ENVIRONMENTAL RISK ANALYSIS;

A DECISION ANALYTIC PERSPECTIVE

A Discussion of Modeling Issues for the Management of Risks from Toxic Substances in our Environment

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The risk arising from toxic materials in our environment has emerged as one of the most important problems of modern civilization. The issues that must be considered for the management of such risks are complex and involve substantial uncertainty. They cut across the fields of biology, engineering, geo-science and philosophy. This report is an overview, from a decision analytic perspective, of the modeling issues involved. Emphasis is on the kinds of mathematical modeling approaches that are used (or might be used), though details of the models are not addressed.

1. Some historical perspective

1.1. The problems are not entirely new.

While the use and synthesis of chemicals has certainly intensified in the latter half of this century, it should not be supposed that the problems are entirely new. An article by Covello and Mumpower (1985) does a nice job of putting the problem into a historical context. Some of the situations quoted by them are amusing in terms of their relevance to modern problems. Consider, for example, the following sequence of events (quoted by Covello and Mumpower):

1. There is a major air pollution problem in a large city due to the use of a polluting fuel. In response to pressure, the government establishes a commission to study the problem.

2. In response to the commission report, industry groups develop a set of voluntary controls.

3. Failure of the voluntary controls leads to governmental banning of the offending fuel.

4. The government sets up a study commission to determine why the ban is not being observed.

THE FUEL	- Soft coal
THE CITY	- London
THE INDUSTRY GROUP	- smiths
THE GOVT. AUTHORITY	- King Edward I
THE DATES	- 1285, 1298 and 1307

Or, consider the following statement:

"…. it seems that water should not be brought in lead pipes if we desire to have it wholesome."

The Roman, Vitruvious
1st century B. C.

Two thousand years later, plumbers were installing plumbing with lead-containing materials.

1.2. What's different now?

Although these problems have been with us for many centuries, there are several factors that are more recent.

1.2.1. Most obviously, human society and industry have developed so that the number and amount of chemicals introduced into our environment increases at a high rate. Current estimates of number of chemicals in current use range from70,000 to 100,000, with about 1,000 new ones being introduced each year.

1.2.2. Second, our expectations about health and life expectancy have increased. The following table shows life expectancy at birth (quoted by Covello and Mumpower)

prehistoric	18 years
Roman Empire	20 to 30
Middle ages	33
1900, U. S.	51 female
	48 male
1975, U. S.	75 female
	66

That is, we *expect* to be safer. In 1900, leading causes of death in the U. S. were infectious diseases. By 1940, the leading causes of death were chronic degenerative adult diseases - heart disease and cancer.

Moreover, we now allow ourselves the luxury of worrying about environmental effects that do not seem in any way to threaten human physical health.

1.2.3. Change and increase in the nature of the hazards:

- nuclear power
- radioactive waste
- synthetic pesticides
- industrial chemicals
- supertanker oil spills
- chemical plant and storage accidents
- recombinant DNA laboratory accidents
- ozone depletion due to emissions of fluorocarbons
- acid rain due to burning of fossil fuels

Most of these hazards are derived from science and technology (contrasted with being "acts of nature or of God").

Most, as Covello and Mumpower point out, are:

- latent (the effects are not immediately obvious)
- effects are long term, and changes to the environment may be irreversible, at least on any reasonable time scale.
- Some are potentially catastrophic, either locally or even globally.
- involuntary

1.2.4. Because of the complexity of modern society, and the degree to which the polluting activities form part of the fabric of that society, regulation and control become extremely complex.

1.2.5. Modern communication and news reporting cause us to be aware of dramatic polluting events, wherever they occur in the world. We hear about chemical spills in Virginia, problems associated with chemical dumps in Love Canal, and of chemical accidents in India. Public awareness is thus increased, but the increase is clearly selective, stressing problems that are deemed "newsworthy".

The following are differences in the way we can address the problem

1.2.6. Modern understanding of cause-effect relationships allows us to formulate and model the problems in a fundamentally and conceptually different way. We are no longer inclined to ascribe bad health effects to supernatural causes.

