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Exposure Assessment Methodologies for Humans and Ecosystems

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ABSTRACT

Health scientists and risk assessment experts are developing approaches to estimate exposure of human populations and ecosystems to environmental contaminants. Ecological scientists are exploring methodologies for estimating the exposure of ecosystems, or subdivisions within an ecosystem, to environmental stresses, while human health scientists are investigating approaches for estimating exposures to contaminants that can affect human health. Exposure assessment methods vary significantly, depending upon factors, such as the scale of the exposure, the measurement focus, and level of biological organization. The paper discusses the elements of ecological and human exposure assessment methodologies. Examples of multiple pathway exposure assessments are provided to illustrate human exposure concepts, and how they may also apply to ecosystem exposure assessments. Ecosystem and human exposure assessment paradigms are compared and contrasted with regard to the level of biological organization, source-receptor relationships, biomarkers, dose, pollutant characteristics, and modeling.

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Introduction

Human exposure is the contact between a "contaminant and the human body" (Sexton and Ryan, 1988). By extension, ecological exposure is the contact between the contaminant and an ecosystem or its components (e.g., communities, species, or individual organisms). The principal elements of exposure are the magnitude of the pollutant concentration, the duration of the exposure, and the frequency of the exposure. Human exposure assessments of these three elements include measuring pollutant concentrations, in the ambient environment, as well as in microenvironments (including outdoor, indoor, transitory, occupational, and personal). Assessments also need to characterize personal exposure scenarios, by describing activity patterns and uptake rates. Ecological exposure is the expression of the magnitude, duration, and frequency of contact between an ecological resource and a "stressor;" i.e., a physical, chemical, or biological entity that can induce an adverse response" (Risk Assessment Forum, 1992).

Risk assessors and other scientists are developing approaches to estimate exposure. Ecological scientists are exploring methodologies for estimating the exposure of ecosystems and their subdivisions to environmental stresses, while human risk assessment analysts are investigating approaches for estimating exposures to contaminants that could affect human health. Exposure assessment methods vary with the spatial and temporal scale of the exposure, the measurement focus, and the level of biological organization. This paper compares ecological and human exposure assessment methodologies concerning the types and scales of monitoring and sampling designs, the availability of models to simulate and estimate exposure, and the components necessary to calculate exposure. The National Research Council of the National Academy of Sciences (1983) developed a risk assessment paradigm with four separate steps: hazard identification; dose-response assessment; exposure assessment; and risk characterization. Regulatory agencies, like the U.S. Environmental Protection Agency (EPA), have applied this paradigm to human health risks. Each of the four steps has been subdivided further. For exposure assessment, EPA

applies the steps shown in Figure 1: source characterization: transport, transformation, and fate; pathways; environmental concentrations; and exposure measurements.

Lipton et al (1993) have questioned the appropriateness of applying the NAS paradigm to ecological risk assessment, since several "intrinsic distinctions" can be drawn between human health and ecological risk assessments. Ecological target receptors may be unknown or ambiguous and the level of biological organization is variable. Exposure assessment, however, is similar for ecological and human risk assessments (Figure 2).

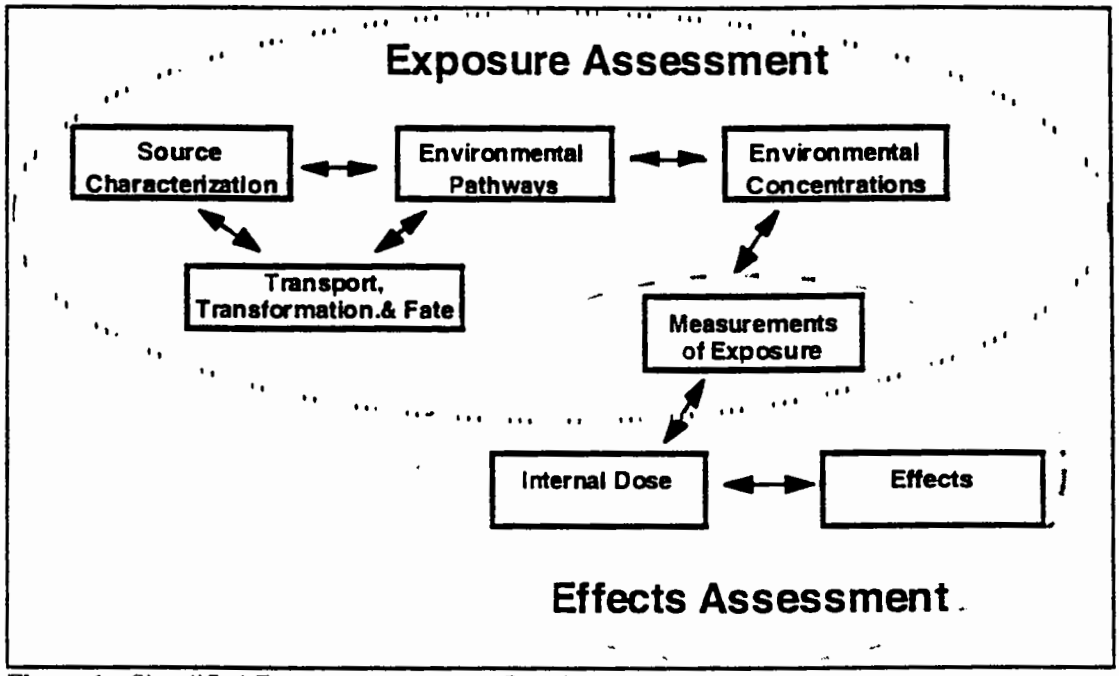

Figure 1: Simplified Exposure Assessment Paradigm.

