PART III

MULTISOURCE MULTIPATHWAY RISK ASSESSMENT

Chapter 9 Overview of Multisource Multipathway Risk Assessment

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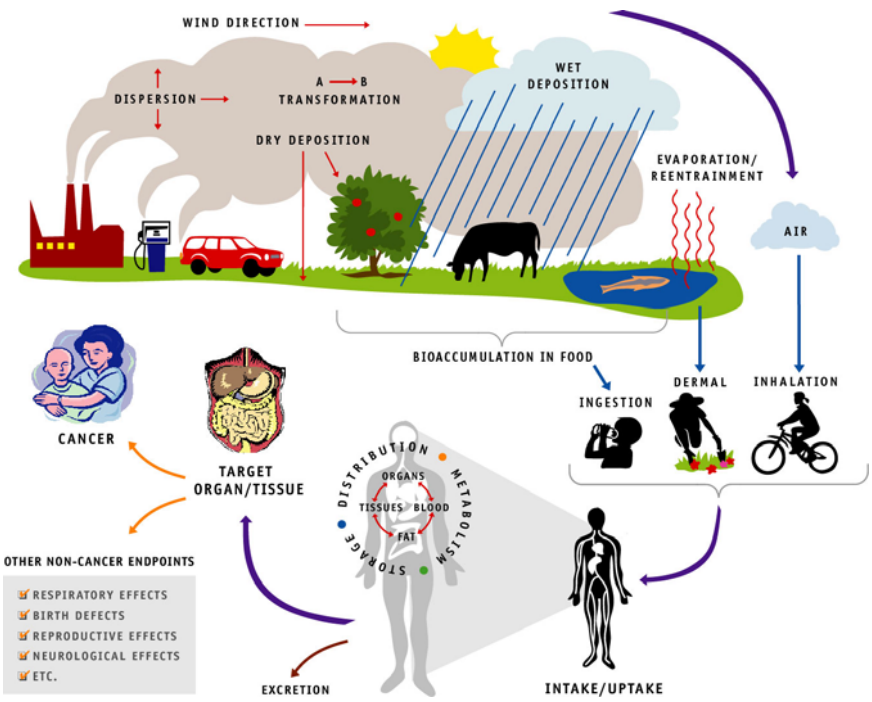
9.0 Introduction

Part II of this Reference Manual discussed how to plan for and conduct a multisource cumulative human health risk assessment via the direct inhalation pathway. Part III provides the same general discussion of the various aspects of the risk assessment process; however, the discussion is focused specifically on multisource *multipathway* (sometimes also called *multimedia*) risk assessment for both humans and ecosystems. Such multipathway risk assessments may be appropriate when air toxics that persist and which may also bioaccumulate or biomagnify are present in releases to ambient air (the HAPs which have these properties are described in the next section). When performed, a multipathway risk assessment will usually involve an assessment of the deposition of air toxics onto soil, plants, and water, any subsequent uptake of the chemicals by biota, and potential exposures by organisms via contact with the contaminated soils, waters, and foods. This is illustrated generically, for a human health multipathway analysis, by the following figure.

Overview of Multisource Multipathway Exposure Pathways



(Note that for human health risk assessments, a multipathway analysis will usually include the inhalation pathway plus any additional relevant exposure pathways. A lack of available data on inhalation exposure pathways for ecological receptors may preclude inclusion of this pathway in ecological multipathway risk assessments.)

The remainder of this chapter presents an overview of multisource multipathway risk assessment for both human and ecological receptors. Detailed information on the various elements of multipathway assessment is provided in ATRA Volume 1, Part III (for human health assessment) and Part IV (for ecological risk assessment).

9.1 Toxic Chemicals That Persist and Which May Also Bioaccumulate

Some toxic compounds have the ability to persist in the environment for long periods of time and may also have the ability to build up in the food chain to levels that are harmful to human health and the environment. More specifically, a chemical that is a persistent bioaccumulator is a substance that partitions to water, sediment, or soil and is not removed at rates adequate to prevent its bioaccumulation in aquatic or terrestrial species.

For example, releases of metals from a source may deposit out of the air onto the ground where they remain in surface soils for long periods of time. Children playing in the area may ingest this contaminated soil through hand-to-mouth behaviors. The chemicals in the soil may also be taken up into plants through the roots and accumulate in foraging animals.

The PBT Chemical Program

PBT pollutants are chemicals that are toxic, persist in the environment and bioaccumulate in food chains and, thus, pose risks to human health and ecosystems. The biggest concerns about PBTs are that they transfer rather easily among air, water, and land, and span boundaries of programs, geography, and generations.

The EPA is forging a new approach to reduce risks from and exposures to priority PBT chemicals through increased coordination among EPA national and regional programs. This PBT chemicals program has been established to overcome the remaining challenges in addressing priority PBT pollutants. EPA is committing, through this program, to create an enduring cross-office system that will address the cross-media issues associated with priority PBT pollutants.

For more information on EPA's PBT Chemical Program, see http://www.epa.gov/pbt/.

EPA's challenge in reducing risks from this category of toxic air pollutants stems from this ability to transfer from air, to sediments, water, land, and food; to linger for long periods of time in the environment; and for some substances, their ability to travel long distances. Many of these chemicals (e.g., DDT) have been banned for use in the U.S. As such, there should be no active air emissions of these chemicals (although releases into the air are still possible, e.g., by resuspension of previously contaminated soil). However some chemicals, such as mercury, are still in use today. A number of lists of these persistent and bioaccumulative chemicals have been developed through international and internal EPA efforts (see Exhibit 9-1). Notably, a number of the HAPs appear on one or more of these lists.

Exposure to persistent and bioaccumulative air toxics through a pathway other than inhalation of contaminated air is termed **an indirect exposure pathway** because contact with the chemical occurs in a medium that is not the original medium to which the chemical was released (i.e., air). In contrast, a **direct exposure pathway** is one in which contact occurs with the chemical in the medium to which it was originally released. When exposure of a organism (e.g., a person, plant, or animal) to a chemical (or chemicals) occurs through more than one pathway, a **multipathway analysis** may be considered. When the releases in question come from multiple sources, the analysis is termed a **multisource multipathway** analysis.

