 91,100 – 224,000 ppb. None of the detected metals exceeded the respective ER-Ms (EA 2006).

In order to characterize the extent and magnitude of contaminated sediments in the Chesapeake Bay, Hartwell and Hameedi (2007) analyzed sediment samples for a suite of metals and metalloids: aluminum (Al), antimony (Sb), arsenic (As), cadmium (Cd), chromium (Cr), copper (Cu), iron (Fe), lead (Pb), mercury (Hg), nickel (Ni), silver (Ag), selenium (Se), tin (Sn), and zinc (Zn). The report documented metals concentrations above background at nearly all locations. The authors noted declining concentration trends with some site-specific exceptions, such as the Patuxent River and several southeastern tributaries that demonstrate high levels of cadmium. According to the report, Baltimore Harbor is one of the most contaminated sites in the Bay. Metals concentrations in sediment near Ft. McHenry, the Patapsco River, and the Potomac River's Swan Point and Mattox Creek are in the top percentile for toxic metals nationwide. The Elizabeth River is also heavily contaminated; concentrations for most constituents are in the upper 75th percentile nationwide. In the upper part of the Bay, concentrations of metals in sediment are higher than in the middle and lower Bay, specifically for manganese, nickel, chromium and lead that are elevated by factors of 1.5 to 2 above background concentrations. Throughout the Bay copper and zinc are elevated by factors of 1.5 to 3.5 above background concentrations. The report cited the Fall Line Toxics Monitoring Report that calculated an enrichment rate of 110 times for cadmium in the Susquehanna River (CBP 1996). Sites near heavily developed areas, such as the Magothy River in Maryland and Broad Bay in Virginia Beach, also exhibited elevated concentrations of metals. Large western tributaries of the Bay had slightly higher concentrations than the lower mainstem. The authors attribute the high degree of variability in metals concentrations from site to site to interactions involving sediment grain size, proximity to sources, and the inherent particle reactivity of the elements.

Fulton et al. (2007) inventoried toxic contaminants in five selected areas of the Bay for which data previously had been insufficient: the Chester River, Nanticoke River, Pocomoke River, lower Mobjack Bay (Poquosin and Back Rivers), and the South and Rhode Rivers. In the study, sediment, water column and benthic analyses were examined to characterize the biological effects of the contaminants. Total metal concentrations in sediment were found to be highest in the lower South River and the middle Chester River. Of the 60 stations, only two had contaminant concentrations that exceeded the ERM for any analyte; in the upper South River concentrations at two stations exceeded the ERM (51.6 ppm) for nickel. The most common metal Effect Range Low (ERL) exceedances were arsenic, nickel and zinc in the Chester River, arsenic and nickel in the Nanticoke River, and arsenic in the Pocomoke River.

Fish and Shellfish

Mercury

The primary method of human exposure to mercury is through ingestion of contaminated fish and shellfish (USGS 2000). States monitor levels in fin fish tissue and issue fish consumption advisories for the species that are found to have elevated levels in specific areas in order to protect human health. An advisory recommends a particular number and size of meals that can be safely consumed by the general public, pregnant women, and children; avoidance recommendations advise that no meals should be eaten regardless of meal size or population demographics.

Maryland currently has fish consumption advisories due to mercury for over 10 species of fish in rivers, lakes and reservoirs within the Chesapeake Bay watershed. The number of recommended meals per month for the general population ranges from one to eight. The State of Maryland reported impairments for approximately 12,500 acres of impoundments. A TMDL report has been completed for nearly 8,200 of those acres (MDE 2010).

The Commonwealth of Virginia currently has fish consumption advisories for mercury for nine species in the Chesapeake Bay and small coastal basins, James River, Rappahannock River, and York River basins. The number of recommended meals for the general population is no more than two per month for all species. Virginia does not have any avoidance recommendations due to mercury in fish tissue at this time. The Commonwealth of Virginia reported a total of approximately 5,980 acres in impoundments, approximately 300 miles in rivers and streams, and approximately 23 square miles in estuaries as being impaired by mercury in fish tissue (VADEQ 2010).

The District of Columbia advises the general population avoid carp, eel, and catfish from the Potomac and Anacostia Rivers; however the cause of advisory is not specified. No impairments specifically attributable to mercury in fish tissue have been reported in DC (DDOE 2010).

The State of New York notes impairment in the Lower Susquehanna River, as well as the Chenango River, a tributary to the Susquehanna. For these waters, the State advises that men over the age of 15 and women over the age of 50 consume no more than one meal per month of walleye longer than 22 inches. The State recommends that women and children eat none. A general fish advisory exists for all other fish of the Susquehanna which recommends that the public consume no more than four one-half pound meals a month of fish, with no more than one meal per week (NYDH 2012).

The State of West Virginia has issued statewide advisories of one to two meals per month for more than 10 species within waters flowing to the Chesapeake Bay as a result of mercury contamination. There is a no consumption recommendation for carp in the Shenandoah River, and a limit of one meal per month for smallmouth bass longer than 12 inches. This recommendation is driven largely by PCB concentrations though mercury is an underlying contaminant. The State of West Virginia did not list any of the free flowing streams to the Chesapeake Bay as impaired for mercury in the 2010 Integrated Report (WVDEP 2010).
The Commonwealth of Pennsylvania has issued advisories for 37 water bodies within the Susquehanna River basin due to mercury contamination; consumption recommendations are for one to two meals per month. One lake within the Potomac River basin has an advisory of two meals of walleye per month as a result of mercury contamination. In the 2010 Integrated Report, the Commonwealth of Pennsylvania reported that approximately 290 miles of rivers and streams within the Chesapeake Bay watershed are considered to be impaired due to mercury in fish tissue. Approximately 2,052 acres of impoundments are considered to be impaired for mercury in fish tissue (PADEP 2010). Pennsylvania is actively monitoring fish tissue mercury levels in the lower Susquehanna River (2006, 2008, 2010, 2011, 2012) and none of the results have resulted in a fish consumption advisory for mercury (P. Buckley, PADEP, personal communication).

The State of Delaware has a statewide advisory of no more than one meal per week for all waters of the state that are not covered by an explicit, waterbody-specific advisory. This advisory is meant to account for mercury and any other contaminants that might be present but not identified through a detailed study (R. Greene, DNREC, personal communication).

The toxicity of mercury to fish is associated with methylmercury concentrations in fish tissue. Methylmercury is a highly toxic compound that crosses biological membranes, accumulates in the exposed species, and can be biomagnified up the food chain (Sandheinrich and Wiener 2011).

State monitoring programs frequently measure only total mercury, and criteria (such as USEPA 2001b) are typically stated as criteria for methylmercury. Because there is little difference in the fish tissue concentrations when both compounds are measured, conversion from total mercury to methylmercury requires a decrease of 10% at most.

The USEPA (2001b) issued a water quality criterion for methylmercury of 0.3 ppm ww fish tissue for the protection of human health. This criterion was developed based on the basis of a review of high-dose exposures (i.e., Minimata Bay, Japan) that resulted in neurotoxic effects including mental retardation, cerebral palsy, deafness, blindness in utero and sensory and motor impairment in adults. Studies of chronic low level mercury exposure have shown developmental effects in children including abnormalities in memory, attention, and language. Other studies have reported cardiovascular and immunological effects.

Within the Bay watershed, Virginia's South River is the area of greatest concern with respect to mercury contamination. Except for stocked trout which spend little time in the river, no fish consumption is recommended. Between 1929 and 1950, mercury was used as a catalyst in fiber production at the DuPont plant in Waynesboro, Virginia. During that time, strict storage and disposal regulations did not exist, and mercury was released into the South River. A serious contamination problem was discovered in the 1970s (South River Science Team 2009); the VDH subsequently issued advisories for fish consumption along the South River and on the South Fork Shenandoah River. As part of a settlement agreement, DuPont paid a penalty and established a State-administered (by VADEQ) trust fund to support a 100-year mercury monitoring program for fish, water, and river sediment administered. The most recent fish sampling data listed on the VADEQ web site are from 2007 (VADEQ 2012c). At several sampling locations downstream from the plant, mercury concentrations frequently exceeded 1.5 ppm (five times the USEPA 2001b criterion) in smallmouth bass (*Micropterus dolomieu*), redbreast sunfish (*Lepomis auritus*), and white sucker (*Catostomus commersoni*).

Sandheinrich and Wiener (2011) reviewed the toxic effects of methylmercury by evaluating the relation between tissue concentrations and toxic effects. Mercury exposure can adversely affect the survival, growth, and reproduction of fish. Wiener and Spry (1996) reported that mortality due to mercury poisoning is observed only at extremely high tissue concentrations (6 - 20 ppm wet weight in muscle). Behavioral effects include hyperactivity and altered shoaling activity (Webber and Haines 2003), which occurred in a laboratory study with golden shiners

(*Notemigonus crysoleucas*). The fish were fed a diet containing 0.96 ppm methylmercury and had whole body concentrations of 0.52 ppm wet weight. Reduced spawning success occurred in fathead minnows (*Pimephales promelas*) at whole body minus gonad concentrations of 0.71 to 0.92 ppm wet weight (Drevnick and Sandheinrich 2003). Methylmercury causes oxidative stress in fish tissues through the formation of radical oxygen species and lipid peroxidation. Organs affected include the brain and liver (summarized in Sandheinrich and Wiener 2011).

