Linking Ecological and Human Health:
The Chesapeake Bay Health Indicators Project

Kristen Chossek Malecki, MPH
Polly Walker, MD, MPH
Thomas A. Burke, PhD, MPH
Beth A. Resnick, MPH

May 2004

The Center for a Livable Future

JOHNS HOPKINS BLOOMBERG SCHOOL of PUBLIC HEALTH

Johns Hopkins Center for a Livable Future

We urgently need to improve human health, prevent disease and disability around the world, and meet basic needs for food, water, and shelter. The challenge of our time is to solve these problems as the world's population and consumer demand continue to increase, while simultaneously protecting the environment, preserving biodiversity, and conserving our finite resources.

Center's Mission: To establish a global resource to develop and disseminate information and to promote policies for the protection of health, the global environment, and our ability to sustain life for future generations.

Contact Information

Center for a Livable Future
Johns Hopkins Bloomberg School of Public Health
615 North Wolfe Street, #W8503, Baltimore, MD 21205-2179
Tel: 410-502-7578 Fax: 410-502-7579 Email: clf@jhsph.edu
http://www.jhsph.edu/environment/

This report will be available in PDF format at:
http://www.jhsph.edu/environment/

Authors

Kristen Chossek Malecki, MPH
Doctoral Candidate, Department of Health Policy and Management
Project Director, Chesapeake Bay Health Indicators Project
Johns Hopkins Bloomberg School of Public Health

Polly Walker, MD, MPH
Associate Director, Center for a Livable Future
Research Associate, Department of Environmental Health Sciences
Johns Hopkins Bloomberg School of Public Health

Thomas A. Burke, Ph.D., MPH
Professor and Associate Chair,
Department of Health Policy and Management
Co-Director, Risk Sciences and Public Policy Institute
Johns Hopkins Bloomberg School of Public Health

Beth A. Resnick, MPH
Associate Director,
Center for Excellence in Environmental Public Health Tracking
Research Associate, Department of Health Policy and Management
Johns Hopkins Bloomberg School of Public Health

Cover printed on recycled paper; 30% post-consumer waste.
Text printed on 35% recycled post-consumer paper.

Tables

Figures

Appendices

Acknowledgements

We would like to thank the Center for a Livable Future and the Chesapeake Bay Foundation for their support and encouragement in this effort, particularly Dr. Robert Lawrence, Kelly Hoban Green, and Kim Coble. In addition, we would like to thank the members of our working group panel for their guidance at individual meetings and their participation in the Chesapeake Bay Health Indicators Project consultative symposium (December 4, 2002). We would also like to thank Joe Beaman at the Maryland Department of the Environment for their guidance and suggestions in indicator development.

Our thanks to:

Tory Ameree, M.D., MPH
Office of Environmental Health Coordination
Maryland Department of Health and Mental Hygiene

Amy Chapin, MPH
Ph.D. Candidate, Department of Environmental Health Sciences
Research Director
Spira/GRACE Project on Industrial Animal Agriculture, Center for a Livable Future
Johns Hopkins Bloomberg School of Public Health

Kim Coble, MSPH
Maryland Executive Director
Chesapeake Bay Foundation

Lynn Goldman, M.D., MPH
Professor
Department of Environmental Health Sciences
Johns Hopkins Bloomberg School of Public Health

Thaddeus Graczyk, M.Sc., Ph.D.
Associate Research Professor
Department of Molecular Microbiology and Immunology
Department of Environmental Health Sciences
Johns Hopkins Bloomberg School of Public Health

Phil Heard, M.D., MPH
Health Advisor
Environmental Health and Risk Assessment Program
Maryland Department of the Environment
Montgomery Park Business Center

Joseph G. Jacangelo, Ph.D.
Director, Center for Water and Health
Department of Environmental Sciences and Environmental Health Engineering
Johns Hopkins Bloomberg School of Public Health

Robert Lawrence, M.D.
Associate Dean, Professional Practice and Programs
Edyth H. Schoenrich Professor of Preventive Medicine
Director, Center for a Livable Future
Johns Hopkins Bloomberg School of Public Health

Dan Morhaim, M.D.
Delegate to Maryland General Assembly
District 11, Baltimore County

Devon Payne-Sturges, DrPH
Public Health and Environmental Policy Team
National Center for Environmental Economics
U.S. Environmental Protection Agency

Frances B. Phillips, RN, MHA
Health Officer
Anne Arundel County Health Department

Beth A. Resnick, MPH
Associate Director
Center for Excellence in Community Environmental Health
Johns Hopkins Bloomberg School of Public Health

Andrew D. Sawyers, Ph.D.
Environmental Justice and Community Development Coordinator
Maryland Department of the Environment

Kellogg J. Schwab, Ph.D.
Assistant Professor
Department of Environmental Health Sciences
Johns Hopkins Bloomberg School of Public Health

Ellen K. Silbergeld, Ph.D.
Professor
Department of Environmental Health Sciences
Johns Hopkins Bloomberg School of Public Health

William P. Stack, P.E.
Chief of Water Quality Management
Baltimore City Department of Public Works
Ashburten Water Treatment Plant

M. Gordon Wolman, Ph.D.
Professor and Chair Emeritus
Department of Geography and Environmental Engineering
Johns Hopkins University

Executive Summary

The Chesapeake Bay has a profound effect on all who reside in the 64,000 square miles of the watershed. More than just an ecological treasure, the Bay has also shaped historical development patterns for homes, industry, agriculture, and transportation. It is a driver of the regional economy, a food source, and receiver of waste waters. From the time when the first settlers arrived on the shores of the Chesapeake Bay in 1603, increased population growth and development within the region have led to continual degradation of the once pristine waterway. Human beings have played a critical role in altering the basic physical, chemical, and biological systems within the Chesapeake Bay and its watershed, and these shifts may ultimately be putting human health at risk.

The Chesapeake Bay Health Indicators Project is part of the ongoing efforts of the Center for a Livable Future (CLF) of the Johns Hopkins Bloomberg School of Public Health and the Chesapeake Bay Foundation to preserve and improve both the human health and the ecological health of populations living in and around the Chesapeake Bay watershed through improved recognition of the linkage between the quality of the environment and the protection of public health.

In the last decade despite significant advancements in the assessing and monitoring of environmental quality, less progress has been made in assessing the relationship between the state of the ecological environment and its impact on human health. The first ecological assessment of the Chesapeake Bay's "health" was completed with the signing of the Chesapeake Bay Agreement in 1983. Since the Chesapeake Bay Agreement was signed, monitoring of these ecological indicators of the Chesapeake Bay has continued, paving the way for better policy decisions and actions taken for the protection of the Bay's fragile ecosystem. However, a parallel set of indicators aimed at tracking risks to the human population within the ecosystem is lacking.

The *State of the Bay Report*, an annual report card measuring environmental quality of the Bay prepared by the Chesapeake Bay Foundation, was the impetus for developing a similar report card for population health. There is a need to develop an equivalent public health tool for tracking the human health impacts of environmental degradation throughout the watershed.

The goals of this project were to examine the relationship between adverse conditions in the ecological environment and health of the human population and to develop a pilot set of regional public health indicators. This paper presents an overview of health and ecological stressors in the Chesapeake Bay region and three case examples of environmental public health indicators that represent major challenges both to the ecology of the watershed and to human health.

Key Findings: An examination of pilot indicators underscores the link between the ecology of the Chesapeake Bay Watershed and the health of its inhabitants. The watershed has been impacted by pollutants that present both acute and chronic health risks.

Pilot indicators examined include measures of:

- **Drinking water protection**—Total trihalomethanes (TTHMs) in public drinking water supplies;
- **Microbial risks in surface waters**—Fecal coliform contamination at bathing beaches and in recreational and urban waterways; and
- **Persistent toxic pollutants**—Mercury and PCB contaminant levels in fish tissue.

These public health indicators can provide a foundation for tracking threats and strengthening efforts to protect and preserve the Bay. While there is still much to learn about the links between human health and the environment, the health of the Bay is vital to the health of the people. It must be emphasized that no immediate threats to public health were revealed through the case examples. At the same time, these indicators underscore the need for continued diligence in protecting and preserving the watershed and recognizing the linkages between human health and the environment. This has been a pilot investigation and these indicators offer only a small sample of potential public health indicators for the region. In the future, measures such as these may be part of a national tracking network of indicators to measure environmental progress, identify emerging hazards, shape research, and strengthen the scientific basis for environmental and health policies.

Case examples of potential environmental public health indicators in the Chesapeake Bay region demonstrate that tracking public health threats over time may be feasible and practical. Within the state of Maryland, there are a number of well-conducted ongoing monitoring efforts that can serve as the foundation for the development of environmental public health indicators. Gaps in these programs highlight several areas for future expansion. Better integration and use of these resources will improve the environmental and public health applications of indicators, and provide new insights into the link between the ecological environment and human health.

The time is right to begin developing Environmental Public Health Indicators (EPHI) for the Chesapeake Bay region. A national momentum for Environmental public health tracking is building and it may become the cornerstone of future environmental health practices. This pilot investigation serves as the first step in developing a public health report card for the Chesapeake Bay. It is hoped that lessons learned from this project will serve as the basis for future expanded efforts on the state and national level. Based on these findings, the following recommendations are presented as next steps to build upon this pilot investigation:

1) Assess information needs to assure effective coverage of the watershed and to meet the data needs of state and county health agencies and environmental officials.

Environmental and health officials should identify additional health and environmental data that is needed to address and prevent emerging public health risks related to environmental quality.

2) Expand the list of indicators to include a broader range of contaminants, additional exposure pathways, and improved measures of population exposure levels.

Indicators could be expanded to include other drinking water contaminants, private well sampling, additional indicators for pathogens, and more direct measurement of actual population exposure levels.

3) Enhance reporting of public health outcomes, such as waterborne and food-borne outbreaks to assure early problem recognition and to safeguard public health.

Improved outbreak surveillance could be developed in order to enhance prevention programs and complement current national efforts to improve identification of emerging population health threats.

4) Coordinate efforts with the EPA Environmental Indicators Initiative and the CDC National Environmental Public Health Tracking Network[i].

These developing national programs may provide opportunities for comparisons with national baseline data, uniform national reporting strategies, and enhanced resources for the development of a national network of environmental public health indicators.

5) Develop a formal strategy for systematic regular reporting of the pilot public health indicators through a public health report card for the Bay region.

This should complement ecological reporting efforts and include mapping and enhanced communication efforts to increase public awareness and provide appropriate public health perspectives regarding potential risks.

The Chesapeake Bay is the region's defining natural resource. Improved tracking of sources of pollution, exposures, and health effects is an essential component of an integrated approach to protecting the Bay and protecting the public's health.

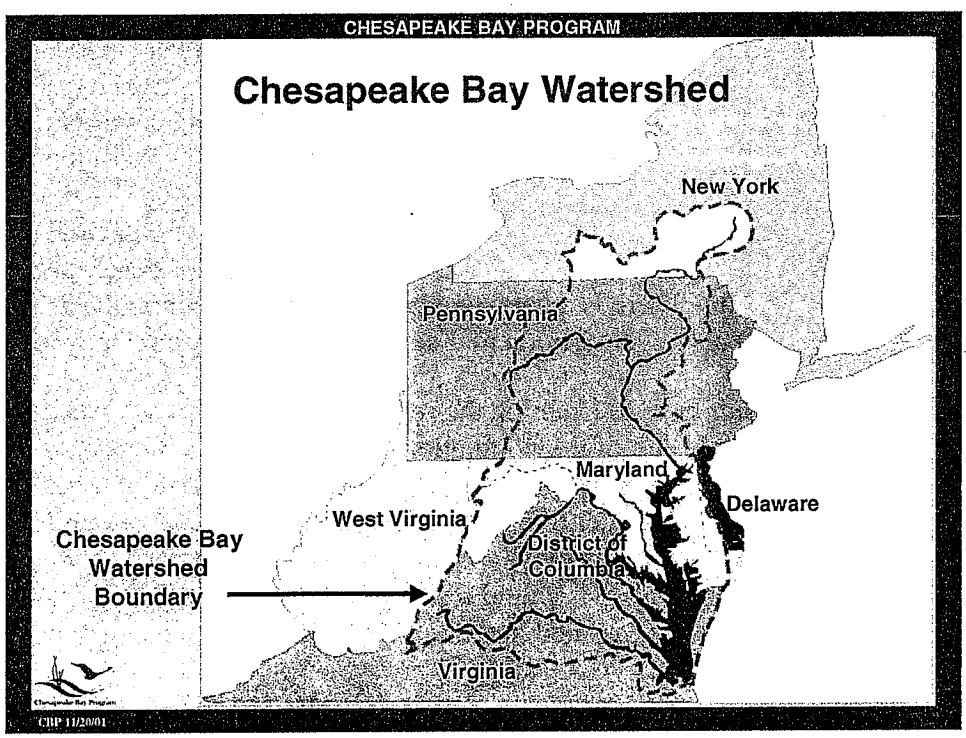
[i] Additional information available from the National Centers for Disease Control National Center for Environmental Health at: *http://www.cdc.gov/nceh/tracking/*

I. Background

The Chesapeake Bay (the Bay) has a profound impact on all who reside in the 64,000 square miles of its watershed. In the words of Major John Wesley Powell, scientist, geographer, river surveyor and former Director of the National United States Geological Service, a watershed is "that area of land, a bounded hydrologic system, within which all living things are inextricably linked by their common water course and where, as humans settled, simple logic demanded they become part of a community."[1,2] More than just an ecological treasure, the Bay has also shaped historical development patterns for homes, industry, agriculture, and transportation. It is a driver of the regional economy, a food source, and receiver of waste waters.

The Chesapeake Bay region encompasses the entire Chesapeake Bay watershed. Over 150 rivers and streams in the watershed are spread throughout six states: New York, Pennsylvania, Maryland, Delaware, Virginia, and West Virginia. The Bay is the largest of the 130 estuaries in the United States, with a watershed that encompasses approximately 11,684 miles of shoreline and is home to more than 3,600 species of plants and animals, including fish and humans.[3] The region serves as a drinking water source for 16 million people and contributes $31.6 billion dollars annually to Maryland and Virginia, making it a primary economic and cultural resource for these states.[4]

Figure 1: The Chesapeake Bay Watershed



Preserving the Bay, its watershed, and its entire ecosystem is essential to protecting the health and well-being of the public within the Chesapeake Bay region. From the time when the first settlers arrived on the shores of the Chesapeake Bay in 1607, increased population growth and development within the region have led to continual degradation of the once pristine waterway.[5,6] Within the region, human health and the environment have been inextricably linked

ever since cholera epidemics wiped out the first colonies established along the banks of the James River. Later, poor sanitation and adverse water quality conditions in Baltimore City led to the foundation of the first Board of Health for the state of Maryland in Baltimore City. [7] Human beings have played a critical role in altering the basic physical, chemical and biological systems within the Chesapeake Bay and its watershed, and these shifts may ultimately be putting human health at risk.[8,9,10,11,12]

In the last decade, despite significant advancements in the assessing and monitoring of environmental quality, less progress has been made in assessing the relationship between the state of the ecological environment and its impact on human health. The first ecological assessment of the Chesapeake Bay's "health" was completed with the signing of the Chesapeake Bay Agreement in 1983. At that time, several key problem areas were identified in relation to diminished wildlife habitat, natural resources such as wetlands and fish species, and the chemical and physical properties of the Bay. Since the Chesapeake Bay Agreement was signed, monitoring of these ecological indicators of the Chesapeake Bay has continued, paving the way for better policy decisions and actions taken for the protection of the Bay's fragile ecosystem. However, a parallel set of indicators aimed at tracking health risks to the human population within the same ecosystem is lacking.

The Chesapeake Bay Health Indicators Project is part of the ongoing efforts of the Center for a Livable Future (CLF) of the Johns Hopkins Bloomberg School of Public Health and the Chesapeake Bay Foundation (CBF) to preserve and improve both the human health and the ecological health of populations living in and around the Chesapeake Bay watershed through improved recognition of the linkage between the quality of the environment and the protection of public health. The 1999 CLF and CBF colloquium "Health of the Bay — Health of People" laid the groundwork for this research by underscoring the need for developing environmental public health indicators to complement the ongoing ecological tracking of the health of the Bay.[10]

The *State of the Bay Report*, an annual report card measuring environmental quality of the Bay prepared by the Chesapeake Bay Foundation, was the impetus for developing a similar report card for population health. There is a need to develop an equivalent public health tool for tracking the human health impacts of environmental degradation throughout the watershed.[13]

The goals of this project were to examine the relationship between adverse conditions in the ecological environment and health of the population and to develop a pilot set of regional public health indicators. Research methods included an assessment of primary threats to the ecology of the Bay and human health, identification of criteria for selection of public health indicators, evaluation of existing data sources, and presentation of key environmental public health indicators specific to Maryland. (Maryland residents represent 5 million of the 16 million regional residents living within the watershed and the majority of residents living along the shores of the Bay.)

This paper presents an overview of health and ecological stressors in the Chesapeake Bay region and three case examples of environmental public health indicators that represent major challenges both to the ecology of the watershed and to human health. It is hoped that the effort may serve as a foundation for future expanded efforts to track progress in improving both population health and the environment in the Chesapeake Bay region and to provide a foundation for other regional and national efforts to link environment and health.

II. Overview of Threats to the Chesapeake Bay Watershed and Human Health

Nutrients, sediments, toxic chemical contamination, air pollution, and landscape changes are the top pollution threats and stressors to the overall Chesapeake Bay ecosystem identified by the Chesapeake Bay Program Office (CPO) of the United States Environmental Protection Agency (USEPA).[14] All of these threats have the potential to adversely affect the public's health. Many are tracked through existing measures that may provide a practical and feasible way to begin monitoring and reporting on the link between public health and the state of the Chesapeake Bay (see Sections III–V). At the same time, many other threats and their potential health outcomes are not systematically monitored and assessed over time, leaving many unanswered questions regarding the relationship between adverse environmental quality in the Bay region and its impact on human health.