Exposure Assessment Methodologies

Routes, magnitude, duration, and frequency of exposure are important considerations for both human and ecosystem exposure assessments. While both paradigms include measurements of pollutant concentration, the major difference rests in measurements of behavior. For humans, exposure is a function of concentration, activity pattern, and uptake (ventilation, consumption, and absorption). For ecosystems, exposure is a function of pollutant concentration in the abiotic and biotic environment, and ecological function and structure (e.g., species migration, bioaccumulation and sequestration rates, bioenergetics, succession, and nutrient cycling). Ecological exposure assessments can be complicated because changes in function and structure are expressions of both exposure and effect: i.e., functions and structures change as a result of the exposure. All pathways, e.g., ingestion, dermal, or inhalation, must be included to express exposure fully. A

single-species exposure assessment (e.g., for an endangered species) can be very similar to the multipathway, human exposure assessment described on the left side of Figure 2.

Multiple pathway field studies are designed to measure concentrations of pollutants in various environmental media. Temporal and spatial distributions of these measurements give an indication of the frequency, magnitude and duration of the exposure. The level of temporal (continuous, hourly, 12-hour, 24 hour, monthly, annual average) and spacial precision of these measurements varies depending upon the field study objectives and methods.

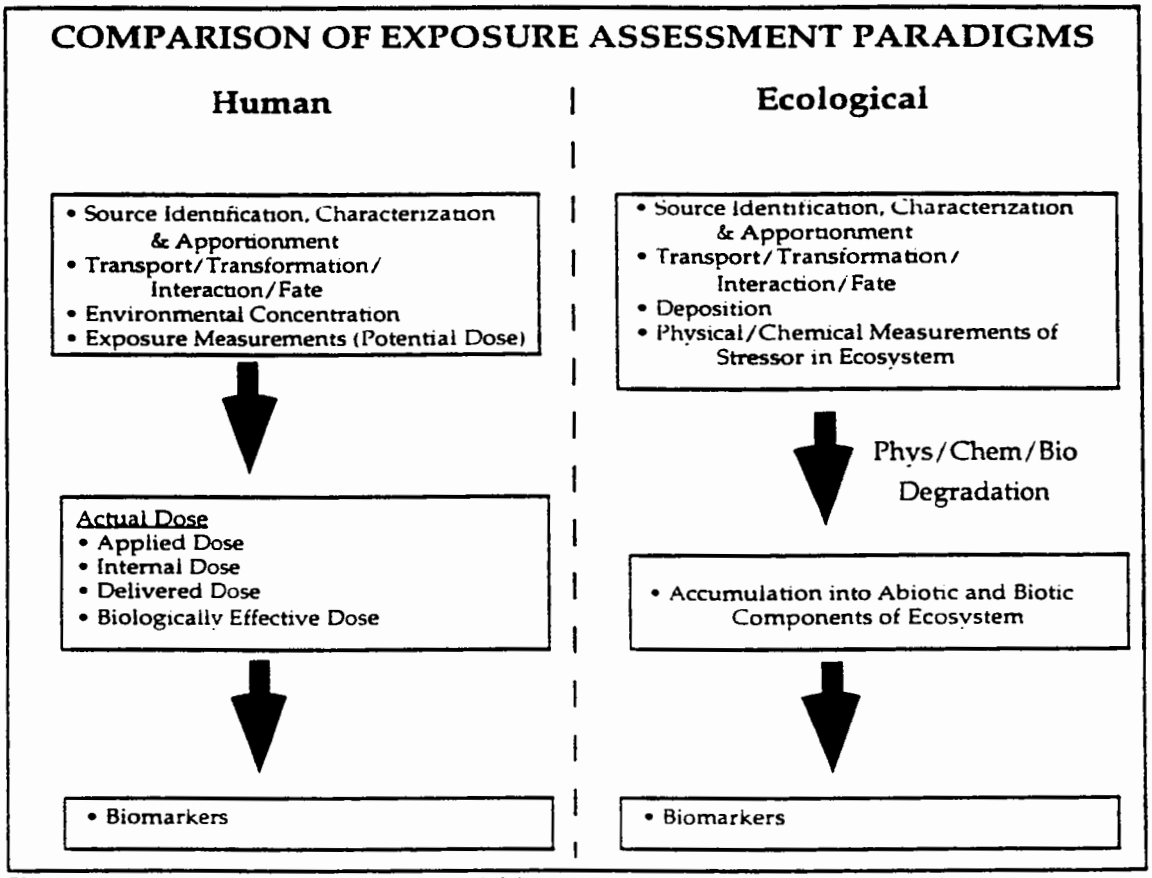

Figure 2: Exposure components of risk paradigms are similar for humans and ecosystems.