On the basis of their literature review, Sandheinrich and Wiener (2011) concluded that effects on fish biochemical processes, damage to cells and tissues, and reduced reproduction have been demonstrated at methylmercury concentrations of about 0.3 to 0.7 ppm in the whole body or 0.5 to 1.2 ppm in muscle tissue. Given the high concentrations present in fish in Virginia's South River, there is a great likelihood that fish in this water body are being adversely affected by methylmercury exposure.

Other Metals and Metalloids

For the assessed waters, the states did not identify any impairments in fish tissue for metals other than mercury. According to the MDE human health screening study of finfish tissue collected from 1985 to 1997, tissue concentrations in finfish from the Maryland portion of the Chesapeake Bay did not exceed the established conservative human health risk-based screening values for arsenic, silver, cadmium, chromium, copper, mercury, nickel, and zinc (MDE, undated). Although concentrations of inorganic arsenic in a few individual bluefish, striped bass, and white perch exceeded the screening values, the MDE noted that those species exhibit migratory behavior and are therefore not considered to be a target species in the Core Monitoring Program (MDE, undated). It should be noted that this report is 15 years old; some screening values may be different now (2012). Additionally, the MDE report focuses on human health criteria only; aquatic life criteria are not examined.

The Commonwealth of Virginia conducted a statewide fish tissue monitoring survey most recently in 2008. A review of the data for waters within the Chesapeake Bay watershed indicates that concentrations of arsenic in 83 of 477 samples from the Bay watershed exceeded the VADEQ screening value for arsenic (VADEQ 2012b). Although arsenic is detected at concentrations in excess of the screening value, VADEQ has not identified these waterbodies as impaired. This is in acknowledgement of the fact that the fish tissue data are based on the concentration of total arsenic, whereas toxicity is associated only with inorganic arsenic. Inorganic arsenic may be a very small portion of the total arsenic value. VADEQ uses this information to identify areas in need of further study and has indicated that a method for measuring inorganic arsenic may be utilized for these efforts (VADEQ 2010). Concentrations in several samples exceeded the detection limits of <0.1 ppm ww for lead and <0.5 ppm ww for selenium (VADEQ 2008b). Because available data were insufficient to identify a threshold associated with a risk of human toxicity, these waters were not identified as impaired. Since 1986, NOAA's Mussel Watch program has measured metal concentrations in oysters (*Crassostrea virginica*) within the Chesapeake Bay and its tributaries at five locations in Maryland and five locations in Virginia as part of a national effort (Kimbrough et al. 2008). Data collected in 2004-2005 from five locations in Maryland and five locations in Virginia were reported in an appendix of the report, with summary data for arsenic cadmium, copper, mercury, nickel, lead, tin, and zinc. For Maryland, data are reported on cadmium, copper, tin, and zinc which were rated as medium or high in at least one location in the national comparison. Cadmium concentrations ranged from 3.2 to 14 ppm, with all except the highest concentration described as medium in the national comparison. The two highest copper concentrations were 301 ppm (rated as medium) and 867 ppm (rated as high). The two highest tin concentrations (0.25 and 0.44 ppm) were both rated as medium. Zinc concentrations of 2550, 2660, and 4570 ppm were all rated as medium and 12,000 ppm was rated as high. In Virginia, concentrations of cadmium, copper, and zinc were rated as medium or high in the national comparison. The three highest cadmium concentrations were 4.8 and 4.8 (medium), and 10 ppm (high). The three highest copper concentrations were 285 and 308 (medium), and 1460 (high). The four highest zinc concentrations (2720, 3200, 3770, and 8110 ppm) were all rated as medium.

Wildlife

Mercury

Mercury concentrations in blood have been measured in several species of birds in the Chesapeake Bay watershed. Levels in osprey nestlings from regions of concern and reference sites ranged from 0.105 to 0.470 (Rattner et al 2008); adult tree swallow samples collected at a contaminated site in the South River (tributary of the Shenandoah River, which flows into the Potomac) had mean concentrations of <3.56 ppm ww (Brasso and Cristol 2008; Hawley et al. 2009), and values were < 0.5 ppm ww in Nelson's sparrow (*Ammodramus nelsoni*) and the saltmarsh sparrow (*A. caudacutus*) from several Bay locations. With the exception of tree swallow samples from the South River, mercury concentrations observed in these species generally were less than the 0.400 ppm ww that has been associated with adverse sublethal effects in birds (Scheuhammer et al. 2007), although recent studies suggest that such concentrations could evoke endocrine disruptive effects in nestlings (Wada et al. 2009).

Mercury is readily incorporated into feathers in growing birds and adults following molt. Mercury levels in feathers of 14- to 16-day-old nestling black-crowned night-herons from Baltimore Harbor and Holland Island did not differ, and ranged only from 0.04 to 0.23 ppm dw (Golden et al. 2003a). Mercury concentrations in feathers of 40- to 45-day-old osprey nestlings were greater in the Elizabeth River (0.26-2.40 ppm dw) compared to Baltimore Harbor and the Patapsco River, the Anacostia and the middle Potomac, and the South, West and Rhode Rivers (values ranged from 0.01-1.53 ppm) (Rattner et al. 2008). Molted feather samples collected from 83 occupied bald eagle nests in the Chesapeake averaged 3.82 ppm dw (Cristol et al. 2012). Feathers from adult tree swallows in the vicinity of the South River in Virginia had average mercury concentrations of 13.55 ppm dw (Brasso and Cristol 2008). With the exception of samples collected in the mercury-contaminated South River location in Virginia, values were generally well below the concentration commonly associated with adverse effects (7.5 ppm dw) in birds (Eisler 1987).

Since the mid-1990s, several studies that examined eggs from ospreys (Rattner et al. 2004), peregrine falcons (USFWS et al. 2004; Clark et al. 2009), common terns (French et al. 2001) and bald eagles (Mojica and Watts 2008, 2011) indicated that mercury concentrations averaged well below 0.5 to 1.5 ppm ww, the generally accepted toxicity threshold for reproductive effects in bird eggs (Wiener et al. 2003).

Other Metals and Metalloids

The Contaminants Exposure and Effects – Terrestrial Vertebrates (CEE-TV) database contains more than 50 records for Chesapeake waterbirds from 1988 to the present (2012) that describe lead concentrations in liver (Rattner and McGowan 2007; B. Rattner, USGS, unpublished data). For 26 of these records (representing 84 individuals including waterfowl, geese, and bald eagles), lead concentration in liver exceeded 2 ppm wet weight, a value associated with subclinical poisoning in waterfowl (Franson and Pain 2011), and ranged up to 183 ppm dry weight in one of the bald eagles (> 10 ppm wet weight is compatible with death; Franson and Pain 2011). Many of these necropsy reports were related to the ingestion of spent lead shot that was historically used for hunting waterfowl but was phased out of use by 1991. Lead can be incorporated into feathers of growing birds (Golden et al. 2003b), and has been proposed as a sensitive minimally invasive indicator of exposure. Feathers of black-crowned night-heron nestlings from Chincoteague Bay and Holland Island were lower (<0.13 ppm dw) than feathers of 14- to 16-day-old nestlings from Baltimore Harbor (0.32 ppm) (Golden et al. 2003a). A similar tendency was noted in feathers of 40-45-day-old osprey nestlings collected from Baltimore Harbor in 2000 (geometric mean:

1.25 ppm dw) compared to nestlings from the South River reference area near Annapolis (0.66 ppm), whereas those samples from the middle Potomac River watershed were intermediate (0.96 ppm) (Rattner et al. 2008). Concentrations in osprey nestling feather samples collected from the Elizabeth River in 2001 (1.47 ppm dw) were significantly greater than the South, West, and Rhode Rivers reference site (0.54 ppm) (Rattner et al. 2008). Although lead concentrations in feathers of waterbirds in the Chesapeake Bay regions of concern are elevated compared to reference sites, it is not possible to relate the concentrations to toxicity thresholds or assess the risk of lead in feathers to overall waterbird health.

Reports of lead in liver of wild mammals in the Chesapeake Bay watershed are far more limited, and focused on military sites with known or suspected contamination. Lead was not detected in liver or kidney of woodchucks (*Marmota monax*) collected in the vicinity of small arms and skeet ranges at Aberdeen Proving Grounds in Maryland, although low levels were detected in blood (mean values < 0.92 ng/ml ww) and bone (mean values < 13 ppm dw) indicating its probable bioavailability (Johnson et al. 2004).

The toxicity threshold for cadmium in liver and kidney has not been adequately established for birds (perhaps >100 ppm ww; Beyer 2000), and seemingly elevated concentrations in individuals may merely reflect that cadmium values increase with age. Cadmium is rarely detected in eggs, and poorly transferred into feathers of nestlings. Cadmium was not detected in blood of osprey nestlings collected in regions of concern, and concentrations in feathers were <0.28 ppm dw (Rattner et al. 2008).

Selenium concentrations in common tern and peregrine falcon eggs averaged <2.6 µg/g dry weight (French et al. 2001; U.S. Fish and Wildlife Service et al. 2004; Clark et al. 2009; J.B. French, USGS-Patuxent Wildlife Research Center, unpublished data), and were clearly less than levels causing embryotoxicity, although this threshold is contentious (Ohlendorf 2003). Selenium was present in blood of nestling ospreys in the range of 2.34 to 18.2 ppm dry weight, and in feathers in the range of 0.71 to 9.73 ppm, neither of which approached levels associated with toxicity (Rattner et al. 2008).