Population Growth and Urban Sprawl

Population growth and related development are the root causes of the environmental changes in the Bay over the past four centuries, impacting virtually every pollution concern throughout the watershed, and affecting the health and quality of life of all in the region. Today, approximately 16 million people live within the Chesapeake Bay watershed. Projections by the Chesapeake Bay Program estimate that the population will grow to 18 million people by 2020. In Maryland alone, a 28% increase in population is projected by 2020.[15] While it is difficult to link population growth or sprawl to specific health endpoints, there is clear evidence of the associated environmental degradation and growing concern about a broad range of potential adverse impacts. Table 1 identifies the pollution sources impacting the health of the Chesapeake Bay and its watershed that arise from sprawl within the Chesapeake Bay region.[10]

Table 1: Sources of Pollution Impacting the Chesapeake Bay

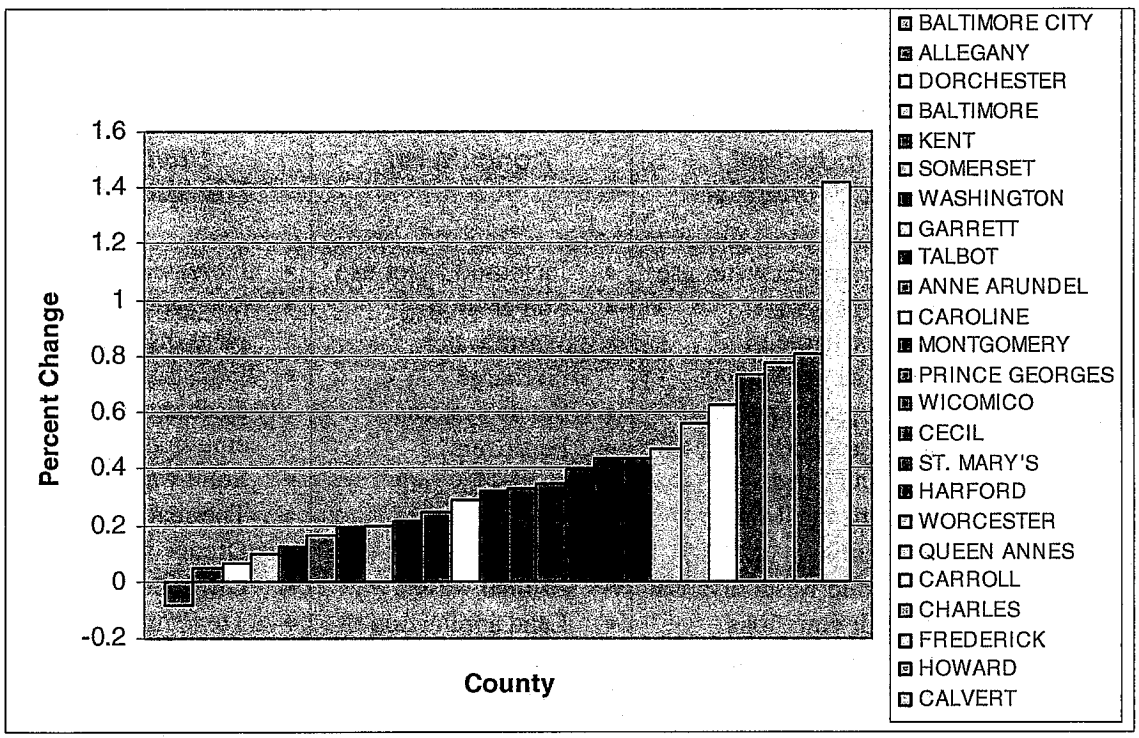
Residential	***Industrial / Commercial***	***Agricultural***	***Utilities / Transportation***
• Increased water use • Municipal sewage • Solid waste disposal • Septic systems • Lawn chemicals • Pesticides • Fertilizers • Increased runoff • Wetlands loss • Increased vehicle traffic	• Increased water use • Waste water discharge to treatment plants • Septic systems • Direct discharge to watershed • Chemical waste • Spills • Solid waste disposal • Air emissions • Increased runoff • Wetlands loss	• Increased water use • Soil erosion • Waste water discharge • Septic systems • Fertilizers • Pesticides and agricultural chemicals • Animal waste • Land application of waste and sludge • Nutrient runoff	• Increased water use • Sewage treatment plant discharge • Industrial waste discharge and emissions • Solid waste disposal • Thermal pollution • Dredge spoils • Oil spills • Toxic material spills • Increased runoff • Airborne nitrogen pollutants

Source: Burke, T. A., J. S. Litt, et al. (2000)[10]

Urban sprawl and its impact on the region's landscape have been designated by the USEPA's Chesapeake Bay Program Office (CBPO) as the leading threats to the survival and recovery of the Chesapeake Bay. Population trends in Maryland show increasing growth in rural areas, with more limited growth in traditional population centers such as Baltimore City. Figure 2 presents a projection of percent population growth within each county from 1990–2020, provided by the CBPO. It is estimated that by the year 2020, Carroll, Charles, Frederick, Howard and Calvert Counties will all have more than a 60% increase in population within a thirty-year time span. At the same time, population growth in urban areas such as Baltimore City is on the decline. This sprawl affects water quality in the entire watershed by increasing the amount of impervious ground cover, increasing non-point discharges, reducing capacity for absorption of nutrients from agriculture, increasing the number of vehicle miles traveled, and increasing waste water pollution. In addition, the habitat degradation resulting from development and deforestation adversely impacts the Bay region's wildlife.[15]

There are several ongoing regional efforts to track population growth and trends, the impact of these changes on urban sprawl, and subsequent effects on the ecological environment. These include projects at the Chesapeake Bay Program office of the EPA, the Chesapeake Bay Foundation, and several other academic institutions.[16] These initiatives are important complements to developing and enriching the utility of environmental public health indicators. All aspects of the ecological health of the Bay are affected by population growth and subsequent development; therefore, tracking trends of growth should accompany future indicator development.

Figure 2: Projected Percent Population Change in Maryland (1990–2020)

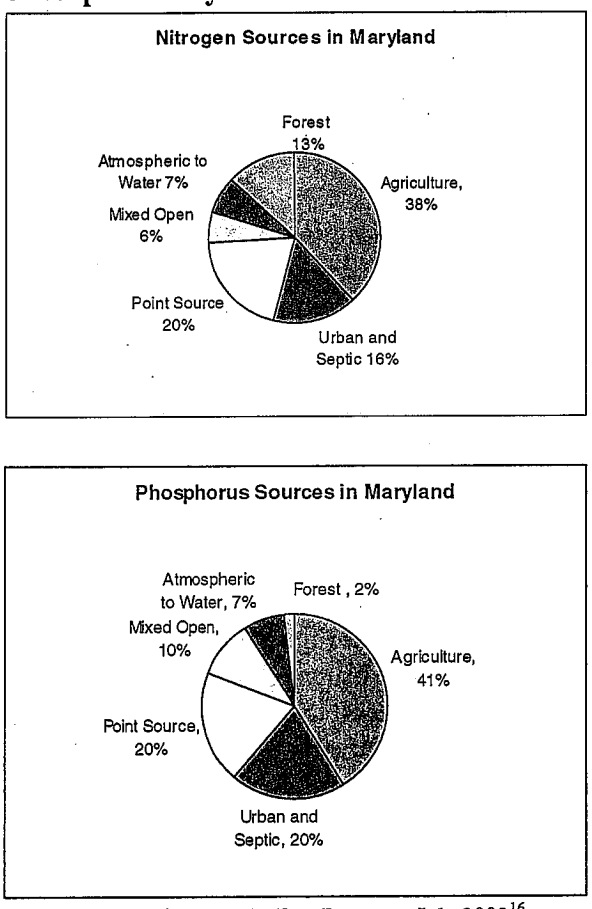


Source: USEPA Chesapeake Bay Program Office[17]

Nutrients and Public Health

Excess nutrients in the Bay have both direct and indirect adverse impacts on the health of people living around the Bay and in the region. While nutrients do not share the same toxicological properties associated with heavy metals and some pathogens, they have devastating effects on the aquatic system. Nutrient overloads in waterways lead to excess growth in algae; the algal blooms block sunlight and settle to the bottom to decompose. Both the algal blooms and decomposition lead to eutrophication, a state of low dissolved oxygen that creates a poor living environment for some organisms and creates ideal living conditions for more invasive species.[18] As a result, the health of finfish, shellfish, and crabs in the Chesapeake Bay is threatened. Currently, blue crabs are at their lowest historical levels. Crabs and shellfish have served as a staple in the diets of Marylanders for centuries. Loss or degradation of these staple foods may have significant public health and economic implications, in particular for commercial shell-fishing and subsistence fishing populations that rely on shellfish for their livelihood.

Figure 3: Maryland Sources of Nitrogen and Phosphorous Loadings into the Chesapeake Bay



Source: USEPA Chesapeake Bay Program, July 2002[16]

Both point and non-point source pollution contribute to excess nutrients in the Bay. Nutrient pollution is primarily due to excess nitrogen and phosphorous. Figure 3 shows the percent contribution of nitrogen and phosphorous sources to nutrient loads in the Chesapeake Bay and tidal tributaries. Agricultural run-off, a non-point source of pollution, is the most

prominent source and contributes approximately 40% of both nitrogen and phosphorous total loadings. Urban and septic, and point source discharges from waste water treatment plants are also prominent sources contributing approximately 20% each.[iii] These sources combined contribute approximately 80% of the excess nutrients in the Bay.[16] The majority of nitrogen and phosphorous pollution is released into lakes, streams, and rivers throughout the watershed. Collectively these waters all flow into the Bay and increase risks to ecological health and potentially human health throughout the region. Consequently, nutrients are an area for future public health indicator development.

In some stances, increased nutrients can also lead to increased growth of harmful algal blooms with toxic consequences for aquatic life and human health. Evidence of this problem in the Bay includes: *Pfisteria* outbreaks of 1997–1998; an increase in mycobacterium leading to malnutrition and lesions in rockfish; and *M. marinum* infection[ii] from recreational exposure to the Bay in Anne Arundel County. [19,20,21,22,23,24] While there is evidence that some of these harmful bacteria are not new to the Chesapeake Bay, increased monitoring of the declining health of the ecosystem and better analytic science reveal that bacterial growth is becoming more prominent in the region. [25,26,27] The Maryland Department of Natural Resources recently discovered *Chantonella*, another algae associated with fish kills in other parts of the country, in the Bay. In addition, harmful microbes like *Cryptosporidium parvum* have been found within the sediment and shellfish in the Bay. [28,29,30] All of these pathogens are potential public health threats in the region and key areas of concern for future indicator development.

Air Pollution in the Chesapeake Bay Region

Air pollution has been associated with health effects ranging from acute and chronic respiratory problems and heart disease to increased mortality. Air quality in the region is a reflection of the increasing development and urban sprawl both within the Chesapeake Bay region itself and within neighboring regions. For instance, a large portion of mercury in Maryland lakes is carried downwind to Maryland from coal-burning power plant sources in the Midwest. This deposition contributes to the bioaccumulation of mercury in fish. [31] In addition, approximately 7% of both nitrogen and phosphorous loadings into Maryland waters are from atmospheric deposition.[iii]

Within the state of Maryland, the most significant air pollution problems identified by the National Ambient Air Quality Standards (NAAQS) are associated with ozone.[32] Ozone emissions exceed NAAQS for a significant portion of the state. Figure 4 identifies those counties for which ozone levels exceed health-based NAAQS as of February 2003. NAAQS are characterized as having non-attainment status when they exceed the standards set by the USEPA. The severity of this status is designated as *moderate*, *serious*, and *severe-15*. Ozone and carbon

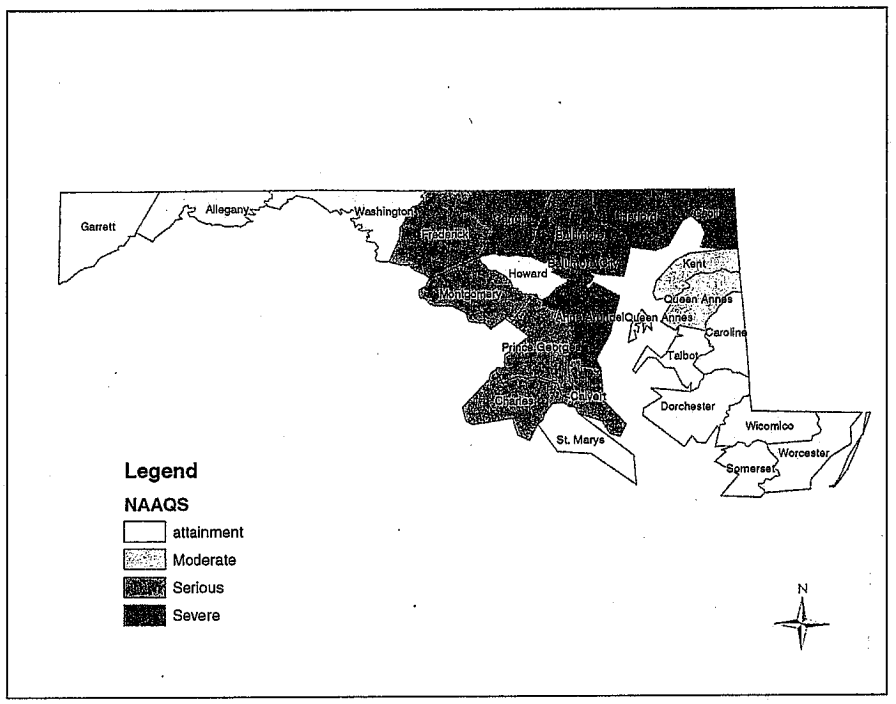
---

[ii] *Mycobacterium marinum* infection in cases results in skin or joint infection in the upper extremities. It was discovered in Anne Arundel County, and a retrospective survey of 41 cases of culture-positive *M. marinum* linked the source of infection to recreational exposure to the Chesapeake Bay and its tributaries. Within Anne Arundel County a positive TB skin test is more likely an indication of *M. marinum* than TB infection.

[iii] These figures measure only the amount of nitrogen and phosphorous that lands directly on the water. They are not a measure of the nitrogen and phosphorous coming from the air that are deposited onto the land, and then flows into the water.

monoxide are the two NAAQS within Maryland that exceed their standards and are non-attainment in several counties. In addition to monitoring NAAQS, the number of bad ozone days and of respiratory health problems within the population can potentially be tracked as environmental public health indicators for the region. Both air quality and deposition of air pollutants into the waterways are a major threat to the Bay and to human health, and they present key areas for future indicator development.

Figure 4: Ozone Non-Attainment Areas for Maryland



Source: National Ambient Air Quality Standards Database, USEPA

Chemical Contaminants and the Bay

Assessment of chemical contaminants in the Chesapeake Bay has been ongoing since the initial assessment was completed in 1983. At that time, Baltimore Harbor in Maryland, the Anacostia River in the District of Columbia, and the Elizabeth River in Norfolk, Virginia, were identified as key areas of concern based on their high levels of chemical contaminants.[33,34] *The Chesapeake Bay 2000* agreement, based upon an overarching goal of a Bay free of toxics, established a strategy to "eliminate the input of chemical contaminants from all controllable sources to levels that result in no toxic or bio-accumulative impact on the living resources that inhibit the Bay or human health."[35] These ongoing monitoring efforts show that traces of toxic chemicals continue to be detected in fish and marine life throughout the Chesapeake Bay watershed, leaving populations living in the region potentially vulnerable to their adverse health effects.[36]

Toxic chemicals in the Bay include both metals and organic chemicals ranging from pesticides to disinfection byproducts. Table 2 lists the "chemical contaminants of concern" for

the Chesapeake Bay. Chemicals of concern are of three kinds: "1) chemical contaminants identified in the USEPA Chesapeake Bay Program (CBP) *1999 Toxics Characterization* that are at levels that may cause toxic impacts to living resources; 2) chemical contaminants responsible for finfish and shellfish advisories; or 3) chemical contaminants responsible for waterbodies being listed as impaired or threatened on the jurisdictions' 303(d) list."

Table 2: Chemicals of Concern for the Chesapeake Bay Region as of September 2000*

1999 Chemicals of Concern for the Chesapeake Bay Tidal Waters[34]		
Arsenic	DDT	Mercury
Cadmium	Dieldrin	Nickel
Chlordane	Kepone	PAHs
Chlorpyrifos	Lead	PCBs
Chromium	Lindane	Zinc
Copper	Malathion	
Fish Consumption Advisories Chemicals of Concern for the Bay Region		
Chlordane	Mercury	Kepone
Mirex	Dioxin	PCBs
Maryland Impaired Tidal and Non-Tidal Waterbodies Chemicals of Concern		
Arsenic	Copper	PCBs
Cadmium	Cyanide	Selenium
Chlordane	Lead	Silver
Chromium	Nickel	Zinc

* *Note:* Virginia, Pennsylvania, and the District of Columbia also have lists of chemical contaminants that include over 70 contaminants with recognized health endpoints. *Source:* USEPA Chesapeake Bay Program Office[34]

The public health consequences of chemical contaminants released into the natural environment are difficult to measure. However, toxicological and epidemiologic research provide evidence that chemicals being released into the environment do pose potential threats to human health. Table 3 (pg. 10) lists the 1999 Chemicals of Concern for the Chesapeake Bay Tidal Waters and their associated health effects as researched by Environmental Defense. (Appendix 1 lists the scientific sources used by ED to develop categories.) Chemicals are designated as recognized toxicants (R) if they have been widely accepted as health hazards by scientific agencies and sources. Chemicals are designated suspected toxicants (S) in a particular health outcome if more limited data exists.[37] Chemicals were analyzed based on their toxicity in twelve different health hazard categories, including cancer, cardiovascular or blood, developmental, endocrine, gastrointestinal or liver, immunotoxicity, kidney, musculoskeletal, neurotoxicity, reproductive, respiratory, and skin or sense organ. Cancer and developmental toxicity are the primary recognized health hazards associated with chemicals of concern for the Chesapeake Bay tidal waters.

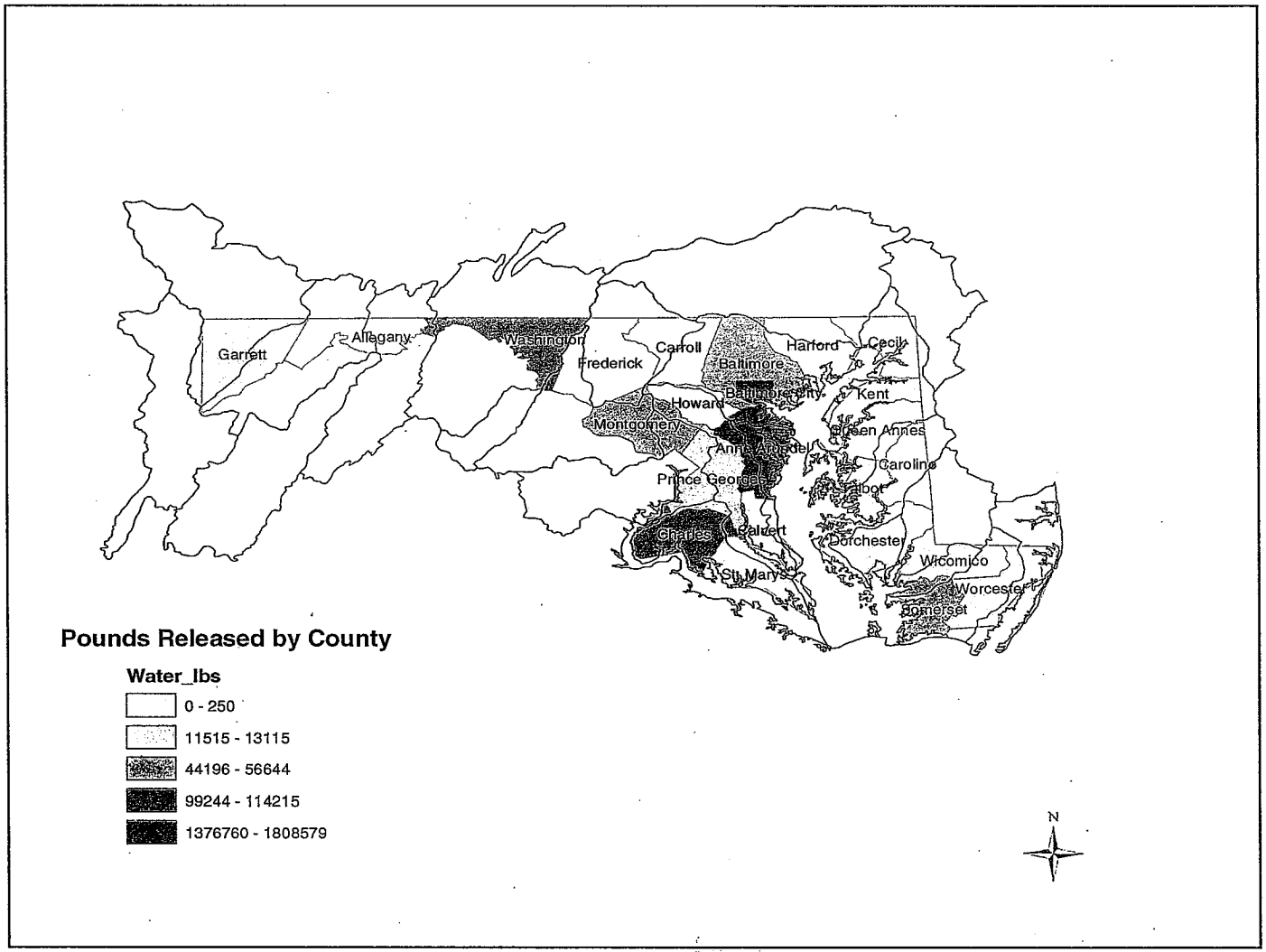
The distribution and concentration of chemical contaminants vary geographically, and the sources of contamination vary for each chemical. Therefore, health threats from toxic chemical pollution vary temporally and spatially throughout the watershed. Industrial growth, agriculture and land-use patterns lead to unequal distribution of chemical loading in the waterways, and some communities are impacted more than others. Figure 5 (pg.11) displays total pounds of USEPA Toxic Release Inventory chemicals released into surface waters by county from industrial point sources reporting to the EPA in 2000. Over 3.5 million pounds of toxic pollutants were released into Maryland waterways.[38] While this is not a measure of actual population exposure, it illustrates areas of both the watershed and the population that are at risk from the discharge of toxics. In addition, the distribution of these chemicals in relation to the social, economic, and demographic distribution of the population is an area for future indicator development.

