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# A Framework for Ecological Risk Assessment: Technical Appendices

March 1997

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CCME Subcommittee on
Environmental Quality Criteria for Contaminated Sites

The National
Contaminated Sites
Remediation Program

Winnipeg, Manitoba

March 1997

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# Sampling Principles

Information on sampling with good statistical design can be obtained from the literature. Some useful sources are provided in the following box.

*Additional reading*

Alldredge, J.R. 1987. Sample size for monitoring of toxic chemical sites. Environ. Monit. Assess. 9:143–154.

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## A.1 SCREENING ASSESSMENT SAMPLING PRINCIPLES

Screening assessments are desktop exercises and so usually do not require sampling. However, a visual inspection of the site is required to get a "hands-on" feeling for the site.

In a Screening Assessment, the data that are available will have to be evaluated and judged whether or not they are representative of the site. The quality of the data will affect the degree of uncertainty surrounding the estimates of risk and provide the rationale for proceeding to a quantitative ecological risk assessment (ERA).

*For example*
If sampling has focused on "hot spots", then information on the spatial distribution may be lacking. This problem is overcome in a Preliminary Quantitative ERA, where representative sampling is required.

Impacts cannot be identified until a determination is made about what kinds and levels of change in environmental parameters are meaningful (Green 1979). This is an important a priori step in sampling a site. Statistical significance has no bearing on the extent to which the change may be either meaningful or important and have ecological consequences. Therefore, statistical information should be tempered with scientific and site-specific concerns.

Meaningful change in measurement endpoints can be determined by comparison of the impacted area with natural or background conditions. For contaminated sites, baseline data do not usually exist, so reference site data are used. Reference sites are areas that are as similar as possible to the area but without pollution impacts. Given that chemistry data are not usually well replicated, statistical comparisons are not usually possible, and comparisons to reference data are usually made qualitatively.

Ideally, the historical data would include

- baseline conditions prior to pollution impact
- measurements of parameters at suspected impact areas
- reference site data

## A.2 PRELIMINARY QUANTITATIVE ERA SAMPLING PRINCIPLES

The objectives of the ERA will define the kind of sampling and level of effort required. Planning should include the development of testable questions or hypotheses. A hypothesis is a further refinement for increased precision of a question by including within it information about the criterion (e.g., a measure of biological impact) and predictor variables (e.g., a measure of impact intensity). The hypothesis should represent the simplest possible answer to the question stated so that it is testable and may be proved false, as with the null hypothesis $H_0$.

Once the questions and hypotheses have been formulated, information gathered in the Screening Assessment may help select the parameters, methodologies, and sampling design techniques that are suitable for the Preliminary Quantitative ERA. Defining contaminant-related impacts depends, in part, on identifying and accounting for natural sources of variability. The most effective sampling designs are stratified according to the dominant pattern of variation of data, which may be seasonal, flow-related, spatial, etc. Such stratification removes the dominant natural variability and allows effective determination of contaminant-related changes. Ideally, the Screening Assessment data will determine the initial stratification scheme, both temporal and spatial, for the Preliminary Quantitative ERA.

Understanding variance is also the key in determining the how many samples are required in order to have a true representation of a variable. Without knowing how much a given parameter varies, it is impossible to determine how many samples are required to characterize it. Preliminary Quantitative ERA sampling should properly characterize the variance of key parameters.

## A.3 DETAILED QUANTITATIVE ERA SAMPLING PRINCIPLES

Detailed Quantitative ERA sampling will likely focus on addressing data gaps. If it was determined that temporal variability was a significant contributor to uncertainty, then a sampling program might be designed to cover critical seasons. Detailed Quantitative ERA sampling requires the same up-front planning, including testable hypotheses, stratification of sampling, and inclusion of reference site sampling.

## REFERENCES

Green, R.H. 1979. Sampling design and statistical methods for environmental biologists. Toronto: John Wiley and Sons.

# Receptor Characterization

## B.1 DEFINITION

A receptor is an ecosystem component that is or may be adversely affected by a pollutant or other stressor emanating from a contaminated site. Receptors may include biological or abiotic (e.g., air or water quality) components. For the purposes of this report, humans are not considered to be an ecological receptor.

From a regulatory standpoint, the preferred strategy would be to always apply a uniform set of standard, rigorous techniques to receptor characterization at any specific location. Unfortunately, due to the natural variability in environmental systems, this is not possible, and receptor characterization relies heavily on expert judgment to cope with site-specific ecological complexity.

Normally, the main focus of receptor characterization is on indigenous populations of resources such as animals and plants. It is also important, though, to identify natural ecosystem processes (e.g., production, decomposition) that may be affected by the stressors, and to consider migratory species.

*For example*
Natural ecosystem processes are important since changes in ecosystem structure or function may, in turn, adversely affect the ability of ecosystems to generate products of value to humans (e.g., fish) or perform vital functions (e.g., flood and erosion protection).

Migratory species, though only passing through an area for a short time, may be highly concentrated in particular habitats (e.g., bird staging areas along a migration route, fish spawning areas), which renders them potentially vulnerable to population level impacts. Contaminant loads in migratory species can not generally be pinpointed to a particular source, unless this source has a unique signature. The juveniles of migratory species that are produced near the contaminated site are more comparable to an indigenous population, and their tissue concentrations are more likely to be the result of local sources. Contaminants can, however, be passed from females to their offspring through eggs, and this type of confounding influence should be considered.

## B.2 OVERVIEW

The following box provides a summary of the information required to characterize receptors.

**Identify receptors**
Identify levels of organization (e.g., individual, population, community, ecosystem)
Evaluate structural and functional attributes
Consider spatial and temporal scale
Consider migration, distribution of species
Ensure receptors are relevant to evaluate remedial alternatives

**Characterize habitat**
Establish physical and chemical attributes
Consider the sensitivity and vulnerability of habitat

### B.2.1 Habitat Characteristics

There are two main objectives for collecting habitat information. The first is to help describe species niches for the populations of concern. The second is to generate background data on structural/physical and chemical environmental attributes that may affect biotic responses to the stressors (See Table B.1). The latter is largely covered as exposure assessment.

### B.2.2 Species and Populations

The scope of most ecological risk assessments (ERAs) is limited to one or several species and occasionally to particular populations. Undoubtedly there are many underlying reasons for this emphasis, but they appear to be related to simplicity and ease, economics, and lack of data characterizing habitat and resident species. Table B.2 provides examples of structural and functional characteristics of species and populations useful in receptor characterization.

It may be unnecessary to develop a *full* inventory of all species present. Instead, it may be more worthwhile to focus on identifying species that are

- potentially sensitive to the stressors from the contaminated site
- recognized by the federal or provincial government as threatened or endangered

**Table B.1 Structural and physical habitat characteristics for characterizing receptors**

| Structural/physical characteristics | Examples |
|---|---|
| Local topography and three-dimensional configuration of the habitat at risk | Elevation<br>Landscape<br>Geographic proximity of each sensitive habitat to the contaminated site |
| Watershed characteristics | Surface cover<br>Soil types<br>Geology<br>Surface and groundwater hydrology |
| Weather and climate data | Temperature regimes<br>Precipitation |
| Physical habitat alterations | Anthropogenic changes (e.g., dammed river) |
| Particularly sensitive habitats | Wetlands potentially retaining released contaminants for long periods<br>Sites with particularly sensitive life history stages (e.g., fish spawning or rearing areas, ground nesting areas of birds)<br>Habitats of local or regional ecological significance (e.g., staging areas for waterfowl) |

**Table B.2 Structural and functional attributes of populations and species for characterizing receptors**

| Structural/functional attributes | Examples |
|---|---|
| Structural attributes | List of species found at and around the contaminated site<br>Presence of rare or endangered species<br>Presence of intolerant species<br>Presence of tolerant species<br>Species historically found at the site but now absent<br>Species historically absent at the site but now present<br>Overall population density<br>Mass of individuals<br>Number and distribution of populations within a community<br>Age-class structure<br>Life history data<br>Proportion of mature females<br>Fecundity per mature female<br>Cumulative probability of survival from the age of reproductive maturity to each future age<br>Individual health: levels of parasitism or disease; skeletal anomalies, lesions, etc |
| Functional attributes | Food requirements<br>Ingestion rates<br>Bioaccumulation potential<br>Intrinsic rate of increase<br>Behavioural capabilities<br>Activity patterns<br>Habitat requirements<br>Natural variability in time and space; e.g., do activity patterns and habitat requirements vary seasonally or with different phases of the life cycle? |

- migratory (birds or fish), where a significant proportion of the population is concentrated in the vicinity of the site during certain periods
- dominant within local biological communities, or functioning as keystone species within nearby ecosystems
- recognized as good indicators or surrogate species
- of aesthetic value or of value to the local human population
- of recreational or commercial importance

### B.2.3 Communities and Ecosystems

If specific ecosystems or communities have been identified as significant to an assessment endpoint, the first step in characterizing them is to provide precise information on their location and specific type. The exact suite of measurements to describe the receptor will vary according to whether the ecosystem is a forest, grassland, wetland, floodplain, agro-ecosystem, stream, river, pond, or lake, and so forth. None of these parameters are easily measured, so before any of them are selected for quantification, there should be a well-designed program to guide data collection and analysis, supported by a distinct need for the data. Structural and functional characteristics of communities and/or ecosystems useful for receptor characterization are provided in Table B.3.

## B.3 QUALITATIVE CHARACTERIZATION

The main purpose of initial screening is to simplify the task of receptor characterization by limiting consideration to the habitats and species most likely to be affected by stressors associated with the contaminated site. Potential receptor habitats and ecosystem components, such as individuals, populations, or communities, are identified through a process involving consideration of spatial and temporal overlaps between stressors from the contaminated site and components of adjacent and nearby ecosystems. Initial screening is usually based on a review of available data and information, field reconnaissance, and a qualitative evaluation of potential effects. During screening, an attempt should be made to catalog all potentially significant or sensitive receptors at or near the contaminated site.

Once vulnerable ecosystems, populations, and processes have been identified, they can be expressed as structured impact hypotheses (Bernard et al. 1990). One purpose of these hypotheses is to clearly illustrate linkages between stressors from the contaminated site and changes in receptors. The process of developing these hypotheses helps in selecting or refining endpoints for the ERA analysis.

**Table B.3 Structural and functional attributes of ecosystems and communities for receptor characterization**

| Structural/functional attributes | Examples |
|---|---|
| Structural attributes | Biodiversity<br>Biomass (by trophic level)<br>Relative abundances<br>Dominance<br>Functional guilds<br>Successional stages present<br>Trophic linkages |
| Functional attributes | Primary production<br>Respiration<br>Decomposition<br>Nutrient cycling<br>Resilience |
| Local or regional significance | Frequency of occurrence of a particular type of ecosystem |

## B.4 QUANTITATIVE CHARACTERIZATION

Quantitative receptor characterization requires field sampling, and the field sampling program should be designed to generate data of sufficient quality and precision to be suitable for the intended type of data analysis and interpretation. Before field work begins, a quality assurance and quality control program should be developed to guide sample collection and analysis.

## B.5 DISCUSSION AND CONCLUSIONS

Even the most comprehensive ERA protocol will have little value if its complex procedures or its extensive data requirements prevent it from being implemented (Parkhurst et al. 1990). As well, the complexity of most ecosystems is an effective barrier to creating a simple, yet thorough, method for characterizing receptors, whether they be ecosystems or individual species. A tiered approach to receptor characterization can lead to the examination of a wider range of species and/or communities, the study of a more extensive area, and/or a more accurate quantitative assessment of measurement endpoints.

All of the parameters listed above can be measured with some degree of success using current methods. As well, some of the methods (e.g., taxonomic surveys) are in routine daily use throughout North America.

## REFERENCES

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Parkhurst, B.R., H.L. Bergmann, M.D. Marcus, C.S. Creager, W. Warren-Hicks, H. Olem, A. Boelter, and J.P. Baker. 1990. Evaluation of protocols for aquatic ecological risk assessment and risk management. WPCF Research Foundation, Technology Assessment Department, Alexandria, VA.

APPENDIX C

# Exposure Assessment

## C.1 DEFINITION

Exposure assessment has been defined as the "co-occurrence of or contact between a stressor and an ecological component" (U.S. EPA 1992). Exposure assessment is an attempt to answer the following questions (adapted from Urban and Cook 1986):

- What are the significant routes of exposure?
- To what amounts of each contaminant are organisms actually or potentially exposed?
- How long is each exposure?
- How often does or will exposure take place?
- What seasonal and climatic variations in conditions are likely to affect exposure?
- What are the site-specific geophysical, physical, and chemical conditions affecting exposure?

Some useful resources for assessing exposure are listed in the following box.

*Additional reading*


Calabrese, E.J., and L.A. Baldwin. 1993. Performing ecological risk assessments. Chelsea, MI: Lewis Publishers.

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## C.2 OVERVIEW

The following box provides a summary of the key elements in assessing exposure.

**Selection of target chemicals**
Review physical, structural, toxicological properties of chemicals

**Contaminant release**
Determine chemical distribution in soil, sediment, water, and biota
Determine background levels and matrix properties

**Transport and fate analysis**
Transport mechanism and fluxes
Breakdown products and transformation rates

**Exposure pathway and analysis**
Identify plausible exposure routes
Direct contact
Water ingestion
Soil or sediment ingestion
Food web

**Aquatic receptor exposure analysis**
Identify maximum accumulation under equilibrium
Characterize accumulation under field conditions
Estimate fraction and rate of chemical uptake
Estimate metabolic elimination rate

**Terrestrial receptor exposure analysis**
Estimate fraction of feeding from site
Estimate feeding rate
Characterize contaminant levels in food
Quantify direct soil uptake
Estimate fraction bioavailability from food and soil
Estimate metabolic elimination rate

**Uncertainty analysis**
Conduct Monte Carlo simulation, sensitivity analysis, calibration with monitoring data

In terms of exposure assessment, the major differences between humans and biota are in the modes of contact, and the spatial exposures to toxic substances. Primary modes of contact for humans are inhalation, dermal exposure, and ingestion of soil, food, or water; plants and animals may have modes of contact that are physiologically very different, such as the transport of

contaminants across the membrane of a fish's gill. Ecological exposure assessments also require a consideration of different parts of the environment than human health exposure assessments.

*For example*
Concentrations of toxic substances in sediments of a deep lake may not be directly hazardous to humans unless they enter drinking water or organisms that are eaten, but they may have significant direct impacts on benthic community structure (e.g., changes in the assemblages of organisms that live in these sediments).

## C.3 SELECTION OF TARGET CHEMICALS

### C.3.1 Qualitative Methods

The objective of this step is to narrow the set of contaminants considered to those that pose either the greatest potential of release or the greatest toxic threat. All contaminants should be considered until they can be excluded based on scientific evidence. The following three general principles for selecting target chemicals for ecological risk assessments (ERAs) can be used:

1. determine the physical/chemical properties of the contaminants stored at the site

2. group contaminants according to their physical/chemical properties, and predominant medium of concern (i.e., air, water, soil, biota)

3. choose one contaminant (or more) within each physical/chemical group that is (are) likely to be the most toxic, based on available criteria, measured concentrations, and available dose-response information. Also consider environmental persistence and potential for bioaccumulation. It is important to consider the hazard data.

The properties of the chemicals will determine the medium of concern (e.g., air, soil, surface or groundwater, animal tissue), and conversely, the properties of the various media will determine the chemicals of concern.

*For example*
Chemicals with a low $K_{ow}$ value and high water solubility could affect organisms that inhabit soil and surface waters but have a low bioaccumulation potential. Transport through surface runoff and groundwater would be key exposure pathways for these chemicals. In contrast, chemicals with high $K_{ow}$ values and low water solubility tend to sorb to particles in soils and surface waters, and have a high bioaccumulation potential. These chemicals may have very different exposure pathways (e.g., adsorption to soil particles, followed by off-site transport through soil erosion or ingestion by terrestrial animals, environmental persistence).

### C.3.2 Quantitative Methods

The selection of target chemicals can be refined and/or restated based on the results of the screening assessment using the same principles but with more accumulated data.

## C.4 EVALUATION OF CONTAMINANT RELEASE, TRANSPORT, AND FATE

Contaminants can be released to the air, surface water, sediment, soil, and groundwater. Common mechanisms for contaminant release from a variety of sites are shown in Table C.1. Figure C.1 provides an overview of environmental fate and transport analysis for exposure assessments.

### C.4.1 Qualitative Methods

Contaminant Release

Initial and qualitative investigations serve to

- identify each potential contaminant release source
- determine the potential environmental media affected by each release
- broadly define the possible extent of the release

Contaminant Transport and Fate

Initial and qualitative investigations serve to

- identify each transport process governing the movement of various contaminants within and among environmental media
- determine the direction and roughly gauge the rate of contaminant movement from the site
- identify areas to which contaminants have been or may be transported

**Table C.1 Common mechanisms for release of contaminants**

| Receiving medium | Release mechanism | Release source |
|---|---|---|
| Air | Volatilization | Surface wastes—lagoons, ponds, pits, spills<br>Contaminated surface water<br>Contaminated surface soil<br>Contaminated wetlands<br>Leaking drums |
| | Fugitive dust generation | Contaminated surface soil<br>Waste piles |
| Surface water | Surface runoff | Contaminated surface soil |
| | Episodic overland flow | Lagoon overflow<br>Spills, leaking containers |
| | Groundwater seepage | Contaminated groundwater |
| Groundwater | Leaching | Surface or buried wastes<br>Contaminated soil |
| Soil | Leaching | Surface or buried wastes |
| | Surface runoff | Contaminated surface soil |
| | Episodic overland flow | Lagoon overflow<br>Spills, leaking containers |
| | Fugitive dust generation/deposition | Contaminated surface soil<br>Waste piles |
| | Tracking | Contaminated surface soil |
| Sediment | Surface runoff, episodic overland flow | Surface wastes—lagoons, ponds, pits, spills<br>Contaminated surface soil |
| | Groundwater seepage | Contaminated groundwater |
| | Leaching | Surface or buried wastes<br>Contaminated soil |

Source: U.S. EPA 1989a

Figures C.2 and C.3 illustrate potential transport routes for contaminants released to soil and groundwater, and to surface water, respectively.

Estimates of the above behaviours can be made from knowing the key physical and chemical properties of the contaminants. Many of these properties can be found in the scientific literature. Examples of significant physical and chemical properties of chemicals are presented in Table C.2.

## C.4.2 Quantitative Methods

### C.4.2.1 *Preliminary Quantitative Analyses*

Contaminant Release

The rate of contaminant release can be computed through a variety of methods and provide the foundation for contaminant fate analysis. Generally, the average release rates to different media are used as input to fate and transport analyses and to ultimately provide measurements and/or predictions of the concentrations of contaminants that organisms are exposed to.