As noted above, in air toxics risk assessment, the inhalation pathway is commonly assessed for human receptors and less frequently for ecological receptors. However, indirect exposure pathways are usually assessed for both humans and ecological receptors for a limited set of chemicals released to the air. EPA has identified a preliminary set of HAPs for which indirect exposure pathway analyses should generally be conducted for situations involving significant

emissions of these chemicals in a study area. This list of chemicals is termed **Persistent Bioaccumulative HAP Compounds (PB-HAP Compounds**; see Exhibit 9-2); however, all of the PB-HAP compounds occur on one or more of EPA's existing lists of PBT chemicals. The designation "PB-HAP" was developed to distinguish this list from the existing lists of PBT chemicals (Exhibit 9-1) and specifically to clarify that chemicals on this new list are:

- HAPs;
- Relatively persistent in the environment; and
- For some chemicals, have a strong propensity to bioaccumulate or biomagnify.

This preliminary list of PB-HAPs was derived primarily on the basis of human health concerns. It does not consider direct contact by plants or inhalation by animals. Additional HAPs may be identified as EPA gains more familiarity with ecological risk assessments for air toxics. Study area-specific circumstances may indicate a need to consider other air toxics posing potential risk via non-inhalation pathways. ATRA Volume 1, Appendix D, more fully describes the process by which EPA identified the list of PB-HAPs.

Some Key Terms for Multipathway Analysis

Persistence refers to the length of time a compound stays in the environment, once introduced. A compound may persist for less than a second or indefinitely.

Bioconcentration is the net accumulation of a substance by an organism as a result of uptake directly from an environmental medium (e.g., net accumulation by an aquatic organism as a result of uptake directly from ambient water, through gill membranes or other external body surfaces).

Bioaccumulation is the net accumulation (storage in tissue and/or organs) of a substance by an organism as a result of uptake from all environmental sources – the medium in which they live, the water they drink, and the diet they consume – over a period of time.

Biomagnification or Biological Magnification is the process whereby certain substances, such as pesticides or heavy metals, transfer up the food chain and increase in concentration. For example, a biomagnifying chemical deposited in rivers or lakes absorbs into algae, which are ingested by aquatic organisms, such as small fish, which are in turn eaten by larger fish, fish-eating birds, terrestrial wildlife, or humans. The chemical tends to accumulates to higher concentration levels with each successive food chain level. Depending on the circumstances, chemicals that biomagnify often are the primary contributors to risk among all the PBT chemicals impacting a study area. Biomagnification is illustrated in ATRA Volume 1, Chapter 23.

Exhibit 9-1. "Lists" of Toxic Chemicals that Persist and Which Also May Bioaccumulate
LRTAP chemicals – The United States signed protocols on Persistent Organic Pollutants (POPs) and heavy metals pursuant to the Convention on Long-Range Transboundary Air Pollution (LRTAP) in June 1998 at a ministerial meeting in Aarhus, Denmark. Sixteen POPs and three metals are regulated (http://www.epa.gov/oppfead1/international/lrtap2pg.htm): 1. aldrin 2. cadmium 3. chlordane 4. dieldrin 5. endrin 6. hexabromobiphenyl 7. kepone (chlordecone) 8. mirex 9. toxaphene 10. hexachlorobenzene 11. heptachlor 12. lead 13. mercury 14. polychlorinated biphenyls (PCBs) 15. dichlorodiphenyltrichloroethane (DDT) 16. lindanedioxins (polychlorinated dibenzo-p-dioxins) 17. furans (polychlorinated dibenzofurans) 18. hexachlorobenzene 19. polycyclic aromatic hydrocarbons
PBT Chemicals – EPA has identified the following priority persistent, bioaccumulative, and toxic (PBT) chemicals and has developed the PBT program to address the cross-media issues associated with these chemicals (http://www.epa.gov/pbt/): 1. aldrin/dieldrin 2. mercury and its compounds 3. benzo(a)pyrene 4. mirex 5. chlordane 6. octachlorostyrene 7. DDT 8. dichlorodiphenyldichloroethane (DDD) 9. dichlorodiphenyldichloroethylene (DDE) 10. PCBs 11. hexachlorobenzene 12. dioxins and furans 13. alkyl-lead 14. toxaphene
Great Lakes Priority Substances. In keeping with the obligations of the Great Lakes Water Quality Agreement, Canada and the United States on April 7, 1997, signed the "Great Lakes Binational Toxics Strategy: Canada-United States Strategy for the Virtual Elimination of Persistent Toxic Substances in the Great Lakes" (http://www.epa.gov/glnpo/p2/bns.html). This Strategy seeks percentage reductions in targeted persistent toxic substances so as to protect and ensure the health and integrity of the Great Lakes ecosystem. The list of "Level 1" substances is identical to EPA's priority PBT pollutants.
Great Waters Pollutants of Concern. The 1990 Clean Air Act Amendments established research and reporting requirements related to the deposition of hazardous air pollutants to the Great Lakes, Lake Champlain, Chesapeake Bay, and certain other "Great Waters." The Program has identified the following pollutants of concern (http://www.epa.gov/airprogm/oar/oaqps/gr8water/index.html): 1. cadmium and cadmium compounds 2. chlordane 3. DDT/DDE 4. dieldrin 5. hexachlorobenzene 6. α-hexachlorocyclohexane 7. lindane (γ-hexachlorocyclohexane) 8. lead and lead compounds 9. mercury and mercury compounds 10. PCBs 11. polycyclic organic matter 12. tetrachlorodibenzo-p-dioxin (dioxins) 13. tetrachlorodibenzofuran (furans) 14. toxaphene 15. nitrogen compounds

Exhibit 9-1 (continued)

TRI PBT chemicals. EPA has published two final rules that lowered the Toxics Release Inventory (TRI) reporting thresholds for certain persistent bioaccumulative and toxic (PBT) chemicals and added certain other PBT chemicals to the TRI list of toxic chemicals (http://www.epa.gov/tri/lawsandregs/pbt/pbtrule.htm). The following PBT chemicals are subject to reporting at lowered thresholds:

1. dioxin and dioxin-like compounds
2. lead compounds
3. mercury compounds
4. polycyclic aromatic compounds
5. aldrin
6. benzo(g,h,i)perylene
7. chlordane
8. heptachlor
9. hexachlorobenzene
10. isodrin
11. lead
12. mercury
13. methoxychlor
14. octachlorostyrene
15. pendimethalin
16. pentachlorobenzene
17. PCBs
18. tetrabromobisphenol A
19. toxaphene
20. trifluralin

Waste Minimization Priority Chemicals. EPA's National Waste Minimization Partnership Program focuses on reducing or eliminating the generation of hazardous waste containing any of 30 Waste Minimization Priority Chemicals (WMPCs). This list replaces the list of 53 chemicals EPA identified in 1998 (*Notice of Availability: Draft RCRA Waste Minimization Persistent, Bioaccumulative and Toxic (PBT) Chemical List*, Federal Register 63(216): 60332-60343, November 9, 1998). Twenty six of the chemicals in the current list were also in the draft list published in 1998. The remaining four chemicals on the current list were added in response to comments and new information EPA received from the public regarding the Agency's methodology for selecting the 53 chemicals in the draft list (http://www.epa.gov/epaoswer/hazwaste/minimize/chemlist.htm).