Table 3: 1999 Chemicals of Concern for the Chesapeake Bay Tidal Waters and Their Associated Recognized (R) and Suspected (S) Health Effects

1999 Toxic Characterization Chemicals of Concern in Tidal Waters	Carcinogen	Cardiovascular or Blood	Developmental	Endocrine	Gastrointestinal or Liver Illness	Immuno-toxicant	Kidney	Musculoskeletal	Neurological	Reproductive	Respiratory	Skin or Sense Organ
Arsenic	R	S	R	S	S		S		S	S	S	S
Cadmium	R	S	R	S		S	S		S	R	S	
Chlordane	R	S	S	S	S				S	S	S	
Chlorpyrifos		S			S				S	S	S	S
Chromium	S				S	S	S			S	S	S
Copper		S	S		S					S	S	
DDT	R	S	R	S	S	S			S	R	S	S
Dieldrin	R	S		S	S	S			S	S	S	
Kepone	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Lead	R	S	R	S	S	S	S			R	S	S
Lindane			R									
Malathion		S		S	S				S	S	S	S
Mercury		S	R	S	S	S	S		S	S	S	S
Nickel	R	S	S			S	S			S	S	S
PAHs	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
PCBs	R		R	S	S	S			S	S	S	S
Zinc		S	S			S				S	S	S
Recognized/Suspected	8R/1S	12S	7R/4S	9S	11S	9S	6S		9S	3R/11S	14S	10S

Sources: Chesapeake Bay Toxic Inventory, update September 26, 2000; and Environmental Defense Scorecard Database; for list of ED scientific sources please see Appendix 1.

Figure 5: Total Pounds of TRI Chemical Releases in Maryland's Surface Waters (2000)



Source: USEPA Toxic Release Inventory[39]

III. Indicator Development

The presence of toxic chemicals and microbial pollutants demonstrates the vulnerability of both ecological and public health in the Chesapeake Bay region. A comprehensive mechanism to track the potential health threats from these hazards is needed. Establishing environmental public health indicators is one way to begin this assessment process.

The following section describes the development and analysis of three case examples of environmental public health indicators applicable in the Chesapeake Bay region. Microbial risks and chemical contaminant risks in both surface and drinking water were selected as the prominent categories of hazards for this initial review of water quality and health.

National Environmental Health Tracking Measures

Tracking indicators of health and environment is emerging as a major component of national environmental policies. The National Center for Environmental Health of the Centers for Disease Control and Prevention (NCEH/CDC) has initiated an environmental public health tracking initiative to integrate environmental hazards, human exposure, and health outcomes data. NCEH/CDC has developed a set of environmental public health indicators to serve as a foundation for the National Environmental Public Health Tracking Network.[40] The State of Maryland is one of twenty states participating in the pilot phase of the National Environmental Public Health Tracking Network, and Johns Hopkins has been funded by CDC as a National Academic Center of Excellence to assist in developing this network. The U.S. Environmental Protection Agency is also developing environmental health indicators as part of the development of a national "State of the Environment" report to describe environmental conditions and human health concerns.[41] Appendix 2 provides definitions and summaries of these initiatives, which will enable ongoing monitoring and dissemination of information on health effect trends and environmental contamination levels, facilitate research on the links between environment and health, and improve the scientific basis for decisions and a sounder public health basis for our national environmental priorities.

Environmental Public Health Indicators

Examining environmental public health indicators (EPHI) for the Chesapeake Bay offers a first step in establishing the link between environment and health by examining those aspects of the environment that also have significant public health implications. EPHI are quantitative measures that can be tracked to describe, summarize, and monitor trends that have the potential to impact both public health and the environment.[42] When indicators are carefully selected, they can be utilized to address complex environment and health relationships by diagnosing problems, tracking changes over time, and guiding policy decisions. For example, the daily air quality index is an existing indicator that summarizes a complex mixture of hazards and provides useful information concerning prevention of adverse health impacts.

Table 4 presents several EPHI that were selected by the NCEH/CDC tracking initiative that have relevance to the Chesapeake Bay region. EPHI range from hazard indicators such as fish and shellfish contamination, to point source discharges into ambient water and health effect indicators measuring outbreaks attributed to contaminated drinking water.

Table 4: Examples of NCEH Environmental Public Health Indicators Relevant to the Chesapeake Bay Health Indicators Project

Hazard Indicators
Chemical spills
Monitored contaminants in ambient drinking water
Point source discharges into ambient water
Contaminants in fish and shellfish
Exposure Indicator (biomarkers of exposure)
Blood lead level
Health Effect Indicators (occurrence of morbidity or mortality attributed to exposure)
Outbreaks attributed to fish and shellfish
Outbreaks attributed to ambient or drinking water contaminants
Intervention Indicators
Activity restrictions in ambient water (health-based)
Compliance with operation and maintenance standards for drinking water systems
Boil-water advisories

Chesapeake Bay Health Indicator Selection

The research approach adopted by the Chesapeake Bay Health Indicators Project included a review of the literature on indicators for both health and environment, an extensive examination of available data to identify indicators, stakeholder outreach to identify priorities for indicators, and an analysis of pilot indicators to choose as illustrative examples. Available monitoring data for the Bay and watershed were evaluated. This included several years of federal data sets as well as state and local monitoring systems. Meetings with environmental groups and environmental and public health agencies were also held. A working group meeting to review draft recommendations and findings was also conducted.

The goals, applications, and limitations of potential indicators were carefully weighed during the selection process. Factors that were considered in the selection of indicators included: availability of data, feasibility for monitoring trends, potential population exposure, scientific basis, public concern, public health importance, and potential for public health interventions. Data sets within the state of Maryland were chosen to facilitate the analysis. Based upon these considerations pilot indicators were examined that include measures of:

- **Drinking water protection**—Total trihalomethanes (TTHMs) in public drinking water supplies;
- **Microbial risks in surface waters**—Fecal coliform contamination at bathing beaches and in recreational and urban waterways; and
- **Persistent toxic pollutants**—Mercury and PCB contaminant levels in fish tissue.

The following section presents three case examples to illustrate the analysis and application of criteria for the selection of environmental public health indicators. The indicators represent chemical and biological measures that provide summary measures of volatile chemicals, pathogens, and persistent bio-accumulating toxic metal pollutants found in Maryland. They also represent multiple population exposure pathways that may be linked to several

potential public heath impacts including both acute and chronic health effects. For each of these indicators, national data are available for future comparisons, and they are also included on the indicator lists developed by the CDC and EPA for national tracking. The data limitations and lessons learned from each pilot indicator are presented, as well as future recommendations for indicator development.

IV. Indicator Findings

Indicator 1: Total Trihalomethanes (TTHMs) in Drinking Water

Source/Hazard—Chlorination of drinking water (chlorine interacts with naturally occurring organic material and organic pollution in the source water and produces several disinfection by-products DBP, including TTHMs). TTHMs may provide an indicator of a broad range of chemical disinfection byproducts.

Exposure—Ingestion of chlorinated drinking water, dermal absorption from bathing and washing, and inhalation of aerosols or volatilized compounds during showering.

Population exposure potential—High. Over 200 million people throughout the United States and close to 5 million in the state of Maryland are served by public water systems.

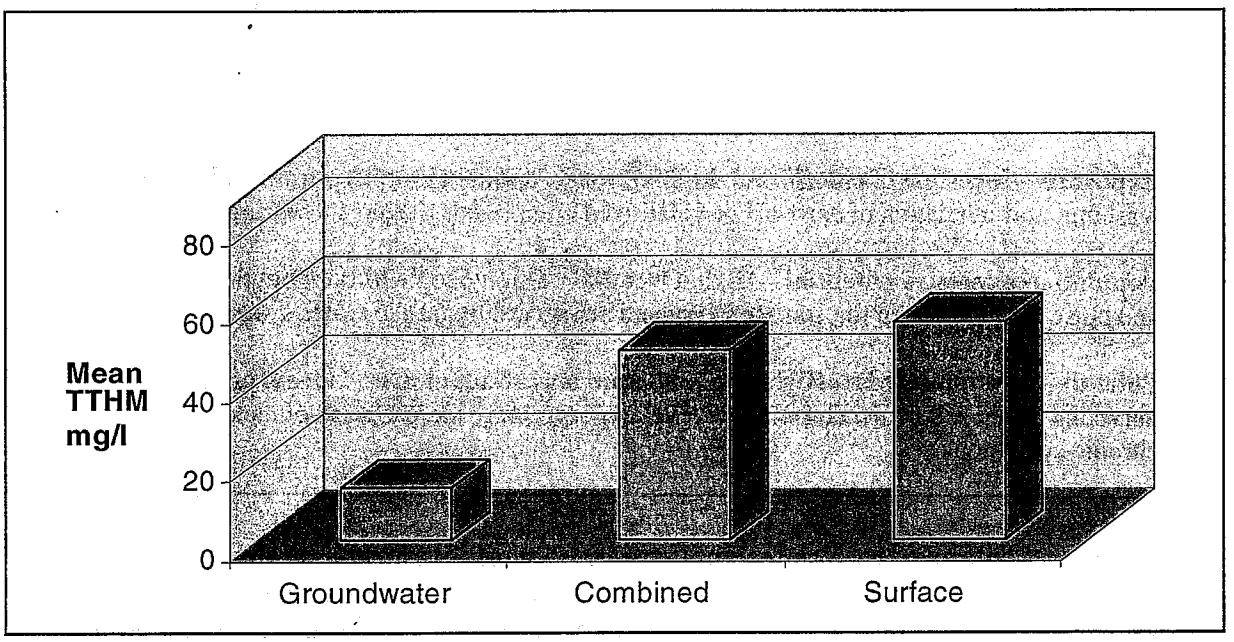
Potential health hazards—High-level exposure may result in liver, kidney, or other central nervous system problems; chronic low level exposure may increase risk of cancer and other chronic conditions.

The use of chlorine as a disinfectant is practiced world-wide, and has been heralded as a major advance in preventing waterborne infectious diseases. Chlorine reacts with any organic matter in the source water, leading to the formation of halogenated byproducts. Some of these byproducts have been associated with negative health effects ranging from bladder cancer to adverse reproductive outcomes. To track and control these potentially harmful byproducts, regular monitoring is mandated by the federal Safe Drinking Water Act (SDWA), and measures to protect surface waters used for drinking water supplies are regulated under the federal Clean Water Act (CWA).

TTHMs (including chloroform, a suspected carcinogen, and other compounds that have toxic and mutagenic properties) provide an effective indicator of potential population exposure to chemical byproducts of drinking water disinfection. Since TTHMs are formed when organic materials in the source water react with chlorine during the chlorination process, they also provide an indicator of total organic carbon (TOC) in source water and potentially of source water quality. It is difficult to distinguish the source of TOC, because it comes from both natural and anthropogenic sources. Measures of surface water quality including both TTHMs and TOC are important to track because of their potential risks to human health. A recent study by Bing-Fang et al. concluded that both TTHMs and acidic properties (Ph) of water are associated with increased rates of certain birth defects in a Norwegian population.[43]

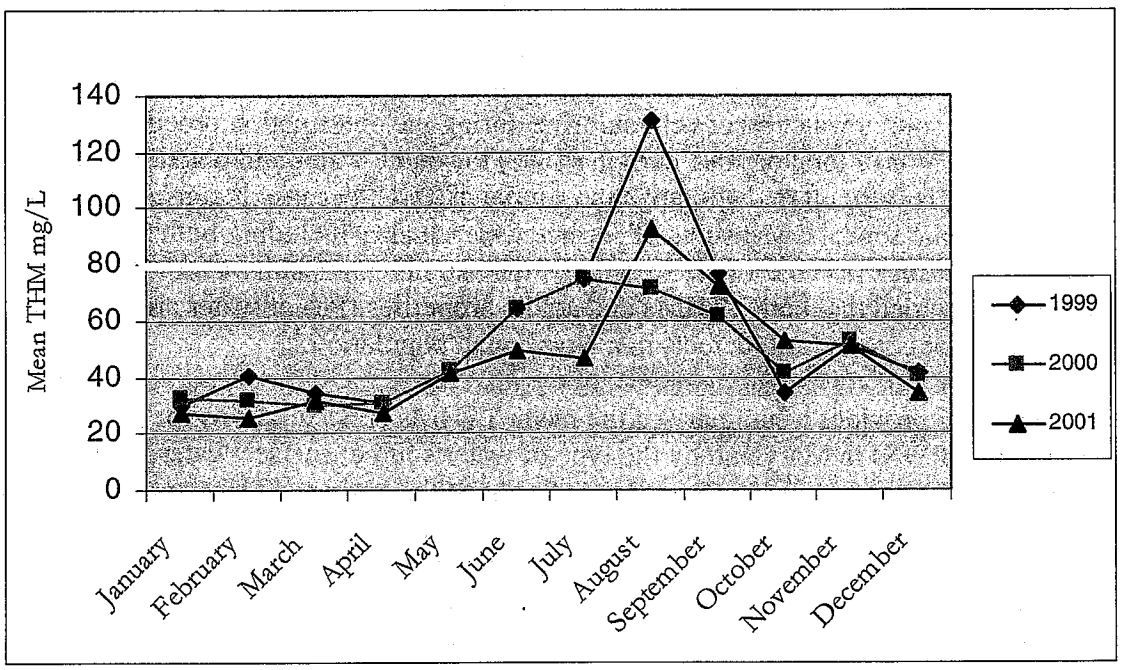
Figure 6 provides a summary of the mean TTHMs concentrations for all major public water systems (PWS) in Maryland by the type of water source; surface water, groundwater or combined. PWS served by surface water supplies have the highest TTHM concentrations due to the increased prevalence of organic material compared to ground water sources. Combined public water systems receive raw water from both groundwater and surface water sources. High levels of TTHMs in combined sources most likely reflect high levels of organic matter in surface waters. Figure 7 shows the monthly variation in TTHMs throughout the year, with the highest levels in the late summer and fall resulting from seasonal variation in levels of organic material in surface waters.

Figure 6: Mean TTHM for Varying Source Water Used in Maryland Public Water Systems*



*** Note: Maximum Contaminant Level (MCL) = 80 mg/L (USEPA's Safe Drinking Water Act standard)**

Figure 7: Annual Monthly Mean TTHM for Surface and Combined Sources in Maryland*



*** Note: Maximum Contaminant Level (MCL) = 80 mg/L, (USEPA's Safe Drinking Water Act standard)**

Table 5 (pg. 18) provides a summary of TTHM monitoring data for all of the Maryland PWS serving greater than 10,000 people for the years 1999 through 2001. It is important to note that all of the state drinking water supplies in Maryland are in compliance with the federal SDWA requirement for TTHMs (Maximum Contaminant Level (MCL) = 80 mg/L). SDWA compliance is determined on an annual basis. If a system has an excursion above the MCL it is noted as a system violation within MDE. Public water systems with violations are given the opportunity to return to compliance status, if this occurs within the appropriate time frame, the system remains in compliance.[44] Over 3.5 million Marylanders consume drinking water from these public water supplies. Virtually all PWS served by surface water supplies in Table 5 have periodic excursions above the recommended health-based standard. These results indicate both the vulnerability of surface sources within the Chesapeake Bay Watershed to organic pollution, and the potential for broad population exposure to chemical contaminants at levels above recommended public health guidance levels. Watershed protection and monitoring of total organic carbon may be the most important step toward lowering levels of exposure to TTHMs and preventing potential adverse health impacts.

TTHMs in Drinking Water—Utility as a Public Health Indicator

In conclusion, TTHMs provide a useful indicator for tracking. Currently the public concern regarding TTHMs is low since the public water supplies are in compliance with USEPA guidelines, but analysis of the data shows that surface supplies are vulnerable to excursions above health-based guidelines. There is a high potential for population exposure because public drinking water supplies serve a large portion of state residents. At the same time, the scientific basis and public health importance of preventing population exposure to low-level chronic risks are balanced by the need for mechanisms to control microbial risks in drinking water. Data on TTHMs are readily available as a result of mandatory reporting under the Safe Drinking Water Act. This systematic reporting also makes the feasibility of monitoring trends relatively easy. There are, however, limitations to TTHMs serving as an indicator because they provide no information on the source and nature of organic precursors. TTHMs represent a good first step in indicator development for tracking population exposures related to drinking water. However, a suite of indicators including TOC, TTHMs, and organic and inorganic contaminants not related to disinfection would provide a better picture of the quality of source water.

Table 5: Total Trihalomethane Exceedences for Public Water Systems in Maryland Serving Greater than 10,000 People (1991–2001).

Public Water Station Name	Total Observations	Exceedences Above MCL [iv](80 mg/L)	Percent Exceedences [v]	Mean (mg/L)	Max (mg/L)	Source Water[vi]	Population Served
Broad Creek	12	0	0.00	6.4	11.4	G	12,000
Cambridge	12	0	0.00	5.8	9.7	G	14,500
City of Annapolis	11	0	0.00	4.5	9.2	G	35,000
City of Bowie	13	0	0.00	3.8	6.4	G	244,500
Crofton-Odenton	14	0	0.00	1.8	3.0	G	31,400
Glen Burnie-Broadneck	38	0	0.00	21.8	68.3	G	249,600
Lexington Park	27	0	0.00	2.5	8.9	G	18,300
Ocean City	14	0	0.00	37.8	61.1	G	30,000
Putuxant Naval Air Station (NEW CAD)	13	0	0.00	1.3	2.6	G	16,600
Waldorf	12	0	0.00	1.7	8.2	G	52,000
Freedom District	75	4	0.05	36.5	95.9	G	21,200
City of Aberdeen	28	5	0.18	45.9	97.7	C	13,000
Fort George Meade	44	1	0.02	27.5	86.0	C	50,000
City of Westminster	88	8	0.09	42.7	120.0	C	23,300
Maryland American Water Company	37	6	0.16	42.5	117.6	C	13,200
Chapel Hill	73	14	0.19	52.5	158.7	C	12,000
Lake Linganore	36	7	0.19	63.3	138.8	C	11,500
New Design Water	50	15	0.30	66.4	185.5	C	14,500
City of Frostburg	42	15	0.36	74.4	131.0	C	11,000
City of Salisbury	26	0	0.00	0.9	4.1	S	25,000
Baltimore City	149	7	0.05	42.0	100.0	S	1,600,000
Harford County D.P.W.	40	2	0.05	37.3	85.8	S	90,000
City of Cumberland	51	4	0.08	31.1	107.8	S	23,600
City of Hagerstown	54	5	0.09	51.0	123.4	S	75,000
City of Frederick	105	15	0.14	53.0	211.3	S	51,000
City of Havre de Grace	19	4	0.21	58.8	139.2	S	10,400
Washington Suburban Sanitary Commission	584	143	0.24	60.6	253.0	S	1,300,000
City of Rockville	70	24	0.34	66.9	193.6	S	48,000
Howard County D.P.W. Distribution	34	6	0.18	51.1	90.7	P	175,000
	Total Population Served by Public Water Systems with at Least One Exceedence						**3,542,700**

[iv] MCL = Maximum Contaminant Level. Safe drinking water standard for TTHMs is MCL = 80 mg/L.