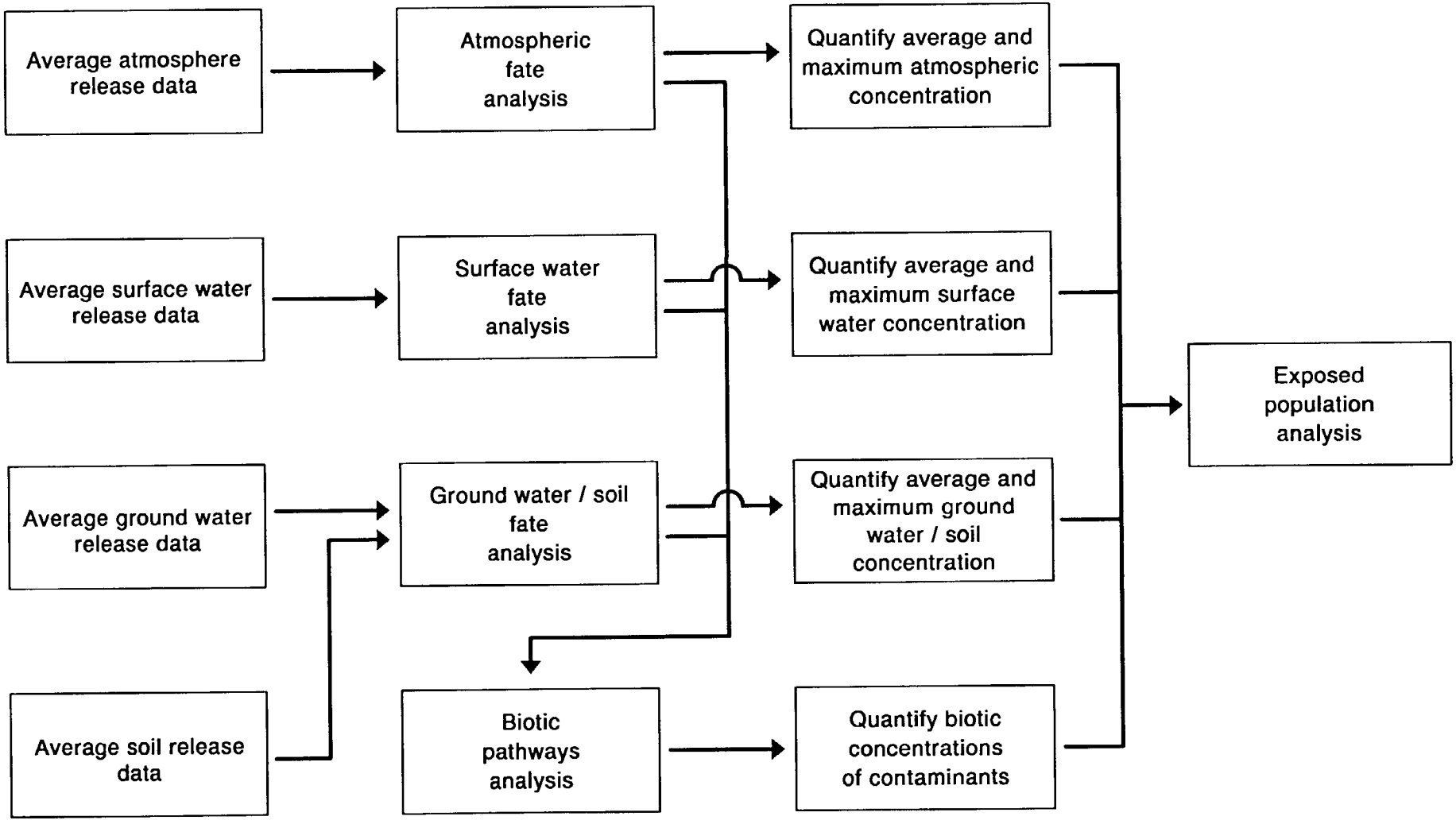


Figure C.1 Overview of environmental fate and transport analysis for exposure assessments. (Adapted from PRC Environmental Management Inc. 1985)

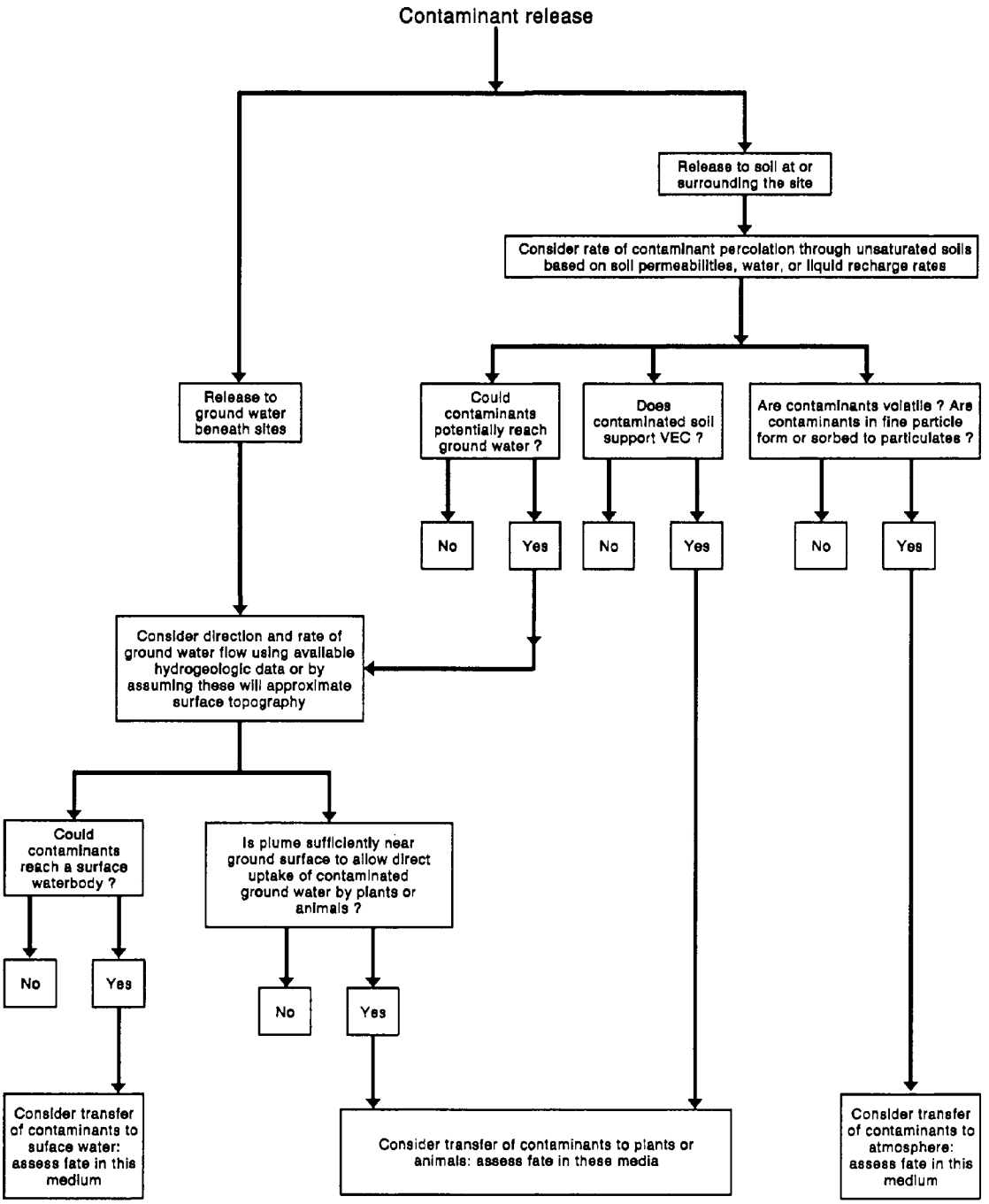


Figure C.2 Fate and transport assessment for contaminants released to soil and groundwater. (Adapted from U.S. EPA 1989c)

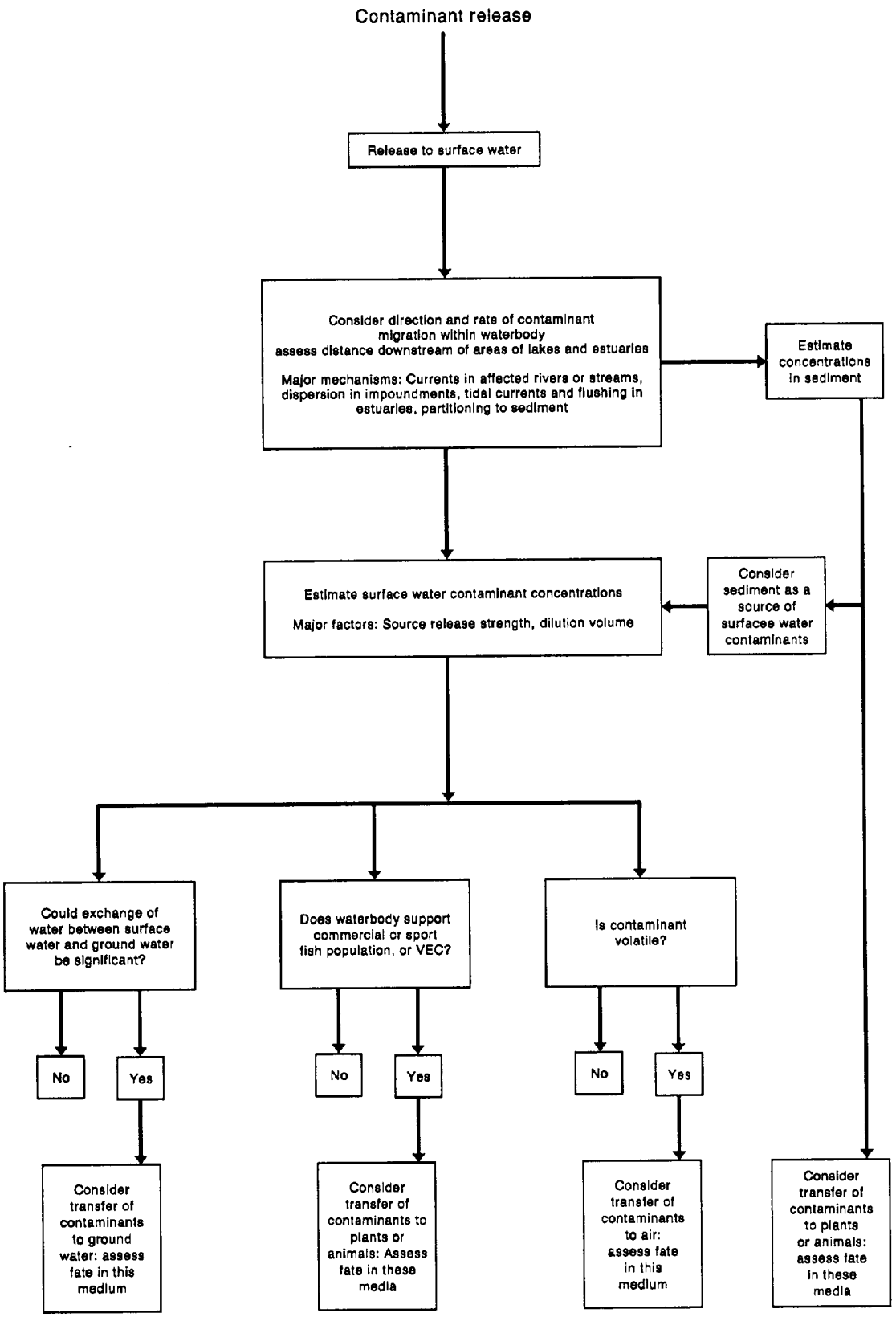


Figure C.3 Fate and transport assessment for contaminants released to surface water. (Adapted from U.S. EPA 1989c)

**Table C.2 Examples of significant physical/chemical properties of chemicals**

| Physical/chemical property | Significance/definition | Sources of data and additional information |
|---|---|---|
| Water solubility (S) | Affects a chemical's distribution between environmental compartments. A chemical with a high water solubility will partition in water.<br>May be used to estimate octanol–water partition coefficient, Henry's law constant, adsorption to soil and bioconcentration factor | Horvath 1982; Kenaga 1980; Riddick et al. 1986; Seidell 1941; Shiu et al. 1990; Wauchope et al. 1992; Worthing and Walker 1987 |
| Partition coefficients ($K_p$)<br>$K_p = [C_s] / [C_w]$<br>$C_s$ = concentration of solute in soil/sediment<br>$C_w$ = concentration of solute in water | $K_p$ measures the partitioning of a chemical substance between the solid phase (soil or sediment) and the solution phase (water) of a two-phase system at equilibrium. Indicates the strength of sorption of chemical to soil, sediment, or biota: the larger the $K_p$ value the more strongly is the chemical sorbed | Kenaga 1980; Meylan et al. 1992; Rao and Davidson 1982; Reinbold et al. 1979; Sabljic 1987 |
| Organic carbon partition coefficient ($K_{oc}$)<br>$K_{oc} = K_p\alpha$<br>$\alpha$ = mg of C per mg of soil or sediment | $K_p$ for a specific compound will vary with the organic or lipid content of the soil or sediment. Normalizing $K_p$ values to the fraction of organic carbon present in the medium provides a more equal basis for comparing distribution constants. | |
| octanol–water partition coefficient ($K_{ow}$)<br>$\log K_{oc} = \log K_{ow} - 0.2$ | The log of the ratio of a chemical's equilibrium concentration in octanol to that in water in a two-phase octanol-water system<br>Strongly correlated with a contaminant's bioaccumulation potential | Mabey et al. 1981 |
| Henry's law constant (H) | The Henry's law constant is the ratio of a chemical's concentration in the gas phase to that in the liquid phase and determines a chemical's volatilization rate from water and moist soil.<br>As the Henry's law constant increases, losses to the atmosphere or the air phase within the soil also increase (Ashworth et al. 1988). | Hine and Mookerjee 1975; Lamarch and Droste 1989; Mabey et.al. 1981; Mackay and Shiu 1981; Shiu and Mackay 1986 |
| Vapour pressure | Affects a chemical's distribution between environmental compartments. A chemical with a high vapor pressure will partition to the atmosphere. | Boublik et al. 1984; Mabey et al. 1981; Riddick et al. 1986; Wauchope et al. 1992; Wilhoit and Zwolinski 1971, 1973; Worthing and Walker 1987 |
| Degradation rates via<br>Hydrolysis | Occurs as a result of the reaction with water | Mabey and Mill 1978 |
| Photolysis | Occurs as a result of the influence of light either directly from the absorption of light or indirectly as a result of an intermolecular reaction between the compound of interest and reactive species formed under the influence of light. | Atkinson 1991; Atkinson and Carter 1984 |
| Microbial degradation | The conversion of an organic compound to inorganic compounds ($CO_2$, $H_2O$, and the oxides or mineral salts of any other elements in the compound) | Alexander 1981, 1985; Dagley 1987; Fewson 1988; Gibson 1984; Grady 1985; Howard and Banerjee 1984; Howard et al. 1987; Klečka 1985; Kobayashi and Rittmann 1982 |

Source: Compiled from Howard and Boethling 1993

On-site monitoring is the most reliable method of estimating most release rates of contaminants and environmental concentrations, though this may not always be possible. Where monitoring is not possible, several preliminary quantitative analyses are available for estimating release rates, which serve as input to environmental fate analyses. These preliminary quantitative methods generally require no field sampling, though they also contain restrictive assumptions (consult U.S. EPA 1988 for further details).

Some of the equations available for calculating release rates and environmental concentrations are presented in the following tables. For more detailed explanations see U.S. EPA (1988) and refer to other texts for additional methods. Tables C.3 and C.4 provide examples of equations to estimate the amount of contaminant released to the atmosphere as particulate matter or as gas and to estimate the amount of contaminant released to surface water and groundwater, respectively.

Contaminant Transport and Fate

The contaminant release rates computed through one of the methods described in Tables C.3 and C.4 or from direct sampling provide the foundation for contaminant fate analysis. Generally, the average release rates to different media are used as input to transport and fate analyses. As with the determination of release rates, direct measurements provide the most accurate estimates. When direct measurements are not possible or are limited, environmental concentrations can be estimated. Tables C.5 and C.6 provide examples of equations that can be used to estimate contaminant concentrations in the atmosphere and in surface water and groundwater, respectively.

*C.4.2.2 Detailed Quantitative Analyses*

Direct measurement of contaminants in environmental media provides the most reliable and accurate information on contaminant release, transport, and fate. However, as in the preliminary quantitative analyses direct measurement may not always be possible. Alternatives to direct measurement include the use models for estimating contaminant concentrations in the environment.

Chemical fate and transport models are often used in exposure assessment to fill in data gaps and to make extrapolations to other exposure scenarios such as future exposures. Models can be used to

- estimate chemical concentrations in each medium of interest under steady state conditions
- estimate degradation rates for the calculation of the environmental residence time of the contaminant
- develop a "mass balance" of the inputs and outputs of contaminants to a defined area or compartment

In order for a model to be successful, high quality data and reasonable assumptions must be used in the model. The model must be validated to compare the model results with direct observations or with expected result suggested by theory. Several statistical techniques are available to measure the goodness of fit between model results and measurements including (Naylow and Finger 1967 cited in Bartell et al. 1992):

- Chi-square tests
- factor analysis
- Kolmogorov-Smirnov test
- nonparametric tests
- regression analysis
- spectral analysis
- Theil's inequality coefficient

***For additional information on verifying and validating models, see***

Bartell, S.M., R.H. Gardner, and R.V. O'Neill. 1992. Ecological risk estimation. Chelsea, MI: Lewis Publishers.

Burns, L.A. 1991. PIRANHA: Pesticide and Industrial Chemical Risk Analysis and Hazard Assessment. Version 2.0. U.S. Environmental Protection Agency, Environmental Research Laboratory, Office of Research and Development, Athens, GA.

**Table C.3 Examples of equations to estimate contaminant releases to the atmosphere**

| Measurement | Equation | Restrictions/assumptions |
|---|---|---|
| Estimate of volatile releases from covered landfills containing toxic materials alone or toxic materials segregated from other landfilled nonhazardous wastes | $E_i = E_i = D_i C_{si} A (P_t^{4/3}) \frac{M_i}{d_{sc}}$<br>emission rate of component $i$ (g/s)<br>$D_i$ = diffusion coefficient of component $i$ in air ($cm^2/s$)<br>$C_{si}$ = saturation vapor concentration of component $i$ ($g/cm^3$)<br>$A$ = exposed area ($cm^2$)<br>$P_t$ = total soil porosity<br>$M_i$ = mole fraction of toxic component $i$ in the waste (gmol/gmol)<br>$d_{sc}$ = effective depth of soil cover (cm) | Restrictions:<br>Total soil porosity is used rather than air-filled soil porosity; the presence of water will decrease the flux rate by effectively decreasing the porosity and also by increasing the complexity and therefore length of the vapor path. |
| Estimate of toxic vapor release from landfills that contain toxic wastes in combination with municipal wastes that generate gases | $E_i = C_i^* V_y A$<br>$E_i$ = emission rate (g/s)<br>$C_i^*$ = concentration of compound $i$ in the soil pore spaces ($g/cm^3$)<br>$V_y$ = mean landfill gas velocity in the soil pore spaces (cm/s)<br>$A$ = area ($cm^2$) | Restrictions:<br>The presence of saturated soils will tend to reduce the rate of volatile release from landfills<br>The degree to which this equation can accurately predict contaminant release rates for gases, especially soluble gases, at site with moist or wet soils is unknown. |
| Estimate of the volatile release from fresh chemical spills on soil where a contaminant pool is visible on the soil surface or where soil is contaminated from the surface down | $E_i = k_{iG} C_i^* A$<br>$Ei$ = emission rate of chemical $i$ (g/s)<br>$k_{iG}$ = gas phase mass transfer coefficient of chemical $i$ (cm/s)<br>$C_i^*$ = vapor concentration of chemical $i$ ($g/cm^3$)<br>$A$ = area ($cm^2$) | Restrictions<br>Does not consider soil-phase mass transfer resistance, so not appropriate for estimating release when spilled contaminants have seeped into surface soils.<br>Only useful for estimating releases of pure compounds |
| Estimate of volatile release from past spills, leaks, or landfarming directly onto or into surface soil . Takes into account contaminant loss over time | $E_i = \frac{2DC_s A}{d + \sqrt{2DC_s t/CB}}$<br>$E_i$ = average emission rate of component $i$ over time $t$ (g/s)<br>$D$ = phase transfer coefficient ($cm^2/s$)<br>$C_s$ = the liquid-phase concentration of contaminant $i$ in the soil ($g/cm^3$)<br>$CB$ = bulk contaminant concentration in soil ($g/cm^3$)<br>$A$ = contaminated surface area ($cm^2$)<br>$d$ = depth of dry zone at sampling time (cm)<br>$t$ = time measured from sampling time (s) | Assumptions:<br>Contaminant concentrations in soil remain constant until all contaminant is lost to the air<br>Contaminant release occurs by the "peeling away" of successive unimolecular layers of contaminant from the surface of the contaminated zone |

**Table C.3 Continued**

| Measurement | Equation | Restrictions/assumptions |
|---|---|---|
| Estimate of volatile releases of low solubility compounds from waterbodies | $E_i = K_i C_s A$<br><br>$E_i$ = emission rate (g/s)<br>$K_i$ = overall mass transfer coefficient (cm/s)<br>$C_s$ = contaminant liquid-phase concentration (g/cm$^3$)<br>$A$ = area (cm$^2$) | Assumptions:<br>Conditions are steady state<br>Diffusion is liquid-state controlled<br>Occurs from a well-mixed water phase to a well-mixed air phase across a stagnant water/air interface |
| Estimates of dust emissions | 1. Estimate the amount of dust generated by wind erosion, using either an equation for annual erosion rates (a function of soil erodibility, climate, soil roughness, field length, and vegetative cover) or the rapid assessment approach of Cowherd et al. (1985) for worst-case daily release rates. | |
| | 2. Adjust rates of total soil loss by wind erosion to reflect the fraction that could be suspended and transported over significant distances by wind. | |
| | 3. Multiply the amount of dust generated by the weight percent of the toxic substances in soil or waste, or (preferably) in dust samples obtained with on-site air monitoring. | |