EPA developed the Total Exposure Assessment Methodology (Wallace, 1987) to estimate total human exposure using personal exposure monitors. Results from these TEAM studies indicate that a person's activities and behavior greatly affect one's actual exposure. Even when ambient concentrations are similar, activity variables, e.g., cleaning, cooking, smoking (active and passive exposure), time spent indoors versus outdoors, and transportation, can introduce considerable variability for most contaminants.

The U.S. EPA's (1990) Non-Occupational Pesticide Exposure Study (NOPES) illustrates the necessary methods to measure and estimate total exposure from the air pathways. NOPES was a multi-season study of pesticides commonly used in and around the home. The study households were selected from stratified random population samples in two urbanized areas. An embedded nine-home pilot study conducted in Jacksonville, Florida found that household dust may be a significant pathway for exposure to previously used pesticides ; e.g., Chlopyrifos, Propoxur, and Chlordane. NOPES extended the findings of other research which found indoor environmental exposures of certain pollutants to be considerably higher than outdoor exposures. Other pathways, such as diet and drinking water, can also be significant pathways for other pesticides. NOPES was successful in estimating exposure levels for populations of two urban areas of the United States, assessing the relative importance of each exposure pathway to the overall level of exposure; characterizing the components of variability in the observed exposure levels, and, in beginning to model the relationships between exposure levels, rates of use, activity patterns, and other factors that could contribute to variation in exposure levels. These results demonstrated that the multi-pathway approach can be applied to nonoccupational exposures through inhalation. The study's probability-sampling design also allowed for inferences about the distribution of exposures for populations.

The objectives tracked well with the approaches recommended by the NAS (1991) for assessing human exposure to airborne pollutants (Figure 3), illustrating the need for data from direct measurements (personal and biomarker monitoring) and from indirect data gathering methods, such as diaries and questionnaires (especially to gain knowledge about activities). NOPES characterized exposure, including seasonal variations, by monitoring and comparing outdoor, indoor, and personal air concentrations. The study also demonstrated that questionnaire-based models may be practical for particular analytes; e.g., certain termiticide concentrations were related to use and application history, age of home, and household inventory of the pesticides.

Scale of Exposure

Exposure studies can range from subcellular exposure to global. Methods for assessing exposure for an individual organism (e.g., one human being) differ from methods used to assess population exposure. Likewise, estimating exposures for a single ecosystem component; e.g., a lake or wetland, will be different from a large-scale exposure assessment of region or biome.

In the case of the small-scale assessment (residential, occupational, farms), a researcher may be able to determine signals of exposure for a wide array of contaminants, and provide detailed and specific information about a subject's activity patterns. Often, however, scientists are asked to estimate exposure of entire populations or target groups, wherein gathering detailed and specific information about the exposure of each individual in a population is scientifically and economically infeasible. Moreover, in the case of ecosystems, detailed information about individuals may have less importance than the interrelationships and diversity of a larger ecological community; true to the adage, "not seeing the forest for the trees." The hypothesis or study objective determines the scale of an exposure assessment.