1.2.7. While uncertainty has always been a fundamental part of the problem, the formalization of the ideas of probability, which began in the 1700's, allows a more disciplined and productive approach to the consideration of risk, and to decision making in the face of risk.

2. International dimensions of the problem.

It is not to be thought that these are uniquely U. S. problems. Problems are reportedly severe in Eastern Europe, although before the demise of the communist distatorships, it was not permissable to recognize the existence of such problems. A Regional Environmental Center has been set up in Budapest to coordinate the study of problems in Eastern Europe. North America

and Western Europe are probably in better shape than other parts of the world because of recognition of the problems, and commitment to at least some action. One of the accomplishments of the "turbulent 60's" in the U. S. was the creation of the EPA.

In developing countries one may find severe problems associated with pesticides and fertilizers, as well as other commonly used chemicals (Pe Benito Claudio 1988). Pesticides and fertilizers are imported and used without adequate controls or even warnings. Sophisticated technologies are imported, introduced in haste without proper safeguards. Technologies as well as chemicals which are deemed unacceptable in more developed nations are transferred to developing countries without regard to the risks.

3. A case history (Kirkwood, 1985)

Let's look at a case study more recent than the one in 13-th century England. The year is 1982, Livingston La., a small town near Baton Rouge. Forty-three cars of a freight train derail and catch fire right along Main street. A mixture of volatile and hazardous petroleum products and other chemical substances is released. The list includes:

metallic sodium
vinyl chloride
perchloroethylene
methyl chloride
styrene monomer
phosphoric acid
toluene diisocyanate.

You can't use water on this mix – it takes several days to put the fire out, while the stuff leaks into the soil. Now what?

Removal of the contaminated soil began, but then it became obvious that this was a very costly endeavor, and might even be physically impossible. What to do and how far to go then became a matter for negotiation between the governmental regulators, and those responsible for cleanup.

3.1. Problem Analysis

- *First step - hazard identification.* Of all the materials present, the most hazardous from a long term standpoint was judged to be perchloroethylene, PCE, suspected of being a carcinogen. It is highly volatile, so it would not persist in the soil surface or surface water long enough to be a threat. The danger was contamination of ground water, which would be tapped by wells for drinking water.

- To assess the *magnitude of this risk,* we need to know
  - ★ The direction and rate of movement of the PCE through the soil to groundwater
  - ★ Movement of PCE with the groundwater to drinking wells.
  - ★ Health effects of drinking water with PCE.

- Modeling *direction and rate of movement* through the soil and groundwater requires detailed consideration of the surface topography (gentle southerly slope), type of layering of the sub-soil layers and the physical characteristics including rainfall infiltration rate, porosity, particle density, etc., etc. The group doing the modeling seemed, from the report, to be fairly well satisfied with the adequacy of the physical models available. However, determination of the values of the specific parameters that applied in this particular case was somewhat more difficult, especially since the relevant area is not uniform.

- Modeling the *health effects* is subject to even greater uncertainty, since at the time, the main studies available were a study on mice, in which PCE caused cancer, and a study on rats, in which it did not. The EPA at the time was using a linear nonthreshold model for PCE with a coefficient of $10^{-6}/0.88$. That is, if p is the added lifetime probability of contracting cancer due to the water, then the relation is

$$p = (10^{-6}/0.88)C,$$

where C is concentration in the water in ppb.

Assuming a lifetime of 70 years, then someone born at time t_b would be subject to an average lifetime exposure based on a concentration of

$$\bar{C} = (1/70) \int_{t_b}^{(t_b+70)} C_t dt.$$

Here, C_t is the concentration in the well water at time t.

- What about the *concentration in the well water*? First, there were no wells at the time that drew water from the contaminated layer. The municipal water came a much deeper well that was not in danger.

  ★ So, one alternative was to simply prohibit wells from being sunk over the next several decades, while the problem would persist.

  ★ A second alternative was to estimate where people were likely to drill wells, and peg the risk analysis to the most likely locations.

  ★ A third alternative, the one chosen, was to say that the risk must be reduced below some acceptable level for a well drilled any place in the area. This meant estimating the worst case location, and gearing the cleanup to that location.