Source: Compiled from U.S. EPA 1988

**Table C.4 Examples of useful measurements and equations for estimating contaminant release rates to surface water and groundwater**

| Measurement | Equation | Restrictions/assumptions/comment |
|---|---|---|
| Modified universal soil loss equation | $Y(S)E = a(V_r q_p)^{0.56} KLSCP$<br>$Y(S)E$ = sediment yield (tonnes/event)<br>$a$ = conversion constant (11.8)<br>$V_r$ = volume of runoff ($m^3$)<br>$q_p$ = peak flow rate ($m^3/s$)<br>$K$ = the erodibility factor<br>$L$ = the slope-length factor<br>$S$ = the slope-steepness factor<br>$C$ = the cover factor<br>$P$ = the erosion control practice factor | Comment:<br>Can use to estimate the amount of hydrophobic compounds released in site runoff when used with sorption partition coefficients derived from the compound's octanol-water coefficient |
| Loading rate to groundwater from landfilled solids | $L_c = q \times A \times C_o$<br>$L_c$ = contaminant loading rate (mass/time)<br>$q$ = percolation rate (length/time)<br>$A$ = area of landfill ($length^2$)<br>$C_o$ = solubility of solid chemical (mass/volume) | Assumptions:<br>Adequate residence time is available for contaminants to reach equilibrium solubility |
| Loading rate to groundwater from lagooned or land-filled liquids<br><br>Volumetric flux leaving the site | $L_c = C_s \times Q_l$<br>$L_c$ = contaminant loading rate (mass/time)<br>$C_s$ = contaminant concentration in lagoon fluid (mass/volume)<br>$Q_l$ = volume loading rate<br>$Q_l = K_s \times i \times A$<br>$Q_l$ = volume loading rate (volume/time)<br>$K_s$ = Darcy's coefficient<br>$i$ = hydraulic gradient (length/time)<br>$A$ = area of lagoon ($length^2$) | Restrictions:<br>Equations are applicable for liquids that are mostly water<br>For lagoons that contain organic fluids, the equations may need to be corrected if the density or viscosity differs from water |
| Loading rate to groundwater from facilities lined with flexible membranes<br><br>Rate of permeation of liquids and gases through polymers | $L_c = P_s \times A \times p/d_l$<br>$L_c$ = contaminant loading rate (mass/time)<br>$P_s$ = permeation rate<br>$A$ = area of liner<br>$p$ = vapor pressure<br>$d_l$ = thickness of the liner<br>$P_s = A_p \varnothing^{-S_H}$<br>$P_s$ = permeation rate<br>$A_p$ = constant solely dependent on the type of polymers used<br>$S_H$ = constant solely dependent on the type of polymers used<br>$\varnothing$ = the polymer "permachor" calculated for each polymer-permeant pair | Comment:<br>Although a flexible membrane liner appears to allow no migration through the barrier, there may be penetration by organic compounds and contaminated water at low rates of permeation. |

Source: Compiled from U.S. EPA 1988

**Table C.5 Examples of equations for estimating atmospheric contaminant concentrations**

| Measurement | Equation | Restrictions/assumptions |
|---|---|---|
| Estimate ground-level atmospheric concentrations of pollutants downwind of the source | $C(x)=Q/\pi\sigma_y\sigma_z\mu$<br><br>$C(x)$ = concentration of a substance at distance x from site (mass/volume)<br>$Q$ = release rate of substance site (mass/time)<br>$\mu$ = mean wind speed (distance/time)<br>$\sigma_y$ = dispersion coefficient in the lateral direction (distance)<br>$\sigma_z$ = dispersion coefficient in the vertical direction (distance)<br>$\pi$ = the value pi = 3.14 | Assumptions:<br>Contaminant released is in a form that can remain airborne indefinitely, either as a gas or as particles less than 20 μm in diameter<br>Steady-state conditions<br>Longitudinal dispersion is negligible<br>All removal and decay processes are disregarded<br>Substance is distributed normally both vertically and in the crosswind direction<br>Air environment is homogeneous |
| Estimate area within which the ground-level concentration of a contaminant is above predetermined critical concentration | $y_{(x)}=2\ln[\frac{C(x)}{C(CL)}]^{1/2}(\sigma_y)$<br><br>$y_{(x)}$ = perpendicular distance from point on plume centerline to the C(CL) isopleth boundary (length)<br>$C(CL)$ = predetermined critical concentration level (mass/volume)<br>$C(x)$ = concentration at plume centerline, X distance from the source (mass/volume)<br>$\sigma_y$ = lateral dispersion coefficient (length) | |

Source: Compiled from U.S. EPA 1988

**Table C.6 Examples of measurements and equations for estimating contaminant concentrations in streams**

| Measurement | Equation | Restrictions/assumptions |
|---|---|---|
| Estimate of the contaminant concentration downstream from a point source release into a flowing waterbody, after dilution of the substance by the receiving waterbody. | $C = C_e Q_e / Q_t$<br>$C$ = concentration of contaminant in stream (mass/volume)<br>$C_e$ = concentration of substance in effluent (mass/volume)<br>$Q_e$ = effluent flow rate (volume/time)<br>$Q_t$ = combined effluent and stream flow rate (volume/time) | Restrictions:<br>Release is into a flowing water body, and concentration is estimated after dilution of the substance by the receiving waterbody<br>Equation does not account for sources of contamination other than the site<br><br>Assumptions:<br>Mixing of contaminants in water is instantaneous and complete<br>All decay or removal processes are disregarded<br>Stream flow and rate of contaminant release to the stream are constant |
| Estimate of stream concentration of contaminant from contaminant release into stream through intermedia transfer from air, soil, groundwater, or nonpoint source. | $C = T_r / Q_t$<br>$C$ = concentration of substance in stream (mass/volume)<br>$T_r$ = intermedia transfer rate (mass/time)<br>$Q_t$ = stream flow rate after intermedia transfer has occurred (volume/time) | |
| Estimate for the length of mixing zone | $MZ = \frac{0.4 w^2 u}{0.6 d \sqrt{g d_n s_l}}$<br>$MZ$ = length of mixing zone (length);<br>$w$ = width of water body (length)<br>$u$ = stream velocity (length/time)<br>$d_n$ = stream depth (length)<br>$s_l$ = slope of stream channel length/length;<br>$g$ = acceleration due to gravity (distance/time$^2$) | Restrictions:<br>Estimates in-stream contaminant concentrations resulting from the site release only ? |
| Estimate of contaminant concentration downstream from site and accounting for overall decay and loss rates affecting the removal of a contaminant from a water body. | $W(x) = W(O) e^{-K(x/u)}$<br>$W(x)$ = concentration at downstream distance x (mass/volume)<br>$W(O)$ = concentration immediately below point of introduction<br>e = 2.71828<br>$K$ = overall decay coefficient (time$^{-1}$)<br>$x$ = distance downstream from point of introduction (length)<br>$u$ = stream velocity (length/time) | Restrictions:<br>Applicable for nonconservative substances<br><br>Assumptions:<br>Mixing is complete |
| Estimate of the distance downstream over which a nonconservative substance remains above a predetermined critical level | $x = -(u/K) \ln[W(CL)/W(O)]$<br>$x$ = distance downstream from point of introduction (length)<br>$u$ = stream velocity (length/time)<br>$K$ = overall decay coefficient (time$^{-1}$)<br>$W(CL)$ = predetermined critical concentration level (mass/volume)<br>$W(O)$ = concentration immediately below point of introduction | Assumptions:<br>Mixing is complete<br>Steady state conditions<br>Longitudinal dispersion is negligible<br>All rate and transfer processes can be described as first order coefficients |

Source: Compiled from U.S. EPA 1988

The selection and use of a model will depend primarily on the purposes for which it is needed and on the following criteria:

- capability of the model to account for important transport, transformation, and transfer processes, which are presented in Table C.7
- model's fit to site-specific and substance-specific parameters
- model's data requirements, compared to the availability and reliability of site information
- form and content of model output
- the level of training of the person using the model
- the computer capabilities available
- the time and resources available for installation, setup, and data organization

A number of environmental fate models have been described in Chapter 5 of Suter (1993). A summary of these models is listed in Table C.8; this should not be construed as a comprehensive list of the fate and transport models that are currently available. Refer to Suter (1993), or the original model reference, for a more detailed explanation of the advantages and disadvantages of each model. The SEAM (U.S. EPA 1988) also provides summaries of the resource requirements and information sources for various surface-water fate models.

## C.5 EXPOSURE PATHWAYS ANALYSIS

Exposure pathways analysis involves the identification of plausible exposure routes for each identified receptor. This analysis views the exposure pathways from the perspective of the organism, rather than that of the contaminated site. For each valued ecosystem component, is exposure likely through direct contact, water ingestion, soil or sediment ingestion, or via the food web? Both direct and indirect pathways should be considered. Ultimately one will need to add up all the different exposure pathways for a given ecosystem component, for both long-term and short-term (extreme) exposure calculations. Often, certain pathways can be quickly eliminated from further consideration through simple calculations.

### C.5.1 Comparison of Aquatic and Terrestrial Exposure Pathways

Aquatic biota are most likely to be exposed to contaminants through direct contact with water or through ingestion of surface water, sediment, and contaminated food. In aquatic systems, organisms are exposed to *concentrations* of contaminants as they are continuously exposed to dissolved contaminants in the water column. Terrestrial animals can also be exposed through ingestion of contaminated surface water, soil, or foods, generally as a *dose*. These foods include plants that can take up contaminants from surface water, groundwater, soil, or air. The exposure for some terrestrial organisms such as plants and soil organisms may also be to a contaminant concentration. Other exposure routes for some terrestrial organisms are ingestion during grooming and preening, absorption through the skin, and inhalation. Overall, there are more exposure routes possible for terrestrial organisms, and the behaviour and spatial distribution of terrestrial organisms is usually more complex than that of aquatic organisms. Figure C.4 illustrates the possible exposure pathways in the terrestrial environment.

## C.6 AQUATIC AND TERRESTRIAL FOOD CHAIN EXPOSURE

***Additional reading***


Barron, M.G. 1990. Bioconcentration. Environ. Sci. Technol. 24:1612–1618.

Bysshe, S.E. 1990. Bioconcentration factor in aquatic organisms. *In* Handbook of chemical property estimation methods. Washington, DC: American Chemical Society.

Connel, D.W., and R.D. Markwell. 1990. Bioaccumulation in the soil to earthworm system. Chemosphere 20:91–100.

Di Toro, D.M., C.S. Zarba, D.J. Hansen, W.J. Berry, R.C. Swartz, C.E. Cowan, S.P. Pavlou, H.E. Allen, N.A. Thomas, and P.R. Paquin. 1991. Technical basis for establishing sediment quality criteria for nonionic organic chemicals using equilibrium partitioning. Environ. Toxicol. Chem. 10:1541–1583.

Kenaga, E.E. 1980. Correlation of bioconcentration factors and soil adsorption coefficients of pesticides and other. Ecotoxicol. Environ. Saf. 4:26–38.

Kenaga, E.E. 1980. Correlation of bioconcentration factors of chemicals in aquatic and terrestrial organisms with their physical and chemical properties. Environ. Sci. Technol. 14:553–556.

Mackay, D., and A.I. Hughes. 1984. Three-parameter equation describing the uptake of organic compounds by fish. Environ. Sci. Technol. 18:439–444.

Veith, G.D., D.L. DeFoe, and B.V. Bergstedt. 1979. Measuring and estimating the bioconcentration factor of chemicals in fish. J. Fish Res. Board Can. 36:1040–1048.

**Table C.7 Transfer and transformation processes in different environmental media**

| Environmental media | Intermedia transfers | Intramedia transformations |
|---|---|---|
| Atmosphere | Dissolution of gases into water droplets<br>Adsorption onto particulate matter<br>Gravitational settling<br>Precipitation | Photolysis<br>Oxidation |
| Surface water | Volatilization<br>Sedimentation<br>Sorption | Photolysis<br>Oxidation<br>Hydrolysis<br>Biodegradation |
| Soil | Volatilization<br>Sorption<br>Leaching | Photolysis<br>Oxidation<br>Reduction<br>Biodegradation |

### C.6.1 Qualitative Methods

Screening assessments of exposure pathways generally involve identifying potential pathways of exposure. Potential pathways can be ascertained by considering the type of environmental media that is contaminated and certain chemical characteristics of the contaminant. Figures C.2 and C.3 provide flow charts that can aid in determining the possible environmental media of concern for contaminant releases to soil and to surface water. The contaminated media will then direct the determination of the possible pathways of exposure. If transport and fate analysis suggests that exposure is possible via the food chain then uptake by biota needs to be assessed. Figures C.5 and C.6 provide conceptual models useful for assessing exposure in aquatic and terrestrial food chains, respectively.

Potential for bioaccumulation within the food chain can also be evaluated with bioconcentration factors (BCF) or bioaccumulation factors (BAF) directly from the literature or estimated from chemical properties as shown in Table C.9. Generally, data for terrestrial BCF values are much scarcer than for aquatic BCF values. BCF values in the terrestrial environment, when studied, are usually for uptake by plants and earthworms, which are both exposed to concentrations of contaminants rather than doses.

BAFs and BCFs range from less than one to several million. The more bioaccumulative a contaminant is, the more important the consumption of accumulating organisms becomes as a potential source of contaminants to wildlife.

***Bioconcentration*** refers to uptake of a chemical by aquatic organisms exposed only to the chemical in water. This is generally a measurement applied to aquatic organisms although in the terrestrial environment it also applies to plants and some soil organisms. A ***bioconcentration factor*** (BCF) is the ratio between the concentration of the chemical in the organism's tissues and the concentration in the water or soil. BCFs are measured in laboratory experiments and are determined as follows:

$$BCF = \frac{\text{Concentration of chemical in organism in the lab}}{\text{Concentration of chemical in water/ soil in the lab}}$$

***Bioaccumulation*** refers to the uptake of a chemical by aquatic organisms from all sources of exposure: from direct contact (as measured with BCF), diet, and bottom sediments, in the case of aquatic organisms. ***Bioaccumulation factors (BAFs)*** are based on field measurements of tissue and water or soil concentrations. A BAF has the same units as a BCF and is determined as follows:

$$BAF = \frac{\text{Concentration of chemical in organism in the field}}{\text{Concentration of chemical in the water/ soil in the field}}$$

***Biomagnification*** is the concentration of certain substances up a food chain. A very important mechanism in concentrating pesticides and heavy metals in organisms such as fish-eating birds and carnivores.

**Table C.8 Environmental models of chemical fate**

| Type of model | Description | Reference |
|---|---|---|
| **Multimedia models**<br>Fugacity models | Vary in complexity from a simple equilibrium distribution of a conservative chemical to steady-state and time-varying descriptions of the fate of reactive compounds. | Mackay 1979, 1991<br>Mackay and Patterson 1982<br>Mackay et al. 1985 |
| GEOTOX | Calculates chemical partitioning, degrading reactions, and diffusive and nondiffusive interphase transport. Consists of the following compartments: air (gas), air (particles), biomass, upper soil, lower soil, groundwater, surface water, and sediments. Represents environmental conditions in the southeastern U.S.. | McKone and Layton 1986 |
| SCMC (Spatial Multimedia Compartmental Model) | Describes the fate of chemicals in a conventional air-water-soil-sediment system under steady or unsteady state conditions. Allows for concentration variation with depth in the soil and sediment. | Cohen 1989<br>Cohen and Ryan 1985<br>Cohen et al. 1990 |
| Enpart (Environmental Partitioning Model) | Estimates the steady-state, equilibrium or dynamic partitioning of organic chemicals among environmental compartments using fugacity estimates | OECD 1989 |
| Toxscreen | Assesses the potential for environmental transport and accumulation of chemicals released to the air, surface water, or soil through a time-dependent multimedia model | Hetrick and McDowell-Boyer 1983 |
| EEP (Environmental Exposure Potential) | Treats multiple or diffuse sources of continuous emissions through a fugacity-based model. Calculates environmental partitioning, environmental concentration, degradation, and accumulation potential in air, water, and soil by employing weighting factors to produce a "fingerprint" of environmental fate. | Klein et al. 1988 |
| Simplesal | Estimates steady-state or time-dependent concentrations of organic chemicals, as well as heavy metals, using a multimedia fugacity model. Determines dominant environmental pathways and processes for contaminants. | OECD 1989 |
| MNSEM (Multi-Phase Non-Steady State Equilibrium Model) | Predicts the fate of organic chemicals under steady-state conditions of continuous loading to the Japanese environment. | Yoshida et al. 1987 |
| WASP4 (Water Quality Analysis Program) | Integrates predictions from other models (e.g., hydrodynamic and sediment transport models) to estimate contaminant concentrations in the water, sediment, and biota. Provides a consistent modeling framework for eutrophication, contaminant transformation and transport, bioaccumulation, and food chain effects. | Ambrose et al. 1990 |
| **Remediation models**<br>AERIS | Estimates environmental concentrations and subsequently, human exposures in the vicinity of contaminated land sites through a multimedia risk assessment model. | Senes Consultants 1989 |
| RAPS (Remedial Action Priority System) | Considers four major pathways of contaminant migration (groundwater, surface water, overland, and atmospheric) to simulate migration and fate from source to receptor by various pathways. | Whelan et al. 1987 |
| **Aquatic models**<br>Persistence | Estimates the fate or organic chemicals (especially pesticides) that are released into the aquatic environment. Calculates both a steady-state and a time-dependent solution for four compartments: water, catch-all (suspended solids, invertebrates, etc.), sediment, and fish. | Asher et al. 1985<br>Roberts et al. 1981 |

**Table C.8 Continued**

| Type of model | Description | Reference |
|---|---|---|
| EXAMS (Exposure Analysis Modelling System) | Simulates the fate and transport of synthetic organic chemicals in aquatic systems. | Burns et al. 1982 |
| QWASI (Quantitative Water Air Sediment Interaction) | Treats the fate of a chemical discharge to a water-air-sediment system using fugacity values. | Mackay et al. 1983 |
| EXWAT | Describes chemical fate in water bodies through a simple, steady-state model. | OECD 1989 |
| Inorganic chemical models (e.g., metals and phosphorus) | Generalizations are difficult to make for inorganic compounds because the chemical properties and speciation tend to be unique. A variety of models are available. | Bonazountas et al. 1988 |
| Speciation models (e.g. MINTEQA1) | Calculates the equilibrium aqueous speciation, adsorption, gas-phase partitioning, solid phase partitioning, saturation states, and precipitation dissolution of 11 metals. Applies to metallic contaminants in surface and groundwater. | Brown and Allison 1987 |
| Sediment chronology models | Examine the variation of contaminant concentrations with depth of burial. | Eisenreich et al. 1989 (for example) |
| **Soil models**<br>SESOIL (Seasonal Soil Compartment Model)<br><br>PRZM (Pesticide Root Zone Model)<br><br>OESTAN (Pesticide Analytical Model)<br><br>"Jury" Model | Describe chemical fate and persistence in soils, especially of pesticides. | OECD 1989<br><br>Carsel et al. 1984<br><br>Enfield et al. 1982<br><br>Jury et al. 1983 |
| **Fish uptake and food chain models**<br>FGETS (Food and Gill Exchange of Toxic Substances) | Simulates the bioaccumulation on non-metabolized organic chemicals in fish. Depends on the physical and chemical properties of the chemicals and on the ecological, morphological, and physiological characteristics of the fish. | Barber et al. 1988<br>Burns 1991 |
| Bioaccumulation model | Predicts chemical concentrations in biota for given chemical concentrations in water sediment. Uses an age-class model for hydrophobic organic chemical bioaccumulation in aquatic food chains. | Thomann and Connolly 1984 |

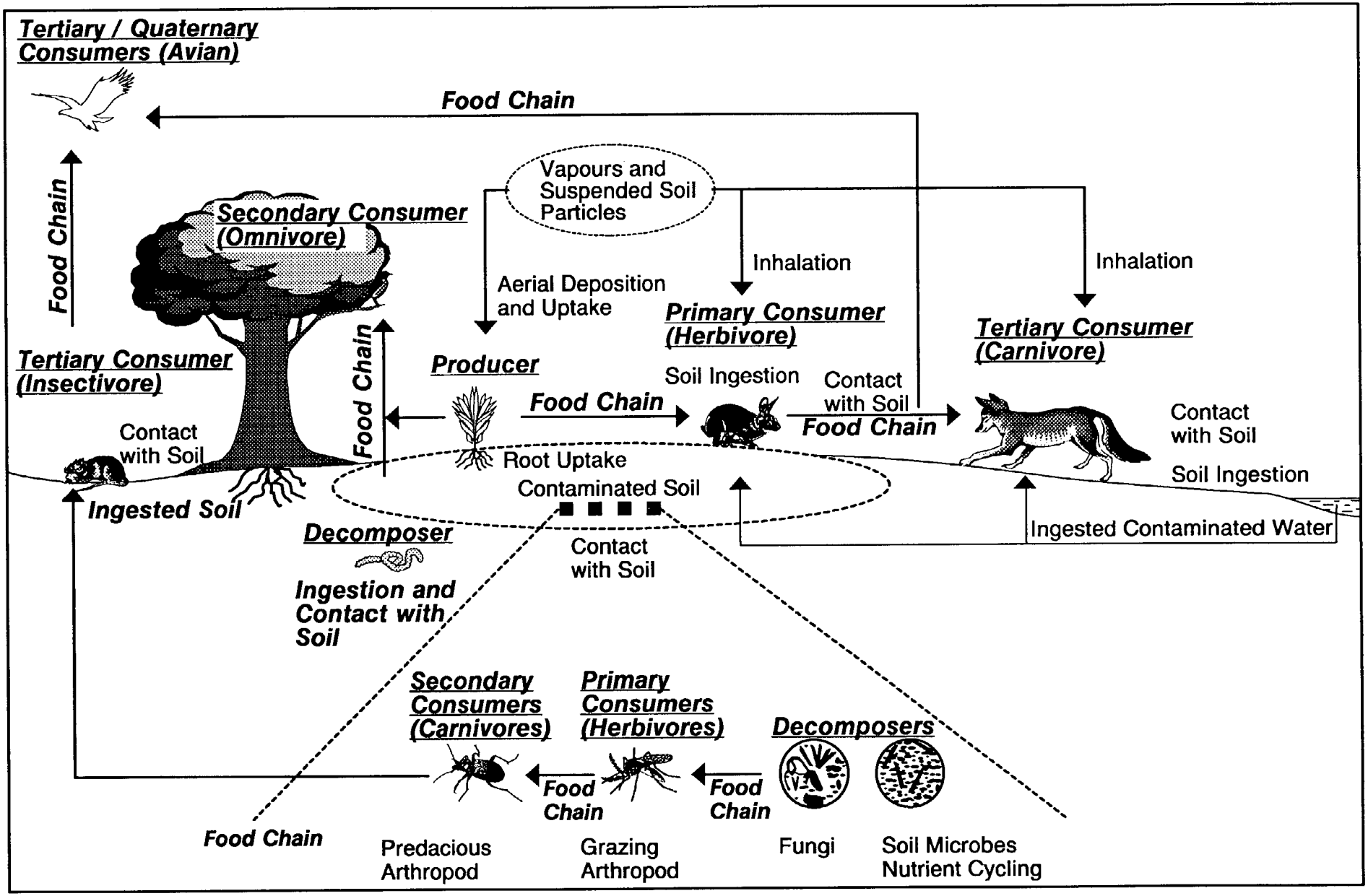


Figure C.4 Simplified diagram of potential ecological receptors and exposure pathways of contaminated soil (CCME 1996).