1. 1,2,4-trichlorobenzene
2. 1,2,4,5-tetrachlorobenzene
3. 2,4,5-trichlorophenol
4. 4-bromophenyl phenyl ether
5. acenaphthene
6. acenaphthylene
7. anthracene
8. benzo(g,h,i)perylene
9. dibenzofuran
10. dioxins/furans
11. endosulfan, alpha and endosulfan, beta
12. fluorene
13. heptachlor and heptachlor epoxide
14. hexachlorobenzene
15. hexachlorobutadiene
16. hexachlorocyclohexane, gamma-
17. hexachloroethane
18. methoxychlor
19. naphthalene
20. PAH group (as defined in TRI)
21. pendimethalin
22. pentachlorobenzene
23. pentachloronitrobenzene
24. pentachlorophenol
25. phenanthrene
26. pyrene
27. trifluralin
28. cadmium and cadmium compounds
29. lead and lead compounds
30. mercury and mercury compounds

Exhibit 9-2. PB-HAP Compounds			
PB-HAP Compound	**Pollution Prevention Priority PBTs**	**Great Waters Pollutants of Concern**	**TRI PBT Chemicals**
Cadmium compounds		X	
Chlordane	X	X	X
Chlorinated dibenzodioxins and furans	X[(a)]	X	X[(b)]
DDE	X	X	
Heptachlor			X
Hexachlorobenzene	X	X	X
Hexachlorocyclohexane (all isomers)		X	
Lead compounds	X[(c)]	X	X
Mercury compounds	X	X	X
Methoxychlor			X
Polychlorinated biphenyls	X	X	X
Polycyclic organic matter	X[(d)]	X	X[(e)]
Toxaphene	X	X	X
Trifluralin			X

[(a)] "Dioxins and furans" ("" denotes the phraseology of the source list)
[(b)] "Dioxin and dioxin-like compounds"
[(c)] Alkyl lead
[(d)] Benzo[a]pyrene
[(e)] "Polycyclic aromatic compounds" and benzo[g,h,i]perylene

9.2 Overview of Multisource Multipathway Human Health Air Toxics Risk Assessment

The multisource multipathway risk assessment for human health is organized in the same way as the multisource cumulative inhalation risk assessment (describe above in Part II) into three general phases:

1. Planning, scoping, and problem formulation;
2. Analysis, consisting of exposure assessment and toxicity assessment; and
3. Risk characterization.

The following sections provide an overview of these various steps in the assessment. Details of the various elements are provided in ATRA Volume 1, Part III.

9.2.1 Planning, Scoping, and Problem Formulation

The planning, scoping, and problem formulation phase of multisource multipathway risk assessment focuses on developing a common understanding of what needs to be included in the risk assessment (beyond the direct inhalation assessment) to assess risks associated with pathways involving deposition (i.e., transfer of the compounds to soil, water, sediment, and biota), bioaccumulation, and biomagnification, and subsequent exposure. Some of the more common exposure pathways considered for a multisource multipathway analysis are illustrated in Exhibit 9-3.

Exhibit 9-3. Human Exposure Pathways Considered for Multisource Multipathway Air Toxics Assessments

Incidental Ingestion Pathways:

air → soil → human
air → surface water → human

Food Chain Pathways:

air → water → human
air → soil → water → human
air → vegetation → human
air → animal → human
air → vegetation → animal → human
air → soil → vegetation → human
air → soil → animal → human
air → soil → water → animal → human
air → soil → water → fish → human
air → soil → water → sediment → fish → human
air → water → animal → human
air → water → fish → human
air → water → sediment → fish → human

In particular, it is noted that the scope of the multisource multipathway risk assessment generally will be more extensive than that for inhalation assessment, and therefore significant additional effort will be likely. EPA is currently working to develop methodologies to support the efficient analysis of multisource multipathway risk assessments and analysts are referred to EPA's Fate, Exposure, and Risk Analysis (FERA) website (http://www.epa.gov/ttn/fera/) and Regional Air Impact Modeling Initiative (RAIMI) website (http://www.epa.gov/Arkansas/6pd/rcra_c/raimi/raimi.htm) for new information in this evolving area.

Keep in mind that, in reality, the planning, scoping, and problem formulation phase for the multisource multipathway assessment would be integrated with that of the inhalation analysis as early as feasible.

It may be necessary to include on the **planning and scoping team** experts in multimedia modeling, bioaccumulation, human exposure factors, and ingestion toxicology. The focus on additional exposure pathways may influence many aspects of the risk assessment, including the size of the study area; emission sources to be considered; the temporal and spatial resolution required; the appropriate level of detail and documentation; trade-offs between depth and breadth in the analysis; QA/QC requirements; analytical approaches to be used; and the staff and monetary resources to commit. The **study-specific conceptual model** would also reflect the specific concerns of air toxics that persist and which also may bioaccumulate. As with the inhalation risk assessment, the planning, scoping, and problem formulation process is an iterative process that reflects changing information and concerns as the multimedia risk assessment unfolds.

9.2.2 Analysis

The analysis phase of the multisource multipathway assessment is divided into two components: exposure assessment and toxicity assessment. **Exposure assessment** for a multisource multipathway analysis is likely to be considerably more complicated than the corresponding inhalation exposure assessment for several reasons:

- People can be exposed to air toxics in many more ways, including in the food they eat, the milk they drink, and the soils on which they play.