Concentrations of aluminum, boron, chromium, copper, iron, manganese, strontium and zinc were often detected in tissues and eggs of Chesapeake Bay waterbirds collected since 1988, and were generally in the range of values for healthy captive birds. Arsenic, barium, beryllium, molybdenum, nickel and vanadium were commonly analyzed for but rarely detected in tissues and eggs (Rattner and McGowan 2007).

Conclusions

Adequate data to evaluate the extent and severity of metals and metalloids exists in the various media as a result of both Federal and routine jurisdiction monitoring. Mercury impairments dominate the 303(d) listings identified for metals with more than 600 river miles and 20,000 acres of impoundments listed for fish tissue impairments. Though impairments in the water column are not as common, both the States of Maryland and West Virginia have identified impairments for aluminum and iron. Pennsylvania has identified hundreds of miles of impairment for metals, nearly all attributable to abandoned mine discharges. In addition, the State of Maryland has identified sediment impairments for chromium, lead and zinc. The impact of mercury contamination on fish tissue is widespread based on documented fish consumption advisories. However, contamination from other metals is more localized, affecting limited areas of the Bay watershed.

In addition to cataloging the extent and severity of toxic effects in the Bay by contaminant class, there is a need to describe studies that indicate the adverse effects of fish exposure to complex mixtures of both legacy contaminants and contaminants of emerging concern (CEC). These complex mixtures can have additive (Silva et al. 2002; Brian et al. 2005; Correia et al. 2007; Vajda et al. 2008) as well as synergistic or antagonistic effects (Micael et al. 2007; Santos et al. 2006; Sárria et al. 2011). In addition, many chemicals, particularly those that affect the endocrine and immune systems, may not elicit the typical dose-response curves. Significant effects may be observed at very low levels, and nonmonotonic (nonlinear) dose responses are increasingly demonstrated (Welshons et al. 2003; Vandenberg et al. 2012). Additionally, most monitoring of chemical concentrations is a snapshot in time, and the results depend on sampling time and numerous environmental factors such as climatic conditions and flow. For instance, atrazine concentrations measured in the Monocacy River (Maryland) in the spring were 100 times those measured in the fall (Alvarez et al. 2009). Short-term exposures, particularly in early life stages, can have long-lasting effects on both the endocrine and immune systems (Leet et al. 2011; Milston et al. 2003; Mcallister and Kime 2003; Liney et al. 2005).

Mouth lesion on a brown bullhead from the South River, Maryland, later diagnosed as squamous cell carcinomas. Photo by Fred Pinkney, USFWS

For these reasons, biological effects monitoring is increasingly recognized as an important factor in assessing the cumulative and interacting effects of toxic chemicals as well as other stressors (such as low oxygen, temperature, and river flow) on ecosystem health (Dubé et al. 2006; Ankley et al. 2010). By collecting data at many levels of biological organization (sub-organismal to populations) along with systematic evaluation of water quality and other stressors, researchers can use these measurements as environmental indicators to document habitat quality and evaluate trends.

The following biological indicators of compromised fish health have been observed within the Chesapeake Bay watershed: increased incidence of infectious disease and parasite infestations contributing to increased mortality; feminization (intersex, plasma vitellogenin) of largemouth and smallmouth bass and other signs of endocrine disruption; reduced reproductive success and recruitment of yellow perch in certain highly urbanized tributaries; and tumors in bottom-dwelling fish. In this section of the report, we describe these effects, and the evidence that suggests possible associations with exposure to toxic chemicals, so that they are considered in developing goal setting recommendations for the CBP.

Infectious Diseases, Parasites, Immune Suppression, and Fish Kills

Worldwide, there have been substantial increases in disease reports for freshwater fishes, amphibians and crayfish from 1970 to 2009 (Johnson and Paull 2010) and anthropogenic drivers, including toxic chemicals may play direct and indirect roles in these increases. Recent studies suggest increased disease incidence can be related to human-induced land use changes (Patz et al. 2004), increased nutrient concentrations (McKenzie and Townsend 2007), climate change (Karvonen et al. 2010), and toxic chemicals (Feingold et al. 2010). Exposure to a range of chemicals has been shown to cause immune system effects that can increase susceptibility, increase the persistence and hence shedding of infectious organisms, and alter the severity of disease (e.g., Arkoosh et al. 1998; Ross 2002; Zelikoff et al. 2002). Hence, understanding disease epizootics in wild populations is complex and requires a multidisciplinary approach.

A number of epizootic diseases have been documented in the Chesapeake Bay in recent decades. Ulcerative skin lesions of menhaden *(Brevoortia tyrannus*) were determined to be caused by the oomycete, *Aphanomyces invadans* (Blazer et al. 1999, 2002; Kiryu et al. 2002), a pathogen responsible for major mortalities in freshwater and estuarine fishes worldwide (Baldock et al. 2005). Skin and internal lesions have also been observed in a high percentage (> 50%) of striped bass (*Morone saxatilis*) from the Bay and its tributaries. Disease in striped bass has been attributed to a variety of *Mycobacteria* spp. (Ottinger and Jacobs 2006; Gauthier and Rhodes 2009). Mycobacteriosis is a chronic disease; however, negative population-level effects have been demonstrated indicating the importance of disease in management and stock assessment (Gauthier et al. 2008). The role of toxic chemicals in susceptibility to these infectious diseases is currently unknown.

Within the Potomac River watershed, in 2002, major mortality of smallmouth bass (*Micropterus dolomieu*) and other freshwater fishes was observed in the South Branch Potomac River. Similar fish kills occurred in the North Fork of the Shenandoah River in 2004, the South Fork Shenandoah River in 2005 and the Monocacy River in 2009. Adult fishes, during the spring were affected and most dead and dying fish exhibited a variety of external skin lesions. A number of potential pathogens, including the bacteria *Aeromonas salmonicida, Flavobacterium columnare, Aeromonas hydrophila* and other motile Aeromonads, internal trematode and myxozoan parasites, as well as external parasites including trematodes and leeches have all been identified or cultured. However, no consistent pathogen or parasite was identified as a single cause of the mortalities at all sites (Blazer et al. 2010). These findings suggest certain sensitive (immunocompromised) species are affected by a variety of opportunistic pathogens eventually leading to lesions and/or death.

Raised skin lesions which microscopically range from hyperplasia (proliferation of normal epidermal cells) to papillomas (benign neoplasia) were observed in adult bass from the Potomac (Blazer et al. 2010) and Susquehanna Rivers (V. Blazer, USGS, personal communication). The cause of these is currently unknown. However, similar lesions have been described in a variety of fish species. In some instances, viral particles have been observed in similar lesions by using electron microscopy (Anders and Möller 1985; Quackenbush et al. 2001). In other cases, investigators have tried to culture and/or visualize viruses using electron microscopy and have not found any indication of a viral etiology. There are a number of reports of increased prevalence of these types of papillomas at sites impacted by industrial and/or sewage effluent (Korkea-aho et al. 2006, 2008) and also by exposure to androgens (Kortnet et al. 2003). The International Council for the Exploration of the Sea (ICES) uses papilloma prevalence as one of suite of indicators to monitor environmental conditions (Bucke et al. 1996).

All of the Potomac River basin watersheds affected with fish kills were also areas where a high prevalence and severity of intersex or testicular oocytes was documented (see below; Blazer et al. 2007a, 2012). The co-occurrence of skin lesions/mortalities and signs of endocrine disruption further suggests that exposure to chemical contaminants and other stressors may contribute to the reduced health of these populations. For instance, whereas exposure to estrogenic compounds, is considered a primary cause of feminization (testicular oocytes and plasma vitellogenin) of male fishes, estrogens have also been shown to modulate disease resistance (Iwanowicz and Ottinger 2009; Robertson et al. 2009). Two other chemicals, atrazine and arsenic, known to affect disease resistance were identified in the fish kill and endocrine disruption research in the Potomac River basin (Blazer et al. 2010; Alvarez et al. 2008, 2009). Exposure to sublethal concentrations of atrazine, a commonly detected herbicide in the Chesapeake watershed, including sites where fish kills occurred (Alvarez et al. 2008, 2009), has been shown to increase susceptibility of silver catfish to *Aeromonas hydrophila* infection (Kreutz et al. 2010). Atrazine exposure is also associated with increased trematode infections in some amphibians (Rohr et al. 2008a, 2008b). Arsenic, used in pesticides as an additive in poultry feed and found naturally, has been reported to modulate the immune response of fishes (Hermann and Kim 2005; Lage et al. 2006) and is also associated with skin lesions in humans (Kazi et al. 2009). Exposure to arsenic was shown to enhance the ability of *A. hydrophila* to colonize and disseminate within exposed catfish (Datta et al. 2009) and inhibit the ability of zebrafish (*Danio rerio*) to clear viral or bacterial infections (Nayak et al. 2007). A significant increase in arsenic concentrations in the skin and anterior kidney (major immune organ of fish) between March and May (leading up to the fish kills) was demonstrated in smallmouth bass from the Shenandoah River (Blazer et al. 2010).

Feminization of Male Bass

Two indicators of feminization of male fishes that have been used worldwide are testicular oocytes (intersex) and vitellogenin. Experimental exposures of various fish species to natural and synthetic estrogens have shown that the most sensitive stage for induction of oocytes within the testes is during sexual differentiation, within two to three weeks after hatching. Exposures at these early life stages can lead to a greater sensitivity to estrogenic exposures later in life (Liney et al. 2005). Plasma and liver vitellogenin mRNA may be indicative of more recent exposure. Estrogens induce vitellogenin production by the liver. Normally this process occurs only in egg-producing females; however, exposure to abnormal concentrations of estrogens can induce immature or male fishes to produce vitellogenin.