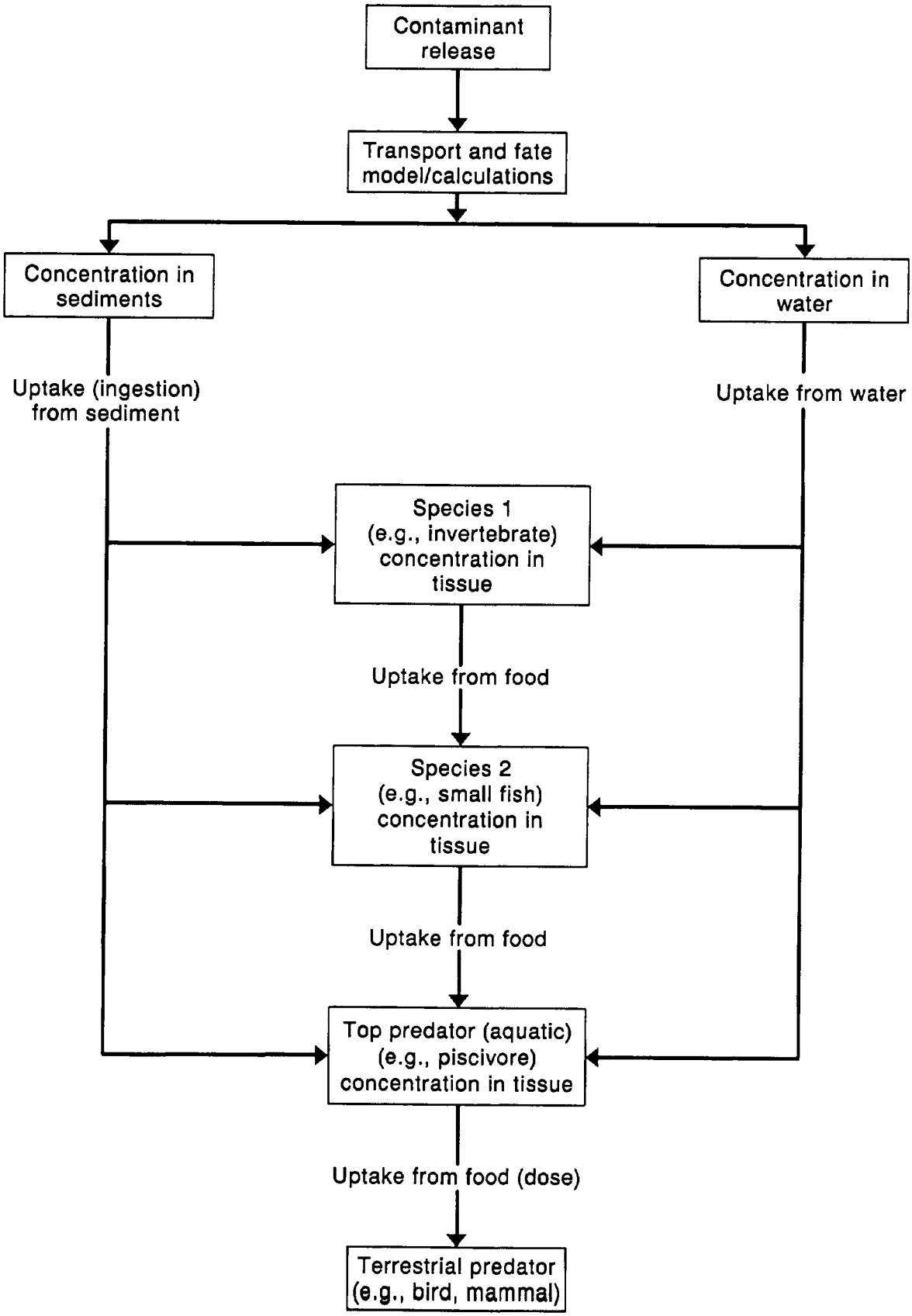


Figure C.5 Exposure in aquatic food chains.

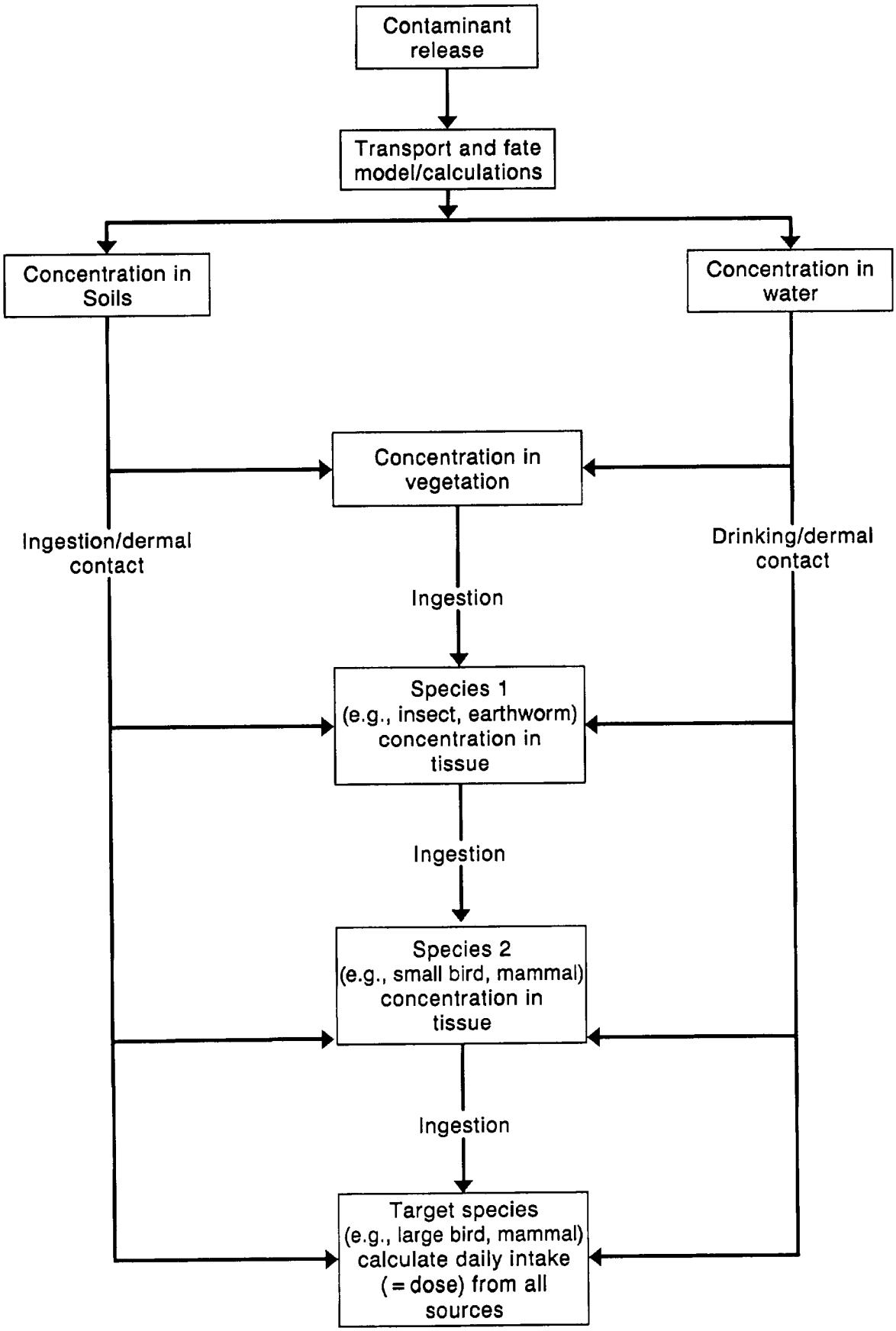


Figure C.6 Exposure in terrestrial food chains.

**Table C.9 Methods of estimating bioconcentration factors**

| Estimation methods |
|---|
| log BCF = 0.76 log Kow - 0.23<br>log BCF = 2.791 - 0.564 log (water solubility)<br>log BCF = 1.119 log Koc - 1.579 |

Source: compiled from Howard and Boethling 1993

Two properties of a chemical that are responsible for high bioaccumulation ratios are

- high partition coefficient, as in lipophilic compounds
- recalcitrance toward all types of degradation

The estimation methods above may be useful for dealing with organic chemicals since bioconcentration is related to a chemical's hydrophobicity. BAFs or BCFs for inorganic chemicals must be measured in the field or the lab.

## C.6.2 Quantitative Methods

*Additional reading*

U.S. EPA (United States Environmental Protection Agency). 1993. Wildlife exposure handbook Vol. 1. Office of Research and Development, Washington DC. EPA/600/R-93/187a. *Provides exposure profiles for selected species, allometric models that can be used to estimate food and water ingestion rates, inhalation rate, surface areas and metabolic rates for wildlife species, and common equations used to estimate wildlife exposure to environmental contaminants.*

U.S. EPA (United States Environmental Protection Agency). 1993. Wildlife exposure handbook. Vol. 2. Appendix: Literature review database. Office of Research and Development, Washington, DC. EPA/600/R-93/187b. *Provides quantitative data for selected species, such as normalizing and contact rate factors, dietary composition, population dynamics, and seasonal activities, to be used in conjunction with Vol. 1.*

U.S. EPA (United States Environmental Protection Agency). 1992. Dermal exposure assessment: principles and applications, interim report. Office of Research and Development, Washington, DC. EPA/600/8-91/001B.

### *C.6.2.1 Preliminary Quantitative Analyses*

Aquatic food chain exposure

Literature values and estimates of BCFs can be a significant source of uncertainty as they do not take into account the influence of site parameters such as temperature, pH, and salinity on the dissolution/bioavailability of the contaminant in water or the influence of organism characteristics such as lipid content, behavior, and ingestion rates that affect contaminant uptake.

Further discrepancies between BCF estimates and laboratory data can occur due to

- errors in physical/chemical measurements, especially for highly soluble chemicals.
- the inability to reach equilibrium experimentally; water insoluble chemicals take a long time and chemicals with log BCF > 6 require more than 20–30 d
- restricted partitioning across membranes if log $K_{ow}$ is above 6
- metabolism of the chemical in the lab studies
- binding of the chemical to dissolved organic matter
- the varying lipid content of aquatic organisms, which can result in different BCFs

Though models have been constructed for particular contaminants and organisms (e.g., Gobas 1993), there are few generalized simple approaches. The most common simple approach (U.S. EPA 1991) is the use of field-measured BAFs based on simultaneous monitoring of water and tissue concentrations or the determination of uptake and depuration rates.

Terrestrial food chain exposure

In terrestrial systems, exposure is generally expressed as dose, or the daily intake of contaminant. Simplified equations and the use of some default values can provide an estimate of exposure for the ingestion exposure pathway which will be the most significant. The proportion of the total exposure attributed to ingestion is the product of the dry matter intake rate (DMIR) and contaminant concentration in that medium. An apportionment factor (AF) of 75% to account for contributions of dry matter intake to the total exposure has been recommended by Walker and MacDonald (1992) in the calculation of tissue residue guidelines for the protection of wildlife consumers of aquatic life.

**Table C.10 Examples of estimates of food and soil ingestion exposures for wildlife**

| Exposure | Equation |
|---|---|
| Estimating the rate of soil ingestion | $SIR = DMIR \times PSI$<br><br>*SIR* = the soil ingestion rate (kg **dw** soil/d)<br>*DMIR* = the dry matter intake rate (kg/d) - *assumes that the DMIR contains only dry matter as food or soil*<br>*PSI* = soil ingestion proportions - *default value of 0.07* |
| Estimating the rate of food ingestion<br><br>If no DMIR information is available, then allometric equations can be used to estimate the FIR (Nagy 1987).<br><br>Allometric equation for mammalian species<br><br><br>Allometric equation for avian species | $FIR = DMIR - SIR$<br><br>*FIR* = the food ingestion rate for the species (kg dw food/d)<br>*DMIR* = dry matter intake rate for the species (kg/d)<br>*SIR* = the soil ingestion rate (kg dw soil/d)<br><br>$F_M = 0.0687 \times (BW)^{0.822}$<br><br>$F_M$ = feeding rate of mammalian species (kg dw food/d)<br>BW = mean body weight of species (kg)<br><br>$F_A = 0.0582 \times (BW)^{0.651}$<br><br>$F_A$ = feeding rate of avian species (kg dw food/d)<br>BW = mean body weight of species (kg) |

Source: CCME 1996

Dry matter is assumed to include only food and soil, and exposures to both of these media can be calculated. To estimate the exposure from soil ingestion only, the percentage of the DMIR attributed to soil must be ascertained. Table C.10 provides examples of simple equations to estimate food and ingested soil exposures.

*C.6.2.2 Detailed Quantitative Analyses*

Food Chain Models

Exposure to both aquatic and terrestrial receptors through the food chain can be estimated with a food web model. Specifying a food chain for model analyses of ecological risk is a compromise between reality and the available data and understanding. Fordham and Reagan (1991) provide the following principles:

- By organizing species with similar feeding habits into groups of key species, bioaccumulation by key species represents bioaccumulation by other organisms in that feeding group.
- By selecting the most sensitive organisms or organisms most likely to accumulate larger levels of contaminants as sink species, results in a conservative approach for developing criteria for bioaccumulative contaminants.
- By using a conservative approach, other less sensitive populations should also be protected.

The simplest models of bioaccumulation in food chains rely on five variables at each trophic level (Fordham and Reagan 1991):

- the concentration of contaminants in prey organisms
- the assimilation efficiency (µg contaminant absorbed/µg contaminant ingested)
- the total daily diet (g food/g body weight/d)
- the depuration or loss rate (per day)
- the fraction of the organism's diet made up by each prey organism

Using this method, one can build up as many trophic levels as necessary, given reliable parameters for each layer.

Aquatic food chain exposure

*Additional reading*

Gobas, F.A.P.C., and D. Mackay. 1987. Dynamics of organic chemical bioconcentration in fish. Environ. Toxicol. Chem. 6:495–504.

Thomann, R.V. 1989. Bioaccumulation model of organic chemical distribution in aquatic food chains. Environ. Sci. Technol. 23:699–707.

Modelling bioaccumulation in aquatic food chains generally begins with planktonic and benthic organisms, or macrophytes, typically using simple approaches. These include the use of BAF, simple pharmacokinetic models (Gobas et al. 1991), or an assumed equilibrium between contaminant concentrations in the organism (e.g., lipid tissue of benthic organisms) and the environment (e.g., contaminant concentrations in sediment organic matter). Assuming that the contaminant concentrations in these benthic/planktonic groups can be estimated or directly measured, the next step is to estimate the average dietary composition of each fish species of interest. This involves specifying the proportions of each benthic/planktonic group in fish diets, by season if necessary.

A considerable body of theory and empirical evidence is available for predicting contaminant bioaccumulation in fish, using pharmacokinetic models (Gobas and Mackay 1987; Thomann 1989). A typical modelling approach for assessing bioaccumulation of hydrophobic contaminants in lakes is that of Gobas (1993). In this model, the change in the fish's contaminant concentration over time is represented by:

$$\frac{dC_F}{dt} = k_1 C_{WD} - k_2 C_D + k_D C_D - k_E C_F - k_G C_F - k_M C_F$$

Eq. C.1

where

$k_1$ is the rate of water uptake through the gill (L/kg/d)

$k_2$ is the rate of elimination via the gills to the water (1/d)

$k_D$ is the rate of food consumption ((kg food/kg fish/d)

$k_E$ is the rate of elimination (1/d)

$k_G$ is the growth rate (/d)

$k_M$ is the rate of metabolic breakdown of the contaminant, which is set to zero for persistent contaminants (1/d)

$C_{WD}$ is the biologically available contaminant concentration in the water (µg/L)

$C_F$ is the contaminant concentration in the fish (µg/kg fish)

$C_D$ is the average contaminant concentration in the fish's diet (µg/kg), calculated from a food-fraction-weighted average of the contaminant concentrations in diet organisms

At steady state this simplifies to:

$$C_F = \frac{(k_1 C_{WD} + k_D C_D)}{(k_2 + k_E + k_G + k_M)}$$

Eq. C.2

or

$$C_F = BCF \times C_{WD} + BMF \times C_D$$

Eq. C.3

where

*BCF* is the bioconcentration factor [ $k_1/(k_2+k_E+k_M+k_G)$ ]

*BMF* is the biomagnification factor [ $k_D/(k_2+k_E+k_M+k_G)$]

Each of the parameters in Equations C.1 and C.2 are derived from empirical equations which hold for many different species, and are related only to a few simple inputs; the mass of the fish, its growth rate and diet preferences, water temperature, and the $K_{ow}$ (octanol-water partition coefficient) of the contaminant (Gobas 1993). This makes these relationships generally applicable.

Once fish contaminant concentrations are estimated, the process is repeated for piscivorous birds and mammals. Though the theory and models of contaminant uptake are not so well developed for these groups, the problem is somewhat simpler in that only biomagnification, and not bioconcentration, needs to be considered. Clark et al. (1988) provide an example of a modelling approach for estimating contaminant concentrations in herring gulls. Monitoring the birds' eggs for contaminants is often the most convenient method to calibrate these models; this assumes that the selected species is sufficiently abundant that sampling will not have a major ecological impact.

***Additional reading***

Pastorok, R.J., and J.R. Sampson. 1990. Review of ecological risk assessment methods to develop numerical criteria for cleanup of hazardous waste sites. Draft. Prepared for Washington Department of Ecology, Olympia, WA. *Provides a food chain model designed for assessing risks from atmospheric emissions, but which could be adapted for exposure via other emissions*

Menzie, C.A., D.E. Burmaster, J.S. Freshman, and C.A. Callahan. 1992. Assessment of methods for estimating ecological risk in the terrestrial component: a case study at the Baird and McGuire superfund site in Holbrook, Massachusetts. Environ. Toxicol. Chem. 11:245–260. *Compares modelling, bioassay, and field methods with regard to assessing conditions and risks to terrestrial biota at a Superfund site contaminated with pesticides.*

Detailed models of contaminant uptake by terrestrial species are rare, and those that do exist have generally been adapted from human exposure models. Table C.11 shows examples of oral exposures for water, diet, and soil or sediment ingestion by wildlife. An uptake model must estimate uptake from most or all exposure routes and therefore requires

- estimates of concentrations in food/water/soil/air. The concentrations in food can be estimated by the model, as food items are generally the species in the food chain. However, these concentrations can also be measured directly in common plants or animals at the base of the food chain

- metabolic parameters (e.g., ingestion rates, clearance rates, and contaminant absorption and depuration rates control the fate of the contaminant in the organism and contaminant transfer between trophic levels)

- behaviour (e.g., food habits or preferences, movement/migration/dispersal, and potential avoidance behaviour can determine the potential for uptake by different routes)

## C.7 UNCERTAINTY ANALYSIS

Sources of uncertainty associated with modelling in ecological risk assessment can include variablility in input parameters (due to spatial variation in parameters and/or lack of data for key parameters), the structure of the model because of simplification and assumptions within the model.

There are several approaches for dealing with these sources of uncertainty. Three common methods are sensitivity analyses, Monte Carlo simulations, and the use of monitoring data for model calibration.

Qualitative and quantitative sensitivity analyses are very important to give the modeler a good understanding of the mathematical sensitivity of his/her model. Sensitivity analysis is the assessment of which parameters of a model have the highest input parameter variation to output parameter variation ratios and which have the lowest. The input parameters that have the greatest effect on the accuracy of the output parameters should have high accuracy. Conversely, the input parameters that have only a small effect on the accuracy of the output parameters can be estimated by less accurate and costly methods.