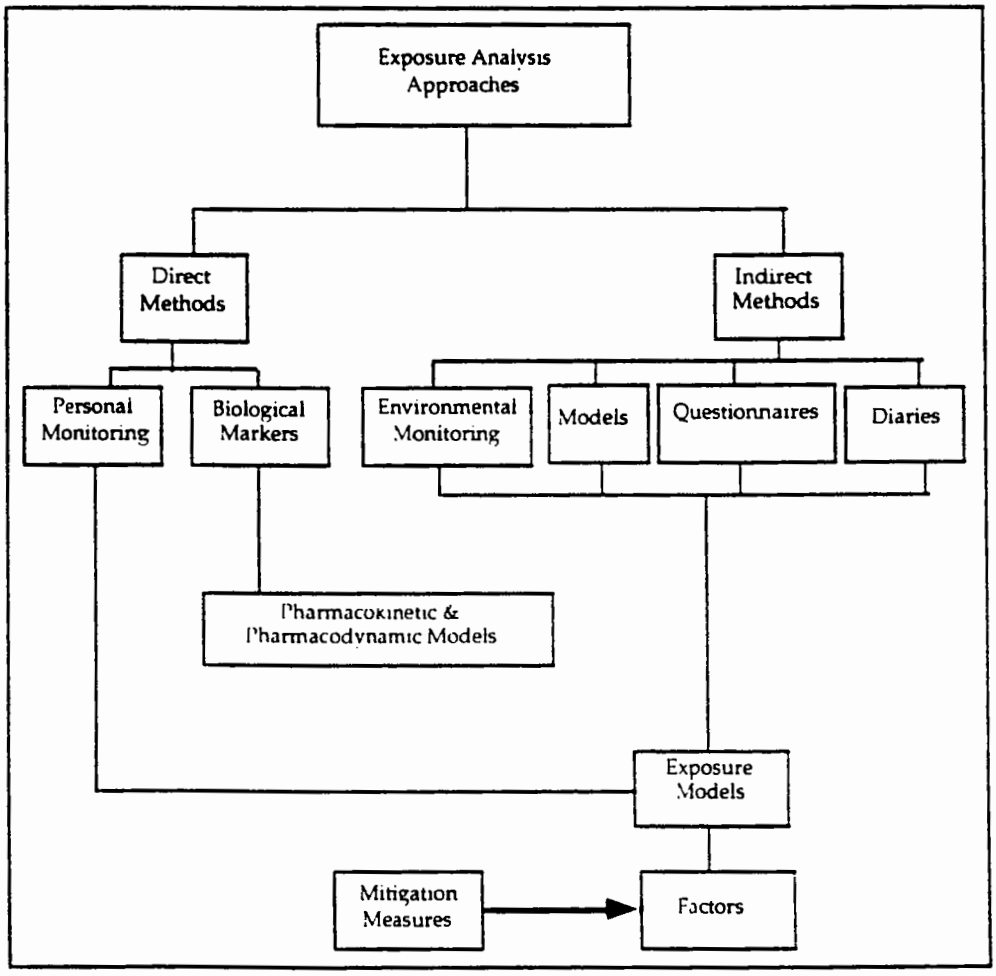

Figure 3 Possible approaches for analysis of air contaminant exposures (National Academy of Sciences, 1991). Author's Note: "Exposure Models" are inputs to "Pharmacokinetic and Pharmacodynamic Models," so a line should connect them.

Focus of Exposure Measurements

Human and ecosystem exposure measurement and assessment methods vary. Studies may be conducted to estimate the exposure of one type of receptor to a single pollutant: e.g., blood lead levels in children. A single pollutant exposure assessment can be conducted for a number of receptors; e.g., bone-lead concentrations in urban and rural school-aged children, lead concentrations in lawns bordering highways, and translocated lead in leafy vegetables downwind from an industrial source. The process is far more complex when a number of pollutants and receptors are included in risk assessment; e.g., ecological and human exposure to dioxins and toxic metals near potential agricultural, industrial, and transportation sources.

The measurement focus varies considerably among exposure assessments. Ambient outdoor, indoor, and personal exposures are directly measured or input into models. "Direct" measurements are usually used to make spatial and temporal inferences about pollutant concentrations, since the measurement is a value at one point for one time period. Stationary monitoring devices provide outdoor and indoor measurements. Passive (diffusion) and active (constant flow) sampling devices are used for personal and microenvironmental measurements. Recently, researchers have deployed these devices to enhance ambient monitoring data and to provide average environmental exposure estimates for ecosystems, especially for forest stands.

Source-Receptor Assessment

Determining source characteristics and the transport and transformation of pollutants is similar for human and ecological exposure assessments. Various methods for identifying and apportioning the sources are available, including emission inventories, source-receptor models, and actual measurements (e.g., stack tests, remote sensing, and continuous emission monitoring). Emission inventories are often derived from calculations of fuel or feedstock and the manufacturing processes taken from emission forms completed by the operator; e.g., incinerator operators provide information about the type of fuel; amount and type of feedstock; a description of the combustion processes; and the types of stacks and vents at the facility, which is used to generate the emission inventory. This information can be highly uncertain and is not sufficiently specific to characterize potential pollutant sources.

Stack tests, such as dilution samplers, are much more reliable than emission inventories, but are costly and require on-site access. Actual measurements of stack emissions are necessary to apportion the sources of pollutants to which a receptor is exposed (Figure 3). Temporally and spatially precise measurements are needed at the

source (i.e., for "source signatures") to be coupled with ambient measurements of chemical species that are "markers" of particular sources. For example, the U.S. EPA's chemical mass balance model (CMB 7.0), developed by Watson et al (1990) is used by the Agency to "identify and the presence of and to quantify source contributions to receptor concentrations." Dispersion models are also useful exposure tools which require emission rates be estimated and combined with meteorology, and transformation algorithms to estimate the relative contribution of sources to measurements of pollutant concentrations at a receptor. If variability and uncertainty are high for emissions, as is common for source information derived from inventories, the dispersion model-derived source-receptor relationship is also highly uncertain and variable.