[v] Percent Exceedence = Total # of exceedences greater than MCL ÷ Total number of observations for each public water system

[vi] G = Groundwater, C = Combined (served by both surface and groundwater), S = Surface water

Indicator 2: Fecal Coliform Levels in Surface Waters

Source/Hazard—Animal and human fecal contamination through non-point source pollution, agricultural run-off, waste water treatment failures, sewage overflows, septic system failures, boat dumping, and storm water run-off.

Exposure—Contact or ingestion of recreational surface water or consumption of shellfish from contaminated areas.

Population exposure potential—Widescale potential for exposure from contact and ingestion of contaminated recreational waters, and microbial contamination of drinking water.

Potential health hazards—Acute gastroenteritis, mild to severe infection possibly leading to serious illness in compromised individuals.

Fecal coliform monitoring serves as a time-tested indicator of potential microbial pollution from sewage, agricultural runoff, and non-point source pollution. Fecal coliform is a bioindicator that provides a summary measure of surface water contamination that may also indicate the presence of other infectious bacterial agents, viruses, and protozoans. These pathogens are responsible for a wide range of infectious outcomes, from acute gastroenteritis to more serious, even fatal, infections such as hepatitis and cryptosporidiosis.[45] Populations in and around the Chesapeake Bay may be vulnerable to microbiologic pathogen exposures through contact with or ingestion of contaminated water, as levels of fecal coliform are at times well above health-based standards.

EPA's current guidance suggests the use of enterococci for marine waters and the use of enterrococi and *E. coli* as bioindicators for fresh water. Fecal coliform counts were used in this analysis because they were used as the standard EPA measurement prior to 2001. There is also a national EPA network of beach water monitoring for microbial pollutants that used fecal coliform. Several Maryland counties have monitored waters for fecal coliform for a number of years. In Anne Arundel County, MD, there are a total of 108 monitoring stations, including six beaches that are part of the national EPA beach monitoring program. An analysis of the county monitoring data for two of these locations was conducted to examine trends and compare levels to current health standards.

Table 6 presents the results of monitoring at the Annapolis City Dock for the years 1991 through 2000. The Annapolis area is one of the most popular spots on the East Coast for sailing and recreational boating. During this time there have been reductions in mean fecal coliform levels, reflecting efforts to reduce waste discharges, including human waste from boat dumping, and to control runoff. Forty-five percent of the samples for this location exceed the health-based guidance level of 200 colonies per 100 ml (200 col/100ml). These results most likely reflect aggressive sampling during high-use periods and after storm events; however, they underscore the need for continued efforts to reduce sewage and microbial contamination. .

Table 7 presents fecal coliform monitoring results for the same 10-year period for Sandy Point State Park Beach. The mean levels have been below the health guidance level since 1992. While there continue to be a small number of samples exceeding the standards each year, the

overall results indicate continued improvement in mean levels. Maximum contaminant levels do not appear to show a similar trend in reduction.

Table 6: Annapolis City Dock Yearly Fecal Coliform Counts

Year	Total Samples	Mean* (col/100 ml)	Min (col/100 ml)	Max** (col/100 ml)	Total Exceedences	Percent Exceedences[vii](%)
1991	19	179.6	9.1	2400	17	89
1992	18	357.3	3.6	2400	5	28
1993	27	556.2	3	2400	18	67
1994	26	365.5	4.4	2400	8	31
1995	22	390.8	11	2400	8	36
1996	20	416.9	4.5	2400	9	45
1997	23	326.3	0	1600	10	43
1998	23	201.9	8	2400	4	17
1999	23	171.6	1.8	1600	6	26
2000	22	224.6	4	1600	10	45

* 200 col/ml = Maximum Contaminant Level (MCL)
** 2400 is the maximum detection level using standard measures.

Table 7: Sandy Point State Park East Beach Yearly Fecal Coliform Counts

Year	Total Samples	Mean* (col/100 ml)	Min (col/100 ml)	Max* (col/100 ml)	Total Exceedences	Percent Exceedence[vii](%)
1991	15	250	3.6	2400	3	20
1992	20	165	3.6	1100	5	25
1993	19	105	3.6	460	3	16
1994	22	121	2	920	3	14
1995	23	39	1.8	170	0	00
1996	15	132	1.8	540	3	20
1997	22	94	2	1000	3	14
1998	28	89	7	540	3	11
1999	27	76	1.8	920	3	11
2000	28	78	2	920	2	7

* 200 col/ml = Maximum Contaminant Level (MCL)
** 2400 is the maximum detection level using standard measures

Results from Anne Arundel County presented in Tables 6 and 7 demonstrate a trend toward improving water quality over the past decade. Mean levels at Sandy Point Beach are well below health guidelines. Levels at the Annapolis City Dock are higher, but also show some improvement. There have been excursions above the standard at both sites, occurring most frequently in the late summer months when recreational use is highest. Trend analysis does not account for real-time exposures on days when excursions occur, as current sampling techniques limit the ability for these types of measures to be monitored.

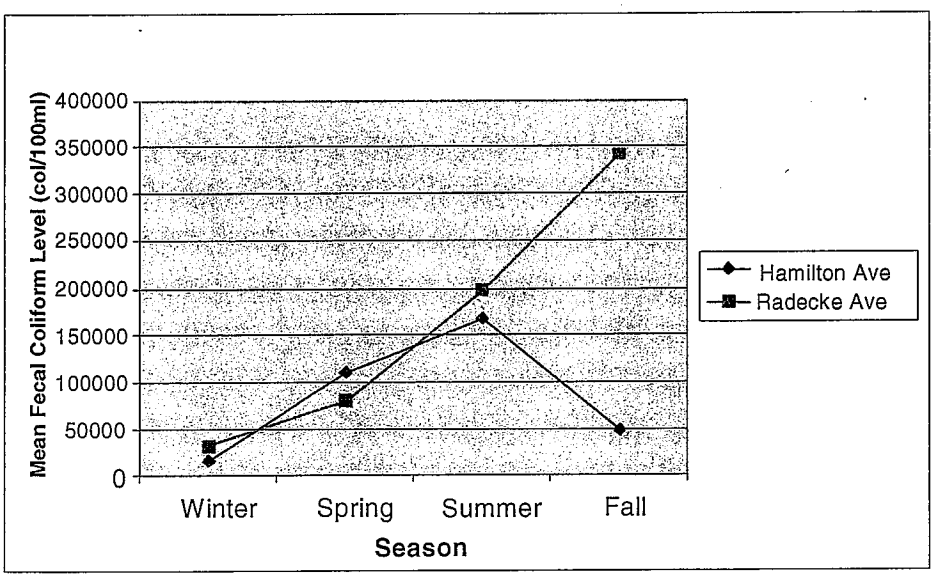
[vii] **Percent Exceedence = Total Exceedences ÷ Total Samples**

To examine conditions in more urbanized and industrialized areas of the watershed, post-storm event fecal coliform monitoring data were examined for Baltimore City from two stations on the Back River. Data for the years 1995 through 2001 were obtained from the Baltimore City Water Quality Management Section (Table 8). All samples exceeded the standard, with levels that were orders of magnitude above the health-based guidance levels. Figure 8 presents a seasonal analysis of the monitoring, showing highest levels during the summer and fall. The data show fecal coliform levels at Redecke Avenue station have an unusual spike in the fall that differs significantly from the Hamilton Avenue station. The exact cause of this variation is unknown. However, these two stations are located at different points along the waterway and may have different non-point source pollution including leaking sewage systems contributing to the variation. This spike might also be the result of an unusual event at one point in time, although this is difficult to determine based on the data alone. These findings indicate the vulnerability of urban waterways within the watershed to bacterial contamination due to runoff, aging waste water infrastructure, water treatment plant malfunctions, and sanitary system overflows. They also indicate the potential for population exposure to pathogenic pollutants through contact or ingestion.

Table 8: Baltimore City Yearly Mean Fecal Coliform Count After Storm Events

Hamilton Avenue			Radecke Avenue		
Year	Mean (col/100ml)	no.Obs	Year	Mean (col/100ml)	no.Obs
1995	88900.8	9	1995	129952.4	4
1996	73320.82	10	1996	73991.17	14
1997	40041.31	15	1997	41286.81	14
1998	49035.62	8	1998	612183.9	11
1999	98216.75	10	1999	78505.39	11
2000	258686.6	13	2000	174450.4	12
2001	64003.71	10	2001	213540.7	9

Figure 8: Fecal Coliform Counts by Seasonal Mean (1995–2001)



Fecal Coliforms—Utility as a Public Health Indicator

In conclusion, fecal coliform in surface water is a good indicator for environmental public health tracking. A national network already exists for monitoring fecal coliform and enteroccoci as water quality measures. Monitoring of microbial pathogens complements the ongoing work of other state agencies related to non-point source pollution control and nutrient reduction plans. Monitoring programs provide data at regular intervals during summer months, making the feasibility of monitoring trends high. Anne Arundel County is able to collect data at relatively low cost with the use of high school students as summer interns. Data indicate that the potential for population exposures to fecal coliforms exceed health-based standards at bathing beaches and in urban and recreational waterways. The scientific basis for monitoring microbial risks is well established, with enteroccoci currently the most feasible indicator for national monitoring efforts. The public health importance of measuring microbial risks is great in spite of limited public concern.

Despite the utility of using fecal coliforms as an indicator, there are currently several limitations in using microbial contaminant data. The collection and availability of monitoring data vary both across the state and nationally. Not all counties within Maryland collect microbial pathogen data. In addition, linking fecal coliform data to health outcomes is limited by the lack of surveillance data for waterborne disease outbreaks. Outbreak data are collected on a voluntary basis and are not collected uniformly either within the state of Maryland or nationally.[46] The current state of infrastructure for water pollution control needs attention. Aging sanitary systems nationally are leading to massive sewage overflows as seen in Baltimore City over the last several years. New science and technology in the fields of microbiology are revealing that more specific microbial indicators may be better at detecting actual risks and sources of microbial contamination than fecal coliform counts. Therefore, as scientific methods improve, more specific indicators of microbial risk may replace fecal coliform and enteroccoci. However, they are currently the most feasible and readily available measures for environmental public health indicator development and tracking.

Indicator 3: Methylmercury and PCB Fish Tissue Contamination

Source/Hazard—Industrial point and non-point source pollution, industrial landfills in and around the Chesapeake Bay region and more distant power plants to the West.

Human Exposure—Ingestion of contaminated fish from Chesapeake Bay tidal waters and tributaries.

Population exposure potential—Over 250,000 licensed anglers in the state of Maryland, unknown number of unlicensed anglers; exposure dependent upon unknown consumption rates among anglers.

Potential health hazards—Mercury exposure is linked to neurological, developmental reproductive, immunological, and cardiovascular effects. PCBs have been linked to increased risk of immunological and neurological effects, and cancer.

Mercury and polychlorinated biphenyls (PCBs) are ubiquitous pollutants throughout the aquatic environment that move through the food chain to impact both ecological and human health. They are a reminder that past, present, and future releases of toxic pollutants have ongoing, long-term public health impacts. While current monitoring of the Chesapeake Bay fish population is limited, the available data provide evidence of persistent toxic pollutants that have well-recognized adverse impacts on human health, including neurological, cardiovascular, and developmental toxicity and cancer. [49] In an effort to limit public exposure to these contaminants, the Maryland Department of Environment (MDE) currently has fish consumption advisories for 14 tidal rivers in the state. Sampling information for fish from Maryland's portion of the Bay watershed is constantly being updated by the MDE. While annual sampling is being conducted, resources and capacity limit the scope of these monitoring efforts.

Sources of Mercury

The primary source of mercury contamination for the people of Maryland is thought to be through the consumption of contaminated fish. Regional estimates of human fish consumption are often unknown. However, the state of Maryland has been monitoring fish tissue concentrations since the 1970's and has used this information for public health interventions. For example, the MDE has issued several fish advisories recommending limits on the amount of recreationally caught fish that individuals should eat per month from select water bodies in the state. Advisories are based on risk calculations using national fish consumption estimates and fish tissue concentrations that vary depending on fish species, fish size and the water bodies that fish were caught in. Currently, MDE has two statewide fish advisories for mercury; one for lakes and reservoirs, and another for freshwater streams. Mercury contamination is most prevalent in freshwater impoundments in the state. As a result, MDE recommends limiting consumption of sport caught fish(in general) from lakes and reservoirs to four 8-oz. meals per month and suggests eating fewer than eight 8-oz. meals per month from freshwater streams. MDE fish advisories distinguish sport caught fish from other types of fish. MDE collects fish tissue samples from both the sport caught fish which are mainly predatory fish (such as small mouth bass and white perch), as well as "accumulator" fish (channel catfish and eel). Accumulator fish provide information that helps to determine the relative amount of contaminants of concern that

are accumulating in the waters and together with the sport caught fish they provide information regarding the potential risk to people who consume recreationally caught fish from Maryland waters.

The EPA states that the primary sources of mercury deposition into lakes and streams in the United States are coal-burning power plants. Risk assessment calculations for fish advisories in Maryland also take into account the widespread atmospheric deposition of mercury. The primary sources of mercury air emissions within Maryland include: power plants (43%), municipal waste combusters (31%), medical waste incinerators (19%), Portland Cement plants (6%), and other sources such as landfills, oil-fired power plants, and other industries (1%).[47,48,49,50,51]

While a person's individual level of fish consumption is often difficult to determine, one way to characterize the potential risk to fish consumers is to use a hazard index (HI) analysis. This analysis compares the estimated intake or dose of mercury for both average and high fish consumers to the current reference dose (RfD) established by EPA (HI = Dose/Rfd).[52][viii] The RfD is an estimate of the daily dose that is likely to be without an appreciable risk of adverse effects during a lifetime. Hazard indices are a simplified quantitative risk characterization measure that can be used to analyze regional hazards over time but they do not estimate the probability of adverse health outcomes within the population. A hazard index above one indicates that population exposures may exceed the RfD. Regions with a hazard index greater than one(HI >1) present a greater potential health risk for fisher people who eat fish from waters in these regions than areas with a low hazard index (HI < 1).

Table 9 presents a summary of Maryland monitoring data of mercury contamination in fish. The data are limited to inland waterways and do not include samples for the main-stem Bay because these monitoring data were not available at the time of analysis. Monitoring data show levels that are below the current FDA guidance level of one part per million. Exposure and risk, however, depend upon both the level of human consumption of fish from these waters and the concentration of the pollutant in the fish. Analysis of the data showed that five of eight regions had an estimated HI over one for high fish consumers and four of these five had excessively high levels. Current fish consumption advisories are supported by these findings. They also underscore the vulnerability of subsistence fisher people and their families who may be high-end consumers to potentially harmful levels of mercury exposure.

[viii] Fish tissue concentrations were based on a mean of all samples for each region. The consumer dose calculations followed the general USEPA equation. The population dose, calculated as an average daily dose (ADD), is an estimation of population intake rates.

$$ADD = \frac{IR*C*ED}{BW*AT}$$

Where IR = Intake rate (g/day), C = concentration in mg/kg fish tissue, BW = body weight (assumed 50 kg), ED = exposure duration over a lifetime, and AT = averaging time in lifetime. For this average daily dose calculation, these two components cancel one another out. Regional fish tissue concentrations were used. Intake rates for average consumers were calculated using EPA estimates of 17.5 g/day, high-end consumers' intake rate was estimated at 100 g/day based on findings from the National Academy of Sciences *Toxicological Effects of Methyl Mercury* report.

Table 9: Mercury Concentrations, Potential Population Exposures, and Hazard Index Scores for Maryland Waterways [viii]

		Average Fish Consumer*		High Fish Consumer*	
Region	Mean Fish Tissue Mercury Concentration (mg/kg)[1]	Dose of Mercury (ug/kg-day)[2]	Hazard Index (Dose/RfD)	Dose of Mercury (ug/kg-day)	Hazard Index (Dose/RfD)
Chester	0.063	0.02205	0.2205	0.126	1.26
Choptank	0.042	0.0147	0.147	0.084	0.84
Elk River	0.003	0.00105	0.0105	0.006	0.06
Gunpowder	0.435	0.15225	1.5225	0.87	8.7
Lower Susquehanna	0.149	0.05215	0.5215	0.298	2.98
Nanticoke/Wicomico	0.172	0.0602	0.602	0.344	3.44
Patapsco	0.233	0.08155	0.8155	0.466	4.66
Pocomoke	0.009	0.00315	0.0315	0.018	0.18

1) FDA guidance concentration = 1 ppm (mg/kg)
2) Mercury RfD = .1 ug/kg bw/day
*It is estimated an average consumer eats approximately 17.5 g/day. A high-end consumer is estimated to eat approximately 100 g/day.

Sources of PCBs

Polychlorinated biphenyls (PCBs) originate entirely from man-made sources. Between 1929 and 1977, approximately 700,000 tons of PCBs were manufactured in the United States. Peak production occurred in the 1970s and during this time, 42,500 tons of PCBs were used in the United States. In 1977 the manufacturing of PCBs was banned. They remain, however, ubiquitous in the environment. The distribution of PCBs in sediments is based on land use and is concentrated in urban areas where storm water facilitates transport these compounds to the aquatic environment and they ultimately end up in the sediment. Bioaccumulation of PCBs in fish tissue is due to PCB concentrations found in sediments that are then re-suspended in the water. Several sources of continued release into the environment are: continued use and disposal of PCB-containing products (e.g., transformers), combustion of PCB-containing materials, recycling of PCB-containing materials (e.g., carbonless copy paper), releases from waste storage and disposal, old consumer goods, and products that may contain PCBs which are not regulated. In addition, spills of PCBs often occur during handling and transport. Between 1989 and 2001 there were 2,611 spills of PCBs in the United States (spills greater than 1 lb. are reported to the EPA National Response Center). [53,54] Due to the nature of these releases, tracking current sources and release of PCBs in the environment is difficult. However, fish tissue concentration of PCBs offers one measure of PCB burden in the natural environment and can also be used to determine a potential level of population risk.

Table 10 presents a hazard index analysis for PCBs in fish samples from tidal tributaries of the Chesapeake Bay. While mean fish tissue concentrations are well below the FDA guidance level of 2 parts per million, the hazard index analysis shows the potential for increased risk of adverse effects for consumers. These concentrations have also resulted in the issuance of consumption advisories based on MDE's risk assessment methodology. Once again, high consumers are at greatest risk, with 14 of the 15 regions having an estimated HI greater than one.