With Monte Carlo analyses, a model is run many times with varying input parameters. Uncertain input parameters can be expressed as distributions rather than fixed values, and can be used to assess the effects of input variable uncertainty. Some data are required to specify the input parameter distributions; otherwise the uncertainty in outputs is purely a function of the assumptions made about the uncertainty of input parameter distributions. When using Monte Carlo analyses one must be careful to consider the correlation among parameters; assuming that all parameter distributions are independent will overestimate the level of uncertainty. Fordham and Reagan (1991) provide an excellent example of the application of Monte Carlo analyses to an ERA at a hazardous waste site.

Monitoring data are invaluable for reducing uncertainty, through model calibration. Biases in model output can also be corrected with monitoring data. However, monitoring data are more useful for reducing

**Table C.11 Examples of estimates of oral exposures for water, diet, and soil or sediment ingestion**

| Exposure | Wildlife dose equations |
|---|---|
| Drinking water<br>• One source of contamination | $ADD_{pot} = C \times FR \times NIR$ |
| • Different sources with varying levels of contamination | $ADD_{pot} = \sum_{i=1}^{n} (C_i \times FR_i) \times NIR$<br>$ADD_{pot}$ = Potential average daily dose (mg/kg)<br>$C$ = Average contaminant concentration in a single water source (mg/L or mg/kg)<br>$FR$ = Fraction of total water ingestion from the contaminated water source (unitless)<br>$NIR$ = Normalized water ingestion rate (fraction of body weight consumed as water per unit time, g/g d)<br>$C_i$ = Average contaminant concentration in the $i$th water source (mg/L)<br>$FR_i$ = Fraction of water consumed from the ith water source (unitless)<br>$n$ = Number of contaminated water sources |
| Dietary exposure<br>• Generic equation for estimating oral doses in contaminants in food for wildlife species | $ADD_{pot} = \sum_{k=1}^{m} (C_k \times FR_k \times NIR_k)$<br>$ADD_{pot}$ = Potential average daily dose (mg/kg)<br>$C_k$ = Average contaminant concentration in the $k$th type of food (mg/kg ww)<br>$FR_k$ = Fraction of the intake of the $k$th food type that is contaminated (unitless)<br>$NIR_k$ = Normalized ingestion rate of the $k$th food type on a ww basis (g/g day)<br>$m$ = Number of contaminated food types |
| Soil or sediment ingestion | $ADD_{pot} = [\sum_{k=1}^{m} (C_k \times FS \times IR_{total} \times FR_k)]/BW$<br>$ADD_{pot}$ = Potential average daily dose (mg/kg)<br>$C_k$ = Average contaminant concentration in soil in the $k$th foraging area (mg/kg dw)<br>$FS$ = Fraction of soil in diet (unitless)<br>$IR_{total}$ = Food ingestion rate on a dw basis (kg/day)<br>$FR_k$ = Fraction of total food intake from the $k$th foraging area (unitless)<br>$BW$ = Body weight (kg)<br>$m$ = Total number of foraging areas |

Source: U.S. EPA 1993

uncertainty in air and surface water modelling than for groundwater models, because of the time lags in groundwater movement. A groundwater model's predictions of future changes in water quality may be correct, but the contaminant plume may not have reached the point of sampling.

## C.8 CONCLUSIONS

The use of successively more sophisticated approaches helps to focus on the critical processes and thereby reduce the uncertainty (and expense) of the overall exposure assessment. Decisions regarding the level of detail of exposure assessments should be made in concert with analogous decisions for receptor and hazard assessments. The levels of precision of different components of an ERA should be more or less congruent. There is no point in having a very detailed quantitative model for exposures if the dose-response relationships used for the hazard assessment have enormous uncertainty. Finally, the modelling of exposure is an evolving science; it is very important that analysts keep abreast of current progress to select the most appropriate approach for the particular contaminants and site of concern.

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# Hazard Assessment

## D.1 DEFINITION

Hazard assessment describes the relationship between the contaminant(s) of concern and the most important ecological endpoints. Within the context of ecological risk assessment (ERA), hazard assessment is usually accomplished by the measurement of toxicity of a substance to one or more species, through toxicity testing, and in some cases augmented with higher level of organization measurements.

## D.2 LEVELS OF ORGANIZATION

Levels of organization of hazard assessment can be categorized as individual, population, and community measurements. Within each of these, there are various levels of stress response, with different time spans and significance. For example, in Figure D.1 the neuroendocrine changes are the most reversible and have the least diagnostic potential of any of the individual level measures; growth or survival are less reversible and more significant.

### D.2.1 Biochemical Responses/Biomarkers

***Biomarkers*** are indicators of exposure to a contaminant that are expressed at a biochemical or cellular level. Most biochemical responses demonstrate exposure, not effects. Although cellular and biochemical responses are the lowest level at which contaminant effects can be detected, these responses are also the most reversible and the least likely to exert effects at the community level. Biomarkers are more useful to look at the mechanisms of toxicity than as indicators of toxicity. They can also serve as an exposure assessment tool. Examples of potential physiological/biochemical endpoints are provided in Table D.1.

Some biochemical responses can provide direct information on contaminant-induced changes while others provide indirect information describing physiological status or nonspecific responses to foreign chemicals.

*For example*
direct responses — the induction of metallothionein or metallothionein-like proteins or changes in blood enzymes related to specific contaminant exposures.

indirect responses — changes in adenylate energy charge, decreases in haematocrit, leucocrit and mean corpuscular volume, or increases in haemoglobin concentration

Criteria for selecting useful biochemical responses in pollution studies are (Widdows 1985)

- they should be sensitive to environmental stress and pollution and have a large scope for response throughout the range from optimal to lethal conditions
- they should reflect a quantitative or otherwise predictable relationship with the toxicant
- they should have a relatively short response time, on the order of hours to weeks, so that the toxicant impact may be detected in its incipient stages
- they should represent nonspecific (general) responses to the sum of environmental stimuli, thus providing measurements of the overall impact of environmental change and complementing the more contaminant-specific responses at the cellular level
- they should be measurable with precision and with a high "signal to noise" ratio so that the effect of pollution may be detected above the "noise" of general variability
- they should have ecological relevance and be shown to be related to adverse or damaging effects on the population

Perhaps the greatest potential weakness in the application of physiological techniques in biological effects monitoring concerns their variability (Bayne 1985). Variability may be attributable to a range of sources such as seasonality, reproductive status, and test conditions. Variability among individuals is not well studied.

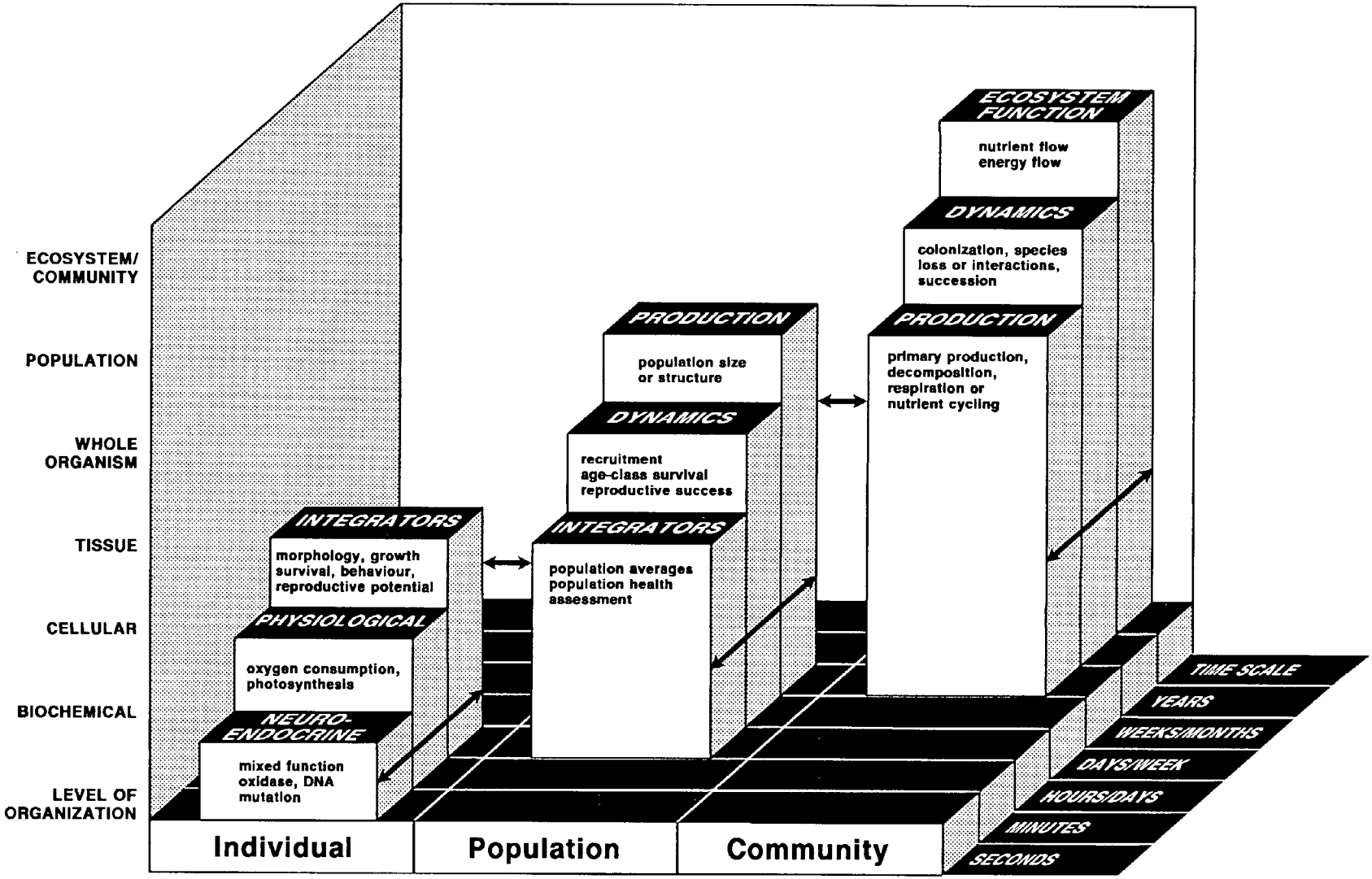


Figure D.1 Hazard assessment components: lowest impact levels, represented by the smallest boxes, respond to a low level of stress, have the fastest response time, the most reversibility, best early warning potential, and worst diagnostic potential. The highest impact levels, represented by the largest boxes respond to the largest amount of stress, have the least reversibility, the longest response time, the best diagnostic potential and the lowest early warning potential. Arrows indicate direction of integration and prediction. (Munkittrick 1988)

**Table D.1 Examples of potential endpoints for hazard assessment**

| Response level | Description | Parameters | Specific examples (where applicable) |
|---|---|---|---|
| Physiological responses | Primary metabolic impact | Enzyme activities | Mixed function oxidase induction |
| | | Respiration | |
| | | Photosynthesis | |
| | | Enzyme activities | |
| | | Excretion | |
| | Primary metabolic responses | Metabolic rate | Adenylate energy charge |
| | | Hematology | Hematocrit, leukocrit, hemoglobin |
| | | Pigmentation | |
| | | Osmoregulation | |
| | | Ionoregulation | |
| | | Hormonal changes | Changes in estradiol, testosterone |
| Individual responses | Survival | | $LC_{50}$, $LD_{50}$, NOEL |
| | Growth | Feeding rate/nutrition | |
| | | Scope for growth | |
| | | Net growth efficiency | |
| | | Body/organ weights | Liver and spleen changes |
| | | Developmental rate/stages | Changes in sexual maturation |
| | Reproduction | Sexual maturation | |
| | | Gamete viability/fertility | |
| | | Larval development | |
| | | Brood size/fecundity | NOEC |
| | | Frequency of reproduction | |
| | Behaviour | Sensory capacity | |
| | | Rhythmic activities | Ventilatory/cough response |
| | | Motor activity | Burrowing |
| | | Learning/motivation | |
| | | Avoidance/attraction | |
| | | Reproductive behavior | |
| | Histopathology | Abnormal growths | |
| | | Abnormal histological changes | Neoplasms/tumors, tissue somatic indices |
| Population, community, and ecosystem Responses | Behaviour | Recolonization/migration | |
| | | Aggression/predation | |
| | | Mating | |
| | Population responses | Age-class survival | |
| | | Extinction | |
| | | Reproductive success | |
| | | Density/abundance | |
| | | Biomass | |
| | | Productive capability | |
| | Community responses | Diversity | |
| | | Pollution indices | |
| | | Species richness | |
| | | Succession | |
| | | Nutrient cycling | |
| | | Energy flow | |
| | | Enzyme activity | Microbial communities |
| | | Oxygen consumption/respiration | Microbial communities |

Source: Adapted from Power et al. 1991

### D.2.2 Individual Responses

The individual level of biological organization is a reasonable compromise in sensitivity and ecological interpretation, relative to the biochemical/cellular level and the population and community levels (Fig. D.1). Individual responses may affect the success of the population, which in turn may cause effects at the community and ecosystem levels.

*Examples of individual responses*

- survival
- growth
- reproduction
- behaviour
- histopathy

#### Survival

Mortality at the individual level can be described as direct (acute) or delayed (chronic). In the field of toxicology the term *survival* has the connotation of acute lethality during a short-term toxicity test, and it is widely recognized that substrates that are not acutely toxic may exert chronic toxicity. The most useful information on site impacts would be field data on survival of individuals residing in a contaminated habitat over an extended time period. However, without marking individuals in a population, it is difficult to measure individual survival rates; therefore, the solution has typically been to measure survival in the laboratory in short-term experiments.

#### Growth

Growth is a fundamental component of fitness and therefore an important index of contaminant effects. Toxicants can affect growth rates indirectly by reducing the food available and directly by impairing metabolic pathways that convert food energy to tissue or by diverting energy from growth to metabolism of the contaminant. The consequences of reduced growth include reduced fecundity, slower maturation, and a reduced ability to compete with other individuals; these have population and community level repercussions.

The growth endpoint is most easily measured in aquatic systems and is not appropriate in systems where populations have a distribution greater than the study area (e.g., birds, mammals). In aquatic systems, growth can be measured in either laboratory or field experiments. Initial investigations should focus on laboratory investigations, as they will indicate the potential for growth effects in the field.

Effects on growth (and reproduction) can best be understood by considering the energy budget of an animal (Widdows 1985), which may be expressed as the *scope for growth* or *net growth efficiency*.

*For example*

The ***scope for growth*** is estimated from the difference between the energy absorbed and the energy expended through respiration and excretion (Widdows 1985). Scope for growth can range from positive values (when there is energy available for growth and the production of gametes) to negative values when the organism is utilizing its body reserves for maintenance metabolism. The scope for growth offers an instantaneous view of sublethal effects that if extended over a period of time would result in death.

***Net growth efficiency*** describes the efficiency with which food is converted into body tissue and is a measure of the energy available for growth, as a proportion of the energy absorbed from the food. A reduction in this value is indicative of a stressed condition, since a greater proportion of the energy absorbed from the food is being used to maintain the animal and consequently a smaller proportion is available for growth. Net growth efficiency values provide a long-term integration of physiological processes.

#### Reproduction

Contaminants can affect reproductive processes in several ways; altering the availability of energy, reproductive behaviour, and reproductive performance and causing metabolic disruption of factors affecting reproductive control. Energy allocation can be affected by decreasing the amount of energy available for reproduction through food limitation or through utilizing energy reserves for dealing with contaminant burdens.

For *fish*, toxicological experiments on reproduction of species with a short life span have been described as the most productive for useful results (Sprague, 1976). This parameter is of ecological importance because it has a direct influence on recruitment and the maintenance of a population. For birds, phenomena such as eggshell thinning have been related to contaminant exposure. Contaminants may also affect the developing embryo in the avian egg. For *invertebrates*, similar perturbations in reproductive processes occur, but less work has been done on the response physiology/biochemistry.

*Examples of reproduction endpoints*

- delays in sexual maturation
- delays in brood release
- egg development time
- brood size
- frequency of reproduction
- complete inhibition of reproduction

Most of the work in this area is toxicity-test oriented, with the endpoints being measures of reproductive processes or success. The repercussions of these reproductive effects are seen at the population and community levels which integrate all of the processes discussed here.

**Behaviour**

Organisms can and do respond to contaminants by altering their behaviour. Basic behavioural patterns such as locomotion and orientation are essential to processes such as prey capture, feeding, predator avoidance movement, migration, courtship, and mating. The integration of these behaviours will, in part, determine the success of each individual and of the population. Behavioural responses to contaminants include a wide range of behaviours such as avoidance, inhibited feeding, and increased random movement.

*Examples of behavioural endpoints*

- spatial selection
- response to food and feeding ability
- predator–prey responses
- aggression, displays
- reproductive behaviours
- feeding response
- ventilatory and cough responses
- preference or avoidance to a variety of stimuli

**Histopathology**

Histopathological effects such as lesions, neoplasms, and tumours in field populations of individuals exposed to contaminants can be used to document effects of contaminants. The presence/absence and numbers of such features have been related to contaminant exposure; however, little information is available on the ecological significance of such growths. For contaminated site assessment, information about histopathology could be collected during field studies, but it should not be a focal endpoint, except in cases where carcinogenicity of the contaminant(s) is suspected.

### D.2.3 Population, Community, and Ecosystem Responses

Evaluation of hazard at the population level and higher requires field assessment. Selection of the optimal level of organization depends on information such as background data, results of toxicity testing, and the specific issues at the contaminated site.

**Population Responses**

Individual level changes related to contaminants (e.g., growth, reproduction) can result in changes in the overall characteristics of populations. These changes are characteristically not easily reversible over a short time span, and if damage is discernable at the community level, the probability for need of remediation of the contaminated site increases. Also, population-level effects of contaminants are considered to be of concern to society, because value is placed on the population level of biological organization (e.g., commercial fisheries, food species, local extinctions).

Some researchers have found that population indicators are more sensitive than individual level measurements, and that population growth may integrate the other parameters as a sensitive indicator of impact. Presence or absence of species in habitats affected by a contaminated site can be used to infer changes associated with the site, particularly where historic data are available on the species' abundance.

Populations change in size through a combination of birth, death, immigration, and/or emigration. Contaminants can affect populations by affecting any of these four processes. Most obvious are decreases in population size related to mortality (e.g., from exposure to lethal concentrations of toxicants, from decreased birth rates, from reduced food supply). Population assessment can be used to field-verify toxicity test data. Evidence linking population decreases with pollutant toxicity in the case of a contaminated site might not be obvious, due to the extended time frame over which adverse changes have occurred (Sheehan 1984); continual gradual contaminant input can lead to slow, gradual changes in population health. Distinguishing pollutant-induced changes from those caused by natural environmental or non-contaminant related anthropogenic factors requires extensive baseline data.

Population dynamics such as recruitment, age-class survival, and reproductive success can be used to characterize population health; however, it is also difficult to ascertain cause and effect in many cases. This level of effort should only be expended if other testing indicates

there is cause for concern, and the evaluation must be carefully designed to screen out unrelated influences. For example, populations may fluctuate in size for reasons completely unrelated to toxicants (e.g., seasonality, competition, food supply).

**Community Responses**

Population interactions, as influenced by contaminants, will affect the dynamics of the exposed communities. Communities fluctuate in their species composition and the relative abundance of each species, and these fluctuations are affected by processes not thoroughly understood. In the absence of a major disruption, a given community can be expected to vary within certain boundaries. Contaminants introduced into the environment significantly affect an exposed community when they create new boundaries. Changes at the community level are difficult to reverse, are only expressed after a considerable time, and allow little opportunity to trace cause and effect. Community-level assessments may take place through field investigations (direct measurement) or through surrogates (e.g., community modelling, microcosms).

*For example*

Some species may decline in abundance, causing others to become more dominant than usual. This will alter the community dynamics and may have effects at the ecosystem level. Professional expertise is required to interpret patterns of species composition and abundance in communities. Such interpretation may be aided by comparisons of contaminated site data to appropriate reference site data.

**Ecosystem Responses**

Although the ecosystem is often the level of biological organization that society wishes to preserve, ecosystem assessment methods are not usually at the level where they can play a significant role in hazard assessment. Ecosystem health is not readily definable or measurable and the ability to determine stability or degradation is complicated by the natural, unknown dynamics of ecosystem processes.

## D.3 QUALITATIVE METHODS

***Hazard identification*** is the first step of hazard assessment and follows from the planning phase of the ERA. Hazard identification qualitatively evaluates the relationship between a stressor and adverse biological effects. Ecological components affected, or potentially affected, by the contaminated site are identified in the receptor characterization. This information is used to select the best method for the hazard assessment. The objective is to link the contaminant (or mixture of contaminants) to the biological response(s). All existing site data should be reviewed with this objective in mind. Literature reviews, scientific publications, and useful sources of information on the toxicity of specific contaminants help guide an investigation to identify the likely mechanisms of toxicity. Literature information is useful for qualitative assessments. Hazard assessment data collected for a specific contaminated site are useful for semiquantitative and quantitative assessments.