- Time is a critical variable. Air toxics that persist and which also may bioaccumulate can slowly build up in soils, sediments, and biota over time. With sufficient time, even relatively small releases have the potential to result in high exposures.

- The spatial distribution of the air toxics can be complex. Chemicals can move away from deposition points due to runoff, erosion, and the movement of contaminated animals. Chemicals deposited over a wide area (e.g., a watershed) can concentrate in smaller areas (e.g., a pond).

- Multimedia models often rely on more extensive list of input variables.

- Sampling and analysis may involve a wider range of media (e.g., soil, sediment) and different types of biota (e.g., fish, shellfish, plants). Each type of sampling and analysis has its own methods, protocols, and QA/QC procedures.

- Whereas the exposure concentration in air is the quantitative metric of exposure for inhalation, intake (usually on a per kg-day basis) is the quantitative metric of ingestion exposure in multipathway analyses. To quantify intake, it is necessary to (1) estimate the concentrations of chemicals of potential concern (COPC) in water, soil, sediment, and/or food items; (2) determine how much water, soil, sediment, and food are ingested; (3) determine the duration and temporal patterns over which ingestion occurs; and (4) adjust for

body weight, to account for the different types of people in the population who interact with the contaminated media. Multipathway exposure assessment uses a number of different **exposure factors** that provide quantitative estimates of the physical and behavioral attributes of potentially exposed populations (e.g., how much fish a person eats per day). Exposure factors can be treated as either constants or variables in the exposure assessment, depending on whether a deterministic or probabilistic analysis is being performed.

The multipathway **toxicity assessment** is similar to the toxicity assessment for inhalation. It considers the same general information: (1) the types of potential adverse health effects associated with chemical exposures; (2) dose-response relationships; and (3) related uncertainties such as the weight of evidence for carcinogenic effects. There are two primary differences:

- A chemical's toxicity is influenced by the route of exposure. That is, the same chemical can result in different toxic effects (and have different dose-response values) depending on whether the chemical is inhaled or ingested. There are a number of reasons why this may occur. For example, when a chemical is inhaled into the respiratory tract, the primary toxic effect may occur in the respiratory tract as a result of inhaling the chemical (a portal of entry effect) and/or the chemical may be absorbed into the bloodstream and subsequently circulated throughout the body (including eventually making its way to the liver). When swallowed, on the other hand, chemicals can also cause a portal of entry effect and/or be absorbed into the bloodstream through the gastrointestinal tract where they are carried directly to the liver. Chemicals in the liver are often metabolized extensively (either to more or less toxic substances) before being transported by the bloodstream to other parts of the body.

- The specific dose-response values used for the ingestion pathway – reference doses (RfDs) for non-cancer (and, in some cases, cancer) effects and oral cancer slope factors (CSFs) – differ in form and derivation from those used for inhalation assessments. Specifically, RfDs and CSFs are developed to match the metric of exposure for ingestion and are expressed (usually) in terms of amount of chemical ingested per unit of body weight per day (i.e., mg/kg-d for RfDs) and risk per amount of chemical ingested per unit body weight per day (i.e., $(mg/kg\text{-}d)^{-1}$ for CSFs).

9.2.3 Risk Characterization

The risk characterization for multipathway assessments also may be more complicated than that for the inhalation risk assessment.

- Ingestion risk estimates are first added across all ingestion pathways and then added to inhalation risk estimates. Where this addition is done across multiple chemicals, the sum is referred to as a cumulative risk estimate. Although the summation process is relatively simple for screening-level analyses, it can become complex for more advanced tiers of risk assessment.

- The uncertainty analysis for multisource multipathway risk assessments may be considerably more complex if multiple sources and pathways are important because many more source characteristics, exposure factors and variables will be involved in the quantification of risk.

As noted earlier, many more specific exposure factors can be treated as variables for probabilistic multipathway risk assessments.

- The uncertainty analysis for multisource multipathway analysis is also much more complex due to the larger number of pathways assessed and the larger number of inputs that are needed.

Analysis of Groundwater Pathways

EPA's Office of Solid Waste has considerable experience in modeling and monitoring the movement of contaminants in groundwater. Much of that experience is based on exposure assessments associated with land-based disposal units (i.e., where the source of contamination is in the subsurface). For example, EPA's Center for Exposure Assessment Modeling (CEAM) distributes multimedia models designed to quantify the movement and concentration of contaminants (from land-based releases at hazardous waste sites) traveling through groundwater, surface water, and food chain media (available at http://www.epa.gov/ceampubl/). In these models, releases to the atmosphere from the subsurface may be considered, but transfer from the air through the subsurface is not.

EPA has limited experience with air toxics multipathway analysis that involve situations in which the groundwater may become contaminated from air releases. EPA's *Methodology for Assessing Health Risks Associated With Multiple Pathways of Exposure to Combustor Emissions* (http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=55525) identifies three site-specific conditions that might lead to greater groundwater impacts:

- Deposition rates that are several times greater than the average;
- The existence of more soluble HAPs in emissions; and
- Higher recharge rates such as would occur in areas with very permeable soil and bedrock near the surface.

9.2.4 Tiered Multisource Multipathway Risk Assessments

EPA guidance generally recommends that a tiered approach to risk assessments be taken to identify the key chemicals, sources, and pathways that contribute most to the risk being evaluated.[(1)] A tiered approach can be particularly valuable for multisource multipathway risk assessments because of the potential complexity commonly associated with such analyses. Often, screening-level analyses assume relatively high exposure factors (e.g., all of the fish a person eats comes from a potentially contaminated pond) to determine whether risk associated with a specific pathway appears to be significant enough to warrant more robust analysis. Subsequent tiers of analysis, using more realistic exposure factors and perhaps involving more complex modeling and perhaps sampling and analysis, are generally undertaken only if lower-tier analyses continue to indicate the potential for evaluated risk. As with inhalation risk assessments, an iterative process of evaluation, deliberation, data collection, work planning and communication is used to decide:

- Whether or not the risk assessment, in its current state, is sufficient to support the risk management decision(s); and

- If the assessment is determined to be insufficient, whether or not progression to a higher tier of complexity (or refinement of the current tier) would provide a sufficient benefit to warrant the additional effort.

More information on tiered approaches to risk assessment can be found in ATRA Volume 1, Section 3.3.3, and ATRA Volume 2.