Table 10: PCB Concentrations, Potential Population Exposures, and Hazard Index Scores for Maryland Waterways[ix]

		*Average Fish Consumer		*High Fish Consumer	
Region	Mean Fish Tissue Concentration (mg/kg)[1]	Dose of PCB (ug/kg-day)	Hazard Index (Dose/MRL)[2]	Dose of PCB (ug/kg-day)	Hazard Index (Dose/MRL)
Bush River	0.08	0.028	1.4	0.16	8
Chester	0.04	0.014	0.7	0.08	4
Choptank	0.1	0.035	1.75	0.2	10
Elk River	0.49	0.1715	8.575	0.98	49
Gunpowder	0.07	0.0245	1.225	0.14	7
Lower Potomac	0.54	0.189	9.45	1.08	54
Lower Susquehanna	0.34	0.119	5.95	0.68	34
Middle Potomac	0.11	0.0385	1.925	0.22	11
Nanticoke/Wicomico	0.02	0.007	0.35	0.04	2
Patapsco	0.28	0.098	4.9	0.56	28
Patuxent	0.08	0.028	1.4	0.16	8
Pocomoke	0.01	0.0035	0.175	0.02	1
Upper Potomac	0.03	0.0105	0.525	0.06	3
Washington Metropolitan	0.98	0.343	17.15	1.96	98
West Chesapeake	0.15	0.0525	2.625	0.3	15

1) FDA guidance concentration = 2 ppm (mg/kg)
2) Agency for Toxic Substances and Disease Registry (ATSDR) minimal risk level (MRL) = .02 ug/kg bw/day
*It is estimated an average consumer eats approximately 17.5 g/day. A high-end consumer is estimated to eat approximately 100 g/day.

These findings support the use of mercury and PCB concentration in fish tissue as indicators of the vulnerability of the Bay and its residents to persistent and bio-accumulating toxic pollutants. They also underscore the importance of improving risk communication about potential hazards of consuming fish from contaminated waters. At the present time there is limited monitoring of fish from the main-stem Bay. Expanded monitoring is needed to track the impacts of these pollutants on the Bay fisheries, and to better understand and control potential health risks to consumers.

Mercury and PCB fish tissue concentration: Utility as a public health indicator

Mercury and PCB fish tissue concentrations are useful indicators for tracking because of their public health significance and strong link to conditions in the ecological environment.[55] Concentrations of toxic pollutants in fish tissue are a priority environmental and public health indicator for national CDC/NCEH tracking networks. The body burden of persistent chemicals in Maryland fish populations indicates the legacy of toxic loading and current air deposition of pollutants into Maryland waterways. The culture of recreational fishing and subsistence fishing habits in the Chesapeake Bay and Maryland waters suggests that population exposure potential may be high. The difficulty of collecting fish tissue data and analyzing metals such as mercury has limited the availability of fish tissue data in the state. MDE environmental assessment initiatives for 2001–2002 and 2003 are currently working to improve data availability through

[ix] See footnote (viii) for Table 9.

better laboratory analysis and increased sampling sites throughout the entire watershed, including the Chesapeake Bay main-stem and its tributaries.

In conclusion, the hazard indices could be used as a long-term measurement for tracking variations in regional risk levels and should complement the current ongoing work at MDE. Fish consumption advisories are an essential tool for real-time public health protection and should be maintained. At the same time, additional information on regional population exposure through fish consumption is needed. The HI approach is different from the consumption advisory approach. Hazard indices provide a simplified method to identify contaminated waterways that pose risk, but they do not provide information on individual risk to consumers. Both risk characterization tools have important implications for public health tracking and provide essential information for prevention efforts to reduce population exposure and limit future toxic loadings into the watershed.

3) Enhance reporting of public health outcomes, such as waterborne and food-borne outbreaks to assure early problem recognition and to safeguard public health.

Improved outbreak surveillance could be developed in order to enhance prevention and complement current national efforts to improve identification of emerging population health threats.

4) Coordinate efforts with the EPA Environmental Indicators Initiative and the CDC National Environmental Public Health Tracking Network.

These developing national programs may provide opportunities for comparisons with national baseline data, uniform national reporting strategies, and enhanced resources for the development of a national network of environmental public health indicators.

5) Develop a formal strategy for systematic regular reporting of the pilot public health indicators through a public health report card for the Bay Region.

This should complement ecological reporting efforts, and include mapping and enhanced communication efforts to increase public awareness and provide appropriate public health perspective regarding potential risks.

The Chesapeake Bay is the region's defining natural resource. Its importance to culture, commerce, real estate, recreation, and the food supply cannot be overstated. Therefore it is important that the findings of this research be recognized as supportive of the many ongoing efforts to preserve this treasure. It is understandable that there may be some resistance to enhanced public awareness of pollution and its potential impacts upon public health. However, scientifically sound indicators provide a foundation for enhancing future efforts to protect the Bay. The linkage between public health and the health of the Bay should serve to underscore the importance of investing in the needed waste water infrastructure, enforcement, and monitoring. Public health indicators can also help to inform the difficult public policy debates concerning development, sprawl, and the future of the Chesapeake Bay.

[1] John Wesley Powell Memorial Museum. Major John Wesley Powell. Accessed 04/02/2003 at http://www.powellmuseum.org/MajorPowell.html.

[2] United States Environmental Protection Agency Watershed Information Network. "What Is a Watershed?" Accessed 10/04/2002 at http://www.epa.gov/win/what.html.

[3] Chesapeake Bay Program Office. "About the Chesapeake Bay—FAQ." Accessed 06/05/2002 at http://www.chesapeakebay.net/info/bayfaq.cfm.

[4] Abbruzzese, H., J. Barmach, S. Jones, K. Klick, J. Maldonado, M. Steinhilber, et al. "Keeping Pace: An Evaluation of Maryland's Most Important Environmental Problems and What We Can Do to Solve Them." Prepared for Senator Brian Frosh, University of Maryland School of Law, Environmental Law Clinic. Baltimore, MD. December 18, 2002.

[5] Chesapeake Bay Program, State of the Bay, 1995. "Land Use, Population, and Pollution." Accessed 10/23/1998 at http://www.chesapeakebay.net/bayprogram/pubs/state95/pop.htm.

[6] Maryland Department of Natural Resources. *Maryland Non-Point Source Management Plan. Chapter 2: Resource Assessment and Monitoring Programs*, pp. 6–7. December 1999. Accessed 03/12/2003 at http://www.dnr.state.md.us/bay/czm/nps/nps_plan/chapter_2.

[7] Shrivers, F.R., S.G. Davison, (Ed) J.Capper, G. Power, J. G. Merwin (contributer), *Chesapeake Waters: Four Centuries of Controversy, Concern, and Legislation*, 2nd edition. Tidewater Publishers, November 1997.

[8] Frumkin, H. "Beyond Toxicity: Human Health and the Natural Environment." *American Journal of Preventive Medicine* 2001; 20(3): pp. 234–240.

[9] Boesch, D.F. (2000) "Measuring the health of the Chesapeake Bay: Toward integration and prediction." *Environmental Research* **82**(2): pp. 134–42.

[10] Burke, T.A., J.S. Litt, and M. Fox. (2000) "Linking public health and the health of the Chesapeake Bay." *Environmental Research* **82**(2): pp. 143–9.

[11] deFur, P. L. and L. Foersom. (2000) "Toxic chemicals: Can what we don't know harm us?" *Environmental Research* **82**(2): pp. 113–33.

[12] Chivian, Eric. "Environment and Health: 7. Species loss and ecosystem disruption—The implications for human health." *CMAJ* 2001:164(1): pp. 66–9

[13] The Chesapeake Bay Foundation. *The State of the Bay Report 2000*. Annapolis, Maryland. 2000.

[14] Chesapeake Bay Program Office. "Reducing and Preventing Pollution." Accessed 02/10/2003 at http://www.chesaepeakbay.net/infor/reduce_pollution.cfm.

[15] Chesapeake Bay Program. "Land and People, Population Trends." Accessed 04/22/2003 at http://www.chesapeakebay.net/pop.htm.

[16] Maryland Institute for Policy Analysis Research. *Is Maryland Growing Smart? A Growth Indicators and Reporting System for Measuring the Achievement of the Goals of Maryland's Smart Growth Policy*. University of Maryland Baltimore County, Catonsville, MD. Accessed 01/2001 at http://www.umbc.edu/mipar/PDFs/SG_indicatorsreport.pdf.

[17] Chesapeake Bay Program Office. "Land and People." Accessed 05/21/2002 at http://www.chesapekaebay.net/land.htm.

[18] Boesch, D.F., R.B. Brinsfield, and R.E. Magnien. (2001) "Chesapeake Bay eutrophication: Scientific understanding, ecosystem restoration, and challenges for agriculture." *Journal of Environmental Quality* Mar-Apr; 30(2): pp. 303–20.

[19] Alavi, M., T. Miller, et al. (2001) "Bacterial community associated with Pfisteria-like dinoflagellate cultures." *Environmental Microbiology* 3(6): pp. 380–96.

[20] Silbergeld, E.K., L. Grattan, D. Oldach, and G.J. Morris. (2000) "*Pfisteria:* Harmful Algal Blooms as Indicator of Human: Ecosystem Interaction" *Environmental Research Section A.* 82, 97-105.

[21] Williams, J. P. "Our Rockfish Need Clean Water." *Chesapeake Notebook.* Annapolis, MD. April 24, 2002.

[22] Huslin, A. "Infection Attacking Rockfish in Chesapeake, Bacterial Disease First Noticed in Region Five Years Ago Now Rampant in the Bay." *The Washington Post,* 04/04/2002, p. B03. Accessed 04/08/2002 at http://www.WashingtonPost.com.

[23] Grattan, L.M., D. Oldach, et al. (1998) "Learning and memory difficulties after environmental exposure to waterways containing toxin-producing *Pfisteria* or *Pfisteria*–like dinoflagellates." *Lancet* 352(9127)532–9.

[24] Joe L., Hall E. (1994) "Mycobacterium marinum disease in Anne Arundel County: 1995 Update." *Maryland Medical Journal.* Dec; 44(12): pp. 1043–6.

[25] Kane, A.S., M.J. Dykstra, et. al. (200) "Etiologies, observations and reporting of estuarine finshfish lesions." *Marine Environmental Research.* 50(1-5): pp. 473–7.

[26] Brush, G. Personal communication.

[27] Heckert, R. A., S. Elankumaran, et al. (2001) "Detection of a new Mycobacterium species in wild striped bass in the Chesapeake Bay." *Journal of Clinical Microbiology* 39(2): pp. 710–5.

[28] Erkenbrecher, C. W., Jr. (1981) "Sediment bacterial indicators in an urban shellfishing subestuary of the lower Chesapeake Bay." *Applied Environmental Microbiology* **42**(3): pp. 484–92.

[29] Graczyk, T. K., R. C. Thompson, et al. (1999). "Giardia duodenalis cysts of genotype A recovered from clams in the Chesapeake Bay subestuary, Rhode River." *American Journal of Tropical Medicine and Hygiene* **61**(4): pp. 526–9.

[30] Graczyk, T. K., R. Fayer, et al. (2000) "Susceptibility of the Chesapeake Bay to environmental contamination with Cryptosporidium parvum." *Environmental Research* **82**(2): pp. 106–12.

[31] Maryland Department of the Environment. "General Mercury Information." Available at http://www.mde.state.md.us/programs/landprograms/hazardous_waste/mercury/mercuryinfo.asp. Copyright, 2003.

[32] United States Environmental Protection Agency. National Ambient Air Quality Standards Database, Classification of NAAQS. Accessed 02/2003 at http://www.epa.gov/oar/oaqps/greenbk/ancl.html#MARYLAND.

[33] USEPA Chesapeake Bay Program Office. "The State of the Bay: A Report to the Citizens of the Bay Region (July 2002)." CBP/TRS 260/02 EPA 903-R-02-002.

[34] United States EPA Chesapeake Bay Program. "Chemical of Concern, Bay Restoration—Reducing and Preventing Toxic Pollution." *Toxics 2000* Strategy Appendix A "Chesapeake Bay Watershed Chemicals of Concern as of September 26, 2000.
http://www.chesapeakebay.net/pubs/subcommittee/tsc/toxics/pdf%20finals/toxics_2000_appendix_a.PDF.

[35] United States EPA Chesapeake Bay Program. *Toxics Strategy 2000—Executive Summary.*
http://www.chesapeakebay.net/pubs/toxics/toxics_exec_summary.pdf.

[36] United States EPA Chesapeake Bay Program Office. "Targeting Toxics: A Characterization Report A Tool for Directing Management Monitoring Actions in the Chesapeake Bays Tidal Rivers." June, 1999 Accessed 10/2002 at: http://www.chesapeakebay.net/pubs/792.pdf.

[37] Environmental Defense. "Scorecard—Health Effects." At: http://www.scorecard.org/health-effects/. Last accessed 02/11/03.

[38] Environmental Defense Fund. "TRI Summary Data Files for Maryland." Accessed 02/11/2003 at http://www.scorecard.org/env-releases/state.tcl?usps_abbrev=MD#pollution_rank_healht_effect.

[39] United States Environmental Protection Agency. "Toxic Release Inventory Explorer Report. (STCH)" Accessed 02/11/2003 at http://www.epa.gov/tri/tridata/index.htm.

[40] Council for State and Territorial Epidemiologists. "Environmental Public Health Indicators. A guide for monitoring the occurrence of environmentally related exposures and diseases." Accessed 10/2002 at: http://www.cste.org/pdffiles/Environmentalpublichealthindicators.pdf.

[41] United States Environmental Protection Agency. "Environmental Indicators Initiative." Accessed 01/20/2003 at http://www.epa.gov/indicate/.

[42] Centers for Disease Control National Center for Environmental Health Assessment Division of Environmental Hazards and Health Effects. *Environmental Public Health Indicators.* Atlanta, Georgia, October 2002.

[43] Bing-Fang Hwang, O.R. Magnus, and J.J.K. Jaakkola. (2002) "Risk of Specific Birth Defects in Relation to Chlorination and the Amount of Natural Organic Matter in the Water Supply. " *American Journal of Epidemiology.* Vol. 156. No. 4: pp. 374–382.

[44] Maryland Department of the Environment Water Supply Program. "Safe Drinking Water Act Annual Compliance Report for Calendar Year 2002." Accessed 02/28/2004 at http://www.mde.state.md.us/assets/document/water supply/water_supply_ar2002.pdf."

[45] Pruss, A. (1998) "Review of epidemiological studies on health effects from exposure to recreational water." *International Journal of Epidemiology*, 27: pp.1–9.

[46] Barwick, R.S.,D.A. Levy, Ph.D., G.F. Craun, M.J. Beach, and R.L. Calderon. "Surveillance for Waterborne-Disease Outbreaks—United States, 1997–1998" *MMWR Surveillance Report.* Vol 49, No SS04:1 05/26/2000.

[47] George, J. "Program turns to mercury and pH loading the Bay." *MDEnvironment.* Published by Maryland Department of the Environment, January 2003: p. 3

[48] U. S. Department of Health and Human Service, Agency for Toxic Substances and Disease Registry. *Toxicological Profile for Mercury.* December, 1989.

[48] Committee on the Toxicological Effects of Methylmercury, Board on Environmental Studies and Toxicology, National Research Council. *Toxicological Effects of Methyl Mercury.* Washington, DC: National Academies Press, 2000.

[50] Gilmour, C.C., and G.S. Riedel. "A survey of size specific mercury concentrations in game fish from Maryland fresh and estuarine waters." *Archives of Environmental Contamination and Toxicology* 2000 Jul:39(1): pp. 53–9.

[51] Committee on the Toxicological Effects of Methylmercury, Board on Environmental Studies and Toxicology, National Research Council. *Toxicological Effects of Methyl Mercury.* Washington, DC: National Academies Press, 2000.

[52] Fan A, Chang L, *Toxicology and Risk Assessment. Principles, Methods, and Applications.* Marcel Dekkar, Inc. New York, NY. 1996.

[53] U. S. Department of Health and Human Service, Agency for Toxic Substances and Disease Registry. *Toxicological Profile for Polychlorinated Biphenyls.* August 1995.

[54] National Research Council. "A Risk Management Strategy for PCBs-Contaminated Sediments." Washington, DC: National Academy Press, 2001: pp. 27–29.

[55] Burger, J., and M. Gochfeld. (2001) "On developing bioindicators for human and ecological health." *Environmental Monitoring and Assessment.* Jan 66(1): pp. 23–46

Appendix 1

EDF Epidemiologic and Toxicology References in Scientific Literature: References for Environmental Defense Toxicology Analysis Listed by Health Effect as of February 2003.

Cancer References

P65: The most current and authoritative list of chemicals that are recognized to cause cancer is California's Proposition 65. Substances are placed on the Proposition 65 list of chemicals "known to the state of California to cause cancer" if an independent science advisory board has concluded they possess sufficient evidence of carcinogenicity in animals or humans, or if an authoritative organization such as the International Agency for Research on Cancer or the National Toxicology Program has reached a similar conclusion, or if a federal regulatory agency requires a cancer warning label. The current Proposition 65 List of Carcinogens (June 2002) can be obtained from http://www.oehha.ca.gov/prop65/prop65_list/Newlist.html.

A number of chemicals have toxicological evidence of carcinogenicity that currently does not satisfy the sufficiency criteria used to list agents under Proposition 65, or that has not yet been finally evaluated by the carcinogen identification processes in the state of California or other authoritative agencies.

Environmental Defense's list of suspect carcinogens is compiled from the following sources:

CPDB: Carcinogenic Potency Database. http://potency.berkeley.edu/app14.html.
Environmental Defense reviewed this compilation of results on carcinogenicity in rats and mice covering 1298 chemicals and added any chemical with positive results in at least two species by a relevant route of exposure to its list of suspect carcinogens.

EDF: See Environmental Defense's Custom Hazard Identification documentation.

EPA-HEN: US EPA, Air Risk Information Support Center. *Health Effects Notebook for Hazardous Air Pollutants*. http://www.epa.gov/ttnatw01/hlthef/hapindex.html.

EPA-TRI: US EPA. Addition of Certain Chemicals; Toxic Chemical Release Reporting; Community Right to Know. Proposed and Final Rules. 59 *Federal Register* 1788 (Jan 12, 1994); 59 *Federal Register* 61432 (November 30, 1994). Summarized in *Hazard Information on Toxic Chemicals Added to EPCRA Section 313 Under Chemical Expansion*. http://www.epa.gov/tri/chemical/hazard_cx.htm.

HAZMAP: A Relational Database of Hazardous Chemicals and Occupational Diseases. Browse Haz-Map by Adverse Effects: Carcinogen (Known, Probable, Possible) and Lung Toxin—Lung Cancer.
http://hazmap.nlm.nih.gov/hazmapadv.html.

IARC: World Health Organization, International Agency for Research on Cancer. *IARC Monographs Programme on the Evaluation of Carcinogenic Risks to Humans*. Lists of Group 1, 2a, and 2b substances can be obtained at http://monographs.iarc.fr/monoeval/grlist.html.

IRIS: US EPA, National Center for Environmental Assessment. Integrated Risk Information System.
http://www.epa.gov/iriswebp/iris/index.html.

NTP-BR: National Toxicology Program. Summary for Agents, Substances, Mixtures or Exposure Circumstances to Be Reviewed in 2001–2002 for Possible Listing in the Report on Carcinogens, Eleventh Edition. NTP Board of Scientific Counselors, NTP, Research Triangle Park, NC. http://ntp-server.niehs.nih.gov/NewHomeRoc/11thConsideration.html.

NTP-C: National Toxicology Program. *10th Report on Carcinogens (2002)*. http://ehis.niehs.nih.gov/roc/toc10.html.

NTP-HS: National Toxicology Program. *Chemical Repository of Health and Safety Data*. http://ntp-db.niehs.nih.gov/NTP_Reports/NTP_Chem_H&S/NTP_Chem8/Radian[Insert CAS#].txt.