## D.4 QUANTITATIVE METHODS

The decision whether to proceed and how best to proceed from a preliminary quantitative hazard assessment to a detailed hazard assessment is based on the data collected up to that point. Studies from the preliminary quantitative hazard assessment may indicate that alternate measurement endpoints are necessary and/or indicate a need to focus on a more specific component.

*For example*

At a contaminated site where leachate drains into a small stream with salmonid fish, the first level may involve collecting the leachate and testing a salmonid species for acute toxicity. If the short-term tests indicate that the fish survive but show behavioural stress responses (e.g., erratic swimming, disequilibrium), the next level may involve a test that looks at behavioural responses as potentially more sensitive measurement endpoints. Alternatively, if severe effects are documented in the first level, there may be no need for further testing to document the problem at the contaminated site.

Initial assessments might involve a recreational fish population survey to determine population health in a potentially impacted stream near a contaminated site versus a reference area. If the survey finds that there are no differences in fish abundance but that the fish downstream of the contaminated site have reduced biomass, the next level of investigation might involve looking at the availability of food supply to the fish. Invertebrate toxicity tests conducted at leachate concentrations similar to those in the field could also be conducted.

The biological level of organization in hazard assessment is usually at the individual level or at the

**Test Battery Approach**

Traditional toxicological investigations have relied heavily on single-species tests, because they provide useful information on dose-response relationships. However, it is extremely difficult to extrapolate population-level effects from individual effects, and single-species toxicity testing is not necessarily protective of ecosystems. No single toxicity test can be used to detect ecosystem impacts, due to the varying target sites and factors that influence sensitivity and differing temporal response times of ecosystem components.

A *toxicity test battery* or *suite of toxicity tests* is preferred, because species sensitivity to toxicants varies between different levels of organization, modes of action, metabolic processes, etc. In general, toxicity tests are chosen for use in a test battery to offer a range of taxa, endpoints, exposure routes, and time spans. The report entitled "A review of whole organism bioassays for assessing the quality of soil, freshwater sediment and freshwater in Canada" (Keddy et al. 1994) recommends a specific battery of tests for each media.

**Toxicity Test Data Analysis and Interpretation**

*Additional reading*

Stevens, D.L., G. Linder, W. Warren-Hicks. 1989. Section 9.0: Data interpretation. *In* Ecological assessment of hazardous sites: A field and laboratory reference, ed. W. Warren-Hicks, B.R. Parkhurst, and S.S. Baker. U.S. Environmental Protection Agency, Environmental Research Laboratory, Office of Research and Development, Corvallis, OR. EPA/600/3-89/013.

The importance of correct data analysis and interpretation cannot be overemphasized and will require separate guidance for national uniformity. In the NCSRP framework, the final products of toxicity testing under hazard assessment are the results of each toxicity test. These data can be used directly in the risk characterization or can be extrapolated to the organisms of concern. Application of safety factors and consideration of risk is discussed under risk characterization in Appendix E. Toxicity data for single chemicals must be used with caution because the contaminants of concern at contaminated sites are often mixtures of chemicals. Since toxicity tests are usually conducted for single chemicals, there are few data for chemical mixtures. When organisms are exposed to two or more chemicals at a time, the effects may be directly additive, synergistic, or antagonistic, depending on the toxicants, the test organisms, and the testing environment. Toxicity testing for contaminated sites involves evaluation of substrates (water, soil, sediment) that likely contain a number of contaminants, and identification of the chemicals of primary concern is not always possible.

**Toxicity Test Data Analysis and Interpretation**

Greene et al. (1989) and Stevens et al. (1989) describe data analysis techniques for toxicity test data and use of toxicity test results. Within this ERA framework, the final products of toxicity testing under hazard assessment are the results of each toxicity test. These data can be used directly in the risk characterization, or can be extrapolated to the organisms of concern.

Toxicity data for single chemicals must be used with caution because the contaminants of concern at contaminated sites are often mixtures of chemicals. Since toxicity tests are usually conducted for single chemicals, there are few data for chemical mixtures. When organisms are exposed to two or more chemicals at a time, the effects may be directly additive, synergistic (more than additive) or antagonistic (less than additive), depending on the toxicants, the test organisms, and the testing environment. Toxicity testing for contaminated sites involves evaluation of substrates (water, soil, sediment), which likely contain a number of contaminants, and identification of the chemicals of primary concern is not always possible.

**Modifying Factors**

***Modifying factors*** are defined as any characteristics of an organism or the surrounding water that affect toxicity, and are usually divided into two descriptive groupings, biotic (intrinsic) and abiotic (extrinsic). Modifying factors can act to either increase or decrease the concentration of a chemical required to produce a biological response, and the impact can vary dramatically between classes of chemicals and the organisms that are exposed. A biological response is detectable when the chemical reaches a sufficient concentration at the target site to affect the measurable performance of the organism. Threshold concentrations vary between chemicals and organisms, and modifying

factors alter the rate at which chemicals reach the target site by changing the availability of the chemical to the organism, or the internal transport rate at which the chemical reaches the target site. The target site can vary with the concentration of chemical affecting the organism.

Both abiotic and biotic modifying factors affect toxicity by altering the external concentration of toxicant required to achieve the threshold internal concentration at that target site, for that chemical, at that dose, and for that organism. Factors affecting chemical activity can interact either within the organism or externally. Internal factors are usually biotic and act to change the manner in which organisms deal with a chemical metabolically. By increasing the rate of metabolic breakdown or the excretion rate of a chemical, the dose (exposure) required to achieve the threshold concentration at the target site increases. External factors are usually abiotic and affect the availability of the chemical for uptake. Chemicals, particularly metals, respond to some modifying factors by changing their speciation state, and some chemical species are able to reach target sites faster than others by crossing membranes more quickly or through preferential uptake by active mechanisms.

*For example*
***biotic modifying factors*** - species, life stage, sex, reproductive state, nutritional status, body size, diet, and acclimation

***abiotic modifying factors*** - temperature, water hardness, alkalinity, humic acid, dissolved oxygen, chelating agents, suspended solids, amino acids, and the presence of organic matter

The hazard assessment design should take into account both abiotic and biotic factors, and recognize their potential contribution to uncertainty. Whenever possible, the effect of modifying factors should be minimized by using appropriate controls, test materials, and test organisms.

### D.4.2 Microcosms

***Microcosms*** provide the opportunity to manipulate experimental conditions and look at population level effects in aquatic systems. These systems allow the study of effects of chemical perturbations on aquatic and terrestrial ecosystems. Through the incorporation of replication in experimental design, microcosms provide data that can be analysed statistically to determine significant changes in ecological structure or function (Sheehan 1989). Microcosm and mesocosms have been most widely implemented under regulatory programs like TOSCA (Toxic Substances Control Act), where new chemicals are being evaluated (Cairns 1979). Such studies have become the backbone of regulatory compliance testing for chemicals such as pesticides and herbicides. They allow investigation at a level of biological organization usually not possible in toxicity testing.Microcosms are not always applicable for hazard assessment at contaminated sites for several reasons. First, setting up the treatments requires dilutions of effluent from the contaminated site, as opposed to spiking with a single chemical. Also, hazard assessment at a contaminated site is usually retrospective, and so conducting a real community assessment would be preferable. Table D.3 presents some of the strengths and weaknesses of microcosm studies.

**Table D.3 Strengths and weaknesses of microcosm studies**

| Strengths | Weaknesses |
|---|---|
| Includes species' interactions | Oversimplifies natural system |
| Relates directly to ecosystem consequences | Lacks components of natural system (biotic and environmental) |
| Can be replicated with adequate controls | Considers cumulative impacts inadequately |
| Bridges single species and field studies | Relevance of test species often is not clear |

Source: Karr 1993

### D.4.3 Field Assessment Methods

*Additional reading*


Kapustka, L.A., T.W. LaPoint, J.F. Fairchild, K. McBee, and J.J. Bromenshenk. 1989. Section 8.0: Field assessments. In Ecological assessment of hazardous waste sites: A field and laboratory reference, ed. W. Warren-Hicks, B.R. Parkhurst, and S.S. Baker. U.S. Environmental Protection Agency, Environmental Research Laboratory, Office of Research and Development, Corvallis, OR. EPA/600/3-89/013.

Plafkin, J.L., M.T. Barbour, K.D. Porter, S.K. Gross, and R.M. Hughes. 1989. Rapid bioassessment protocols for use in streams and rivers: benthic macroinvertebrates and fish. U.S. Environmental Protection Agency, Office of Water. EPA/444/4-89/001.

Warren-Hicks, W., B.R. Parkhurst, and S.S. Baker, editors. 1989. Ecological assessment of hazardous waste sites: a field and laboratory reference. U.S. Environmental Protection Agency, Environmental Research Laboratory, Office of Research and Development, Corvallis, OR. EPA/600/3-89/013.


The use of field assessment methods depends, in part, on the approach that the ERA team has taken. The importance of going to the contaminated site and collecting field data cannot be over emphasized. Toxicity testing serves only to model the field situation and is not truly representative of the dynamics of populations and communities. However, the level of effort required to obtain useful field data usually means that investigators try other, simpler means of hazard assessment.

The advantages of collecting field data for hazard assessment include (adapted from Kapustka et al. 1989)

- impacts of contaminated site on indigenous species are measured
- direct measurements are taken (extrapolations from toxicity data are not required)
- results are interpretable
- results are more easily understood by decision makers and the general public
- the information can feed into the receptor characterization

However, field assessment data may be highly variable, reflecting natural fluctuations in ecological components with season, weather, time of day, etc. As a result of this high variability, field programs must be designed so that effects related to a contaminated site are actually detectable.

*For example*
Investigators are usually concerned with the probability ($\alpha$) of declaring an effect significant when it is not (= Type I error). This is a reasonable concern for routine scientific practice, as it focuses attention and resources on phenomena that are likely to be real, and weeds out phenomena whose existence is equivocal or doubtful. However, environmental scientists must also consider $\beta$, or the probability that an effect could be detected. The costs and consequences of Type II errors, or failing to detect an effect which actually exists, may be much greater than the costs and consequences of Type I errors (Peterman 1990). For this reason, impact and hazard assessments should include power analysis (power = $1-\beta$) and ensure that sample sizes are adequate to detect effects considered biologically significant. There is also no reason why power analysis should be restricted to field studies; toxicity tests may also show high variability and consequently have surprisingly little power (Barnthouse et al. 1986; Suter et al. 1987).

It is critical that the field methods selected (measurement endpoints) match the assessment endpoints that were set during the planning stage of the ERA, or which evolved during the course of the investigation. One of the temptations of collecting field information is to collect too much or the wrong kind; discipline must be practised in the design of field programs for contaminated sites.

### D.4.4 QSARs

***QSAR*** (Quantitative Structure Activity Relationship) models are mathematical equations derived to estimate the toxicity or other property of a chemical from its structure. Each substructure of a molecule contributes to its toxicity in a specific way, and the QSAR equation describes this contribution. Models of this type have proven to be successful in estimation of carcinogenicity, mutagenicity, and toxicity to rat, mouse, daphnid, and fathead minnow. QSARs are usually applied to predict the toxicity of new chemicals and in the case of contaminated sites with multiple contaminants, it would be best to actually test the toxicity of the contaminated site, as opposed to predicting it. QSARs might have a role at a site where organisms are being exposed to a chemical about which little is known.

## D.5 EXTRAPOLATIONS OF HAZARD ASSESSMENT DATA

*Additional reading*

Aldenberg, T., and W. Stob. 1993. Confidence limits for hazardous concentrations based on logistically distributed NOEC toxicity data. Ecotoxicol. Environ. Saf. 25:48–64.

Barnthouse, L.W., G.W. Suter II, A.E. Rosen, and J.J. Beauchamp. 1986. Extrapolation of population responses. *In* User's manual for ecological risk assessment, ed. L.W. Barnthouse and G.W. Suter II. Oak Ridge National Laboratory, Oak Ridge, TN. ORNL-6251.

Parkhurst, B.R., G. Linder, K. McBee, G. Titton, B.J. Dutka, and C.W. Hendrichs. 1989. Section 6.0: Toxicity tests. *In* Ecological assessment of hazardous waste sites: a field and laboratory reference, ed. W. Warren-Hicks, B.R. Parkhurst, and S.S. Baker. U.S. Environmental Protection Agency, Environmental Research Laboratory, Office of Research and Development, Corvallis, OR. EPA/600/3-89/013.

One of the largest sources of uncertainty in hazard assessment is data extrapolation. This section is not intended to provide in-depth information on extrapolation but to familiarize the reader with the kinds of data extrapolation that are used for hazard assessment. Barnthouse et al. (1986) discuss the analysis of extrapolation error and provide practical examples to demonstrate that species-to-species and taxa-to-taxa extrapolations can work.

### Species-to-Species Extrapolation

Toxicity tests conducted in the laboratory should use species representative of the ecosystem being assessed. There has been a great deal of discussion in Canada about the use of native species versus standardized toxicity-test organisms in laboratory assessments. On the one hand, data generated using species that live, or are expected to live, within the contaminated site will be directly applicable to the site and will not require as much extrapolation to predict effects. On the other hand, the success rate with adapting standardized tests to native species is not good; control survival problems and high variability plague such laboratory work and confound data interpretation.

The most viable option at this point appears to be to use standardized toxicity tests, at least initially, and to extrapolate from these results to the species of concern for the site. Selection of toxicity-test organisms should be made with consideration of the sensitivity of the species, mode of action of the stressor, expected exposure period of natural populations, etc. Uncertainty factors in hazard assessment have been shown to be greatest for between-species comparisons; on the order of 1 000 to 10 000 for acute toxicity and 100 to 1 000 for chronic toxicity (U.S. EPA 1991). Also, as taxonomic similarity decreases, the extrapolation uncertainty increases.

The most common method for species-to-species extrapolation is to compile toxicological data for organisms in similar taxa (e.g., same family or class) and develop confidence intervals or a range of effects concentrations. Assuming that the untested species have a similar sensitivity to the test species, the untested species are expected to fall within the same range (Mayer et al. 1986; also, this assumption was the initial basis for development of water quality criteria). Species within a similar taxa can have a wide range of response concentrations, but the more data one compiles, the more confidence one can place in the range or interval. For contaminated sites, this approach would be suitable, but the level of effort in testing is usually not practical. What happens in practice is that the relatively few toxicity tests that are available (relative to the number of species in existence) are used to represent a host of native species. For example, the earthworm test represents soil invertebrates, the rainbow trout test represents freshwater fish, and the domestic poultry test represents waterfowl. There is a heavy dependency on the assumption that standard toxicity-test organisms are sensitive.

### Endpoint-to-Endpoint Extrapolation

Given that it is relatively easy to collect acute toxicity data and that few true chronic-toxicity tests are standardized, methods to extrapolate from acute to chronic endpoints have been developed. For example, no-observed-effect concentrations (NOECs) can be developed from an $LC_{50}$. First, an analysis of acute to chronic ratios or regression analysis is conducted for species that have been tested, to determine the relationship from empirical data for similar species. Then, the relationship derived can be used for other species for which only acute data are available. One must assume that the ratio or relationship of acute to chronic toxicity remains similar between species. These extrapolations should be made only for the same types of tests conducted under the same conditions (e.g., water quality, life stage) (Parkhurst et al. 1989).

Due to the nature of toxicity data, acute to chronic ratios often have high variability. Wherever possible (i.e., where a higher tier of investigation is warranted) chronic testing or field assessments should be conducted for contaminated sites. Investigators must evaluate the uncertainty that endpoint-to-endpoint extrapolation will be introduced into the risk assessment, and determine whether it is acceptable on a site-specific basis.

In addition to acute to chronic ratios, short-term tests such as early life-stage tests can be used as predictors of chronic toxicities. By using sensitive life stages, good estimates of chronic toxicity endpoints can be obtained in much less time, at much less cost than full life-cycle tests (Rand and Petrocelli 1985).

**Laboratory-to-Field Extrapolation**

Field surveys are useful to identify deleteriously affected populations and communities, and possibly identify specific environmental effects (e.g., reproductive problems in a fish population by examining age-class structure and size of individuals). However, the link of cause and effect must be established through experimentation, usually in the laboratory, although field experiments can also be conducted. Ideally, investigators will link the design of laboratory experiments to the field data, permitting extrapolation from the laboratory to the field.

One of the most frequently raised concerns is that single-species toxicity testing in the laboratory does not measure higher level effects at the community and ecosystem level. The best must be done with the tools that are available, and toxicity tests provide useful information to identify the potential for toxicity from samples collected at a contaminated site.

To maximize extrapolation from the lab to the field, the test conditions should be as similar as possible to those in the field. Modifying factors such as water hardness, temperature, and organic carbon should be considered when setting up toxicity tests, so that appropriate controls are conducted. However, despite the best intentions of investigators, the responses of organisms exposed in the laboratory often differ from those exposed under natural conditions; laboratory-to-field extrapolation provides some indication of the direction and magnitude of those differences.

Toxicity and field survey data can be compared using exploratory data analysis techniques (Parkhurst et al. 1989). These preliminary analyses should show the relationship between the field-collected and laboratory-collected data, and suggest cause-effect relationships. For complex mixtures, which will often exist in contaminated sites, it may be impossible to determine which chemical or chemicals is causing the toxicity (Parkhurst et al. 1989).

If the laboratory-to-field extrapolation appears to be the major component of uncertainty in an assessment, further field studies may be warranted to pinpoint the actual hazard.

> ***For example***
> If a field survey shows there are reduced numbers of benthic invertebrates, but the toxicity testing indicates that the leachate from a contaminated site is not toxic at field concentrations, a more in-depth field study may be required to look at
> - substrate characteristics (a determinant of benthic community structure)
> - toxicity of field-collected water to native benthic species

## D.6 UNCERTAINTY IN HAZARD ASSESSMENT DATA

The extrapolations discussed contribute largely to uncertainty in hazard assessment. Models have been developed for extrapolating among taxa, endpoints, and laboratory and field data with known degrees of uncertainty (see U.S. EPA 1991). However, the ability to reduce uncertainty may be limited by the following (U.S. EPA 1991):

- variations in physical and chemical environmental factors (e.g., modifying factors)
- chemical interactions
- physical-chemical interactions
- nonchemical stresses
- biotic interactions
- indirect biological effects which are not explicitly determined in laboratory tests

Uncertainty for field assessments has traditionally been difficult to quantify. With statistical approaches such as power analysis, techniques for predicting and/or monitoring uncertainty are beginning to be developed. However, whether the level of uncertainty in field studies is acceptable is another issue. Regardless, it is

clear that direct measurement of toxicity to the organisms of concern, combined with focused field assessment, provides the risk assessor with the optimal combination of information for hazard assessment.

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APPENDIX E

# Risk Characterization

## E.1 DEFINITION

Risk characterization is the process of estimating the magnitude and probability of effects (e.g., Norton et al. 1988; Parkhurst et al. 1990; Pastorok and Sampson 1990). Risk characterization combines the results of exposure assessment, which estimates the concentrations of contaminants in the environment, and hazard assessment, which estimates the effects associated with various concentrations. If the endpoints and target species or communities are properly chosen, the risk characterization will make an ecologically important statement. Risk characterizations should also include a summary and discussion of strengths, limitations, and uncertainties arising from the data and models used to provide conclusions.

In many cases, it is difficult to define the boundary between risk characterization and other components of the risk assessment, especially hazard assessment. Hazard assessment and the other ecological risk assessment (ERA) components should be as objective as possible, and include only the assumptions and calculations necessary to fulfil their objectives as well as provide specific statements or distributions of measured or expected effects: "species X will suffer 10% mortality at concentration Y".

In contrast, the risk characterization should include additional assumptions and calculations, particularly those related to uncertainties, and the steps such as dividing by a safety factor used to account for various uncertainties. If this division is adopted, the results of the hazard assessment and other components can be used at other sites, by other investigators, and with different risk characterization methods. Any new effects data from subsequent monitoring or toxicity testing can easily be applied to the hazard assessment. The risk characterization will then contain the most contentious assumptions, including those specific to the method or approach adopted. If these assumptions are shown to be untrue, or if another approach or method of risk characterization is used, only the risk characterization process needs to be repeated.

## E.2 UNCERTAINTY IN RISK CHARACTERIZATION

The sources and magnitudes of uncertainties in risk characterization should be identified and reduced whenever possible. Barnthouse and Suter (1986) considered three sources of uncertainty:

- ***inherent variability*** is the natural variability in ecological systems and in the measurement of ecological parameters such as variability in discharge, and measurement and sampling error. Measurement and sampling error can be reduced by more precise measurements and proper sampling designs. Natural variability cannot be reduced, but can be quantified by providing variances as well as means, and by using these variances to calculate probabilities of effects.

- ***parameter uncertainty*** is the uncertainty associated with estimating parameters such as the estimation of chronic benchmark concentrations from $LC_{50}$s, and estimation of toxicity from chemical structure or activity. Parameter uncertainty can be reduced by developing more precise estimation procedures (e.g., regressions) or by directly measuring the parameter of interest.