9.3 Multisource Multipathway Ecological Risk Assessment

A Note to the Reader

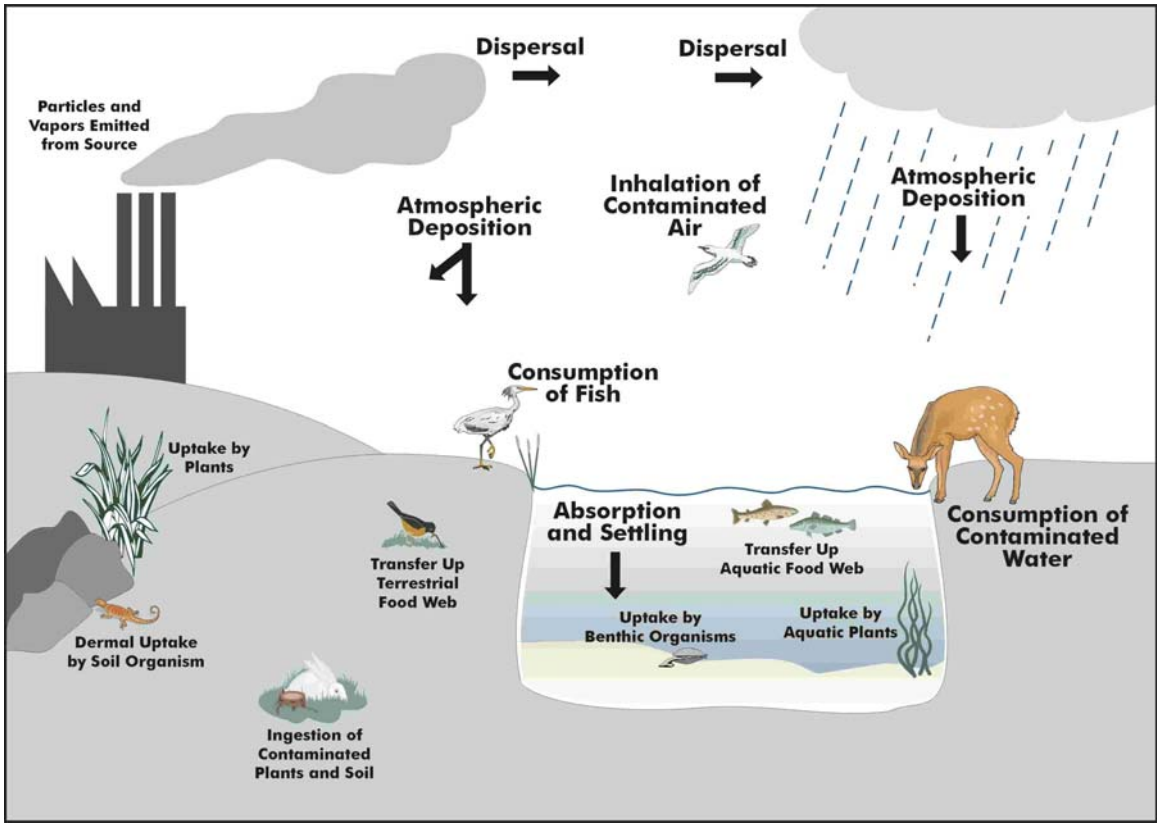
ATRA Volume 1, Part IV (and this Section 9.3), constitutes a snapshot of EPA's current thinking and approach to the adaptation of the evolving methods of ecological risk assessment in the context of Federal and state control of air toxics. While inhalation risk assessment has been increasingly used in regulatory contexts over the last several years, ecological risk assessment tools for air toxics are less well developed and field tested in a regulatory context. The information provided in the ATRA reference library should be considered a living document for review and input. By publishing this information in its current state of development, EPA continues to solicit the involvement of persons with experience in this field to help improve assessment methods for use in a future regulatory context. EPA anticipates ongoing revision to ecological risk assessment methods provided in ATRA.

Section 9.2 discusses how to plan for and conduct a multisource multipathway human health risk assessment when air toxics that persist and which may also bioaccumulate are released to the air. These substances may also pose risks to ecological receptors from exposure to contaminated media or through exposure via aquatic and terrestrial food chains (see Exhibit 9-4).

This section introduces the basic concepts of ecological risk assessment and describes their application to air toxics. The discussion of ecological risk assessment follows the same general framework as that presented for human health in Section 9.2 since the overall concept is the same; however, there are several important differences between the terminology of human health and ecological risk assessment with which readers are encouraged to become familiar (see text box below and ATRA Volume 1, Part IV). In addition, professional expertise will always be required to apply the ecological risk assessment principles and tools identified in this document to specific assessment areas or problems. This document is not a substitute for a working familiarity with ecological principles, their application, and the field of ecological risk assessment.

In addition to the information presented here, readers are also encouraged to become familiar with EPA's *Guidelines for Ecological Risk Assessment* (http://cfpub.epa.gov/ncea/raf/recordisplay.cfm?deid=12460) which provides a more complete understanding of EPA's recommended approach to ecological risk assessment. Interested readers are also referred to *EPA's Ecological Risk and Decision Making Workshop* materials which provide detailed information on the definition of ecological risk assessment, how it relates to human health assessment, the ecosystem protection place-based approach, and the basis for ecological protection and risk assessment at EPA.[2]

Exhibit 9-4. Air Toxics Exposure Pathways of Potential Concern for Ecological Receptors



This graphic illustrates some of the potential multimedia pathways of concern for air toxics exposure to ecological receptors. Air toxics released from a source disperse through the air and eventually fall to the earth (atmospheric deposition) via settling and/or precipitation. Air toxics deposited to soil may be absorbed or ingested by plants and soil invertebrates (uptake). Terrestrial animals may be exposed to air toxics via ingestion of contaminated plants and soil, or by consuming contaminated terrestrial animals (for those air toxics that bioaccumulate and transfer up the terrestrial food web). Air toxics deposited to water may be dissolved in the water column and/or may settle and be absorbed into aquatic sediments. Air toxics in sediments may be absorbed or ingested by benthic organisms (uptake); those in sediments and the water column may be absorbed by aquatic organisms (uptake). Aquatic organisms (e.g., fish) may be exposed directly to air toxics in the water column and/or by consuming contaminated aquatic organisms (for those air toxics that bioaccumulate and transfer up the aquatic food web). Terrestrial animals may be exposed to air toxics by eating contaminated fish or shellfish and/or by drinking contaminated water. Note also that, while in the atmosphere, air toxics may also have direct impacts on plants (direct exposure) and terrestrial animals (inhalation).