Studies in the Chesapeake Bay watershed have concentrated on smallmouth and largemouth (*M. salmoides*) black bass species, although species comparisons have been conducted in some areas. Results of the U.S. Geological Survey Biomonitoring of Environmental Status and Trends program showed black bass to be sensitive species for reproductive endocrine disruption studies. In surveys conducted from 1995 to 2004 in nine river basins (Mississippi, Columbia, Rio Grande, Colorado, Yukon, Pee Dee, Apalachicola, Savannah, and Mobile), intersex was most prevalent in smallmouth and largemouth bass (Hinck et al. 2009). Largemouth bass were collected at 52 of the 111 sites sampled and at least one male with testicular oocytes was collected at 23 (44%) of these sites, with an overall prevalence of 18%. Smallmouth bass were collected at 16 sites and at 7 (44%) at least one male with testicular oocytes was observed, with an overall prevalence for smallmouth bass of 33% (Hinck et al. 2009). Similarly, largemouth bass within the Potomac River drainage also show signs of feminization, but may be less sensitive than smallmouth bass (Blazer et al. 2007; Iwanowicz et al. 2009). The prevalence of intersex in smallmouth bass in the Potomac River is considerably higher than that observed in the national survey, ranging from 50 to 100% (Blazer et al. 2007, 2012).

With respect to reproductive endocrine disruption, most studies to date have focused on the prevalence and severity of, and factors that contribute to feminization (intersex and plasma vitellogenin in males). Because aquatic organisms may be exposed to complex mixtures of chemicals that can have additive, synergistic, or antagonistic effects, it is difficult to identify one chemical or one source of the estrogenic compounds. Hence, biological effects, as well as chemical monitoring, were used to determine the extent of reproductive endocrine disruption throughout the Potomac River and nearby drainages, to identify potential causes, sources, and associated land-use practices and to document effects. Initial biological effects monitoring (Blazer et al. 2007) indicated that the prevalence of testicular oocytes varied from 100% at some sites in the Shenandoah River (Virginia) to low or background levels (10-14%) at selected out-of-basin sites such as the Gauley, Tygart, and Cheat Rivers (West Viginia). Prevalence in the South Branch of the Potomac River in West Virginia was intermediate, with 50% to 75% of the male bass affected. These surveys indicated a probable gradient of intersex in West Virginia and Virginia associated with human population and agricultural land use in the counties containing the sites. Although sample sizes of male smallmouth bass were small at some sites, there was an indication that, even within out-of-basin rivers such as the Greenbrier River, prevalence of intersex at downstream sites increased with human population and agricultural land use (Blazer et al. 2007).

Feminization of male fishes, in other parts of the world, has most commonly been associated with exposure to human wastewater-treatment plant (WWTP) effluents or other point sources. Hence, an early study in the Potomac River watershed to identify chemical compounds and associated point sources focused on sites upstream and downstream from WWTPs on the Monocacy River and Conococheague Creek, Maryland (Alvarez et al. 2009; Iwanowicz et al. 2009). Although some biomarkers such as gonadosomatic index and female plasma vitellogenin concentrations were adversely affected downstream from these WWTPs, the prevalence of males with testicular oocytes or plasma vitellogenin was not consistently higher downstream. A similar lack of increased prevalence of intersex downstream from WWTPs was found in the Susquehanna River drainage in Pennsylvania (V. Blazer, USGS, personal communication). Whereas prevalence was not significantly different, severity increased downstream, indicating that other sources may be inducing intersex early in development but that WWTP effluent may contribute to increased severity. The

Pennsylvania project also demonstrated that white sucker (*Catostomus commersonii*) collected at the same sites as smallmouth bass had no signs of testicular oocytes, but male suckers did show vitellogenin.

These findings led to investigation of other sources such as agriculture. A spring spawning study (Blazer et al. 2012; Kolpin et al. 2013) was conducted in which fish were collected just prior to spawning for a suite of reproductive endpoints. Sediment samples were collected from the smallmouth bass nests, integrative samplers were placed near nesting areas to bracket spawning, and discrete water samples were collected at the time of fish sampling. Because many of the chemicals currently of concern have biological effects at very low levels–commonly close to or below analytical capabilities–two approaches were used to assess water samples in conjunction with the biological effects in the fish. First, integrative passive samplers were deployed for approximately 30 days at the various sites, allowing the capture not only of cumulative base flow but also runoff events that may occur during the same time period. Second, extracts of discrete water samples as well as the integrated samples were tested for total estrogenicity. Results of the biological effects monitoring over two spring spawning seasons demonstrated a reduction in sperm count and in the motility of the sperm in bass from the Potomac River with respect to those at the reference site in the Gauley River, and an inverse relation between testicular oocyte severity and sperm motility. Second, a seasonal difference in intersex and vitellogenin in male bass was found. Third, for the various land-use characteristics (human population density, number of WWTPs, WWTP flow, and various agricultural attributes), intersex prevalence was associated only with percent agriculture and animal density in the catchment above the site. Intersex severity was associated with these two variables, as well as total animal feeding operations (AFOs), number of poultry houses, and WWTP flow (Blazer et al. 2012), also indicating that factors associated with agricultural runoff may be responsible for induction of intersex in these areas, whereas multiple sources increase intersex severity over the life of the fish.

Chemical analyses results obtained during the spawning study indicated that 39 of a total of 201 target chemicals were detected in at least one discrete water sample. Atrazine, its degradate, and simazine (herbicides), iso--chlorotetracycline (antibiotic) and caffeine (stimulant) were the most frequently detected. In contrast, 100 compounds, including four biogenic hormones were detected in extracts from the integrated samplers. The concentration of atrazine in the discrete water samples and total hormone and sterols in the sediment were significantly related (positively correlated) with the prevalence and severity of testicular oocytes (Kolpin et al. 2013).

Ciparis et al. (2012) conducted a landscape-scale study in smaller watersheds of the Shenandoah River basin, assessing effects of WWTP and AFOs on nutrients and estrogenic activity, using the bioluminescent yeast estrogen screen. Eighteen sites representing a gradient of influence from AFOs combined with presence/absence of WWTP discharge were sampled during both low and high flow periods. Strong relationships were found between watershed densities of AFOs and estrogen equivalents at all sampling times. The authors concluded that there was no consistent relationship between estrogenicity and WWTP discharges.

Reproductive Impairment of Yellow Perch

Several Chesapeake Bay tributaries, including the Severn and South Rivers, were closed to yellow perch fishing for nearly 20 years. Despite both recreational and commercial fishing regulations, a continued lack of recruitment has occurred. Hence, the streams were reopened for recreational fishing in 2009. The rationale for reopening the Severn, South, and West Rivers was that these watersheds are extremely degraded habitat, reproductive output is low, and, therefore, recreational harvest would not affect the total reproductive capacity (Maryland Fisheries Service 2012).

Larval presence, L_p, is defined as the proportion of 0.5-meter plankton tows with larvae during the peak weeks from late March through early May, and is used as an indicator of year class strength. The L_p index integrates egg production, egg hatching success, and survival of first-feeding larvae. Brackish systems with small watersheds and high levels of development (South, Severn, and Magothy Rivers) have exhibited a persistent depression in L_p to below a reference minimum since 2002. Regression analyses indicated that development (percent impervious surface in the watershed--i.e., pavement, rooftops, and compacted soils) was negatively related to L_p. Other systems may exhibit wide variation in larval presence, but low levels similar to those seen in these urbanized subestuaries are not common; however, since 2008 spawning site loss for anadromous fishes is evident in the urbanizing Mattawoman Creek watershed (Uphoff et al. 2011).

Despite numerous studies, the contributing factors to the lack of reproductive success have not been identified. Neither excessive adult mortality nor reduced growth has been observed in Severn River yellow perch. Severn River broodstock induced to spawn in the hatchery produced visually abnormal egg chains and too few viable eggs to support hatchery production. Hence, the depressed egg and larval survival are hypothesized to be critical factors suppressing resident yellow perch populations in western shore subestuaries whose watersheds are subject to high levels of development (Uphoff et al. 2005). In recent surveys that assessed a suite of bioindicators, a number of biological effects were found in yellow perch from the Severn and South Rivers and, to a lesser degree, Mattawoman Creek. An apparent lack of final maturation, abnormal yolk, and zona pellucida (egg coat) were noted in ovaries collected during the spawning run. In the males, abnormal proliferation of Leydig cells was noted (Blazer et al. in press).

Skin and Liver Tumors in Bottom-Dwelling Fish

Although liver tumors have been clearly associated with sediment exposure to polycyclic aromatic hydrocarbons (PAHs) in bottom-dwelling fish (see Section 2.3), it is likely that exposure to other chemicals contributes either as an initiator or a promoter. Evidence that polychlorinated biphenyls (PCBs) and estrogens are believed to promote liver tumors is summarized in Sections 2.1 and 2.9, respectively.

In contrast, the causes of skin tumors in brown bullheads are more uncertain and the subject of intense investigation. These tumors are commonly grotesque in appearance and alarming to the public. From 1992 to 2008, the USFWS conducted surveys in the following low salinity waters of the Chesapeake Bay watershed: Potomac River (Quantico Embayment), Anacostia River, Rhode River, South River, Severn River, Back River, Furnace Creek, Farm Creek, Marumsco Creek, Neabsco Creek, Choptank River, Little Blackwater River, and Tuckahoe River (Pinkney et al. 2009, 2011). An early goal of the tumor surveys was to systematically evaluate tumor prevalence in bullheads from the Anacostia River in Washington, DC, where there was anecdotal evidence of fish with visible mouth lesions. The linkage between liver tumors and PAHs in this river is described in Section 2.3, as are studies relating liver tumors and PAH exposure in mummichogs in the Elizabeth River.