- ***model errors*** are broad-scale sources of uncertainty and would include errors associated with using few variables to represent many complex phenomena, using inappropriate functional relationships, and using inappropriate boundaries to define the system of study. These model errors are very difficult to quantify or even identify, and are consequently difficult to reduce, because they deal with the "unknown" (true uncertainties).

The relative importance of these sources of uncertainty may vary among methods or approaches. For example, inherent variability may be the most important source of uncertainty for retrospective and perhaps empirical approaches, whereas parameter uncertainty may be more important for predictive and theoretical approaches. Although the term *model error* suggests that this is an important source of uncertainty only for theoretical models, it is actually important for all risk characterization methods. All methods rely on a reduced set of variables, make some assumptions about functional relationships (or ignore them), and place boundaries on the system to be studied.

Uncertainty from different sources may also be correlated. A precise measure of some parameter will not only reduce inherent variability, but will also increase the precision of any other parameters estimated from that parameter. There is usually a trade-off between parameter uncertainty and some model errors. Including more variables in a model or characterization, and expanding the boundaries, increases the summed contribution of parameter uncertainties. The same consideration applies to empirical regression models. Increasing the number of variables increases the proportion of variance accounted for by the regression, but the residual mean square (which will determine the prediction or confidence intervals) may actually increase because of the reduction in degrees of freedom. Even retrospective analyses such as analysis of variance (ANOVA) in an impact assessment can rapidly become unmanageable if too many factors are included.

Verification, calibration, and validity are necessary to increase the precision of risk characterizations and to increase confidence in the final output of risk assessment studies. Verification of specific predictive risk characterizations by subsequent observation is important, although rarely done (Norton et al. 1988; Parkhurst et al. 1990; Pastorok and Sampson 1990). Methods that are verifiable, and that have been successful in past studies, are more credible than those which are not verifiable or have not been verified in the past. Methods, especially those dealing with extrapolation or estimation, should also be based on valid or reasonable assumptions about relationships or processes.

## E.3 QUALITATIVE METHODS

Qualitative methods do not quantify the magnitude or probability of effects but may rank or categorize the severity of effects as in high, moderate, or low risk. In many cases, qualitative methods depend on professional judgment and are used as a preliminary means of identifying sites or areas of concern. The most obvious Canadian example is the method used by the CCME (1991) to classify contaminated sites. Useful resources for more information on qualitative risk estimates are provided in the box below, and Table E.1 provides some examples of methods in use.

***Additional reading***


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## E.4 QUANTITATIVE METHODS

### E.4.1 Quotient Methods

Most quotient methods apply to single chemicals and to the individual level. These restrictions are major

**Table E.1 Examples of qualitative risk characterization methods**

| Agency/method | Description/comments |
|---|---|
| **Canada**<br>CCME (1991) National Classification System | Screening method/scoring system for contaminated sites<br>Based on contaminant characteristics, exposure pathways, and receptors |
| **USA**<br>U.S. EPA Office of Water Regulations and Standards<br>(U.S. EPA 1983) | Verbal risk characterization of sites<br>Expert judgment<br>Based on combinations of key species, chemicals, locations |
| U.S. EPA Office of Solid Waste<br>(U.S. EPA 1987a) | Based on proximity to sensitive environments<br>risk= inverse of distance to nearest sensitive environment<br>= number of sensitive environments nearby<br>Oil and gas/mining activities |

limitations of the methods, and attempts to remove the restrictions are discussed in detail below. Otherwise, quotient methods can be applied to any species, chemical, or site for which a Benchmark Concentration (BC) and Expected Environmental Concentration (EEC) can be calculated. The quotient method identifies the presence of potential risk, but does not characterize its magnitude. The the following equation is for the quotient method and Table E.2 lists the advantages and limitations of its use. Examples of quotient methods used in various jurisdictions are provided in Table E.3.

Quotient = EEC/BC
where,
EEC = expected environmental concentration, either from direct measurement, predicted through modeling, or back-calculated to set a certain "safe" concentration
BC = benchmark concentration, derived from the hazard assessment
Quotient < 1 indicates low or extremely low risk or probability of effect
Quotient ≥ 1 may indicate potential risk or effects

**Quotient Method for Multiple Chemicals**

Summing quotients is one method used to deal with multiple chemicals (e.g., U.S. EPA 1987b; this method is used by the U.S. EPA Office of Solid Waste). The sum is then interpreted in the same way as a quotient for a single chemical; if the sum is ≥ 1, a risk is assumed to exist The underlying assumption is that toxicities (actually 1/BC) are additive. This is a reasonable assumption for lethal effects concentrations such as an $LC_{50}$, and it forms the basis for the use of toxic units (which are $EEC/LC_{50}$) (see U.S. EPA 1985 for a discussion of toxic units). However, the same assumption of additivity may not apply to sublethal effects concentrations such as NOEC or MATC.

Any summing should be part of the risk characterization, rather than the hazard or exposure assessment (i.e., one should sum quotients rather than calculate a BC for a specific mixture). If an existing or predicted mixture of chemicals is used for hazard assessment, and a BC calculated for that mixture, then that BC applies only to that specific mixture and cannot be used to generate remediation criteria. As well, the composition of any mixture may vary among media and over time. However, if the hazard assessment is restricted to calculating BC for individual chemicals, these individual BC can be used for risk characterization of any mixture, existing or targeted.

One important exception might be effluents of a reasonably constant composition, if exposure were largely restricted to waterborne contaminants. In that case, the hazard and risk characterization could deal with the effluent as a whole, and remediation criteria could be based on whole effluent toxicity or measured in-stream effects (i.e., the criterion or objective might be an effluent NOEC greater than the minimum concentration expected in-stream).

**Table E.2 Advantages and limitations of the quotient method**

| Advantages | Limitations |
|---|---|
| Simplicity, ease of implementation and low cost | Predicted risks are semiquantitative and nonprobabilistic. |
| The hazard data required (usually $LC_{50}$ or MATC) are more available, or more easily estimated, than other types of data | The magnitude of effect is often not specified |
| The actual risk characterization is trivial and produces a single number (quotient) that can easily be used to rank priorities in terms of contaminants or species of concern. | The probability distribution of the quotient is rarely specified |
| Establishing remediation criteria is also simple, using the BCs, possibly adjusted by a safety factor | The probability distribution of different effect sizes is, by definition, never specified |
| The methods and associated assumptions could easily be verified using large data sets comparing predicted and observed effects. | The predictions of quotient methods at specific sites will be virtually unverifiable even if follow-up monitoring is conducted. |
| | The widespread use of safety factors to express uncertainty is often arbitrary, may vary among methods, and is sometimes concealed in the hazard assessment, reducing the validity and utility of that assessment. |

**Table E.3 Examples of quotient risk characterization methods**

| Agency/method | Scope | Description | Comments |
|---|---|---|---|
| **Canada**<br>CCME Water Quality Guidelines (CCREM 1987) | Aquatic; single chemical<br>Could be applied to other media/ecosystems | Guidelines are BC/SF<br>SF vary depending on chemical properties, data available | Basic quotient/criteria method |
| **USA**<br>U.S. EPA Office of Pesticide Programs Standard Evaluation Procedure (Urban and Cook 1986) | Aquatic/terrestrial<br>Single chemical/exposure pathway<br>Scope could be expanded by modifying method | Risk = EEC/BC<br>SF (actually AF) applied if BC based on $LC_{50}/LD_{50}$<br>No SF applied if BC based on NOEC<br>Used as part of pesticide registration | Basic quotient method<br>Programs available for personal computer |
| Chemical Migration Risk Assessment (Onishi et al. 1982,1985) | Aquatic; single chemical<br>Potentially adaptable to other ecosystems/chemical mixtures | BC fixed; EEC expressed as distribution<br>Risk = probability of exceeding BC<br>Several BC often used (e.g., acute, chronic) | Strength is exposure assessment<br>Computer program (FRANCO) available and adaptable |
| Office of Water Regulations and Standards, Natl. Water Quality Criteria (U.S. EPA 1986) | Aquatic (extension to wildlife under consideration)<br>Single chemical<br>Exposure through water; but some consideration of dietary uptake | Risk = EEC/BC<br>BC applies to lowest 5th percentile of species ranked by sensitivity | Basic quotient/criteria method<br>Programs available for personal computer |
| Waste Load Allocations (U.S. EPA 1985, 1987c) | Aquatic<br>Single chemicals/effluents | Expressed in loads ($wt.\cdot d^{-1}$) rather than concentration<br>SF applied to acute waste allocation<br>Chronic allocation based on low flow (7Q10) | Useful for effluents |
| Office of Solid Wastes Risk-Based Variance (U.S. EPA 1987b) | Waste from hazardous waste tanks<br>Aquatic; terrestrial<br>Multiple chemicals | Risk = $\sum$ (EEC/BC) for multiple chemicals<br>BC are EPA water quality criteria or MATC/SF | Basic method, but sums quotients |
| Risk-Cost Analysis Model (U.S. EPA 1984) | Aquatic/terrestrial<br>Hazardous wastes<br>Multiple chemicals present, but only most toxic used<br>Allegedly applicable to community/ecosystem | Based on quotient(s) from most sensitive species<br>Quotient(s) subject to further manipulation to give qualitative score estimating risk to community/ecosystem<br>BC may be EPA water quality criteria<br>Complex set of SF applied | Some empirical support for method used to extrapolate individual/single species to community<br>Difficult to describe and classify<br>Program available for personal computer<br>Also relies on extensive computer database (inventory of U.S. habitats) |

**Table E.3 Continued**

| Agency/method | Scope | Description | Comments |
|---|---|---|---|
| Ohio EPA (1987a, b, 1988)<br>Biological Criteria | Community level<br>Aquatic<br>Indirectly addresses multiple chemicals/exposure pathways | Based on 3 indices:<br>Index of Biotic Integrity for fish<br>Index of Well-Being for fish<br>Invertebrate Community Index<br>Risk = Observed Index Value/Background or Criterion Index Value<br>Risks/criteria for water quality are based on effects not concentration | Empirical method<br>Requires data on Background Index Values<br>Effective in setting remediation objectives or criteria, and in measuring progress towards meeting those objectives or criteria<br>Probably the most highly developed quotient method available for higher-level (community, ecosystem) effects |
| New York Dept. Ecology and Conservation<br>Niagara River Fish Flesh Criteria (NY DEC 1987) | Piscivorous wildlife<br>Single chemical | Risk = EEC/BC<br>EEC refers to expected or observed tissue residue in fish<br>BC refers to dose for birds/ mammals<br>Various SF applied to BC | Takes advantage of large data set available for BC for birds/ mammals |
| Washington Dept. Ecology<br>Apparent Effects Thresholds (AET) (Washington DOE 1991) | Aquatic; sediments<br>Single chemicals<br>Multiple exposure pathways? | Risk = EEC/BC<br>BC = empirically derived AET, with SF often applied<br>AET = highest concentration associated with no effect in toxicity tests, benthic communities | Empirical method<br>Requires large data set to establish AET |
| Oak Ridge Nat'l Laboratory<br>Analysis of Extrapolation Error<br>(Suter et al. 1986) | Aquatic; adaptable to terrestrial<br>Single chemical/exposure pathway | EEC, BC expressed as distribution<br>Prediction limits for quotient<br>No SF applied to BC | Shown in Figure E.1 (c) |

AF = Application Factor (acute → chronic); BC = Benchmark Concentration: EEC = Expected Environmental Concentration; SF = Safety Factor

**Risk of effects to higher levels.**
Within the hazard assessment, effects of contaminants to individuals may have been extrapolated to higher levels of biological organization such as the use of sensitive species, with the assumption that protection of these species will protect the remainder of the community or the development of empirical databases relating higher-level effects to contaminant concentrations (e.g., Apparent Effects Threshold methods). If such data are to be used when characterizing risk, all assumptions and/or extrapolations must be noted so that a complete estimate of uncertainty can be made.

A more quantitative and probabilistic approach is that used by some Europeans (e.g., Wagner and Lokke 1991). BCs are obtained (measured or estimated) for selected species representing the community. This sample of BCs is assumed to follow some distribution, usually lognormal, and lower statistical tolerance limits are calculated. Thus, the lower 95% tolerance limit would protect 95% of the species in the community. The advantages of this method, relative to simply selecting the BC for the most sensitive species, are that tolerance limits are less variable and more precise than extremes such as minima and that the tolerance limits are quantitative and probabilistic.

**Magnitude of Effects**
The simplest quotient methods make no statement about uncertainty or probability. Either an effect will (quotient $\geq 1$) or will not occur (quotient $< 1$). If the BC is an NOEC or MATC, it may not even correspond to a specified magnitude of effect. However, it is possible to make risk characterizations produced by the quotient method more quantitative and probabilistic by specifying the effect as a specific quantile (e.g., $EC_{10}$ or $LC_{10}$) and by attaching prediction or tolerance limits to the BC or EEC or both.

*For example*
The analysis of extrapolation error method described by Suter et al. (1986) considers uncertainty associated with both the BC and EEC, to estimate the probability that EEC>BC. This method corresponds with Figure E.1 (part c), although the uncertainty about BC refers only to the error in extrapolating from acute to chronic effects or between species.

Another alternative described in Barnthouse and Suter (1986) and in U.S. EPA (1987b) is the use qualitative categories for quotients:

- <0.1 = no risk
- 0.1 to <10 = possible risk
- >10 = high risk

**Uncertainty**
Quotient methods usually deal with uncertainty by establishing qualitative categories for quotients or by applying safety factors to the BC or less commonly, the EEC. Often a distinction is made between application factors, which are used to convert acute or lethal effects concentrations to chronic or sublethal effects concentrations, and safety factors, which are used to provide some unspecified margin of safety. Application factors are considered part of hazard assessment, as they usually have some empirical support (Barnthouse et al. 1986). Safety factors are part of risk characterization because they are a substitute for probabilistic statements.

Canada uses safety factors in establishing water quality guidelines, as do many other agencies (CCREM 1987). In the standard procedure used by the U.S. EPA Office of Pesticide Programs, a number of different safety factors are applied to the BC (Urban and Cook 1986). For example, a safety factor of 2 is applied for aquatic organisms because they are considered less able to limit their exposure by migration or other behaviour, and safety factors of 10 and 20 are used for endangered terrestrial and aquatic species, respectively. Suter (1986) proposed that establishing categories for quotients is preferable to applying safety factors to BCs. The categories can be (and often are) based on the same considerations and numerical values as are safety factors; the point is that any adjustments of this type should be clearly stated in the risk characterization rather than potentially concealed in the hazard assessment.

The assumptions of quotient methods discussed above have rarely been verified, partly because more verifiable and/or better supported assumptions have been deliberately classified as part of hazard assessment.

**Verifying Data**
The semiquantitative and nonprobabilistic nature of most quotient methods does not pose serious problems for verification using large data sets (i.e., the methods and their assumptions are verifiable). If large numbers of cases are available, both predicted and observed responses can be expressed as yes/no, effect/no effect responses for comparison. The power of such comparisons comes not from the precision of the individual responses but from the generality of including many cases. This type of comparison can even be conducted when the predicted and observed responses represent different endpoints or different levels of organization; for example,

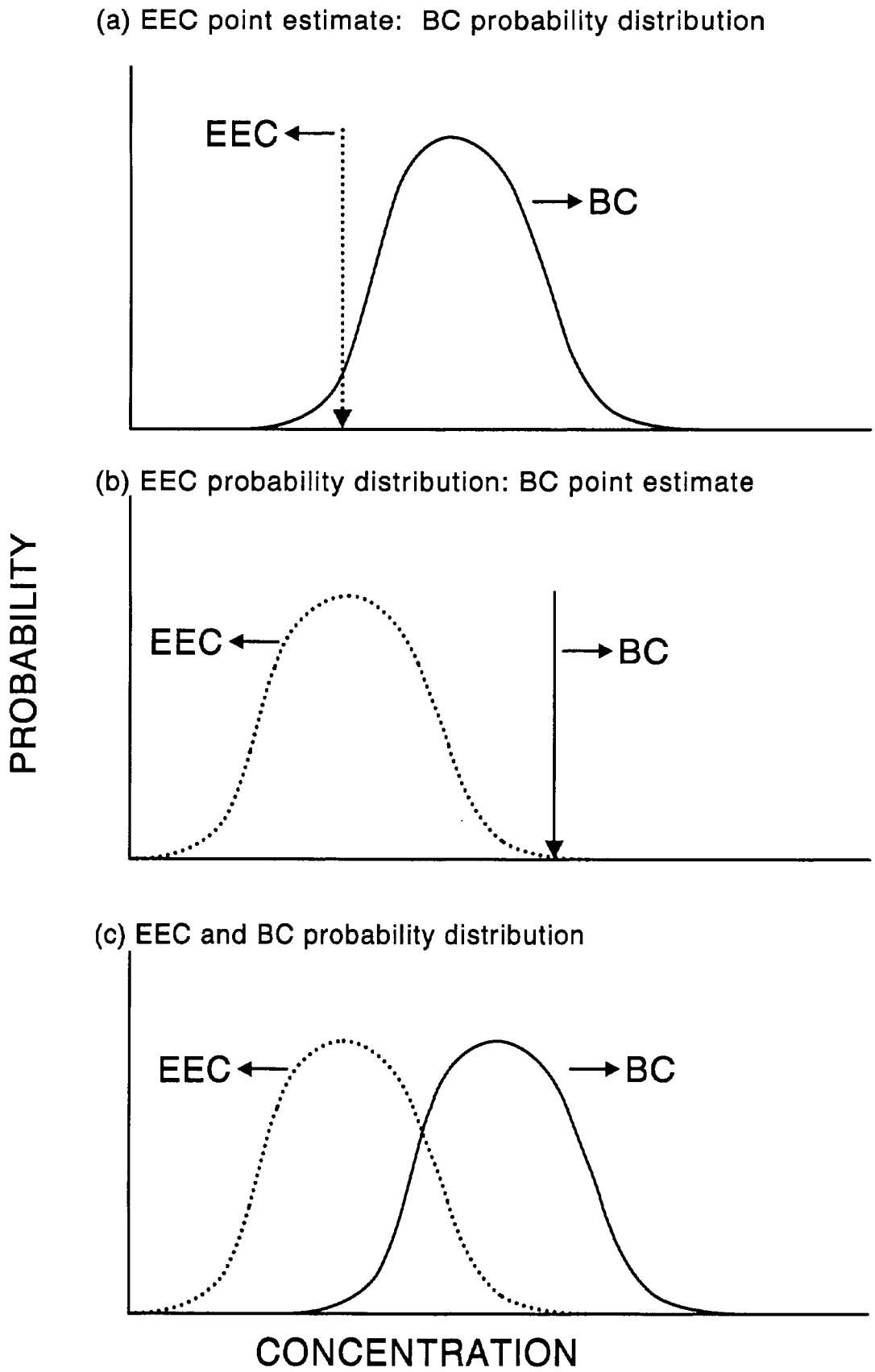



Figure E.1 Degrees of quantification of uncertainity/probability for quotient risk characterization methods. BC = Benchmark Concentration; EEC = Expected Environmental Concentration.

when predictions based on single species toxicity tests were compared with observed in-stream macroinvertebrate community responses by Eagleson et al. (1990). Unfortunately, the methods and assumptions of quotient methods are potentially unverifiable in cases involving one or a few studies.

*For example*
Consider the case of a projected risk calculated for a site with an endangered or rare species, which is to be followed up and verified by monitoring studies because of concern over the species. There may be few or no other sites at which risk projections for this species could be verified. Under these circumstances, a risk projection expressed as a quotient is virtually untestable. To illustrate, suppose the quotient EEC/BC were 0.1, categorized as no or low risk. Follow-up studies indicate a statistically significant 15% reduction in mean growth rate of the individuals. Arguably, the method failed; specifically by underestimating risk (predicting no effect when one was observed). However, if the BC were equivalent to an $EC_{40}$, an investigator might conclude instead that the method was successful since a 40% reduction in growth rate was not observed (if the observed 15% reduction were significantly different from 0%, it would almost certainly be significantly different from 40%). In reality, most investigators would want to compare the confidence limits for the magnitude of the observed response (easily calculated but probably narrow) with the prediction or tolerance limits for the *magnitude* of the predicted effect. In other words, the observed effect should be stated as **e.g.,** 15±5%, and the predicted effect as e.g., 5±20%. The prediction limits for the projected effect can only be obtained from continuous exposure-response relationships that account for uncertainty (variance) in both the EEC and the expected effect. Based on the overlapping confidence and prediction limits provided above, an investigator would conclude that the prediction was either successful or too imprecise to provide a meaningful test.