9.3.1 Overview of Air Toxics Ecological Risk Assessment

The ecological risk assessment process has three main steps that broadly correspond to the four basic steps in human health risk assessment methodology (Exhibit 9-5; see http://cfpub.epa.gov/ncea/raf/recordisplay.cfm?deid=12460):

- **Problem formulation**, which corresponds to the problem formulation step of the human health risk assessment methodology (planning and scoping activities similar to human health risk assessment are also integrated with this step; however, they are discussed separately below to maintain the operational structure of the ecological risk assessment as described in EPA's ecological risk assessment guidelines);

- **Analysis**, which corresponds to the exposure assessment and toxicity assessment steps of the human health risk assessment methodology; and

- **Risk characterization**, which corresponds to the risk characterization step of the human health risk assessment methodology.

Some Important Differences Between Ecological Risk Assessment and Multipathway Human Health Risk Assessment

- **Planning and scoping**. The ecological risk assessment requires more preliminary analysis and deliberation regarding endpoints to be assessed and toxicity values to be used because ecological systems are more complex and are not as well understood biologically as human health systems. The planning and scoping team should include individuals with specific expertise in ecological risk assessment.

- **Assessment area**. It may be necessary to evaluate additional portions of the assessment area that are not of concern from a human health perspective.

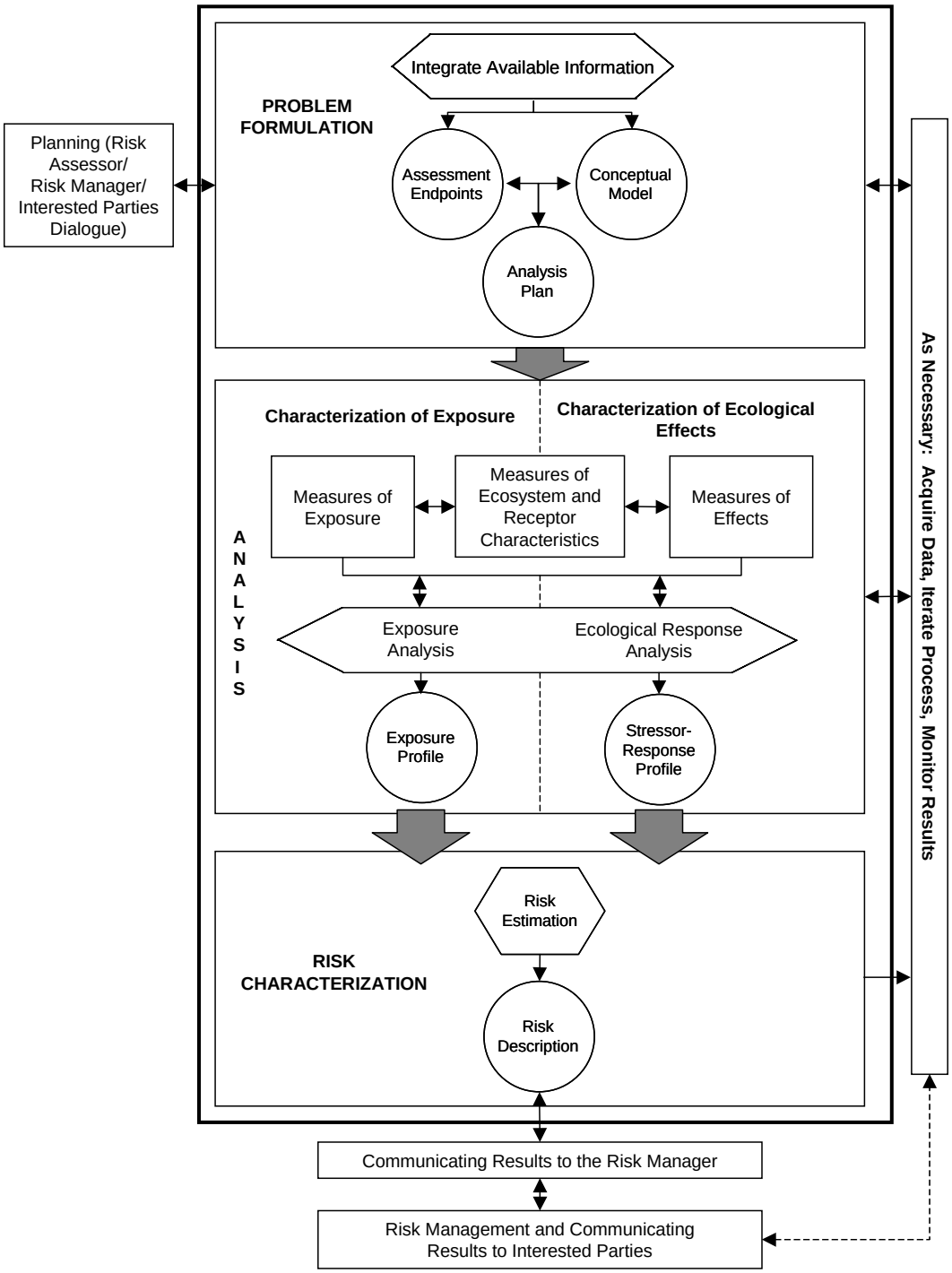
- **Potentially exposed populations**. The focus shifts from potentially exposed individual humans to potentially exposed populations and species of ecological receptors of concern. In many cases, the exposure assessment may need to address multiple species and life-stages, many of which have physiological and biochemical processes that differ significantly from humans. (When threatened or endangered species are present, the assessment may also include an evaluation of those organisms as individuals.)

- **Exposure pathways and exposure routes**. It may be necessary to assess different exposure pathways and routes that are not of concern for human health.

- **Ecological effects assessment**. Ecological systems have traits and properties that are different from humans and, thus, the ecological effects assessment (comparable to toxicity assessment for human health) may consider a wider range of potential causal relationships.

- **Risk characterization**. While risks may be assessed at multiple levels of ecological organization (i.e., organism, population, community, and ecosystem), they generally are assessed at the population level in air toxics assessments. (Nevertheless, when appropriate, consideration should be given to assessments at high levels of ecological organization, such as at the landscape level.)