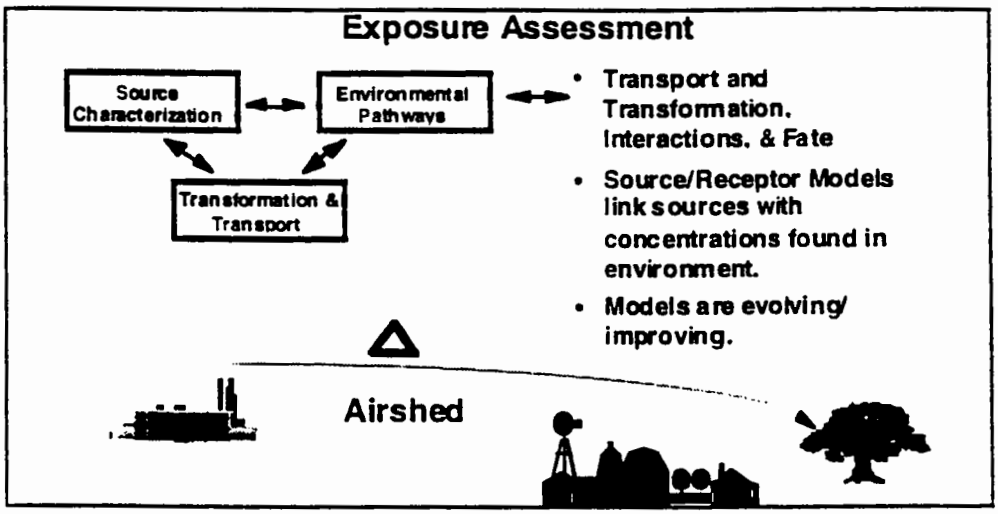

Figure 4: After emissions are released, they undergo physical and chemical transformation before being deposited. Receptors can be human or ecological. The level of biological organization can be subcellular to regional. Human exposure assessments are often conducted at the population or subpopulation level (e.g., cancer risk per million in the United States). Ecological exposure assessments are conducted at many different levels, but regulatory and natural resources agencies often are interested in community level risk (e.g., loss of biological diversity in forest stands or wetlands).

Some promising chemical markers and their associated source categories are shown in Table 1. The total, upper-bound contribution of the potential source on the measured ambient concentration can be obtained by multiplying the measured ambient concentration of the marker species by the characteristic factor (i.e., the reciprocal of the marker's per cent abundance in the source's emission (i.e., listed in "Source Profiles," such as the U.S. EPA's VOC/Particulate Matter Speciation Data System, Version 1.4.). For example, acetylene is one of the common volatile organic compounds (VOCs) found in motor vehicle tailpipe emissions. On average, acetylene represents 4 ± 2 % of total VOCs in exhaust in

the U.S. If ambient acetylene is measured to be 3 $\mu g/m^3$, then the upper bound estimate = 3 $\mu g/m^3$ (25 ± 12) = 75 ± 36 $\mu g/m^3$. Therefore, if total ambient VOCs = 150 $\mu g/m^3$, the greatest possible motor vehicle contribution is about one-half (75/150) of all VOC sources at this ambient site.

Aerosols	**Dominant Source**
Na, Cl	Marine
K (soil-corrected), Cl, ^{14}C	Wood Combustion
Al, Si, K, Ca, Ti, Fe	Soil
Zn, Pb, Sn, Sb, Cl	Incinerators
V	Electric Utility Oil Combustion
Gases	
CO, various VOCs	Motor Vehicles
xylene	Industrial Solvents
ethane, propane	Natural Gas
isoprene, a-pinene, b-pinene, ^{14}C	Biogenic Emissions

Table 1: Selected examples of presently available chemical marker species. The dominant source is airshed dependent; i.e., in addition to indicating a dominant source, measurements of marker concentrations in ambient air may represent products of transformation or background concentrations. For example, Na and Cl-rich particles not near marine water bodies may be indicators of extraction or transportation activities that emit salt. High concentrations of Fe and Al may not be re-entrained dust, but may be indications of smelting activities. Therefore, an inventory of source types in the airshed should complement the receptor modeling.

The physical and chemical characteristic must also be considered when determining potential sources of measured ambient contaminant concentrations. Figure 5 illustrates three different idealized bimodal distributions for particles. The distributions can provide weight-of-evidence for whether the particles are anthropogenic or natural in origin. "Routine screening of certain indicators" in ecosystems provides an estimate of "the actual threats to the condition" of those ecosystems (Messer, 1990). Such screening for the presence of pollutants can be an indication of ecosystem exposure; however, the chemical and physical characteristics of a contaminant can ultimately determine actual exposure. For example, outdoor concentrations of fine particles near a home can be similar to fine particle concentrations inside the home, but ozone (O_3) concentrations may be much lower inside, because O_3 readily absorbs on surfaces. Aerosol acidity may be lower indoors due to higher concentrations of ammonia that buffer the acid.

Physical characterization techniques, such as scanning electron microscopy and X-ray fluorescence, can help to verify linkages to source categories because particles emitted by different types of combustion display unique morphologies (e.g., spheres, chains, and clusters). Analyzed together, chemical composition and physical characterization can provide weight-of-evidence for linking source emissions to measured ambient concentrations.