### E.4.2 Continuous Exposure-Response Methods

Continuous exposure-response methods do not rely on a single BC but use the entire relationship between concentration or dose and one or more responses. Thus, the risks of a broad range of effect magnitudes (e.g., 1, 10, 25 and 50% reduction in survival) are considered and prediction limits are calculated. These relationships are derived from toxicity data in the hazard assessment. Procedures used to calculate prediction limits should account for variance or uncertainty in the independent variable (exposure) as well as in the dependent variable (response) (Barnthouse et al. 1986). The spatial scope of these models is usually broad, but site-specific models have been developed (Appendix D in U.S. EPA 1991). The RAMAS series of models can deal specifically with the effects of spatial scale and differing spatial distributions (i.e., many small isolated populations versus a few large populations). Models also deal with a longer time span than do quotient methods.

Table E.4 lists the advantages and limitations of continuous exposure-response methods and examples of continuous exposure-response risk characterization methods are provided in Table E.5. The relationship shown in Figure E.2 (a), if it referred to a single species, would represent a risk characterization at the individual level.

Theoretically, continuous exposure-response methods can be applied to any species, chemical, or level of biological organization (individual, population, community/ecosystem). In practice, the number of species or chemicals for which continuous exposure-response data are available will restrict the scope of the methods or increase uncertainty if extrapolations from species to species or chemical to chemical are used. Also, in practice, exposure-response relationships at the individual level for a number of species or endpoints serve as input for models predicting higher level effects or risks and the use of community/ecosystem models has largely been restricted to aquatic systems.

Some authors (e.g., Parkhurst et al. 1990) consider these methods as applying to the population as well as individual level. However, in this report, the category of population level methods is reserved for methods (usually population models) that predict effects for more than one generation and consider population-level effects such as the probability of extinction. Similarly, higher-level (community, ecosystem) methods predict effects above the population level.

#### Individual

Methods applying to the individual level do not consider effects beyond those considered in most bioassays and toxicity tests reductions in survival, growth, or reproduction of individuals, usually of a single species. Barnthouse et al. (1986, 1987) provide a method for estimating parameters of continuous functions from point estimates.

#### Populations, communities, ecosystems

By definition, population and higher-level models attempt to estimate the magnitude and uncertainties of higher-level effects. It follows that these models will be useful when

- these higher-level effects exist, and are large
- additional uncertainties are identified, quantified, and *subsequently reduced*

**Table E.4 Advantages and limitations of the continuous exposure-response method**

| Advantages | Limitations |
|---|---|
| Can quantify a range of effect magnitudes and their uncertainties such as density dependent effects through multiple simulations and sensitivity analysis. | Requires continuous exposure-response data which is usually less available<br>Lack of suitable models for many non-commercial species and most ecosystems |
| Predictions of these continuous measures are easier to verify at a specific site than are the predictions of quotient methods | Increased uncertainty associated with additional parameters |
| Models can also be useful for investigating alternative scenarios | Difficulties in verifying long term predictions |
| | Use of population and higher-level models requires considerable expertise and effort beyond that usually required for the hazard and exposure assessment |

Models have identified effects that would not be predicted by individual-level methods.

*For example*
The SWACOM model indicated that algal biomass may increase, even if contaminants negatively affect individual algae, because of greater effects on grazers and alteration of algal community composition (O'Neill et al. 1983, 1986). The model also indicated that effects could differ with timing of exposure initiation (spring versus fall). Barnthouse et al. (1987, 1990) used their fish population model to estimate and compare various sources of uncertainty. The greatest source of uncertainty was associated with estimation of long-term toxic effects from short-term effects or QSAR. Finally, both the SWACOM and fish population models indicated that risks at higher levels were greater than those at the individual level.

Deterministic linear population models are the most common method used to predict population level effects. These models have traditionally been used in fish, wildlife, forestry, and pest management (see Getz and Haight 1989; Emlen 1989 for reviews). The models are usually age-, stage- or size-specific, tracking abundance of different age- or size-classes or ontogenetic stages separately. A bookkeeping approach is usually followed, with birth, death, and growth rates applied to age-, stage-, or size-class abundances to predict abundances at the next time interval (Figure E.3). The most common interval is one year, because of the annual seasonal cycle of processes such as birth, but the interval may be shorter for smaller organisms with short life cycles. Modifications of basic population models include stochastic and nonlinear models.

Stochastic models include variability in model parameters, an obvious desideratum for risk characterization. Nonlinear models provide an alternative to the traditional assumption that relationships between births or deaths and numbers are linear (i.e., constant birth or death rate). Thus, these nonlinear models can account for density-dependent processes.

Although population and higher-level models do account for some effects beyond the individual level, they cannot account for all such effects and are open to the criticism that important effects have been excluded. Reviews have suggested that density dependent effects on mortality, growth, and reproduction may be the most important effects excluded from existing models (e.g., Barnthouse et al. 1986; Norton et al. 1988; Parkhurst et al. 1990; Pastorok and Sampson 1990). There are models available that include density dependent effects, and there is evidence for the existence of these effects (Getz and Haight 1989). However, in most cases, the exclusion of density dependent processes is conservative (i.e., overestimates risk). Density dependent processes tend to move successive age- or size-classes towards a fixed abundance or biomass.

*For example*
Food availability in a stream might limit the number of available territories and therefore the recruitment of juveniles regardless of the number of eggs or alevins produced in any year (Elliott 1987). If a contaminant affected primarily the survival of younger stages, the population density might remain relatively stable. The surviving juveniles would enjoy better growth and survival because they would have a better chance of securing territories and food. This type of compensatory growth or mortality would be especially important in migratory species, with only one life stage exposed to contaminants.

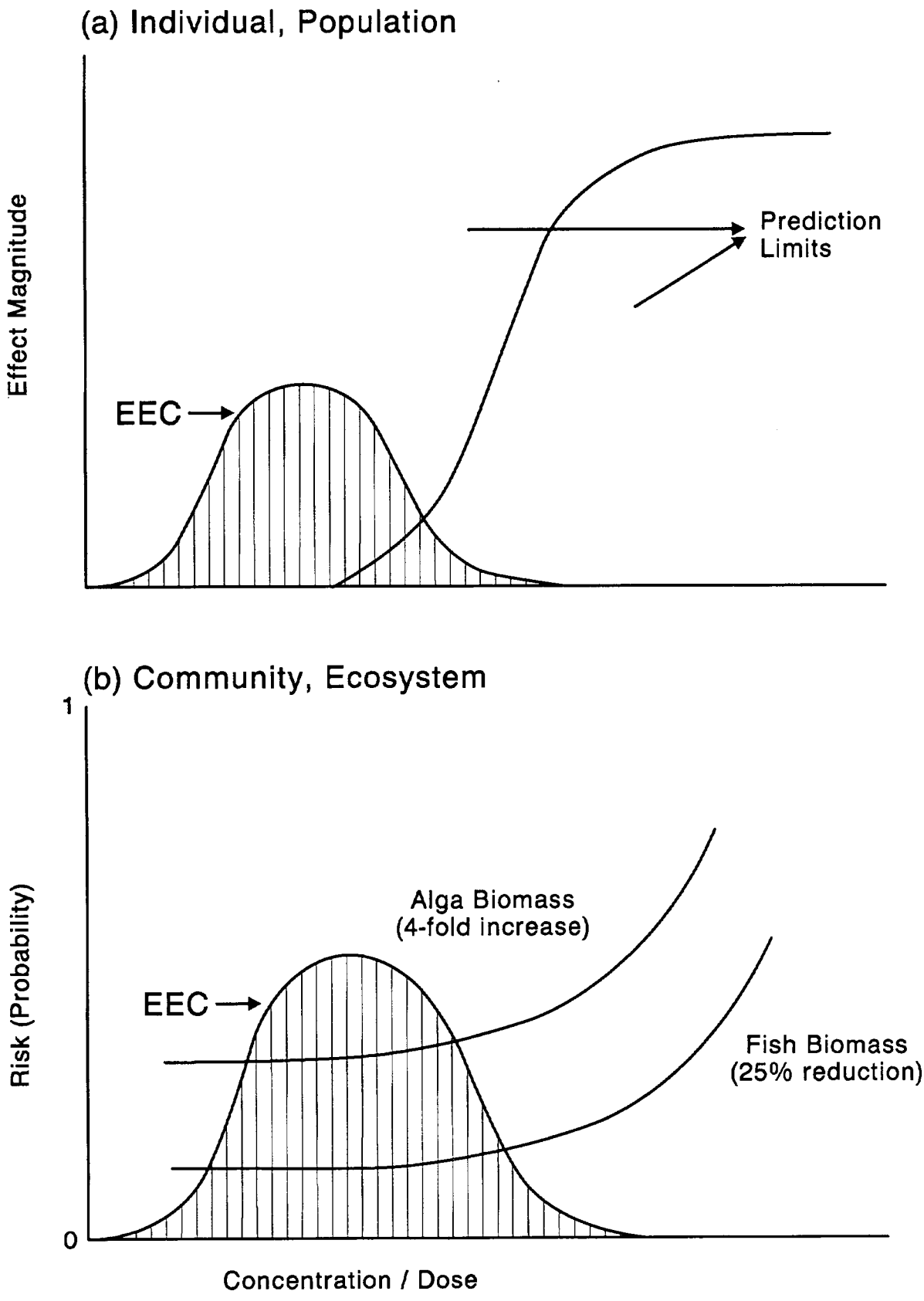
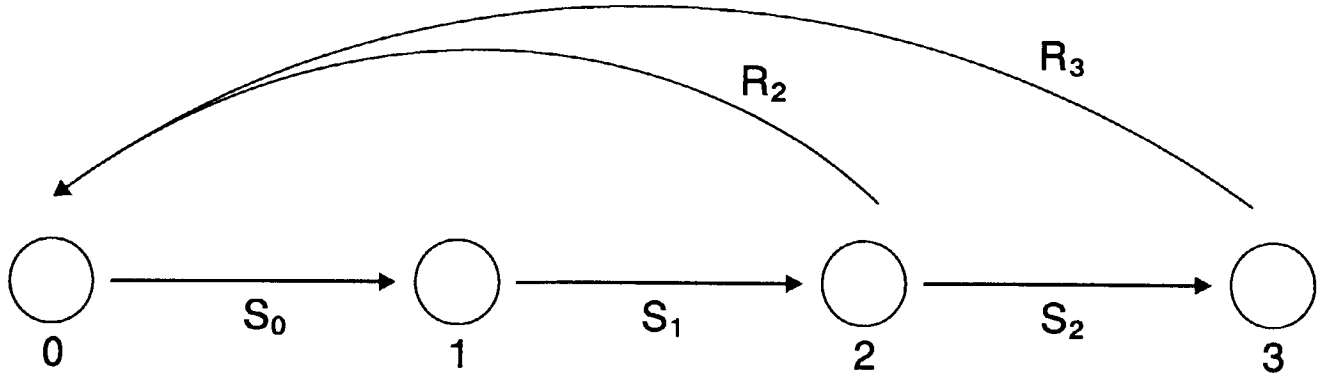



Figure E.2 Risk charcterization from continuous exposure-response methods. The distribution of Expected Environmental Concentrations (EEC) has been superimposed on the response curves. Adapted from Barnthouse *et al.* (1986).




| Age | Abundance at time: | |
|---|---|---|
| | $t$ | $t + 1$ |
| 0 | $N_0$ | $S_1N_1R_2 + S_2N_2R_3 + \ldots$ |
| 1 | $N_1$ | $S_0N_0$ |
| 2 → | $N_2$ | $S_1N_1$ |
| 3 | $N_3$ | $S_2N_2$ |
| . | | |
| . | | |
| . | | |
| etc. | | |

Figure E.3 Basic age or stage-specific linear deterministic population model. $S_i$ are survival rates and $R_i$ are reproductive rates, for the $i$th age class. The hypothetical organism shown does not reproduce until age 2, and reproduction occurs at the end of the interval from time $t$ to $t + 1$.

**Table E.5 Examples of continuous exposure-response risk characterization methods**

| Agency/method | Scope | Description/comments |
|---|---|---|
| **Population**<br>Oak Ridge National Laboratory<br>(Barnthouse et al. 1986) | Aquatic—fish<br>Single chemical/exposure pathway | Linear<br>Output is female reproductive potential<br>• if reproductive potential averages 1, then each female will replace herself and abundance will remain constant<br>• if reproductive potential is < 1 then the population will decline<br>• if reproductive potential is > 1, then the population will increase<br>Adapted from models used in assessment of power plant impacts<br>Requires data on survival, reproduction |
| **Community/ecosystem**<br>U.S. Dept. Interior<br>CERCLA Damage Assessment<br>(U.S. DOI 1987) | Aquatic<br>Oil spills, hazardous wastes | Deterministic, linear<br>Retrospective (damage assessment), but also predicts long-term impacts<br>Basically population models, but can pass on effects from algae to zooplankton<br>No estimates of uncertainty |
| Oak Ridge National Laboratory<br>SWACOM model<br>(O'Neill et al. 1986) | Aquatic<br>Applies to the pelagic zone of north temperate **dimictic** lakes<br>Single chemical / exposure pathway | Transfers effects through trophic levels<br>Can include species interactions such as competition<br>Presents results as the probabilities of a four-fold reduction in algal biomass and a 25% reduction in game fish biomass but other effects measures or magnitudes can easily be generated.<br>Simulations provide uncertainty analysis |

Fisheries and wildlife management depends on the assumption that compensatory mortality and growth will counteract the effects of increased mortality from exploitation up to a certain level. In fact, exploitation will in some cases increase biomass or production. Even density-independent mortality from changes in climate or discharge may completely override toxic effects. Thus, inclusion of density dependent effects is likely to reduce estimates of risk. Exceptions would occur in cases of reverse density dependence; for example, when low densities lead to an increased probability of failing to find a mate. If density-dependent processes are to be included in models, the objective should be to identify the critical contaminant concentrations and effects beyond which compensation is no longer effective.

### Multiple Chemicals

Continuous exposure-response methods, and specifically population, community, and ecosystem models, predicting effects of multiple chemicals or exposure pathways, have not been developed. Survival probabilities for exposure to several chemicals or pathways can easily be combined by multiplication into an overall survival. However, combining effects on reproduction or growth might be considerably more difficult as these effects are rarely expressed as binomial probabilities. It is suspected that developing models addressing multiple chemicals or exposure pathways is theoretically possible but might be technically difficult in practice and would require making and then verifying a number of assumptions about how exposure-response relationships should be combined. Population models can integrate effects on several different endpoints such as survival, growth and reproduction, and higher-level methods can integrate effects on different species.

### Uncertainty

Individual-level continuous exposure-response methods provide measures of uncertainty in the form of prediction limits about the exposure-response relationships (Figure E.2). These prediction intervals can be based on uncertainty about environmental concentrations as well as about effects. As discussed in the evaluation of quotient methods, the inclusion of prediction intervals makes it much easier to compare

predictions with observed effects at specific sites. However, the prediction intervals address only a limited range of uncertainties, usually those related to extrapolations or assumptions in the hazard and exposure assessments. Higher-level continuous-exposure methods attempt to deal with other sources of uncertainty, particularly, of course, higher-level effects.

The most common approach to analysing uncertainty in population and higher-level models consists of multiple simulations followed by sensitivity analysis (O'Neill et al. 1986). Monte Carlo simulation involves repeated runs of the model with parameter values randomly selected from probability distributions. These simulations indicate the uncertainty about model predictions or output but do not indicate the major sources of uncertainty. The major sources of uncertainty are identified by sensitivity analysis, which determines which parameters have the greatest effect in determining the value of the output measure (see O'Neill et al. 1986; U.S. EPA 1991 for descriptions of some specific methods). Sensitivity analysis is very important if models are to be used in risk assessments, because otherwise the models will only add additional uncertainty (and quantify the usually depressing effects of that additional uncertainty). There should also be some follow-up to the sensitivity analysis, through additional hazard and exposure assessment and further model refinement to focus on and reduce the major sources of uncertainty.

In general, the best means of ensuring the validity of model results is to use models that have been applied previously and are credible to the scientific community, and to calibrate the models through an iterative process of simulation, sensitivity analysis, and direct measurement.

**Verifying Data**

The input parameter values used in models can often be verified or calibrated by direct measurement. The particular processes included in the model, such as transfer of energy from one trophic level to another, should also be verifiable through direct measurement or valid in terms of being based on similar processes observed in the literature. Output measures, particularly those related to longer-term effects such as the probability of extinction, may be more difficult to verify.

## E.5 CURRENT PRACTICES AND STATE OF THE ART

Table E.6 summarizes current risk assessment practices in U.S. federal and state agencies, taken from Appendices E and F in U.S. EPA (1991). The survey indicated that most agencies use qualitative and quotient methods, and rely strongly on professional judgment. Quantification of uncertainty is rare. In fact, the consensus among the state agency personnel was that the EPA should omit any reference to quantitative uncertainty analysis and statistical significance of the final risk in guidelines produced for risk assessment. This consensus is in sharp contrast to the recommendations of reviewers (e.g., Norton et al. 1988; Parkhurst et al. 1990; Pastorok and Sampson 1990), who argued for increased levels of quantification in risk characterization. The question of when or even whether the increased complexity and costs of quantification of uncertainty and use of higher-level models is justified is probably the major issue in risk characterization. The U.S. EPA (1991) survey and the other reviews cited agreed that qualitative and quotient methods are adequate for an initial assessment of risk and for ranking the relative risks associated with different chemicals, sites or species. Continuous exposure-response methods and models can be used for a more refined risk characterization and to explore higher-level effects.

The following two factors, unrelated to the scientific merits of qualitative/quotient versus more quantitative methods, which probably contribute to the widespread use of the less quantitative methods:

- many agencies use risk assessments to establish criteria or assist regulatory decisions
- most toxicologists are not familiar with population and ecosystem models

Dichotomous (effect/no effect) risk characterizations are simpler to apply in a regulatory framework or in establishing criteria than are continuous values. More generally, simple risk characterizations are easier to understand and communicate to others. Even though the more quantitative methods can give a wide range of effects and associated uncertainties, the risk characterizations are usually expressed as the probability of only one or a few effect magnitudes.

**Table E.6 Risk characterization methods used by U.S. state and federal agencies**

| Agency | Method(s) used |
|---|---|
| States | |
| Michigan Department of Natural Resources | Use water quality criteria; compare with existing concentration<br>Aquatic Chronic Value<br>Terrestrial Life Cycle Safe Concentration<br>Goal: protect 95% of species for 80% of chemicals |
| New Jersey Department of Environmental Protection | Currently developing methods<br>Have considered:<br>• Analysis of Extrapolation Error (quotient)<br>• Toxicity Quotient (basic quotient method)<br>• Mink and mallard risk assessments (quotient) |
| Ohio Environmental Protection Agency | Numerical biocriteria<br>Compare observed effects with predictions from basic quotient method |
| Washington Department of Ecology | Qualitative methods for ranking priorities<br>Qualitative risk estimates derived from models (dredge disposal)<br>AET (quotient) |
| Wisconsin Department of Natural Resources | Focus on aquatic wildlife; fish in surface waters program<br>Based on state water quality criteria (quotient)<br>Have modeled contaminant uptake for birds (exposure assessment) |
| Federal agencies | |
| Food and Drug Administration (FDA) | Basic quotient method<br>BC divided by SF |
| National Marine Fisheries Service (NMFS) | Focus is on physical rather than chemical stressors<br>Extensive use of existing models planned, but have also used qualitative methods<br>Qualitative method has survived court appeals |
| Army | Effect-based approach; often retrospective<br>Quotient or qualitative<br>Exploring demographic models |
| Fish and Wildlife Service (FWS) | Stress retrospective/field assessments<br>Rely on individual level<br>Interested in biomarkers for exposure assessment<br>Quotient/qualitative |
| National Ocean and Atmospheric Administration (NOAA) | Primarily retrospective (sediments) |
| Forest Service | Quotient; SF used<br>Must protect entire forest community/ecosystem; considering methods of doing so |
| Department of Energy | Quotient with SF<br>Superfund requires only proof of adverse effect, regardless of level<br>Therefore, higher level effects not priority |

Source: U.S. EPA 1991; BC = benchmark concentration; SF = safety factor

However, the U.S. EPA Science Advisory Board has recommended that the expression and communication of risks should be kept separate from the actual risk characterization (U.S. EPA 1991). Thus risks can still be quantified in the main body of a risk assessment report, even if simplified in conclusions or summaries.

Ecological models have only recently entered into the toxicological field from other fields. Thus, lack of familiarity may be a major reason for toxicologists' reluctance to use models. The National Marine Fisheries Service, which uses population models extensively for other purposes, was one of the few agencies in Table E.6 that indicated a desire to use these models in risk characterization. Population models have been widely used in assessments conducted for power plants, and it is not surprising that researchers at the Oak Ridge National Laboratory, particularly Barnthouse and his collaborators, have adapted those models for use in assessing the effects of synthetic fuel technologies.

Other current issues and deficiencies in risk characterization identified for future research include (Norton et al. 1988; Parkhurst et al. 1990; Pastorok and Sampson 1990):

- quotient methods for higher-level effects
- multiple chemicals and exposure pathways
- density-dependent effects
- the lack of models and methods for terrestrial ecosystems
- the need for more empirical models and methods
- verifying and comparing existing methods

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