OEHHA-TCD: California EPA, Office of Environmental Health Hazard Assessment. *Toxicity Criteria Database—OEHHA Cancer Potency Values*. http://www.oehha.ca.gov/risk/pdf/CancerPotDec2002.pdf.

OPP-CAN: US EPA, Office of Pesticide Programs. List of Chemicals Evaluated for Carcinogenic Potential (5/10/2002). OPP, Washington, DC. http://www.epi.uci.edu/valleycenter/EPAListCarcinogenicChemicals.pdf.

ORD-SF: US EPA, Office of Research and Development, Superfund Health Risk Technical Support Center. *Risk Assessment Issue Paper for: Toxicity Information and Provisional Oral Slope Factor for Nitroglycerin*. ORD, Washington, DC.

P65-CAND: California EPA, Office of Environmental Health Hazard Assessment. Prioritization Notices. http://www.oehha.org/prop65/CRNR_notices/state_listing/prioritization_notices/index.html.
Chemicals on Candidate List for consideration under Proposition 65 due to high carcinogenicity concern (through November 1999).
Chemicals under consideration for listing via the authoritative bodies mechanisms (through November 2000).

P65-MC: Hazard identification based on an extension of a Proposition 65 listing. Substance is either a member of a class that is a recognized Proposition 65 hazard, or is a class that contains a member that is a recognized Proposition 65 hazard. See Environmental Defense's Member Class Hazard Identification documentation.

P65-PEND: California EPA, Office of Environmental Health Hazard Assessment. Notices of Intent to List. http://www.oehha.org/prop65/CRNR_notices/admin_listing/intent_to_list/index.html.
Chemicals noticed for listing under Proposition 65 because a body considered to be authoritative by the state's qualified experts has formally identified it as causing cancer (through August 2002).

SCDM: US EPA, Office of Emergency Response and Remediation. *Superfund Chemical Data Matrix*. http://www.epa.gov/superfund/resources/scdm/index.htm.

Developmental Toxicity References

P65: The most current and authoritative list of chemicals that are recognized to cause developmental toxicity is California's Proposition 65. Substances are placed on the Proposition 65 list of chemicals "known to the state of California to cause reproductive toxicity" if an independent science advisory board has concluded they possess sufficient evidence of such toxicity in animals or humans, or if an authoritative organization such as the National Toxicology Program has reached a similar conclusion, or if a federal regulatory agency requires a reproductive toxicity warning label. The Proposition 65 list identifies whether a chemical is a developmental toxicant. The current Proposition 65 List of Carcinogens (June 2002) can be obtained from http://www.oehha.ca.gov/prop65/prop65_list/Newlist.html.

A number of chemicals have toxicological evidence of developmental toxicity that currently does not satisfy the sufficiency criteria used to list agents under Proposition 65, or that has not yet been finally evaluated by hazard identification processes in the state of California or other authoritative agencies. Environmental Defense's list of suspect developmental toxicants is compiled from the following sources:

CAA-AQC: US EPA, Office of Research and Development. *Air Quality Criteria for Oxides of Nitrogen, Volume III.* Washington, DC. August 1993. http://www.epa.gov/ttn/naaqs/standards/nox/s_nox_index.html.
US Environmental Protection Agency, Office of Research and Development. *Air Quality Criteria for Carbon Monoxide.* Washington, DC. December 1991. http://www.epa.gov/ttn/naaqs/standards/co/s_co_index.html.

CERHR: National Toxicology Program Center for the Evaluation of Risks to Human Reproduction. NTP-CERHR Briefs and Expert Panel Reports. http://cerhr.niehs.nih.gov/.

EDF: See Environmental Defense's Custom Hazard Identification documentation.

EPA-HEN: US EPA, Air Risk Information Support Center. *Health Effects Notebook for Hazardous Air Pollutants.* http://www.epa.gov/ttnatw01/hlthef/hapindex.html.

EPA-SARA: US EPA. *Roadmaps to Sources of Information on Chemicals Listed in the Emergency Planning Community and Community Right-to-Know Act (Also Known as SARA Title 3), Section 313 Toxic Release Inventory (for Microcomputers).* (Report Number EPADFDK92040). 1991. Data file distributed in 2 diskettes by Office of Pollution, Prevention, and Toxics, Environmental Protection Agency, Washington, DC. NOTE: Datasource no longer being maintained by EPA; not currently available online.

EPA-TRI: US EPA. Addition of Certain Chemicals; Toxic Chemical Release Reporting; Community Right to Know. Proposed and Final Rules. 59 *Federal Register* 1788 (Jan 12, 1994); 59 *Federal Register* 61432 (November 30, 1994). Summarized in *Hazard Information on Toxic Chemicals Added to EPCRA Section 313 Under Chemical Expansion.* http://www.epa.gov/tri/chemical/hazard_cx.htm.

JANK: Jankovic, J. "A Screening Method for Occupational Reproductive Health Risk." *American Industrial Hygiene Association Journal.* 57: 641-649. 1996.

NTP-R: Chapin, R.E., and R.A. Sloane. "NIEHS/NTP Reproductive Assessment by Continuous Breeding: Evolving Study Design and Summaries of Ninety Studies." *Environmental Health Perspectives* 105, Supplement 1: 199–394. 1997. http://ehpnet1.niehs.nih.gov/docs/1997/Suppl-1/chapin.html.

OEHHA-AREL: California EPA, Office of Environmental Health Hazard Assessment. *Acute Reference Exposure Levels (RELs), Averaging Times, and Toxicologic Endpoints.* Includes all Acute Reference Exposure Levels (ARELs) developed by OEHHA through May 2000 http://www.oehha.org/air/acute_rels/allAcRELs.html

OEHHA-CREL: California EPA, Office of Environmental Health Hazard Assessment. *Air Toxics Hot Spots Program Risk Assessment Guidelines, Part III: Technical Support Document "Determination of Noncancer Chronic Reference Exposure Levels."* Includes all Chronic Reference Exposure Levels (CRELs) adopted by OEHHA as of

September 2002 (http://www.oehha.ca.gov/air/chronic_rels/AllChrels.html), plus draft CRELS proposed through September 2002 (http://www.oehha.ca.gov/air/chronic_rels/index.html).

P65-CAND: California EPA, Office of Environmental Health Hazard Assessment. Chemicals under consideration for possible listing via the authoritative bodies mechanisms (through November 2001). http://www.oehha.ca.gov/prop65/CRNR_notices/admin_listing/requests_info/.

P65-MC: Hazard identification based on an extension of a Proposition 65 listing. Substance is either a member of a class that is a recognized Proposition 65 hazard, or is a class that contains a member that is a recognized Proposition 65 hazard. See Environmental Defense's Member Class Hazard Identification documentation.

P65-PEND: California EPA, Office of Environmental Health Hazard Assessment. Notices of Intent to List. http://www.oehha.org/prop65/CRNR_notices/admin_listing/intent_to_list/index.html. Chemicals noticed for listing under Proposition 65 because a body considered to be authoritative by the state's qualified experts has formally identified it as causing reproductive toxicity (through August 2002).

Reproductive Toxicity References

P65: The most current and authoritative list of chemicals that are recognized to cause reproductive toxicity is California's Proposition 65. Substances are placed on the Proposition 65 list of chemicals "known to the state of California to cause reproductive toxicity" if an independent science advisory board has concluded they possess sufficient evidence of such toxicity in animals or humans, or if an authoritative organization such as the National Toxicology Program has reached a similar conclusion, or if a federal regulatory agency requires a reproductive toxicity warning label. The Proposition 65 list identifies whether a chemical is a male or female reproductive toxicant. The current Proposition 65 List of Carcinogens (June 2002) can be obtained from http://www.oehha.ca.gov/prop65/prop65_list/Newlist.html.

A number of chemicals have toxicological evidence of reproductive toxicity that currently does not satisfy the sufficiency criteria used to list agents under Proposition 65, or that has not yet been finally evaluated by hazard identification processes in the state of California or other authoritative agencies. Environmental Defense's list of suspect reproductive toxicants is compiled from the following sources:

ATSDR: Agency for Toxic Substances and Disease Registry. "Minimal Risk Levels for Hazardous Substances." December 2001. http://www.atsdr.cdc.gov/mrls.html.

CERHR: National Toxicology Program Center for the Evaluation of Risks to Human Reproduction. NTP-CERHR Briefs and Expert Panel Reports. http://cerhr.niehs.nih.gov/.

EDF: See Environmental Defense's Custom Hazard Identification documentation.

EPA-HEN: US EPA, Air Risk Information Support Center. *Health Effects Notebook for Hazardous Air Pollutants.* http://www.epa.gov/ttnatw01/hlthef/hapindex.html.

EPA-SARA: US EPA. *Roadmaps to Sources of Information on Chemicals Listed in the Emergency Planning Community and Community Right-to-Know Act (Also Known as SARA Title 3), Section 313 Toxic Release Inventory (for Microcomputers).* (Report Number EPADFDK92040). 1991. Data file distributed in 2 diskettes by Office of Pollution, Prevention, and Toxics, Environmental Protection Agency, Washington, DC. NOTE: Datasource no longer being maintained by EPA; not currently available online.

EPA-TRI: US EPA. Addition of Certain Chemicals; Toxic Chemical Release Reporting; Community Right to Know. Proposed and Final Rules. 59 *Federal Register* 1788 (Jan 12, 1994); 59 *Federal Register* 61432 (November 30, 1994). Summarized in *Hazard Information on Toxic Chemicals Added to EPCRA Section 313 Under Chemical Expansion.* http://www.epa.gov/tri/chemical/hazard_cx.htm.

FRAZIER: Frazier, L. and M. L. Hage (eds.). *Reproductive Hazards of the Workplace.* Wiley Europe, 1998. Table 10 (Partial List of Reproductive Toxicants) available at http://www.pharmacy.ohio-state.edu/homepage/safety/chemhygiene_table_repro.pdf.

HAZMAP:A Relational Database of Hazardous Chemicals and Occupational Diseases. Browse Haz-Map by Adverse Effects: Other Tissue Toxin—Reproductive Toxin. http://hazmap.nlm.nih.gov/hazmapadv.html.

JANK: Jankovic, J. "A Screening Method for Occupational Reproductive Health Risk." *American Industrial Hygiene Association Journal.* 57: 641-649. 1996.

KEMI-DAN: Hass, U. et al. *Reproductive Toxicants in the Working Environment (In Danish.).* Reproduktionsskadende kemiske stoffer i arbejdsmiljoet. AMI-rapport Nr. 35/1991. National Institute of Occupational Health, Copenhagen, DK. 1991.

NTP-R: Chapin, R.E., and R.A. Sloane. "NIEHS/NTP Reproductive Assessment by Continuous Breeding: Evolving Study Design and Summaries of Ninety Studies." *Environmental Health Perspectives* 105, Supplement 1: 199-394. 1997. http://ehpnet1.niehs.nih.gov/docs/1997/Suppl-1/chapin.html.

OEHHA-AREL: California EPA, Office of Environmental Health Hazard Assessment. *Acute Reference Exposure Levels (RELs), Averaging Times, and Toxicologic Endpoints*. Includes all Acute Reference Exposure Levels (ARELs) developed by OEHHA through May 2000 http://www.oehha.org/air/acute_rels/allAcRELs.html.

OEHHA-CREL: California EPA, Office of Environmental Health Hazard Assessment. *Air Toxics Hot Spots Program Risk Assessment Guidelines, Part III: Technical Support Document "Determination of Noncancer Chronic Reference Exposure Levels."* Includes all Chronic Reference Exposure Levels (CRELs) adopted by OEHHA as of September 2002 (http://www.oehha.ca.gov/air/chronic_rels/AllChrels.html), plus draft CRELS proposed through September 2002 (http://www.oehha.ca.gov/air/chronic_rels/index.html).

P65-CAND: California EPA, Office of Environmental Health Hazard Assessment. Chemicals under consideration for possible listing via the authoritative bodies mechanisms (through November 2001). http://www.oehha.ca.gov/prop65/CRNR_notices/admin_listing/requests_info/.

P65-MC: Hazard identification based on an extension of a Proposition 65 listing. Substance is either a member of a class that is a recognized Proposition 65 hazard, or is a class that contains a member that is a recognized Proposition 65 hazard. See Environmental Defense's Member Class Hazard Identification documentation.

P65-PEND: California EPA, Office of Environmental Health Hazard Assessment. Notices of Intent to List. http://www.oehha.org/prop65/CRNR_notices/admin_listing/intent_to_list/index.html. Chemicals noticed for listing under Proposition 65 because a body considered to be authoritative by the state's qualified experts has formally identified it as causing reproductive toxicity (through August 2002).

Endocrine Toxicity References

There is no generally accepted source for an authoritative list of chemicals that are recognized to cause endocrine toxicity.

Environmental Defense's list of suspect endocrine toxicants is compiled from the following sources:

ATSDR: Agency for Toxic Substances and Disease Registry. "Minimal Risk Levels for Hazardous Substances." December 2001. http://www.atsdr.cdc.gov/mrls.html.

BKH: BKH/European Commission. "Towards the establishment of a priority list of substances for further evaluation of their role in endocrine disruption: Preparation of a candidate list of substances as a basis for priority setting. Final report—November 2000." http://europa.eu.int/comm/environment/docum/01262_en.htm#bkh. Category 1 chemicals from Annex 1: Candidate list of 553 substances. http://europa.eu.int/comm/environment/docum/bkh_annex_01.pdf.

BRUC: Brucker-Davis, F. "Effects of Environmental Synthetic Chemicals on Thyroid Function." *Thyroid*. 8(9): 827-856. 1998.

EDF: See Environmental Defense's Custom Hazard Identification documentation.

EPA-HEN: US EPA, Air Risk Information Support Center. *Health Effects Notebook for Hazardous Air Pollutants*. http://www.epa.gov/ttnatw01/hlthef/hapindex.html.

EPA-SDWA: UUS EPA. "Announcement of the Draft Drinking Water Contaminant Candidate List; Notice 62." *Federal Register* 52193-52219 (October 6, 1997). (Table 6). http://www.epa.gov/safewater/ccl/dwccl.pdf.

EPA-TRI: US EPA. Addition of Certain Chemicals; Toxic Chemical Release Reporting; Community Right to Know. Proposed and Final Rules. 59 *Federal Register* 1788 (Jan 12, 1994); 59 *Federal Register* 61432 (November 30, 1994). Summarized in *Hazard Information on Toxic Chemicals Added to EPCRA Section 313 Under Chemical Expansion*. http://www.epa.gov/tri/chemical/hazard_cx.htm.

GUIL: Guillette, L. J., and E. Guillette. "Environmental Contaminants and Reproductive Abnormalities in Wildlife: Implications for Public Health?" *Toxicology and Industrial Health*. 12(3): 537-550. 1996.

IL-EPA: Illinois EPA. Endocrine Disruptors Strategy. 1997. (Table 1: Preliminary List of Chemicals Associated with Endocrine System Effects in Animals and Humans or In Vitro).
http://www.nihs.go.jp/hse/environ/illiepatable.htm.

JNIHS: Japanese National Institute of Health Sciences. Lists of Paradigmatic Chemicals.
http://www.nihs.go.jp/hse/endocrine-e/paradigm/paradigm.html.

KEIT: Keith, L.H. (ed.). *Environmental Endocrine Disruptors*. New York: John Wiley & Sons, 1997.
http://www.wileyeurope.com/cda/product/0,,0471191450%7Cdesc%7C3037,00.html.

NJ-FS: New Jersey Department of Health Services. Right to Know Program, NJDOH, Trenton, NJ.
http://www.state.nj.us/health/eoh/rtkweb/rtkhsfs.htm.

OEHHA-CREL: California EPA, Office of Environmental Health Hazard Assessment. *Air Toxics Hot Spots Program Risk Assessment Guidelines, Part III: Technical Support Document "Determination of Noncancer Chronic Reference Exposure Levels."* Includes all Chronic Reference Exposure Levels (CRELs) adopted by OEHHA as of September 2002 (http://www.oehha.ca.gov/air/chronic_rels/AllChrels.html), plus draft CRELS proposed through September 2002 (http://www.oehha.ca.gov/air/chronic_rels/index.html).

RTECS: National Institute for Occupational Safety and Health's Registry of Toxic Effects of Chemical Substances. See Environmental Defense's Suspect Hazard Identification documentation.

WWF: World Wildlife Fund. Our Stolen Future. Widespread Pollutants with Endocrine-disrupting Effects. http://www.ourstolenfuture.org/Basics/chemlist.htm. The WWF list is derived from references detailed at http://www.ourstolenfuture.org/Sources/chemsources.htm and was originally published in: Colborn, T., F.S. vom Saal, and A.M. Soto. "Developmental Effects of Endocrine-Disrupting Chemicals In Wildlife and Humans." *Environmental Health Perspectives* 101(5): 378-384. 1993.

Gastrointestinal or Liver Toxicity References

There is no generally accepted source for an authoritative list of chemicals that are recognized to cause liver or gastrointestinal toxicity.

Environmental Defense's list of suspect liver or gastrointestinal toxicants is compiled from the following sources:

ATSDR: Agency for Toxic Substances and Disease Registry. "Minimal Risk Levels for Hazardous Substances." December 2001. http://www.atsdr.cdc.gov/mrls.html.

ATSDR-FAQ: Agency for Toxic Substances and Disease Registry. ToxFAQs. http://www.atsdr.cdc.gov/toxfaq.html.

CAA-AQC: US EPA, Office of Research and Development. *Air Quality Criteria for Ozone and Related Photochemical Oxidants, Volume III.* Washington, DC. July 1996. http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_index.html.

CARB-TAC: California Air Resources Board. *Toxic Air Contaminant Fact Sheets.* http://www.arb.ca.gov/toxics/tac/tac.htm.

DIPA: DiPalma, J.A., J. Cunningham, J. Herrera, T. McCaffery, and D. Wolf. "Occupational and Industrial Toxin Exposures and the Gastrointestinal Tract." *American Journal of Gastroenterology.* 86(9): 1107-1117. 1991. (Table 2: Selected Agents with Purported Digestive System Injury.)

DOSS: Dossing, M. and P. Skinhoj. "Occupational Liver Injury: Present State of Knowledge and Future Perspectives." *International Archives of Occupational and Environmental Health.* 56:1-21. 1985. (Table 2: Chemically induced liver injury: Morphologic features and examples of confirmed and suspected causative agents.)

EDF: See Environmental Defense's Custom Hazard Identification documentation.

EPA-HEN: US EPA, Air Risk Information Support Center. *Health Effects Notebook for Hazardous Air Pollutants.* http://www.epa.gov/ttnatw01/hlthef/hapindex.html.

EPA-TRI: US EPA. Addition of Certain Chemicals; Toxic Chemical Release Reporting; Community Right to Know. Proposed and Final Rules. 59 *Federal Register* 1788 (Jan 12, 1994); 59 *Federal Register* 61432 (November 30, 1994). Summarized in *Hazard Information on Toxic Chemicals Added to EPCRA Section 313 Under Chemical Expansion.* http://www.epa.gov/tri/chemical/hazard_cx.htm.

EXTOX: EXtension TOXicology NETwork. Pesticide Information Profiles (PIPs). http://ace.ace.orst.edu/info/extoxnet/pips/pips.html.

HAZMAP: A Relational Database of Hazardous Chemicals and Occupational Diseases. Industrial Chemicals Associated with Toxic Hepatitis. http://www.haz-map.com/heptox1.htm.

KLAA: Klaassen, C., M. Amdur, and J. Doull (eds.). *Casarett and Doull's Toxicology. The Basic Science of Poisons,* 5th Edition. New York: Pergamon Press, 1996. (Table 13-2: Types of Hepatic Injury.)