A second goal of the bullhead surveys has been to understand the relation between skin tumors and exposure to environmental contaminants. High prevalence of skin tumors has been observed in the Anacostia, South, Severn, Little Blackwater, and Neabsco Rivers (Pinkney et al. 2009, 2011). The skin tumor prevalence in the South River, in particular, attracted widespread attention as a result of the grotesque appearance of several individuals. Results of the first survey in 2005, revealed a 53% prevalence of skin tumors (Pinkney and Harshbarger 2005), as high as any reported in North America. A follow-up study was conducted with an archived sample from 2004 and multiple samples from 2007 and 2008, as well as samples collected from the nearby Rhode and Severn Rivers (Pinkney et al. 2011). Skin tumor prevalence in the South River bullheads ranged from 19 to 58%. Prevalence of liver tumors was low in most collections, ranging from 0 to 6% in all collections but one, in which prevalence was 20%. As part of the study, biliary PAH-like metabolites as well as polycyclic aromatic compound (PAC)-DNA adducts in skin and liver tissues were measured. The authors also investigated the concentrations of alkyl-DNA adducts, which arise from interactions of alkylating agents such as nitrosamines with DNA. South River sediment total PAH concentrations averaged only 3.6 ppm within two kilometers of the collection site; concentrations from the other rivers were smaller. Neither the sediment PAH concentrations, the PAH-like metabolite data, nor the PAC-DNA adduct data show a linkage between exposure to PAHs and liver or skin tumors in these rivers. The alkyl-DNA adduct data also were not consistent with liver- or skin-tumor prevalence (Pinkney et al. 2011). Therefore, no chemical class has been associated with the high skin-tumor prevalence in the South River. On a broader scale, Pinkney et al. (2009) produced scatterplots of skin tumor percentages in relation to sediment total PAH concentrations near the Chesapeake Bay bullhead collection sites and found no relationship between prevalence and concentrations.

A third goal was to use logistic regression to statistically analyze the Chesapeake Bay bullhead tumor database to determine which covariates must be considered when comparing tumor prevalence among collections and locations. Based on 1992 through 2008 Chesapeake bullhead tumor data, Pinkney et al. (2009) reported that length and sex (with females having a higher ratio) were significant covariates for liver tumors, and length was a significant covariate for skin tumors. This statistical analysis, enhances the utility of bullhead tumors as an environmental indicator as defined by USEPA (2006), since it allows researchers to make unbiased comparisons over space and time.

Both the bullhead and mummichog tumor surveys meet the criteria proposed by USEPA (2006) to select environmental indicators. The use of mummichog liver histopathology as an environmental indicator, especially in the Elizabeth River Program monitoring studies (Chapter 2.3, e.g., Vogelbein and Unger 2011), make it a useful complement to the bullhead for higher salinity waters of the Chesapeake Bay watershed.

This section compiles and summarizes wildlife information in Chapter 2 to provide an overall assessment of contaminant exposure and responses in wildlife in the Chesapeake Bay watershed. The vast majority of the studies focus on birds.

On the basis of earlier reviews (Ohlendorf and Fleming 1988; Heinz and Wiemeyer 1991; Rattner and McGowan 2007) and the present analysis, tissue concentrations of organochlorine pesticides and their metabolites have declined and widespread adverse reproductive effects on Chesapeake Bay waterbirds have subsided. Nevertheless, in a few locations concentrations of organochlorine pesticides remain elevated, eggshell thinning associated with *p,p'*-DDE is apparent, reproduction may be impaired, and organochlorine pesticide-linked lethality has even been observed in high-trophic-level predatory birds (reviewed in Rattner and McGowan 2007). In numerous instances, organophosphorus and carbamate pesticides have been linked to avian die-off events in agricultural regions of the Chesapeake Bay watershed (Rattner and McGowan 2007), although use of the most hazardous anticholinesterases (e.g., granular formulations of carbofuran and diazinon, parathion) has been curtailed. Residue data in wildlife are not available for many categories of pesticides (e.g., herbicides, fungicides, rodenticides), likely because most have very low potential to bioaccumulate and have short environmental half-lives. The toxicological effects of chronic low-level exposure to newer pesticides and mixtures are unknown.

Concentrations of PCBs in tissues of many species of Chesapeake Bay wildlife have not declined since the final USEPA rule restricting the manufacture, processing, and distribution became effective in 1979 (Rice et al. 2003). In some urbanized regions, exposure to PCBs appears to be substantial and molecular effects are apparent; these compounds may contribute to localized reproductive problems (Rattner et al. 1997; Rattner and McGowan 2007; Mojica and Watts

2008, 2011). Concentrations of dioxins and dibenzofurans, which evoke toxicity by a similar mechanism to coplanar PCB congeners, are low in peregrine falcon eggs (Clark et al. 2009), but limited sampling-location data and the absence of exposure information in other species do not permit Bay-wide conclusions.

Moderate to high concentrations of polybrominated diphenyl ether flame retardants (PBDEs) have been detected in eggs from predatory birds in the Chesapeake Bay watershed, and approach the lowest-observed-adverse-effect level for pipping and hatching success (Rattner et al. 2004; Chen et al. 2010). Because of their documented persistence and tendency to bioaccumulate, along with concerns about potential toxicity, certain penta- and deca-BDE formulations were phased out in 2004 and deca-BDE formulations will be phased out by the end of 2013 (USEPA 2012a). Exposure and effects data for other groups of new flame retardants (e.g., hexabromocyclododecane and organophosphate flame retardants including tris(3,5-dimethylphenyl)phosphate and tris(1,3-dichloro-2-propyl)phosphate) are lacking for Chesapeake Bay wildlife. Some exposure monitoring has been undertaken with surfactants, such as alkylphenols and ethoxylates, and concentrations of perfluorinated compounds were found to be low, and seemingly well below effect thresholds in wildlife (Rattner et al. 2004).

On average there are about 500 reportable oil spill events in the Chesapeake Bay annually, but no major spill events have resulted in large-scale wildlife die-offs. Small spills and seepage of oil from marine vessels, and possibly pump facilities and industrial sites, are thought to be pervasive. The effects of chronic exposure to aliphatic and aromatic hydrocarbons in Chesapeake Bay wildlife have not been monitored, and are unknown.

Despite nationwide interest and concern about potential effects of household products, personal care products, and pharmaceuticals released from wastewater-treatment plants, septic systems, combined sewer outflows, and landfills, there have been no studies examining exposure and potential effects of these chemicals on Chesapeake Bay wildlife. Efforts are currently underway that are examining a suite of compounds (analgesics, antibiotics, anti-inflammatories, antihypertensives, antihistamines, antilipemics, antiseizures, anticoagulants, stimulants, psychostimulants, parasiticides, antidepressants, and antiseptics) in blood of osprey nestlings (Lazarus et al. 2012). The extent of exposure and potential effects of these compounds on wild birds is as yet unknown.

Although use of lead shot for hunting waterfowl was phased out by 1991, lead exposure and its resulting adverse effects are not uncommon in the Chesapeake Bay watershed, particularly in industrialized regions. However, exposure to other metals, including cadmium, mercury, and arsenic, do not seem to be a significant stressor to wildlife in most of the Chesapeake Bay and its tributaries (Rattner and McGowan 2007).

Interest in the effects of biotoxins, particularly those associated with harmful algal blooms (HABs), on wildlife has increased. Since 2001, a protracted series of heron die-offs that may be linked to microcystins, a potent hepatotoxin from cyanobacteria found in harmful algal blooms, has occurred (Rattner et al. 2006). Global climate change could cause an increase in HABs in the Chesapeake Bay watershed, and adverse effects on waterbird health may be a prominent issue in the foreseeable future (O’Neill et al. 2012).

Summary of Extent and Severity of Occurrence of Contaminants

Based on the information in Chapter 2, ten contaminant groups are summarized in terms of extent and severity.

Extent: Extent was characterized as "widespread, localized, or uncertain" and depended on a series of considerations using readily available information. If information on the occurrence of a contaminant group was acquired, an assessment of local versus widespread extent was made. However, if information was limited, the assessment of extent could only be classified as localized or uncertain.

- **Widespread extent:** For polychlorinated biphenyls (PCBs), polycyclic aromatic hydrocarbons (PAHs), herbicides (primarily atrazine, simazine, metolachlor, and their degradation products), and mercury, available information indicates widespread extent of occurrence throughout the Bay watershed.
- **Localized extent:** For dioxins/furans, petroleum hydrocarbons, some chlorinated insecticides (aldrin, chlordane, dieldrin, DDT/DDE, heptachlor epoxide, mirex), and some metals (aluminum, chromium, iron, lead, manganese, zinc), available information indicates localized extent of occurrence.
- **Uncertain extent:** For pharmaceuticals, household and personal-care products, polybrominated diphenyl ether (PBDE) flame retardants, some pesticides, and biogenic hormones, available information is insufficient to determine extent of contamination. However, the widespread distribution of known sources of these contaminants (e.g., wastewater effluents, agricultural runoff, etc.) in the watershed and the summarized occurrence data indicate that some contaminants from each of these groups may have the potential to be found in many locations throughout the Bay watershed.