Exhibit 9-5. Ecological Risk Assessment Framework



From: *Guidelines for Ecological Risk Assessment.* U.S. Environmental Protection Agency, Risk Assessment Forum, Washington, DC, EPA/630/R095/002F, 1998 (http://cfpub.epa.gov/ncea/raf/recordisplay.cfm?deid=12460)

9.3.2 Problem Formulation

Problem formulation provides the foundation for the entire ecological risk assessment. This step includes:

- Identifying risk management goals from an ecological perspective, ecological receptors of concern (e.g., wetlands, fish populations, keystone species that impact the overall ecosystem), and assessment endpoints (explicit expression of the environmental value that is to be protected, operationally defined by an ecological entity and its attributes);

- Developing the ecological risk part of the conceptual model as necessary to account for ecological exposure pathways and receptors; and

- If necessary, developing the sampling and analysis plan and associated quality assurance project plan to collect data on exposures and measures of effects that are needed to support the ecological risk assessment.

As with human health risk assessments, problem formulation is often an iterative process, in which substantial re-evaluation may occur as new information and data become available. Data collection in subsequent iterations often is triggered by identification of major data gaps and uncertainties in the risk characterization that prevent confident decision-making by risk managers.

The problem formulation process for ecological risk assessment for air toxics focuses on developing a common understanding of what needs to be done to assess *ecological* risks associated with pathways involving the transfer of compounds from the air to soil, water, sediment, and biota, and subsequent exposure. While the ecological risk assessment may build on the foundation of the human health multipathway assessment (e.g., using the same emissions data and multimedia models), the problem formulation step is particularly critical for the ecological risk assessment because of the effort needed to understand and identify important ecological receptors, exposure pathways, endpoints, and management goals. The ecological risk assessment is not simply an "add-on" to the human health multipathway risk assessment. The problem formulation effort will need to consider a wide variety of possible ecological receptors that are not similar to humans. For example:

- Different species (and life stages) may have very different responses to the same exposure. Therefore, knowledge of the exposure-response of many species, including those that may be particularly sensitive to the air toxic, is needed.

- There may be many different types of ecosystems present in the assessment area, and sensitivity likely varies among them. Therefore, the particular features of the ecosystem(s) that occur in areas where high exposures are predicted may be particularly important.

Planning and Scoping the Multisource Ecological Risk Assessment

To ensure that the ecological risk assessment will provide information useful to the risk managers who will be making the risk management decisions, EPA's *Guidelines for Ecological Risk Assessment* recommends a planning and scoping dialogue occur between the risk assessors, risk managers, and where appropriate, interested stakeholders at the very start of the risk assessment process. The outcome of the planning and scoping phase is an agreement on the basic goals, scope, and timing of the risk assessment. Important goals of the dialogue are the identification of the risk management goals and risk management options that the risk assessment will be designed to inform. This 'kick-off' dialogue sets the stage for the problem formulation phase, when the plans for the ecological risk assessment are finalized. Ultimately, the planning and scoping process is a helpful tool to get the right people involved and talking so that the risk questions, expectations, and plans in place to make the overall assessment go smoothly and in a scientifically responsible manner.

9.3.3 Analysis

Analysis includes two principal steps – **characterization of exposures** and **characterization of ecological effects** to the **contaminants of potential ecological concern** (COPECs).

9.3.3.1 Characterization of Exposures

During this step, the analysts will characterize the spatial and/or temporal pattern of stressor concentrations in environmental media (including certain body burden levels) and analyze the level of contact or co-occurrence between the stressors and the ecological receptors. This often is done using the multimedia models such as those identified in ATRA Volume 1, Chapter 18; however, different models or approaches may be appropriate.

It should be noted that the ecological exposure characterization is likely to differ significantly from the corresponding multipathway exposure assessment for human health. For example:

- In addition to food chain (ingestion) exposures, many ecological receptors can be exposed to air toxics via direct contact with contaminated soils (e.g., earthworms) or sediments (e.g., sediment-dwelling invertebrates, bottom-feeding fish); direct exposure to surface water (e.g., free-swimming invertebrates and fish); or direct exposure to contaminated air via inhalation (e.g., birds), dermal contact (e.g., amphibians), deposition to plant surfaces, etc.

- Particular geographic areas of concern may differ because ecological receptors may occur in areas rarely used by human populations (e.g., large wetland areas, ponds where people rarely fish).

- Sampling and analysis may involve a wider range of media (e.g., sediment) and different types of biota (e.g., earthworms, aquatic invertebrates). Each type of sampling and analysis has its own methods, protocols, and Quality Assurance/Quality Control (QA/QC) procedures.

- Quantitative metrics of exposure may include both direct and indirect exposures for ecological receptors, including an evaluation of ambient concentrations of COPECs in soil, water, sediment, and or food stuffs. Quantification of exposure via ingestion is similar to

that for human health ingestion analyses, except that different food items may be involved, and the appropriate ecological **exposure factors** (e.g., diet, body weight) will be different. As with human health analyses, many exposure factors can be treated either as constants or as distributions in the exposure assessment. Ecological exposure assessments for ingestion pathways frequently use bioenergetic models to more explicitly relate intake to adverse effects.[3]

9.3.3.2 Characterization of Ecological Effects

Characterization of ecological effects includes identifying the types of effects that different stressors may have on ecological receptors, along with characterizing the **stressor-response relationship** (the relationship between the level of exposure to the stressor and the expected biological or ecological response). A common result is the identification of **ecological toxicity reference values (TRVs)**, which are concentrations of chemicals in environmental media (including biota such as fish tissues) below which no significant ecological effects are anticipated. TRVs are similar, in concept, to RfDs (reference doses) and RfCs (reference concentrations) for human health noncancer evaluations. TRVs may be screening level (i.e., conservative, generic values) or more refined values for use in higher levels of analysis. They may be point values, ranges, or developed using more advanced probabilistic methods (such as Monte Carlo techniques).

There are two primary differences between the characterization of ecological effects for ecological risk assessment and toxicity assessment for human health risk assessment.