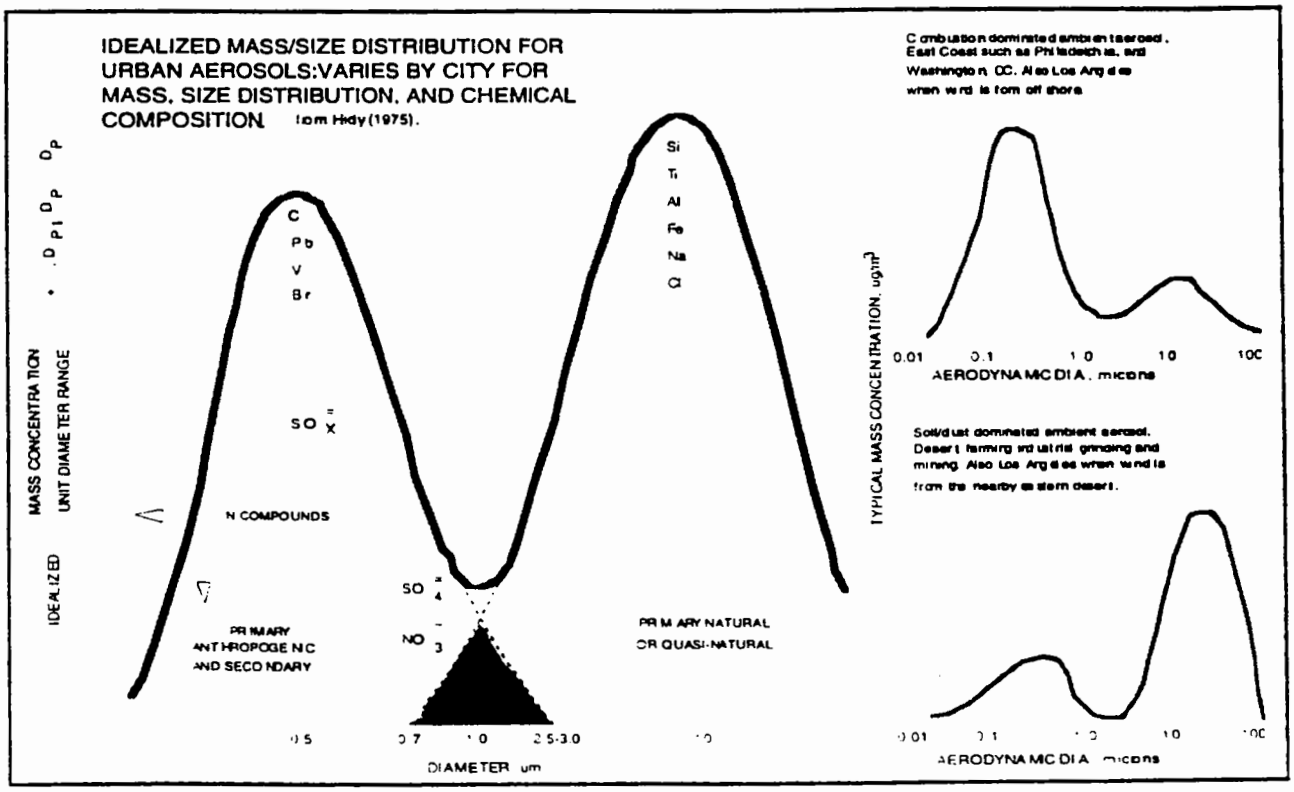

Figure 5: Particles can have a bimodal distribution, originate from multiple sources, show dynamic growth and reactivity, and are carriers of other pollutants (Hidy, 1975). The upper right diagram is typical for a anthropogenic-sources, while the bottom distribution is typical for natural (e.g., re-entrained soil and mining activities).

Dose

The nexus between exposure and effect is "dose." For human exposure assessments, the U.S. Environmental Protection Agency (1992) classifies dose as: potential dose (D_P); applied dose (D_A); internal dose (D_I); delivered dose (D_D); and biologically effective dose (D_{BE}). Figure 11 shows the pathway from an organism's first contact with a substance (D_P) to its intake, absorption, and metabolism (D_A, D_I and D_D) to its effect on the target organ (D_{BE}). Measurements of D_P can often provide a reasonable

estimate of exposure: i.e., the concentration of a contaminant around an organism. For airborne contaminants, D_A is a function of concentration, time, and ventilation. It is difficult or impossible to measure D_{BE} directly, so D_A, D_I and D_D are most often expressed by biomarkers, i.e., "indicators of changes or events in human biological systems" (NAS, 1991). Biomarkers may either be the contaminant itself or metabolites indicating exposure to the contaminant; e.g., increased concentration of cotinine (a metabolite of nicotine)in blood resulting from exposure to tobacco smoke. Similarly, biomarkers in ecosystems are "biochemical, physiological, or histological indicators of either exposure to or effects of xenobiotic chemicals at the suborganismal or organismal level" (Huggett, et al, 1992).