Severity: Contaminant groups (or constituents) having impairments identified at many locations were considered widespread. Contaminants with impairments in a few locations are classified as having localized severity. All other contaminant groups are classified as uncertain.

- **Widespread severity:** For PCBs and mercury, impairments have been identified in many locations in the watershed, largely in response to concentrations in sediments and in fish tissues that frequently result in the need for fish-consumption advisories.
- **Localized severity:** For dioxins/furans, PAHs, petroleum hydrocarbons, some chlorinated pesticides (aldrin, chlordane, dieldrin, DDT/DDE, heptachlor epoxide, mirex), and some metals (aluminum, chromium, iron, lead, manganese, zinc), the report identifies localized severity on the basis of impairments in a limited number of areas in the Bay watershed.
- **Uncertain severity:** For atrazine, some pharmaceuticals, some household and personal-care products, some PBDEs, and biogenic hormones, severity as defined in this report could not be assessed. However, recent peer-reviewed research has documented sublethal effects for some compounds at environmentally relevant concentrations, raising concerns about the potential for adverse ecological effects.

The availability of Chesapeake watershed monitoring data and established toxicity benchmarks is variable for the toxic contaminant groups assessed in this report. For some pollutants such as PCBs, and mercury, abundant data indicate extensive environmental distribution at concentrations that result in impairments and fish consumption advisories. PAHs and herbicides are also widespread throughout the system. Impairments for PAHs exist in some jurisdictions and therefore the severity was defined as localized. For herbicides severity, as defined in this report, is classified as uncertain although the published literature has reported effects on organisms exposed to concentrations detected in the watershed. For an additional group of pollutants-dioxin and furans, petroleum hydrocarbons, chlorinated insecticides and some metals-localized areas of contamination have been identified at concentrations resulting in impairment designations. For other contaminant groups such as certain pharmaceuticals, household products, personal care products, flame retardants and biogenic hormones, there are data available that suggest that these contaminants are in the Bay system; however, more monitoring data and effects research would be needed to assess the severity of effects. Finally, there are groups of toxic contaminants such as certain pesticides and pharmaceuticals for which monitoring data gaps exist.

The assessment made through this report has noteworthy limitations. It relies on available data from state and Federal environmental monitoring, most of which is short term in nature and does not allow for temporal assessment of trends. While the report focused mainly on data specific to Chesapeake Bay published after 2000, peer-reviewed literature that included data from outside the Chesapeake Bay watershed was used to provide additional perspective for contaminants that were either not monitored in the Chesapeake Bay watershed or where monitoring data were spatially insufficient. The cost of analytical tests for detection, quantification, and biological effects of toxic contaminants is significant and, therefore, the spatial coverage of the data is limited even where monitoring programs exist. The method that underlies the report's conclusions is single-stressor oriented (or groups of chemically similar stressors such as congeners of PCBs) and does not consider the risk presented by additive, synergistic, or antagonistic effects of multiple chemical, biological, or physical stressors, nor does it address the growing body of toxicological research on sublethal effects. The latter is a major limitation to present paradigms in adequately addressing the severity of toxic contaminants in the environment. Where appropriate, references to this limitation have been discussed and relevant published research has been cited.

In conclusion, sufficient assessment and occurrence data were available to characterize a limited number of contaminant groups assessed as "widespread" or "local" in extent and severity. For these contaminant groups, this report can be used by the CBP partnership and others as an information resource to help inform decisions regarding whether to establish reduction goals and strategies. For other contaminants, further monitoring and research would better inform the understandings of the extent and(or) severity of effects. The summary table below (Table 2) illustrates these conclusions.

Table 2. Summary of extent, severity, and monitoring and research gaps for specific contaminant groups in the Chesapeake Bay watershed.

Quantity of Information (available throughout the watershed or limited)	Geographic Extent (localized, widespread, or uncertain)	Severity (localized, widespread, or uncertain)	Monitoring and Research Gaps
Polychlorinated Biphenyls			
Information available throughout the Chesapeake Bay watershed	Widespread. Detected in fish tissue and sediment.	Widespread. Impairments in all jurisdictions and multiple water bodies in most jurisdictions are subject to fish consumption advisories.	None* (see footnote)
Dioxins and Furans			
Limited information to define extent.	Localized. Detected in Elizabeth River, North Branch of the Potomac (WVA and MD) and in C&D Canal (DE).	Localized. Impairments in VA (Elizabeth River) and DE (C&D Canal) based on fish consumption advisories.	Monitoring data are limited to determine if extent is more widespread.
Polycyclic Aromatic Hydrocarbons			
Information available throughout the Chesapeake Bay watershed	Widespread. Detections mostly in sediment in all jurisdictions including tidal tributaries.	Localized. Impairments limited to two areas (Patuxent River segments and DC stream segments). Evidence of adverse effects (such as liver tumors in fish) in several locations (Anacostia River and Elizabeth River).	None* (see footnote)
Petroleum Hydrocarbons			
Limited information for the Chesapeake Bay watershed	Localized. Mostly associated with areas with heavy boating or shipping activities.	Localized. Impairments in DC and PA (based on oil and grease).	Monitoring data are limited to determine if extent is more widespread. Concentration data are typically present in the form of a surrogate parameter "oil and grease".

Quantity of Information (available throughout the watershed or limited)	Geographic Extent (localized, widespread, or uncertain)	Severity (localized, widespread, or uncertain)	Monitoring and Research Gaps
Pesticides			
Information available throughout the Chesapeake Bay watershed for some compounds. Others compounds have limited information.	Widespread. Herbicides that are in use, primarily atrazine, simazine, metolachlor and their degradation products, which have been detected in streams and the Bay waters. Localized. Pesticides not currently in use, primarily aldrin, chlordane, dieldrin, DDT/DDE, heptachlor epoxide, mirex, and their degradation products. Detected mostly in sediment and fish tissues. Uncertain. Limited monitoring information for many other pesticides including some insecticides and fungicides. However, the widespread extent of known or suspected sources of these contaminants and the existing occurrence data suggest the potential to be found in many locations throughout the watershed.	Localized. Organochlorine pesticides not in use (see extent) still causing impairments at a few locations. Uncertain. Water quality standards not available to determine impairments for some compounds. Some research shows sublethal effects of some compounds (e.g. atrazine) at environmentally relevant concentrations.	Monitoring data for many current use pesticides (e.g. some insecticides and fungicides) are not widely available and represent a data gap. Understanding the sub-lethal effects of individual (e.g., simazine and metolachlor) and mixtures of pesticides, degradation products, and the adjuvants used in their formulations on resources in the Bay and its watershed is a research gap.
Pharmaceuticals			
Limited information for the Chesapeake Bay watershed.	Uncertain. Limited monitoring information for pharmaceuticals. However, the widespread extent of known or suspected sources of these contaminants (e.g. human and animal waste management activities and infrastructure) and the existing occurrence data suggest the potential to be found in many locations throughout the watershed.	Uncertain. Water quality standards not available to determine impairments for these compounds. A growing number of studies show sublethal effects of some compounds (e.g. some antidepressants, ciprofloxacin, sulfamethoxazole, and synthetic hormones) at environmentally relevant concentrations.	Monitoring data for pharmaceuticals are not widely available and represents a data gap. Understanding the effects of individual and mixtures of pharmaceuticals, degradation products, and the adjuvants used in their formulations on resources in the Bay and its watershed is a research gap.

Quantity of Information (available throughout the watershed or limited)	Geographic Extent (localized, widespread, or uncertain)	Severity (localized, widespread, or uncertain)	Monitoring and Research Gaps
Household and Personal Care Products (HPCPs)			
Limited information for the Chesapeake Bay watershed.	Uncertain. Limited monitoring information for HPCPs. However, the widespread extent of known or suspected sources of these contaminants (e.g. human waste management activities and infrastructure) and the existing occurrence data suggest the potential to be found in many locations throughout the watershed.	Uncertain. Water quality standards not available to determine impairments for some compounds. Some research shows sublethal effects of some compounds (e.g. surfactants, fragrances, triclosan and triclocarban) at environmentally relevant concentrations.	Monitoring data for HPCPs are not widely available and represents a data gap. Understanding the effects of individual and mixtures of HPCPs and their degradation products on resources in the Bay and its watershed is a research gap.
Polybrominated Diphenyl Ether Flame Retardants			
Limited information for the Chesapeake Bay watershed.	Uncertain. Limited monitoring information does not allow determination of extent.	Uncertain. Water quality standards not available to determine impairments. Some research shows sublethal effects at environmentally relevant concentrations.	Limited monitoring information does not allow determination of extent. Water quality standards do not exist and research is limited on effects on resources in the Bay and its watershed.
Biogenic Hormones			
Limited information for the Chesapeake Bay watershed.	Uncertain. Limited monitoring information for biogenic hormones. However, the widespread extent of known or suspected sources of these contaminants (human and animal waste management activities and infrastructure) and the existing occurrence data suggest the potential for them to be found in many locations throughout the watershed.	Uncertain. Water quality standards not available to determine impairments for these compounds. Some research shows sublethal effects of biogenic hormones at environmentally relevant concentrations.	Monitoring data for biogenic hormones are not widely available and represents a data gap. Understanding the effects of individual and mixtures of biogenic hormones and their degradation products on resources in the Bay and its watershed is a research gap.