- Adverse effects of concern generally focus at the population, community, or ecosystem level. With rare exceptions (e.g., threatened or endangered species), effects to individual organisms are not the primary concern. Note, however, that ecological risk assessments often use estimates of impacts to individual organisms (e.g., mortality, reproductive effects) to infer impacts at higher levels of organization because exposure-response data for populations, communities, or ecosystems often are lacking. Some approaches are available, however, for incorporating population-level analysis in ecological risk assessments.[4]

- A distinction is made between **assessment endpoints**, which are the environmental values to be protected, and **measures of effects**, which are the specific measures used to evaluate risk to the assessment endpoints (assessment endpoints and measures of effects are defined in ATRA Volume 1, Section 23.3.4.2).

9.3.4 Ecological Risk Characterization

Similar to human health risk characterization, ecological risk characterization combines information concerning exposure to chemicals with information regarding effects of chemicals to estimate risks. Human health risk assessments consider health effects in the bodies of individual people. Ecological risk assessments consider various “health” issues that can range from actual health effects in the bodies of individual ecological receptors to something more attuned to the “health” of the ecosystem as measured by species richness and diversity.

An Ecological Risk Assessment Case Study: Ozone Risks To Agroecosystems

The case study summarized here provides an example of how EPA has assessed environmental risks from an air pollutant (ozone) as part of EPA's effort to promulgate National Ambient Air Quality Standards (NAAQS) for criteria air pollutants (see ATRA Volume 1, Chapter 2). Note that this example is for ozone, a criteria air pollutant; however, the concepts presented here are relevant to air toxics risk assessment. In addition, an agroecosystem, such as the system discussed here, is more of a human construct than a natural ecosystem and is provided here only for illustration of general principles. An actual air toxics ecological risk assessment of a natural system would have to consider site-specific characteristics of the system in question.

Problem Formulation. Pursuant to the Clean Air Act (CAA), EPA is required to set NAAQS for "any pollutant which, if present in the air, may reasonably be anticipated to endanger public health or welfare and whose presence in the air results from numerous or diverse mobile and/or stationary sources." EPA develops public health (primary) and welfare (secondary) NAAQS. According to section 302 of the CAA, the term welfare "includes ... effects on soils, water, crops, vegetation, manmade materials, animals, wildlife, weather, visibility, and climate, damage to and deterioration of property, and hazards to transportation, as well as effects on economic values ...." A secondary standard, as defined in section 109(b)(2) of the CAA, must "specify a level of air quality the attainment and maintenance of which in the judgment of the Administrator, based on such criteria, is requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of such air pollutant in the ambient air."

This case study focuses on an assessment endpoint for agricultural crops (e.g., the prevention of an economically adverse reduction in crop yields). Yield loss is defined as an impairment of, or decrease in, the value of the intended use of the plant. This concept includes a decrease in the weight of the marketable plant organ, reduction in aesthetic values, changes in crop quality, and/or occurrence of foliar injury when foliage is the marketable part of the plant. These types of yield loss can be directly measured as changes in crop growth, foliar injury, or productivity, so they also serve as the measures of effect for the assessment.

Exposure Analysis. EPA used ambient ozone monitoring data across the U.S. and a Geographic Information System (GIS) model to project national cumulative, seasonal ozone for the maximum three month period during the summer ozone season. This allowed EPA to project ozone concentrations for some rural parts of the country where no monitoring data were available but where crops were grown, and to estimate the attainment of alternative NAAQS scenarios. The U.S. Department of Agriculture's (USDA's) national crop inventory data were used to identify where ozone-sensitive crop species were being grown and in what quantities. This information allowed the Agency to estimate the extent of exposure of ozone-sensitive species under the different scenarios.

Ecological Effects Analysis. Stressor-response profiles describing the relationship between ozone and growth and productivity for 15 crop species representative of major production crops in the U.S. (e.g., crops that are economically valuable to the U.S., of regional importance, and representative of a number of crop types) had already been developed from field studies conducted from 1980 to 1986 under the National Crop Loss Assessment Network (NCLAN) program. The NCLAN studies also included secondary stressors (e.g., low soil moisture and co-exposure with other pollutants like sulfur dioxide), which helped EPA interpret the environmental effects data for ozone.

Risk Characterization. Under the different NAAQS scenarios, the Agency estimated the increased protection from ozone-related effects on vegetation associated with attainment of the different NAAQS scenarios. Monetized estimates of increased protection associated with several alternative standards for economically important crops were also developed. This analysis focused on ozone effects on vegetation since these public welfare effects are of most concern at ozone concentrations typically occurring in the U.S. By affecting commercial crops and natural vegetation, ozone may also indirectly affect natural ecosystem components such as soils, water, animals, and wildlife.

Source: U.S. Environmental Protection Agency. 1999. *Residual Risk Report to Congress*. Office of Air Quality Planning and Standards, Research Triangle, NC, March 1999. EPA-453/R-99-011.

Other Key Ecological Risk Assessment Resources

- NCEA's Ecological Risk Assessment webpage http://cfpub.epa.gov/ncea/cfm/ecologic.cfm
- The Oak Ridge National Laboratory Ecological Risk Assessment webpage on tools, guidance, and applications http://www.esd.ornl.gov/programs/ecorisk/ecorisk.html
- The Superfund Ecological Risk Assessment Program http://www.epa.gov/oswer/riskassessment/ecorisk/ecorisk.htm
- Navy Guidance for Conducting Ecological Risk Assessments http://web.ead.anl.gov/ecorisk/
- EPA's Watershed Ecological Risk Assessment program http://cfpub.epa.gov/ncea/cfm/weracs.cfm?ActType=default

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1. U.S. Environmental Protection Agency. 2001. *Risk Assessment Guidance for Superfund (RAGS). Volume III - Part A. Process for Conducting Probabilistic Risk Assessment*. Office of Emergency and Remedial Response, Washington, D.C., December 2001. EPA/540/R-02/002. Available at: http://www.epa.gov/oswer/riskassessment/rags3adt/index.htm

2. U.S. Environmental Protection Agency. 1995. *Ecological Risk and Decision Making Workshop.* December 1995. EPA 230/B96/004B.

3. Moore, D.W.J., et al. 1999. A probabilistic risk assessment of the effects of methylmercury and PCBs on mink and kingfishers along East Fork Poplar Creek, Oak Ridge, Tennessee, USA. *Environmental Toxicology and Chemistry* 8:2941-2953.

4. Suter II, G.W., et al. 2000. *Ecological Risk Assessment of Contaminated Sites*. Lewis Publishers, Boca Raton, FL. (see pages 228-231).