LADO: LaDou, J. (ed.). *Occupational Medicine.* Norwalk, CT: Appleton & Lange, 1990. (Table 20-1: Chemical Agents associated with occupational liver disease; Table 20-4: Agents causing acute hepatic injury.)

MALA: Malachowsky, M.J. *Health Effects of Toxic Substances.* Government Institutes. Rockville, MD 1995. (Tables 7-2&3: Hepatotoxic Agents).

NJ-FS: New Jersey Department of Health Services. Right to Know Program, NJDOH, Trenton, NJ. http://www.state.nj.us/health/eoh/rtkweb/rtkhsfs.htm.

OEHHA-AREL: California EPA, Office of Environmental Health Hazard Assessment. *Acute Reference Exposure Levels (RELs), Averaging Times, and Toxicologic Endpoints*. Includes all Acute Reference Exposure Levels (ARELs) developed by OEHHA through May 2000 http://www.oehha.org/air/acute_rels/allAcRELs.html.

OEHHA-CREL: California EPA, Office of Environmental Health Hazard Assessment. *Air Toxics Hot Spots Program Risk Assessment Guidelines, Part III: Technical Support Document "Determination of Noncancer Chronic Reference Exposure Levels."* Includes all Chronic Reference Exposure Levels (CRELs) adopted by OEHHA as of September 2002 (http://www.oehha.ca.gov/air/chronic_rels/AllChrels.html), plus draft CRELS proposed through September 2002 (http://www.oehha.ca.gov/air/chronic_rels/index.html).

RTECS: National Institute for Occupational Safety and Health's Registry of Toxic Effects of Chemical Substances. See Environmental Defense's Suspect Hazard Identification documentation.

STAC: Stacey, N.H. *Occupational Toxicology*. Taylor & Francis. 1995. (Table 3.23: Clinical manifestations of chemical-induced gastrointestinal injury.)

ZIMM: Zimmerman, H.J. and J.H. Lewis. "Chemical- and Toxin-Induced Hepatotoxicity." *Gastroenterology Clinics of North America*. 24(4): 1027-1045. 1995. (Table 3: Forms of environmental hepatic injury.)

Immunotoxicity References

There is no generally accepted source for an authoritative list of chemicals that are recognized to cause immunotoxicity.

Environmental Defense's list of suspect immunotoxicants is compiled from the following sources:

ATSDR: Agency for Toxic Substances and Disease Registry. "Minimal Risk Levels for Hazardous Substances." December 2001. http://www.atsdr.cdc.gov/mrls.html.

ATSDR-FAQ: Agency for Toxic Substances and Disease Registry. ToxFAQs. http://www.atsdr.cdc.gov/toxfaq.html.

CAA-AQC: US EPA, Office of Research and Development. *Air Quality Criteria for Oxides of Nitrogen, Volume III.* Washington, DC. August 1993. http://www.epa.gov/ttn/naaqs/standards/nox/s_nox_index.html.

EDF: See Environmental Defense's Custom Hazard Identification documentation.

EEC: European Economic Community. Sensitizing Substances in the EEC List of Dangerous Substances. Annex I to Council Directive 67/548/EEC. http://www.kemi.se/default_eng.cfm?page=klass_mark/klasshem_eng.htm.

EPA-HEN: US EPA, Air Risk Information Support Center. *Health Effects Notebook for Hazardous Air Pollutants.* http://www.epa.gov/ttnatw01/hlthef/hapindex.html.

EPA-TRI: US EPA. Addition of Certain Chemicals; Toxic Chemical Release Reporting; Community Right to Know. Proposed and Final Rules. 59 *Federal Register* 1788 (Jan 12, 1994); 59 *Federal Register* 61432 (November 30, 1994). Summarized in *Hazard Information on Toxic Chemicals Added to EPCRA Section 313 Under Chemical Expansion.* http://www.epa.gov/tri/chemical/hazard_cx.htm.

HAZMAP: A Relational Database of Hazardous Chemicals and Occupational Diseases. Browse Haz-Map by Adverse Effects: Dermatotoxin—Skin Sensitizer or Photoallergic Contact Dermatitis. http://hazmap.nlm.nih.gov/hazmapadv.html.

IPCS: International Programme of Chemical Safety Environmental Health Criteria 180. *Principles and Methods for Assessing Direct Immunotoxicity Associated with Exposure to Chemicals.* Geneva: World Health Organization, 1996. (Table 1: Examples of compounds that are immunotoxic for humans or rodents.)

NAP: Nordic Council of Ministers (Nordic Allergy Project). Kriteriedokumenter fra nordisk allergiprojekt - vurdering af 50 kemiske stoffers evne til at forarsage allergi, en litteraturgennemgang. Nord 1991:51. Copenhagen, 1991.

NTP-I: National Toxicology Program. Summary Reports on Immunotoxicity. http://ntp-server.niehs.nih.gov/htdocs/pub-IT0.html.

OEHHA-AREL: California EPA, Office of Environmental Health Hazard Assessment. *Acute Reference Exposure Levels (RELs), Averaging Times, and Toxicologic Endpoints.* Includes all Acute Reference Exposure Levels (ARELs) developed by OEHHA through May 2000 http://www.oehha.org/air/acute_rels/allAcRELs.html.

OEHHA-CREL: California EPA, Office of Environmental Health Hazard Assessment. *Air Toxics Hot Spots Program Risk Assessment Guidelines, Part III: Technical Support Document "Determination of Noncancer Chronic Reference Exposure Levels."* Includes all Chronic Reference Exposure Levels (CRELs) adopted by OEHHA as of September 2002 (http://www.oehha.ca.gov/air/chronic_rels/AllChrels.html), plus draft CRELS proposed through September 2002 (http://www.oehha.ca.gov/air/chronic_rels/index.html).

RTECS: National Institute for Occupational Safety and Health's Registry of Toxic Effects of Chemical Substances. See Environmental Defense's Suspect Hazard Identification documentation.

SNCI: Swedish National Chemicals Inspectorate. Labelling of chemical products, Swedish regulations, and general advice with regard to the classification and labelling of chemical products hazardous to health, flammables, and explosives. List of Allergenic Substances. National Chemicals Inspectorate, Solna, Sweden: 1987, 1990–1992.

Kidney Toxicity References

There is no generally accepted source for an authoritative list of chemicals that are recognized to cause kidney toxicity.

Environmental Defense's list of suspect kidney toxicants is compiled from the following sources:

AEGL: US EPA, National Advisory Committee for Acute Exposure Guideline Levels for Hazardous Substances. Notices. 62 *Federal Register*: 58839-58851 (October 30, 1997).

ATSDR: Agency for Toxic Substances and Disease Registry. "Minimal Risk Levels for Hazardous Substances." December 2001. http://www.atsdr.cdc.gov/mrls.html.

CARB-TAC: California Air Resources Board. *Toxic Air Contaminant Fact Sheets*. http://www.arb.ca.gov/toxics/tac/tac.htm.

EDF: See Environmental Defense's Custom Hazard Identification documentation.

EPA-HEN: US EPA, Air Risk Information Support Center. *Health Effects Notebook for Hazardous Air Pollutants*. http://www.epa.gov/ttnatw01/hlthef/hapindex.html.

EPA-TRI: US EPA. Addition of Certain Chemicals; Toxic Chemical Release Reporting; Community Right to Know. Proposed and Final Rules. 59 *Federal Register* 1788 (Jan 12, 1994); 59 *Federal Register* 61432 (November 30, 1994). Summarized in *Hazard Information on Toxic Chemicals Added to EPCRA Section 313 Under Chemical Expansion*. http://www.epa.gov/tri/chemical/hazard_cx.htm.

HAZMAP: A Relational Database of Hazardous Chemicals and Occupational Diseases. Browse Haz-Map by Adverse Effects: Other Tissue Toxin—Nephrotoxin. http://hazmap.nlm.nih.gov/hazmapadv.html.

KLAA: Klaassen, C., M. Amdur and J. Doull (eds.). *Casarett and Doull's Toxicology. The Basic Science of Poisons*, 5th ed. New York: Pergamon Press, 1996. (Table 14-2: Examples of Nephrotoxic Therapeutic Agents: Table 14-3: Examples of Environmental Nephrotoxicants.)

LAND: Landrigan, P.J., Goyer, R.A. Clarkson, T.W., Sandler, D.P., Smith, J.H., Thun, M.J., and R. Wedeen. The Work-Relatedness of Renal Disease. *Archives of Environmental Health*. 39(3): 225–230. 1984. (Table 2: Estimated Numbers of Workers in the United States with Potential Occupational Exposures to Known or Suspect Nephrotoxins.)

NJ-FS: New Jersey Department of Health Services. Right to Know Program, NJDOH, Trenton, NJ. http://www.state.nj.us/health/eoh/rtkweb/rtkhsfs.htm.

OEHHA-AREL: California EPA, Office of Environmental Health Hazard Assessment. *Acute Reference Exposure Levels (RELs), Averaging Times, and Toxicologic Endpoints*. Includes all Acute Reference Exposure Levels (ARELs) developed by OEHHA through May 2000 http://www.oehha.org/air/acute_rels/allAcRELs.html.

OEHHA-CREL: California EPA, Office of Environmental Health Hazard Assessment. *Air Toxics Hot Spots Program Risk Assessment Guidelines, Part III: Technical Support Document "Determination of Noncancer Chronic Reference Exposure Levels."* Includes all Chronic Reference Exposure Levels (CRELs) adopted by OEHHA as of September 2002 (http://www.oehha.ca.gov/air/chronic_rels/AllChrels.html), plus draft CRELS proposed through September 2002 (http://www.oehha.ca.gov/air/chronic_rels/index.html).

RTECS: National Institute for Occupational Safety and Health's Registry of Toxic Effects of Chemical Substances. See Environmental Defense's Suspect Hazard Identification documentation.

STAC: Stacey, N.H. *Occupational Toxicology*. Taylor & Francis. 1995. (Table 3.8: Examples of workplace exposures that have resulted in renal toxicity.)

Musculoskeletal Toxicity References
There is no generally accepted source for an authoritative list of chemicals that are recognized to cause musculoskeletal toxicity.

Environmental Defense's list of suspect musculoskeletal toxicants is compiled from the following sources:

ATSDR: Agency for Toxic Substances and Disease Registry. "Minimal Risk Levels for Hazardous Substances." December 2001. http://www.atsdr.cdc.gov/mrls.html.

EDF: See Environmental Defense's Custom Hazard Identification documentation.

EPA-HEN: US EPA, Air Risk Information Support Center. *Health Effects Notebook for Hazardous Air Pollutants.* http://www.epa.gov/ttnatw01/hlthef/hapindex.html.

EPA-TRI: US EPA. Addition of Certain Chemicals; Toxic Chemical Release Reporting; Community Right to Know. Proposed and Final Rules. 59 *Federal Register* 1788 (Jan 12, 1994); 59 *Federal Register* 61432 (November 30, 1994). Summarized in *Hazard Information on Toxic Chemicals Added to EPCRA Section 313 Under Chemical Expansion.* http://www.epa.gov/tri/chemical/hazard_cx.htm.

OEHHA-CREL: California EPA, Office of Environmental Health Hazard Assessment. *Air Toxics Hot Spots Program Risk Assessment Guidelines, Part III: Technical Support Document "Determination of Noncancer Chronic Reference Exposure Levels."* Includes all Chronic Reference Exposure Levels (CRELs) adopted by OEHHA as of September 2002 (http://www.oehha.ca.gov/air/chronic_rels/AllChrels.html), plus draft CRELS proposed through September 2002 (http://www.oehha.ca.gov/air/chronic_rels/index.html).

RTECS: National Institute for Occupational Safety and Health's Registry of Toxic Effects of Chemical Substances. See Environmental Defense's Suspect Hazard Identification documentation.

Neurotoxicity References

There is no generally accepted source for an authoritative list of chemicals that are recognized to cause neurotoxicity.

Environmental Defense's list of suspect neurotoxicants is compiled from the following sources:

AEGL: US EPA, National Advisory Committee for Acute Exposure Guideline Levels for Hazardous Substances. Notices. 62 *Federal Register*: 58839-58851 (October 30, 1997).

ATSDR: Agency for Toxic Substances and Disease Registry. Minimal risk Levels for Hazardous Substances. December 2001. http://www.atsdr.cdc.gov/mrls.html.

CAA-AQC: US EPA, Office of Research and Development. *Air Quality Criteria for Carbon Monoxide*. Washington, DC. December 1991. http://www.epa.gov/ttn/naaqs/standards/co/s_co_index.html.

CARB-TAC: California Air Resources Board. *Toxic Air Contaminant Fact Sheets*. http://www.arb.ca.gov/toxics/tac/tac.htm.

DAN: Nordic Council of Ministers and Danish National Institute of Occupational Health. Neurotoxic Substances in the Working Environment (Danish ad hoc list). List originally published in *Neurotoxic Substances in the Work Environment*. Danish Working Environment Service, At-report Nr. 13/1990.

DPR-CIP: California EPA, Department of Pesticide Regulation. Summary of Pesticide Use Report Data 2001 Indexed by Chemical: Use Trends of Cholinesterase Inhibiting Pesticides. http://www.cdpr.ca.gov/docs/pur/pur01rep/chmrpt01.pdf.

EDF: See Environmental Defense's Custom Hazard Identification documentation.

EPA-HEN: US EPA, Air Risk Information Support Center. *Health Effects Notebook for Hazardous Air Pollutants*. http://www.epa.gov/ttnatw01/hlthef/hapindex.html.

EPA-SARA: US EPA. *Roadmaps to Sources of Information on Chemicals Listed in the Emergency Planning Community and Community Right-to-Know Act (Also Known as SARA Title 3), Section 313 Toxic Release Inventory (for Microcomputers)*. (Report Number EPADFDK92040). 1991. Data file distributed in 2 diskettes by Office of Pollution, Prevention, and Toxics, Environmental Protection Agency, Washington, DC. NOTE: Datasource no longer being maintained by EPA; not currently available online.

EPA-TRI: US EPA. Addition of Certain Chemicals; Toxic Chemical Release Reporting; Community Right to Know. Proposed and Final Rules. 59 *Federal Register* 1788 (Jan 12, 1994); 59 *Federal Register* 61432 (November 30, 1994). Summarized in *Hazard Information on Toxic Chemicals Added to EPCRA Section 313 Under Chemical Expansion*. http://www.epa.gov/tri/chemical/hazard_cx.htm.

EVAN: Evangelista, A.M. "Behavioral Toxicology, Risk Assessment, and Chlorinated Hydrocarbons." *Environmental. Health Perspectives*. 104 (Supplement 2): 353-360. 1996. (Table 1: Comparison of behavioral toxicity of chlorinated hydrocarbons and related compounds.)

FELD: Feldman, R.G. "Role of the Neurologist in Hazard Identification and Risk Assessment." *Environmental Health Perspectives*. 104 (Supplement 2):227-237. 1996. (Table 1: Neurologic symptoms and associated exposures.)

HAZMAP: A Relational Database of Hazardous Chemicals and Occupational Diseases. Browse Haz-Map by Adverse Effects: Neurotoxin. http://hazmap.nlm.nih.gov/hazmapadv.html.

KLAA: Klaassen, C., M. Amdur, and J. Doull, eds. *Casarett and Doull's Toxicology. The Basic Science of Poisons*, 5th ed. New York: Pergamon Press, 1996. (Table 16-1: Compounds Associated with Neuronal Injury; Table 16-2: Compounds Associated with Axonal Injury; Table 16-3: Compounds Associated with Injury of Myelin.)

LU: Lu, F.C. *Basic Toxicology*. 2nd Edition. 1991. (Appendix 16-1: Selected Neurotoxicants.)

MASL: Massachusetts Department of Public Health. Commonwealth of Massachusetts. 105CMR 670.000 Administrative Bulletin Concerning Massachusetts Substance List for "Right to Know" Law, M.G.L. 111F. 4/24/93. (Appendix A: Massachusetts Substance List.)

NJ-FS: New Jersey Department of Health Services. Right to Know Program, NJDOH, Trenton, NJ. http://www.state.nj.us/health/eoh/rtkweb/rtkhsfs.htm.

NTP-HS: National Toxicology Program. *Chemical Repository of Health and Safety Data*. http://ntp-db.niehs.nih.gov/NTP_Reports/NTP_Chem_H&S/NTP_Chem8/Radian[Insert CAS#].txt.

OEHHA-AREL: California EPA, Office of Environmental Health Hazard Assessment. *Acute Reference Exposure Levels (RELs), Averaging Times, and Toxicologic Endpoints*. Includes all Acute Reference Exposure Levels (ARELs) developed by OEHHA through May 2000 http://www.oehha.org/air/acute_rels/allAcRELs.html.

OEHHA-CREL: California EPA, Office of Environmental Health Hazard Assessment. *Air Toxics Hot Spots Program Risk Assessment Guidelines, Part III: Technical Support Document "Determination of Noncancer Chronic Reference Exposure Levels."* Includes all Chronic Reference Exposure Levels (CRELs) adopted by OEHHA as of September 2002 (http://www.oehha.ca.gov/air/chronic_rels/AllChrels.html), plus draft CRELS proposed through September 2002 (http://www.oehha.ca.gov/air/chronic_rels/index.html).

RTECS: National Institute for Occupational Safety and Health's Registry of Toxic Effects of Chemical Substances. See Environmental Defense's Suspect Hazard Identification documentation.

STAC: Stacey, N.H. *Occupational Toxicology*. Taylor & Francis. 1995. (Table 3.9: Occupational diseases—brain and spinal cord; Table 3.10: Occupations associated with an excess of brain cancer; Table 3.11: Agents capable of producing brain cancer in experimental animals; Table 3.12: Occupational diseases—mental disorders; Table 3.14: Agents causing occupational neuropathy.)

TANN: Tanner, C. "Occupational and Environmental Causes of Parkinsonism." *Occupational Medicine* 7(3): 5-3-513. (Table 2: Occupational and Environmental Causes of Parkinsonism.)

ZAKR: Zakrzewski, S.F. *Principles of Environmental Toxicology*. American Chemical Society, Washington, DC. 1997. (Table 7.4: TLV-TWA Values of Some Neurotoxins).

Respiratory Toxicity References
There is no generally accepted source for an authoritative list of chemicals that are recognized to cause respiratory toxicity.

Environmental Defense's list of suspect respiratory toxicants is compiled from the following sources:

AEGL: US EPA, National Advisory Committee for Acute Exposure Guideline Levels for Hazardous Substances. Notices. 62 *Federal Register*: 58839-58851 (October 30, 1997).

ATSDR: Agency for Toxic Substances and Disease Registry. "Minimal Risk Levels for Hazardous Substances." December 2001. http://www.atsdr.cdc.gov/mrls.html.

CAA-AQC: US EPA, Office of Research and Development.
Air Quality Criteria for Oxides of Nitrogen, Volume III. Washington, DC. August 1993.
http://www.epa.gov/ttn/naaqs/standards/nox/s_nox_index.html.
Air Quality Criteria for Ozone and Related Photochemical Oxidants, Volume III. Washington, DC. July 1996.
http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_index.html.
Air Quality Criteria for Particulate Matter, Volume III. Washington, DC. April 1996.
http://www.epa.gov/ttn/naaqs/standards/pm/s_pm_index.html.
Air Quality Criteria for Particulate Matter and Sulfur Oxides, Volume III. Washington, DC. December 1982.
http://www.epa.gov/ttn/naaqs/standards/so2/s_so2_index.html.

CARB-TAC: California Air Resources Board. *Toxic Air Contaminant Fact Sheets.*
http://www.arb.ca.gov/toxics/tac/tac.htm.