Quantity of Information (available throughout the watershed or limited)	**Geographic Extent (localized, widespread, or uncertain)**	**Severity (localized, widespread, or uncertain)**	**Monitoring and Research Gaps**
Metals and Metalloids			
Information available throughout the Chesapeake Bay watershed	Widespread. Mercury Localized. Aluminum, chromium, iron, lead, manganese, zinc.	Widespread. Mercury impairments noted primarily in freshwater fish tissue. Localized. Aluminum, chromium, iron, lead, manganese, zinc. Freshwater and sediment impairments in several jurisdictions and some exceedances of State standards.	None* (see footnote)

** "None" for research and monitoring gaps indicates that available information is sufficient to define extent and severity. Additional monitoring could be used to define sources and further research may be helpful to address sublethal effects on resources in the Bay and its watershed.*

Biological Effects of Toxic Contaminants on Fish and Wildlife

Additional supporting information on the toxic effects of contaminants on fish and wildlife is summarized to contribute to further understanding severity. This information provides insights for assessing the cumulative and interacting effects of toxic chemicals as well as other stressors on fish and wildlife.

The following indicators of compromised fish health have been observed within fish populations in the Chesapeake Bay watershed: increased incidence of infectious disease and parasite infestations contributing to increased mortality in several species of fish; feminization (intersex, plasma vitellogenin) of largemouth and smallmouth bass and other signs of endocrine disruption; reduced reproductive success and recruitment of yellow perch in certain highly urbanized tributaries; and tumors in bottom dwelling fish. The evidence for associations between toxic contaminant exposure and these indicators of compromised fish health is discussed.

For wildlife in the Chesapeake Bay watershed, there are also indications of responses to contaminant exposure, primarily in wild birds. In a few locations, eggshell thinning associated with *p,p'*-DDE is apparent and reproduction may be impaired. There are also cases where organochlorine pesticide concentrations in eggs of predatory birds are at concentrations associated with embryo lethality. Several studies are cited in which PCB concentrations in addled bald eagle eggs may have been high enough to contribute to the failure to hatch. Detectable concentrations of PBDEs have been found in eggs from predatory birds that approach the lowest-observed-adverse-effect level for pipping and hatching success.

Considerations for the Development of Federal-State Toxic Reduction Goals and Strategies

The findings in this report will be used during 2013 by the CBP partnership as an information resource to inform decisions regarding whether to establish new goals for reducing concentrations of toxic contaminants. If goals are established, this information resource can be used to inform decisions related to development of strategies by 2015 to achieve the goals. Considerations for development of goals and reduction strategies could also include:

Sources – Individual contaminants have unique profiles with regard to the origin of the ongoing inputs and bioavailability in the watershed. Some contaminant groups, such as pesticides, have overlapping and varied sources and pathways to the environment. Developing a basic understanding of the relative magnitude of sources as well as the nature of their environmental pathways for individual and groups of contaminants is critical in determining the extent to which reductions can be achieved. Without this first step, reduction goals and actions might be limited to addressing the symptoms of the problem rather than the root sources.

Regulatory and Voluntary Controls – For each contaminant group and pathway, consideration of the current regulatory and/or voluntary controls that can be applied will allow for informed decisions regarding the strengths and limitations of specific reduction goals and actions.

Technology Limitations and Opportunities – In many cases, technology limitations, green chemistry, sustainable agricultural and other sustainable practices, wastewater and drinking water treatment, and best management practices will influence the extent to which reductions can be expected. Therefore, opportunities for developing and applying new technologies with an emphasis on prevention could be considered and promoted through goals and strategies if developed. However, technological advances alone are not likely to completely reduce the risk of toxic contaminants and therefore adequate attention to socioeconomic and cultural aspects related to increases in existing and new sources of these toxic contaminants could be acknowledged and addressed.

Resource Limitations and Opportunities – Both state and federal entities charged with identifying pollutants responsible for impairments and pursuing policies and programs to address those pollutants face significant resource limitations. This is why the CBP, with its high level of both Federal and State leadership and authorities, represents a unique opportunity for resource allocation decisions that would allow progress to be made in reducing inputs of the pollutants identified in this report. Goals and strategies, if developed, should be considerate of resource limitations but also not underestimate the opportunities that exist for creative solutions that may result in the application of new resources.

Competing Priorities – In the Chesapeake Bay and its watershed, there are ongoing efforts of a historically large scale to address other high priority pollutants (e.g., nutrients and sediments) that impact the ecological success of the system. Goals and strategies would be attenuated by this fact; however, this report suggests it is appropriate for CBP to consider reaffirming goals for a subset of the toxic contaminants that are also compromising the system, particularly those that are widespread and occurring at concentrations that are likely to have adverse ecological effects. High level policy decisions would be needed on priorities. Depending on the pattern of contaminant distribution in the system, there may be a need for support of all phases of goal setting and strategy implementation across jurisdictional boundaries.

Summary of Monitoring and Research Gaps

Monitoring gaps were identified for the following contaminant groups: dioxins and furans; petroleum hydrocarbons, some pesticides currently in use (e.g. insecticides and fungicides), pharmaceuticals, household and personal care products, flame retardants, and biogenic hormones. Biological monitoring at many levels of biological organization (sub-organisms to populations) along with systematic evaluation of water contaminants and other stressors would allow for more effective documentation of extent and severity.

Research that accounts for the complexities of mixture and multiple stressor effects, sublethal effects, nonlinear dose response curves, the role of contaminant exposures in immune response and subsequent pathogenic disease, would better define relations between contaminant exposures and potential effects in fish and wildlife. In addition, research gaps were identified that limit understandings of the relations between sources of these contaminants, their pathways to the environment, and exposures to receptor organisms.

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Table A-1: Water column standards - Aquatic life protection (µg/L)

	Jurisdiction Partner						
Parameters	**DC**	**DE**	**MD**	**NY**	**PA**	**VA**	**WV**
PCBs							
Polychlorinated Biphenyls PCBs	FC: 0.014	FC: 0.014 SC: 0.03	FC: 0.014 SC: 0.03		FC: 0.014	FC: 0.014 SC: 0.03	FC: 0.014
PAHs							
Acenaphthene	FC:50				FA: 83 FC: 17		
Benzidine	FC: 250			FA: 0.1 FC:0.1	FA: 300 FC: 59		
Benzo(a)Anthracene					FA. .05 FC: .01		
2-Chloronaphthalene	FC: 1600						
Fluoranthene	FC: 400				FA: 200 FC: 43		
Naphthalene	FC: 600 FA: 1.1				FC: 140 FA: 43		
Phenanthrene					FA: 5 FC: 1		
Pesticides and Chlorinated Compounds							
4,4'-DDD	FA: 1.1 FC: 0.001				FA: 1.1 FC: 0.001		
4,4'-DDE	FA: 1.1 FC: 0.001				FA: 1.1 FC: 0.001		
4,4'-DDT	FA: 1.1 FC: 0.001		FA: 1.1 FC: 0.001 SA: 0.13 SC: 0.001		FA: 1.1 FC: 0.001	FA: 1.1 FC: 0.001 SA: 0.13 SC: 0.001	FA: 1.1 FC: 0.001
DDT and metabolites		FA: 1.1 FC: 0.001 SA: 0.13 SC: 0.001					
Aldrin	FA: 3.0 FC: .04	FA: 3.0 SA: 1.3	FA: 3.0 SA: 1.3		FA: 3.0 SA: 0.1	FA: 3.0 SA: 1.3	FA: 0.003
alpha-BHC			SC: 0.026				
alpha-Endosulfan	FA: 0.22 FC: 0.056		FA: 0.22 FC: 0.056 SA: 0.034 SC: 0.0087		FA: 0.22 FC: 0.056	FA: 0.22 FC: 0.056 SA: 0.034 SC: 0.0087	
beta-BHC					FA: 0.22 FC: 0.056		
beta-Endosulfan	FA: 0.22 FC: 0.056		FA: 0.22 FC: 0.056 SA: 0.034 SC: 0.0087			FA: 0.22 FC: 0.056 SA: 0.034 SC: 0.0087	
Chlordane	FA: 2.4 FC: 0.0043	FA: 2.4 FC: 0.0043 SA: 0.09 SC: 0.004	FA: 2.4 FC: 0.0043 SA: 0.09 SC: 0.004		FA: 2.4 FC: 0.0043	FA: 2.4 FC: 0.0043 SA: 0.09 SC: 0.004	FA: 2.4 FC: 0.0043
Chlorpyrifos		FA: 0.083 FC: 0.041 SA: 0.011 SC: 0.0056	FA: 0.083 FC: 0.041 SA: 0.011 SC: 0.0056			FA: 0.083 FC: 0.041 SA: 0.011 SC: 0.0056	
p-Cresol					FA: 800 FC: 160		
Demeton		FC: 0.10 SC: 0.10		FC: 0.1		FC: 0.10 SC: 0.10	

Table A-1: Water column standards - Aquatic life protection (µg/L)