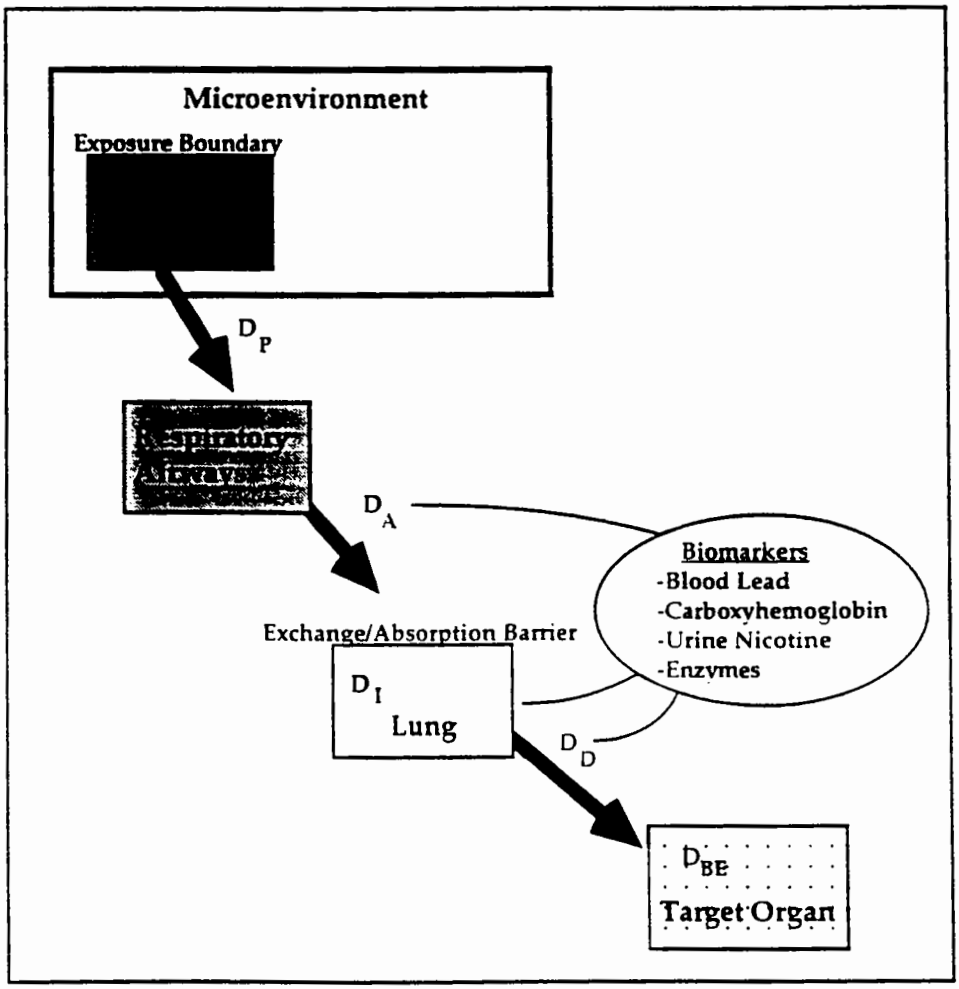

Figure 6: U.S. Environmental Protection Agency s Schematic of Dose and Exposure for Airborne Substances (Modified by McCurdy, Draft in Process). Biomarkers can be substances to which the organism is exposed or metabolites (e.g., enzymes) indicating exposure.

Biomarkers can also apply to ecological exposure, although they are not often classified as measures of dose ("biotic and abiotic accumulation" in Figure 2). For

example. Hunsaker, et al (1990) suggested measuring cholinesterase levels and porphyrin accumulation to indicate the level of ecosystem exposure.

Comparison of Human and Ecological Assessments

A major difference between human and ecological exposure paradigms is their level of biological organization; i.e., population exposure for one species (human) versus community (several species), association, and population exposure for ecological risk assessments. Human risk assessments express the likelihood that an adverse outcome will result from a given hazard; e.g., 10^{-6} chance of ovarian cancer in a population exposed to a particular pollutant. Ecological risk assessments are also expressions of the likelihood of an adverse outcome, but the expression depends upon the "environmental value" of concern; e.g., biological diversity, sustainability, and aesthetics (Environmental Monitoring and Assessment Program, 1993). Scientists are currently debating the usefulness of ecological risk assessments, with many instead favoring ecological benefits assessments. That is, benefits, can be gained or lost, depending on regulatory, management, and other decisions. Both risk and benefit assessments, however, require exposure assessments.

A number of similarities exist between human and ecological exposure assessments. Both are often concerned with sensitive subpopulations, many pollutants are both human and ecological stressors, and ambient measurements for some pollutants can be indicators of both human and ecosystem exposure (e.g., ozone).

Passive monitors may improve useful data for both human and ecosystem exposure assessments, since they provide an inexpensive means of gaining coverage over large areas with reasonable accuracy for several gaseous pollutants ($\pm$ 20% for nitric oxide, ozone, and sulfur dioxide). The use of passive devices may even provide greater potential for ecosystems than for human exposure, since the need for more temporal precision may often be less for ecosystems than for human; i.e., if accumulation and degradation of a contaminant are the major areas of concern, a weekly average may be sufficient, whereas, hourly averages may be critical for human exposure assessments.

Both assessments can benefit from the use of models, although modeling ecosystem exposure pathways can be highly complex and includes much uncertainty. However, an increased understanding of fluxes and cycling of nutrients and contaminants, bioenergetics, and bioaccumulation will improve the application of ecosystem models.

Human Microenvironmental Exposure Models

Exposure models vary by scale (personal, microenvironmental, indoor, site-specific, regional), and type. Table 2 compares 20 human exposure models insofar as they incorporate ventilation rates, outdoor and indoor microenvironmental concentrations, and human activity patterns. Presently, new models are being used for carbon monoxide, oxides of nitrogen, ozone, lead, particulate matter, sulfur dioxide, and hazardous pollutants. The table illustrates that many have not yet been validated, or have been validated for limited microenvironments; e.g., within an automobile. However, the application of human exposure models is expanding rapidly and their reliability is being improved.