EDF: See Environmental Defense's Custom Hazard Identification documentation.

EEC: European Economic Community. Sensitizing Substances in the EEC List of Dangerous Substances. Annex I to Council Directive 67/548/EEC. http://www.kemi.se/default_eng.cfm?page=klass_mark/klasshem_eng.htm.

EPA-HEN: US EPA, Air Risk Information Support Center. *Health Effects Notebook for Hazardous Air Pollutants.*
http://www.epa.gov/ttnatw01/hlthef/hapindex.html.

EPA-TRI: US EPA. Addition of Certain Chemicals; Toxic Chemical Release Reporting; Community Right to Know. Proposed and Final Rules. 59 *Federal Register* 1788 (Jan 12, 1994); 59 *Federal Register* 61432 (November 30, 1994). Summarized in *Hazard Information on Toxic Chemicals Added to EPCRA Section 313 Under Chemical Expansion.* http://www.epa.gov/tri/chemical/hazard_cx.htm.

FOTH: Foth, H. "Role of the Lung in Accumulation and Metabolism of Xenobiotic compounds—Implications for Chemically Induced Toxicity." *Critical Reviews in Toxicology.* 25(2): 165-205. 1995. (Table 1:Toxic Damage of Lung by Foreign Compounds.)

HAZMAP: A Relational Database of Hazardous Chemicals and Occupational Diseases. Browse Haz-Map by Adverse Effects: Lung Toxin and Other Poison - Chemical Asphyxiant, Simple Asphyxiant .
http://hazmap.nlm.nih.gov/hazmapadv.html. Chemicals Associated with Occupational Asthma. http://www.haz-map.com/OA1.html.

KLAA: Klaassen, C., M. Amdur and J. Doull (eds.). *Casarett and Doull's Toxicology. The Basic Science of Poisons,* 5th Edition. New York: Pergamon Press, 1996. (Table 15-1: Industrial Toxicants that Produce Lung Disease.)

LADO: LaDou, J. (ed.). *Occupational Medicine.* Norwalk, CT: Appleton & Lange, 1990. (Table 39-1: Major pollutants associated with adverse pulmonary effects.)

LU: Lu, F.C. *Basic Toxicology*. 2nd Edition. 1991. (Appendix 11-1: Site of Action and Pulmonary Disease Produced by Selected Occupationally Inhaled Toxicants.)

NEME: Nemery, B. Metal Toxicity and the Respiratory Tract. *European Respiratory Journal*. 3(2): 202-219. 1990. (Table 1: Summary of pulmonary toxicity of metals.)

NJ-FS: New Jersey Department of Health Services. Right to Know Program, NJDOH, Trenton, NJ. http://www.state.nj.us/health/eoh/rtkweb/rtkhsfs.htm.

NTP-HS: National Toxicology Program. *Chemical Repository of Health and Safety Data*. http://ntp-db.niehs.nih.gov/NTP_Reports/NTP_Chem_H&S/NTP_Chem8/Radian[Insert CAS#].txt.

OEHHA-AREL: California EPA, Office of Environmental Health Hazard Assessment. *Acute Reference Exposure Levels (RELs), Averaging Times, and Toxicologic Endpoints*. Includes all Acute Reference Exposure Levels (ARELs) developed by OEHHA through May 2000 http://www.oehha.org/air/acute_rels/allAcRELs.html.

OEHHA-CREL: California EPA, Office of Environmental Health Hazard Assessment. *Air Toxics Hot Spots Program Risk Assessment Guidelines, Part III: Technical Support Document "Determination of Noncancer Chronic Reference Exposure Levels."* Includes all Chronic Reference Exposure Levels (CRELs) adopted by OEHHA as of September 2002 (http://www.oehha.ca.gov/air/chronic_rels/AllChrels.html), plus draft CRELS proposed through September 2002 (http://www.oehha.ca.gov/air/chronic_rels/index.html).

RTECS: National Institute for Occupational Safety and Health's Registry of Toxic Effects of Chemical Substances. See Environmental Defense's Suspect Hazard Identification documentation.

Skin or Sense Organ Toxicity References

There is no generally accepted source for an authoritative list of chemicals that are recognized to cause skin or sensory organ toxicity.

Environmental Defense's list of suspect skin or sensory organ toxicants is compiled from the following sources:

AEGL: US EPA, National Advisory Committee for Acute Exposure Guideline Levels for Hazardous Substances. Notices. 62 *Federal Register*: 58839-58851 (October 30, 1997).

ATSDR: Agency for Toxic Substances and Disease Registry. "Minimal Risk Levels for Hazardous Substances." December 2001. http://www.atsdr.cdc.gov/mrls.html.

CARB-TAC: California Air Resources Board. *Toxic Air Contaminant Fact Sheets.* http://www.arb.ca.gov/toxics/tac/tac.htm.

EDF: See Environmental Defense's Custom Hazard Identification documentation.

EEC: European Economic Community. Sensitizing Substances in the EEC List of Dangerous Substances. Annex I to Council Directive 67/548/EEC. http://www.kemi.se/default_eng.cfm?page=klass_mark/klasshem_eng.htm.

EPA-HEN: US EPA, Air Risk Information Support Center. *Health Effects Notebook for Hazardous Air Pollutants.* http://www.epa.gov/ttnatw01/hlthef/hapindex.html.

EPA-TRI: US EPA. Addition of Certain Chemicals; Toxic Chemical Release Reporting; Community Right to Know. Proposed and Final Rules. 59 *Federal Register* 1788 (Jan 12, 1994); 59 *Federal Register* 61432 (November 30, 1994). Summarized in *Hazard Information on Toxic Chemicals Added to EPCRA Section 313 Under Chemical Expansion.* http://www.epa.gov/tri/chemical/hazard_cx.htm.

HARV: Harvell, J., M. Bason and H. Maibach. "Contact Urticaria and Its Mechanisms." *Food Chemistry and Toxicology* 32(2): 103-112. 1994. (Table 2: Substances identified as capable of causing contact urticaria.)

HAZMAP: A Relational Database of Hazardous Chemicals and Occupational Diseases. Browse Haz-Map by Adverse Effects: Dermatotoxin. http://hazmap.nlm.nih.gov/hazmapadv.html.

KLAA: Klaassen, C., M. Amdur and J. Doull (eds.). *Casarett and Doull's Toxicology. The Basic Science of Poisons*, 5th Edition. Pergamon Press, NY. 1996. (Table 18-2: Selected Chemicals Causing Skin Burns; Table 18-3: Common Contact Allergens; Table 18- 6: Selected Phototoxic Agents; Table 18-8: Causes of Chloracne.)

LADO: LaDou, J. (ed.). *Occupational Medicine*. Norwalk, CT: Appleton & Lange, 1990. (Table 17-1: Occupational phototoxic dermatitis: Causes and workers affected: Table 17-4: Common causes of occupational allergic contact dermatitis and typical occupational groups affected: Table 17-5: Established causes of occupational vitiligo: Table 17-8: Occupational cutaneous carcinogens and occupations with significant exposure.)

LOCK: Lock, E.A., and E. Harpur. "Toxicology of Sensory System: A Perspective." *Human and Experimental Toxicology*. 11(6): 442-448. 1992. (Table 1: Chemicals that induce olfactory lesions in experimental animals by either inhalation and/or non-inhalation routes.)

LU: Lu, F.C. *Basic Toxicology*, 2nd Edition. 1991. (Appendix 15-1: Cataractogenic Chemicals.)

NJ-FS: New Jersey Department of Health Services. Right to Know Program, NJDOH, Trenton, NJ. http://www.state.nj.us/health/eoh/rtkweb/rtkhsfs.htm.

NTP-HS: National Toxicology Program. *Chemical Repository of Health and Safety Data.* http://ntp-db.niehs.nih.gov/NTP_Reports/NTP_Chem_H&S/NTP_Chem8/Radian[Insert CAS#].txt.

OEHHA-AREL: California EPA, Office of Environmental Health Hazard Assessment. *Acute Reference Exposure Levels (RELs), Averaging Times, and Toxicologic Endpoints.* Includes all Acute Reference Exposure Levels (ARELs) developed by OEHHA through May 2000 http://www.oehha.org/air/acute_rels/allAcRELs.html.

OEHHA-CREL: California EPA, Office of Environmental Health Hazard Assessment. *Air Toxics Hot Spots Program Risk Assessment Guidelines, Part III: Technical Support Document "Determination of Noncancer Chronic Reference Exposure Levels."* Includes all Chronic Reference Exposure Levels (CRELs) adopted by OEHHA as of September 2002 (http://www.oehha.ca.gov/air/chronic_rels/AllChrels.html), plus draft CRELS proposed through September 2002 (http://www.oehha.ca.gov/air/chronic_rels/index.html).

RTECS: National Institute for Occupational Safety and Health's Registry of Toxic Effects of Chemical Substances. See Environmental Defense's Suspect Hazard Identification documentation.

TIMB: Timbrell, J.A. *Introduction to Toxicology.* New York: Taylor and Francis, 1995. (Table 5.1: Types of skin sensitizers.)

Appendix 2

Summary of National Indicator Initiatives: CSTE Environmental Public Health Indicators and US EPA State of the Environment Report.

Defining Environmental Public Health Indicators

The Council for State and Territorial Epidemiologists (CSTE) identifies four types of environmental public health indicators related to hazard, exposure and outcome tracking:

- ***Hazard indicators***—"a condition or activity that identifies the potential for exposure to a contaminant or hazardous condition."
- ***Exposure indicators***—"a biologic marker in tissue or fluid that identifies the presence of a substance or combination of substances that may potentially harm the individual."
- H***ealth effect indicator***—"a disease or condition that identifies the occurrence of an adverse effect from exposure to a known or suspected environmental hazard."
- ***Intervention indicator***—"Implementation of a program or official policy that minimizes or prevents an environmental hazard, exposure, or health effect."

In addition to defining each type of environmental public health indicator, CSTE identifies several categories of core indicators ranging from toxics, waste, and pesticides to disasters including ambient water and drinking water[1,2].

Categories of Core Indicators:

Air, Ambient (Outdoor)	Noise	Toxics and Waste
Air, Indoor	Pesticides	Water, Ambient
Disasters	Sentinel Events	Water, Drinking
Lead (PB)	Sun and Ultraviolet Light	

[1] CSTE brochure. "Environmental Public Health Indicators: A guide for monitoring the occurrence of environmentally-related exposure and diseases." http://www.cste.organization.

[2] Centers for Disease Control National Center for Environmental Health Assessment Division of Environmental Hazards and Health Effects. *Environmental Public Health Indicator*. Atlanta, Georgia. October, 2002.

CSTE Environmental Public Health Indicators by Type: Hazard, Exposure, Health Effect, and Intervention

Hazard Indicators	*Exposure Indicator*	*Health Effect Indicators*	*Intervention Indicators*
▪ Monitored contaminants in shellfish and sport commercial fish	▪ Blood lead levels in children	▪ Deaths attributed to extremes in ambient temperature	▪ Programs that address motor vehicle emissions
▪ Point source discharges into ambient water		▪ Lead poisoning in children	▪ Alternate fuel use in registered motor vehicles
▪ Monitored contaminants in ambient and drinking water		▪ Heating loss in adults who have no occupational exposure to noise	▪ Availability of mass transit
▪ Criteria air pollutants in ambient air		▪ Pesticide-related poisoning and illness	▪ Policies that address indoor air hazards in schools
▪ Hazardous substances or toxics released in the ambient air		▪ Individual cases of illnesses or conditions such as asthma death, food-borne outbreaks (including seafood consumption) and outbreaks attributed to ambient or drinking water contaminants	▪ Laws pertaining to smoke-free indoor air
▪ Residence in non-attainment areas (for criteria air pollutants)			▪ Complaint-related indoor air inspections
▪ Tobacco smoke in homes with children		▪ Carbon monoxide poisoning	▪ Emergency preparedness, response, and mitigation training programs, plans, and protocols
▪ Chemical spills		▪ Melanoma	▪ Annual multi-institutional exercises for disaster response
▪ Motor vehicle emissions		▪ Possible child poisoning resulting in consultation or emergency department visit	▪ Compliance with pesticide workers training standards
▪ Residence in flood plains			▪ Health-based activity restriction in ambient water
▪ Pesticide use and patterns			▪ Implementation of sanitary surveys
▪ Residual pesticide or toxic contaminants in foods			▪ Compliance with operation and maintenance standards for drinking water systems
▪ Ultraviolet radiation			▪ Boil-water advisories

EPHI Indicators can be used for:

1) **Surveillance of health status or trends in order to:**

- Prevent and/or detect existing or new, known or suspected adverse public health events associated with environmental exposures
- Provide efficient and consistent reporting mechanisms

2) **Program planning activities such as:**

- Tracking program goals and objectives
- Supporting existing programs
- Guiding research initiatives
- Developing new program initiatives

3) **Building core environmental public health capacity with other agencies**

CTE Five-Step Approach for Selecting Environmental Public Health Indicators

1) **Classify the link between the proposed indicator and the public health issues. Preference should be given to direct measures.**

2) **Determine the Public Health impact of using the proposed indicator.**

3) **Evaluate the feasibility of using the proposed indicator. Data exist on the local and state levels for the monitoring of trends.**

4) **Assess the ability to incorporate the proposed indicator into the public health interventions and environmental regulations.**

5) **Consider the following criteria for determining priority**
   - Scientific basis
   - Public health importance
   - Public concern (fish, bacteria, pfisteria)
   - Public health capacity (feasibility)
   - Degree to which exposure can be avoided

All materials adapted from: Centers for Disease Control National Center for Environmental Health Assessment Division of Environmental Hazards and Health Effects. *Environmental Public Health Indicators.* Atlanta, Georgia. October, 2002.

Adapted from CSTE- http://www.cste.org/pdffiles/Environmentalpublichealthindicators.pdf.

Additional information available from the National Centers for Disease Control National Center for Environmental Health at: *http://www.cdc.gov/nceh/tracking/.*

EPA's Report on the Environment

The State of the Environment Report will be using available national-level data and indicators to describe environmental conditions and human health concerns. A draft report was released in the spring of 2003 for public review and comment. It uses existing data and indicators to describe current national environmental conditions and trends. It also identifies data gaps and research needs and discusses the challenges remaining to overcome these gaps. Technical support information on indicator development is also provided.

The five "theme areas" to covered in the report include; human health, ecological condition, clean air, pure water, and better protected land use. Human health examines trends in human exposure to environmental pollutants, and environmentally related diseases. Ecological condition looks at living and natural resources, current pressures and stressors on these resources and future sustainability. Clean air examines the impact of indoor air quality on human health and ecosystems. Pure water examines aspects of drinking water, recreational water use, the condition of the nation's water resources and the living resources sustained by them. Better protected land use assesses the land use and activities that affect the condition of the American landscape, including agricultural practices, pesticide programs, waste management, emergency response and preparedness and recycling.

Adapted from: http://www.epa.gov/indicate/. Last accessed 01/20/03.

V. Conclusions and Recommendations

This investigation and pilot analysis has shown that public health indicators are a critical tool for tracking threats to ecology and health. The pilot case examples highlight major warning signs for public health in relation to the vulnerability of our drinking water sources, microbial risks in surface waters and the presence of persistent toxic pollutants in Maryland waters of the Chesapeake Bay watershed.

Drinking Water

Analysis of TTHMs in public drinking water supplies found that all major Maryland drinking water supplies are in compliance with the federal Safe Drinking Water Act requirement for TTHMs. However, virtually all surface water supplies have periodic excursions above the recommended health-based standard especially during the late summer months and early fall. Over 3.5 million Marylanders consume drinking water from these supplies. Consequently, TTHMs indicate both the vulnerability of surface drinking water sources within the Chesapeake Bay watershed to organic pollution from both natural and anthropogenic sources and the potential for broad population exposure to chemical contaminants at levels above recommended public health guidance levels.

Microbial Risks

Measurements of fecal coliform in surface waters at recreational beaches and within urban waterways suggest that populations living in and around the watershed are vulnerable to toxic pathogen exposure. At various times, levels of monitored fecal coliform (or equivalent pathogenic organisms) are orders of magnitude above health-based standards. The vulnerability of urban and recreational waterways to microbial pollution within the watershed is due to non-point source runoff, as well as to an aging waste water infrastructure, water treatment plant malfunctions, and overflows. Bioindicators like fecal coliform levels denote the need for improvements in overall sanitary infrastructure and reductions in non-point source pollution throughout the Chesapeake Bay region. In addition, the potential for population exposure either through contact and ingestion of contaminated fish or through recreational dermal contact and ingestion of surface water indicates that public health is threatened by the presence of microbial pathogens in the watershed.

Toxic Pollutants

Concentrations of mercury and PCB contaminants in fish tissue indicate that past, present and future releases of persistent toxic pollutants have long term public health implications. Toxic pollutants impact every waterway in the state of Maryland. Actual levels of population consumption of impacted fish in the state are unknown; however, hazard indices reveal that contamination levels of PCBs and mercury may put some high fish consumers at risk of adverse health effects, including children and women of childbearing age. Major health hazards that have been associated with the USEPA Cheasapeake Bay Program "chemicals of concern" for the Bay include cancer and adverse developmental and reproductive health outcomes. The findings for these persistent pollutants underscore the need to work for better long-term pollution control to maintain both ecological integrity and public health within the region.

It must be emphasized that no immediate threats to public health were revealed through the case examples. At the same time, these indicators underscore the need for continued diligence in protecting and preserving the watershed and recognizing the linkages between

human health and the environment. This has been a pilot investigation and these indicators offer only a small sample of potential public health indicators for the region. In the future, measures such as these may be part of a national tracking network of indicators to measure environmental progress, identify emerging hazards, shape research, and strengthen the scientific basis for environmental and health policies.

These case examples of potential environmental public health indicators in the Chesapeake Bay region reveal that tracking public health threats over time is feasible and practical. Within the state of Maryland, there are a number of well-conducted ongoing monitoring efforts that can serve as the foundation for the development of environmental public health indicators. Gaps in these programs highlight several areas for future expansion. First, there is a need to expand health outcome monitoring in the region; data sets were chosen primarily from hazard databases because comparable health outcome data sets were not available. Second, there is a need to improve data accessibility; a fragmented system of data collection and overlaps in water quality monitoring systems within the state make finding appropriate data sets difficult. Third, there is a need for improved mapping and communication of findings within state, local, and federal agencies, as well as with the public. Better communication of findings will help in developing more targeted and effective prevention and intervention programs. Finally, there is a need for better integration and use of health and environmental monitoring data on the state level. Better integration of these resources will improve the environmental and public health applications of indicators and provide new insights into the link between the ecological environment and human health.

In conclusion, the time is right to begin developing Environmental Public Health Indicators (EPHI) for the Chesapeake Bay region. A national momentum for environmental public health tracking is building and it may become the cornerstone of future of environmental health practices. This pilot investigation serves as the first step in developing a public health report card for the Chesapeake Bay. It is hoped that lessons learned from this project will serve as the basis for future expanded efforts on the state and national level. Based on these findings the following recommendations are presented as next steps to build upon this pilot investigation:

1) Assess information needs to assure effective coverage of the watershed and to meet the data needs of state and county health agencies and environmental officials.

Environmental and health officials should identify additional health and environmental data that is needed to address and prevent emerging public health risks related to environmental quality.

2) Expand the list of indicators to include a broader range of contaminants, additional exposure pathways, and improved measures of population exposure levels.

Indicators could be expanded to include other drinking water contaminants, private well sampling, additional indicators for pathogens, and more direct measurement of actual population exposure levels.