	Jurisdiction Partner						
Parameters	**DC**	**DE**	**MD**	**NY**	**PA**	**VA**	**WV**
Diazinon				FC: 0.08	FC: 0.17 FC: 0.17	FC: 0.17 FC: 0.17 SA: 0.82 SC: 0.82	
Dieldrin	FA: 0.24 FC: 0.056	FA: 0.24 FC: 0.056 SA: 0.71 SC: 0.0019	FA: 0.24 FC: 0.056 SA: 0.71 SC: 0.0019	FA: 0.24 FC: 0.056	FA: 0.24 FC: 0.056	FA: 0.24 FC: 0.056 SA: 0.71 SC: 0.0019	FA: 0.24 FC: 0.056
Endosulfan	FA: 0.22 FC: 0.56	FA: 0.22 FC: 0.56 SA: 0.034 SC: 0.0087		FC: 0.009			
Endrin	FA: 0.086 FC: 0.036	FA: 0.086 FC: 0.036 SA: 0.037 SC: 0.0023	FA: 0.086 FC: 0.036 SA: 0.037 SC: 0.0023	FA: 0.086 FC: 0.036	FA: 0.086 FC: 0.036	FA: 0.086 FC: 0.036 SA: 0.037 SC: 0.0023	FA: 0.18 FC: 0.0023
Endrin Aldehyde							
gamma - BHC (Lindane)	FA: 0.095 FC: 0.08		FA: 0.095 FC: 0.16	FA: 0.095	FA: 0.095	FA: 0.095 FC: 0.16	FA: 2.0 FC: 0.08
Guthion	FC: 0.01	FC: 0.01 SC: 0.01				FC: 0.01 SC: 0.01	
Heptachlor	FA: 0.52 FC: 0.0038	FA: 0.52 FC: 0.0038 SA: 0.053 SC: 0.0036	FA: 0.52 FC: 0.0038 SA: 0.053 SC: 0.0036		FA: 0.52 FC: 0.0038	FA: 0.52 FC: 0.0038 SA: 0.053 SC: 0.0036	
Heptachlor Epoxide	FA: 0.52 FC: 0.0038		FA: 0.52 FC: 0.0038 SA: 0.053 SC: 0.0036		FA: 0.5 FC: 0.0038	FA: 0.52 FC: 0.0038 SA: 0.053 SC: 0.0036	FA: 0.52 FC: 0.0038
Hexachlorocyclohexane		FA: 0.095 FC: 0.08 SA: 0.16					
Malathion	FC: 0.1			FC: 0.1		FC: 0.1 SC: 0.1	
Methoxychlor	FC: 0.03	FC: 0.03 SC: 0.03		FC: 0.03		FC: 0.03 SC: 0.03	FC: 0.03
Mirex	FC: 0.001	FC: 0.001 SC: 0.001				FA: ZERO FC: ZERO	
Parathion	FC: 0.013	FA: 0.065 FC: 0.013		FA: 0.065		FA: 0.065 FC: 0.013	
Toxaphene	FA: 0.73 FC: 0.0002	FA: 0.73 FC: 0.0002 SA: 0.21 SC: 0.0002	FA: 0.73 FC: 0.0002 SA: 0.21 SC: 0.0002	FA: 1.6 FC: 0.005	FA: 0.73 FC: 0.0002	FA: 0.73 FC: 0.0002 SA: 0.21 SC: 0.0002	FA: 0.73 FC: 0.0002
Tributyltin (TBT)	FA: 0.46 FC: 0.072		FA: 0.46 FC: 0.072 SA: 0.42 SC: 0.0074			FA: 0.46 FC: 0.072 SA: 0.42 SC: 0.0074	
Household and Personal Care Products							
Nonylphenol	FA: 28 FC: 6.6					FA: 28 FC: 6.6 SA: 7.0 SC: 1.7	
Inorganics							
Aluminum		FA: 750* FC: 87*		FC: 100	FA: 750		FA: 750 FC: 750
Antimony					FA: 1100 FC: 220		
Arsenic	FA: 340 FC: 150	FA: 340 FC: 150 SA: 69 SC: 36	FA: 340 FC: 150 SA: 69 SC: 36	FA: 340 FC: 150	FA: 340 ^ FC: 150 ^	FA: 340 FC: 150 SA: 69 SC: 36	FA: 340 ^ FC: 150 ^

Table A-1: Water column standards - Aquatic life protection (µg/L)

	Jurisdiction Partner						
Parameters	**DC**	**DE**	**MD**	**NY**	**PA**	**VA**	**WV**
Barium					FA: 21,000 FC: 4100		
Beryllium				FC: 11/1,100#			FA: 130
Cadmium	FA: 2.0** FC: 0.25**	FA: 2.0** FC: 0.25** SA: 40 SC: 8.8	FA: 2.0** FC: 0.25** SA: 40 SC: 8.8	FA: 3.8** FC: 2.1**	FA: 2.0** FC: 0.25**	FA: 3.9** FC: 1.1** SA: 40 SC: 8.8	FA: 2.0** FC: 0.25**
Chromium III	FA: 570** FC: 74**	FA: 570** FC: 74**	FA: 570** FC: 74**	FA: 570** FC: 74**	FA: 570** FC: 74**	FA: 570** FC: 74**	FA: 570** FC: 74**
Chromium Vi	FA: 16 FC: 11	FA: 16 FC: 11 SA: 1100 SC: 50	FA: 16 FC: 11 SA: 1100 SC: 50		FA: 16 FC: 11	FA: 16 FC: 11	FA: 16 FC: 11
Copper	FA: 13** FC: 9.0**	FA: 13** FC: 9.0** SA: 4.8 SC: 3.1	FA: 13 FC: 9.0 EA: 6.1 SA: 4.8 SC: 3.1	FA: 13** FC: 9.0**	FA: 13** FC: 9.0**	FA: 13** FC: 9.0** SA: 9.3 SC: 6.0	FA: 13** FC: 9.0**
Iron	FA: 1000	FC: 1000					FC: 1.5
Lead	FA: 65** FC: 2.5**	FA: 65** FC: 2.5** SA: 210 SC: 8.1	FA: 65** FC: 2.5** SA: 210 SC: 8.1	FA: 97** FC 3.8**	FA: 65** FC 2.5**	FA: 120** FC: 14** SA: 240 SC: 9.3	FA: 65** FC 2.5**
Mercury	FA: 1.4 FC: 0.077	FA: 1.4 FC: 0.077 SA: 1.8 SC: 0.94	FA: 1.4 FC: 0.77 SA: 1.8 SC: 0.94	FA: 1.4 FC: 0.77	FA: 1.2^^ FC: 0.77^^		FA: 2.4@
Methylmercury							FC: 0.012
Nickel	FA: 470** FC: 52**	FA: 470** FC: 52** SA: 74 SC: 8.2	FA: 470** FC: 52** SA: 74 SC: 8.2	FA: 470** FC: 52**	FA: 470** FC: 52**	FA: 180** FC: 20** SA: 74 SC: 8.2	FA: 470** FC: 52**
Selenium	FA: 20 FC: 5.0	FA: 20 FC: 5.0 SA: 290 SC: 71	FA: 20 FC: 5.0 SA: 290 SC: 71	FC: 4.6	FC: 4.6	FA: 20 FC: 5.0 SA: 290 SC: 71	
Silver	FA: 3.2**	FA: 3.2** SA: 1.9	FA: 3.2** SA: 1.9	FA: 4.1** SA: 0.1	FA: 3.2**	FC: 3.4** SA: 1.9	FA: 3.2**
Thallium				FA: 8 FC: 20	FA: 65 FC: 13		
Zinc	FA: 120** FC: 120**	FA: 120** FC: 120** SA: 90 SC: 81	FA: 120** FC: 120** SA: 90 SC: 81	FA: 120** FC: 83**	FA: 120** FC: 120**	FA: 120** FC: 120** SA: 90 SC: 81	FA: 120** FC: 120**

* Aluminum Standard: pH 6.5-9.0
^ Arsenic III
** hardness = 100 mg/L CaCO3
^^ Mercury II
@Total Mercury

#11 µg/L when hardness is ≤ ppm; 1,100 µg/L when hardness is > 75 ppm
FA: Freshwater Acute Standard
FC: Freshwater Chronic Standard
SA: Saltwater Acute Standard
SC: Saltwater Chronic Standard

Table A-2: Sediment Screening Guidelines (ppb dry weight) (Buchman 2008)

	Freshwater		Estuarine Waters	
Parameters	**TEC**	**PEC**	**ER-L**	**ER-M**
PCBs				
Polychlorinated Biphenyls PCBs	59.8	676	22.70	180
PAHs				
Low MW PAHs			552	3,160
High MW PAHs			1,700	9,600
Total PAHs	1,610	22,800	4,022	44,792
Acenaphthene			16	500
Acenaphthylene			44	640
Anthracene	57.20	845	85.30	1,100
Benzo(a)Anthracene	108	1,050	261	1,600
Benzo(a)Pyrene	150	1,450	430	1,600
Chrysene	166	1,290	384	2,800
Dibenzo(a,h)Anthracene	33		63.40	260
Fluoranthene	423	2,230	600	5,100
Fluorene	77.40	536	19	540
2-methyl naphthalene			70	670
Naphthalene	176	561	160	2,100
Phenanthrene	204	1,170	240	1,500
Pyrene	195	1,520	665	2,600
Pesticides and Chlorinated Compounds				
4,4'-DDD	4.88	28	2	20
4,4'-DDE	3.16	31.30	2.20	27
4,4'-DDT	4.16	62.90	1	7
DDx, total	5.28	572	1.58	46.10
Chlordane	3.24	17.60	0.50	6
Dieldrin	1.90	61.80	0.02	8
Endrin	2.22	207		
gamma-BHC (Lindane)	2.37	4.99		
Heptachlor Epoxide	2.47	16		
Inorganic				
Arsenic	9,790	33,000	8,200	70,000
Cadmium	990	4,980	1,200	9,600
Chromium (total)	43,400	111,000	81,000	370,000
Copper	31,600	149,000	34,000	270,000
Lead	35,800	128,000	6,700	218,000
Mercury	180	1,060	150	710
Nickel	22,700	48,600	20,900	51,600
Silver			1,000	3,700
Zinc	121,000.00	459,000	50,000	410,000