Conclusions

Exposure assessment can be similar for humans and ecosystems, although the level of biological organization is often different for the two types of receptors. Data gathered from the field may be used for both human and ecosystem exposure assessments. This seems to indicate a likelihood for an increase in the number of combined human/ecosystem exposure studies. The information about both receptor types would be enhanced, and the understanding of the interrelationships between humans and ecosystems may be better understood. Data and assessments may become more interchangeable insofar as they are used to interpret to protect both public health and the environment.

New methods for measuring, modeling, and assessing exposure are presently being developed. Passive monitors may prove to be valuable for ecosystem exposure estimates. beyond their uses in human microenvironmental monitoring, since even large averaging times may sufficient for many ecosystem exposure scenarios. The body of knowledge is growing beyond simple ambient measurements to personal and indoor monitoring. Although the science has emerged relatively recently, models are increasingly providing more reliable exposure information for a greater number of microenvironments. This trend may lead to greater certainty in characterizing and predicting exposures.

Enhancements in exposure assessment should lead to improved, scientifically-based mechanisms and programs to reduce exposures of humans and ecosystems to harmful substances and other stresses.

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Name of Model	Maintain Time Series?	Account for V_E?	Outdoor µe Conc.	Indoor µe Conc.	Human Activity/ V_E	Validated Model?	User Friendly?	Citations
CARBON MONOXIDE								
Convolution	No	No	3	5	B	No	No	Duan (1989)
CO/Regression	No	No	3	5	A	No	Yes	Schwab (1989)
SHAPE	No	No	3	5	B	Limited	No	Ott (1984)
pNEM/CO	Yes	Yes	2,3	7	F	Limited	No	Johnson, et al (1992)
NITROGEN DIOXIDE								
SIMSYS	No	No	3	5	B	No	No	Ryan (1986)
REHEX	No	Yes	2	6	D	No	No	Lurmann, et al (1989)
NO2/Regression	No	No	3	5	A	No	Yes	Drye, et al (1989)
OZONE								
SAI/NEM	Yes	Yes	1	7	D	No	No	Hayes, et al (1984)
REHEX	No	Yes	2	7	D	No	No	Lurmann, et al (1989)
pNEM/O3	Yes	Yes	2	7	D	Limited	No	Johnson, et al (1993)
EPEM	No	Yes	2	-	D	No	No	Johnson, et al (1992)
LEAD								
Pb-NEM	No	No	4	6	A	No	No	OAQPS (1989)
IEUBK	No	No	4	6	A	No	Yes	Lead Work-group (1994)
RESPIRABLE PARTICULATES								
THEM	Yes	No	2	7	C	No	No	Klepeis (1994)
SULFUR DIOXIDE								
SO2-NEM	No	Yes	1	-	A	No	No	Biller et al (1986)
HAZARDOUS POLLUTANTS								
HEM	No	No	4	6	A	No	Yes	Radian (1985)
HAPEM	No	No	2	6	C	No	No	Johnson (1992)
AERAM	No	Yes	4	-	-	No	No	Eschenroder et al (1985)
SHEAR	No	No	4	-	-	No	No	Anderson & Lundberg (1983)
BEAM	No	Yes	1	6	-	No	Yes	Behar et al (1994)

Table 2: Attributes of selected air exposure models (After McCurdy Draft in Process).
Notes: OUTDOOR µe: 1 = Use fixed site values as a surrogate. 2 = Use "adjusted" fixed site µe values. 3 = Monitor outdoor µe concentrations. 4 = Model outdoor µe concentrations. INDOOR µe: 5 = Measure indoor µe concentrations. 6 = Use indoor/outdoor ratios + indoor sources (if any). 7 = Use mass-balance model that includes indoor sources (if any). 8 = Use regression equations developed from indoor µe measurements. HUMAN ACTIVITY/ VENTILATION: A = Use of aggregate data and/or V_E. B = Simulate transitions: ignore V_E C = Sample from activity data: ignore V_E. D = Sample from joint activity/:V_E data.

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16. ABSTRACT

Scientists and risk assessment experts are developing approaches to estimate exposure of human populations and ecosystems to environmental contaminants. Ecological scientists are exploring methodologies for estimating the exposure of ecosystems and their subdivisions to environmental stresses, while risk analysts are investigating approaches for estimating exposures to contaminants which could affect human health. Exposure assessment methods vary significantly, depending upon factors, such as the scale of the exposure, the measurement focus, and whether the measurements are actual expressions of exposure or part of an algorithm to indicate exposure. The paper discusses the elements of ecological and human exposure assessment methodologies. The Nonoccupational pesticide Exposure Study provides an example of multiple pathway exposure assessment. Ecosystem and human exposure assessment paradigms are compared and contrasted with regard to the level of biological organization, source-receptor relationships, biomarkers, dose, pollutant characteristics, and modeling.

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