- What *criterion* to use?

  Management and the regulators agreed that a target would be to reduce concentration to the point at which an individual who took all of his or her drinking water from a well at the worst area would be subject to an increased lifetime probability of cancer of at most 10^{-6}.

- This would mean, using the given dose/response relation, an average concentration of 0.88 ppb over the next 70 years. However, evaluating the trajectory of C as a function of time over 70 years is subject to staggering uncertainty. Possibly the most uncertain value in the analysis is related to the health effect. This value, however, was the one accepted without examination. Uncertainties about the hydrogeological parameters were evaluated and described with probability distributions, which were then used as the basis for Monte Carlo simulation. Whre uncertainties were too great, additional data was gathered.

- **The outcome.**

The then current levels of the PCE were clearly in excess of the health criterion. Two management options were considered:

★ Excavate material from the site until a sufficient amount was removed.

★ Construct a containment system around the site which would prevent the material from moving into the groundwater.

The first alternative was ruled out because it was much more expensive, and, according to the author of the report, was possibly even infeasible. The decision was made to implement the second alternative.

3.2. Note the sequence of steps:

- Identify the hazardous chemicals
- Model transport and distribution in space and time
- Model the exposure: concentration in well water and amount consumed
- Model the health effect as a function of the exposure
- Identify decision alternatives
- Identify decision criteria

4. Decision analytic perspective.

Although it is often not made explicit, the purpose of a risk analysis or risk assessment is to help make some decision. It will therefore be useful to introduce some of the fundamental concepts of decision analysis.

4.1. Components of a decision.

The essential components of a *decision* about the system might be listed as follows (Gold, 1989):

- A set of decision alternatives $\{d\}$.
- A set of possible outcomes (consequences, resulting scenarios, ...), $\{c\}$
- A value or utility function, which assigns a relative degree of desirability to each outcome, $c \rightarrow v$

- A probability distribution over the set $\{c\}$, or, equivalently, a probability distribution over the values for v.

  Note that this probability distribution depends on choice of d. We can not choose the outcome c; ***through the choice of d, we choose a probability distribution on $\{c\}$.***

- Which probability distribution is best?

  It turns out that we can define the value or utility function U in such a way that it becomes reasonable to order the desirability of probability distributions based on the associated expected value $\mathcal{E}(U)$ (DeGroot, 1970).

- The final ingredient in the process is some way of handling new information. There are basically two things we want to do with information

  ★ determine in advance if it is likely to be worth the time, effort and cost of getting it, and

  ★ once we have it, use it to revise our probability distributions.

4.2. Hazard vs. risk.

The literature on risk analysis makes an important distinction between hazard and risk. Both of these terms specifically refer to possible bad or adverse outcomes.

The term hazard usually refers to the existence of some danger. For example, a large rock in a body of water might be termed a boating hazard. It is not a risk, unless there are boats in the area. Moreover, the risk might be reduced or eliminated by creating some sort of boundary which keeps boats away. The rock would still be a hazard, however.

Formally, we can define the hazard as the set of possible bad outcomes, or sometimes (Kaplan and Garrick, 1981) as the set of pairs,

$$H = \{(c_i, v_i)\}.$$

The *risk*, would be defined by adding the probability; that is, as the set of triples,

$$R = \{(c_i, v_i, p_i)\}.$$

[In both of these, the inclusion of the c_i is actually redundant.]

The essential point to notice is that without the element of uncertainty, there is no risk. A disaster which will occur for certain is not a risk. Moreover, we have not defined the risk until we have evaluated the uncertainties.

4.3. If this set of triples (of ordered pairs, (v_i, p_i)), focusing on adverse consequences, defines the risk, the decision analytic perspective extends the framework in two ways.

- First, it extends the framework to include good as well as bad outcomes, since we are often in the situation of asking, "is the risk worth taking?"

- Second, it adds consideration of a criterion of telling when one probability distribution is better than another.

4.4. Structuring a decision analytic risk study. With this notation, a decision-based risk analysis would need to go through the following phases (see Figure 1):

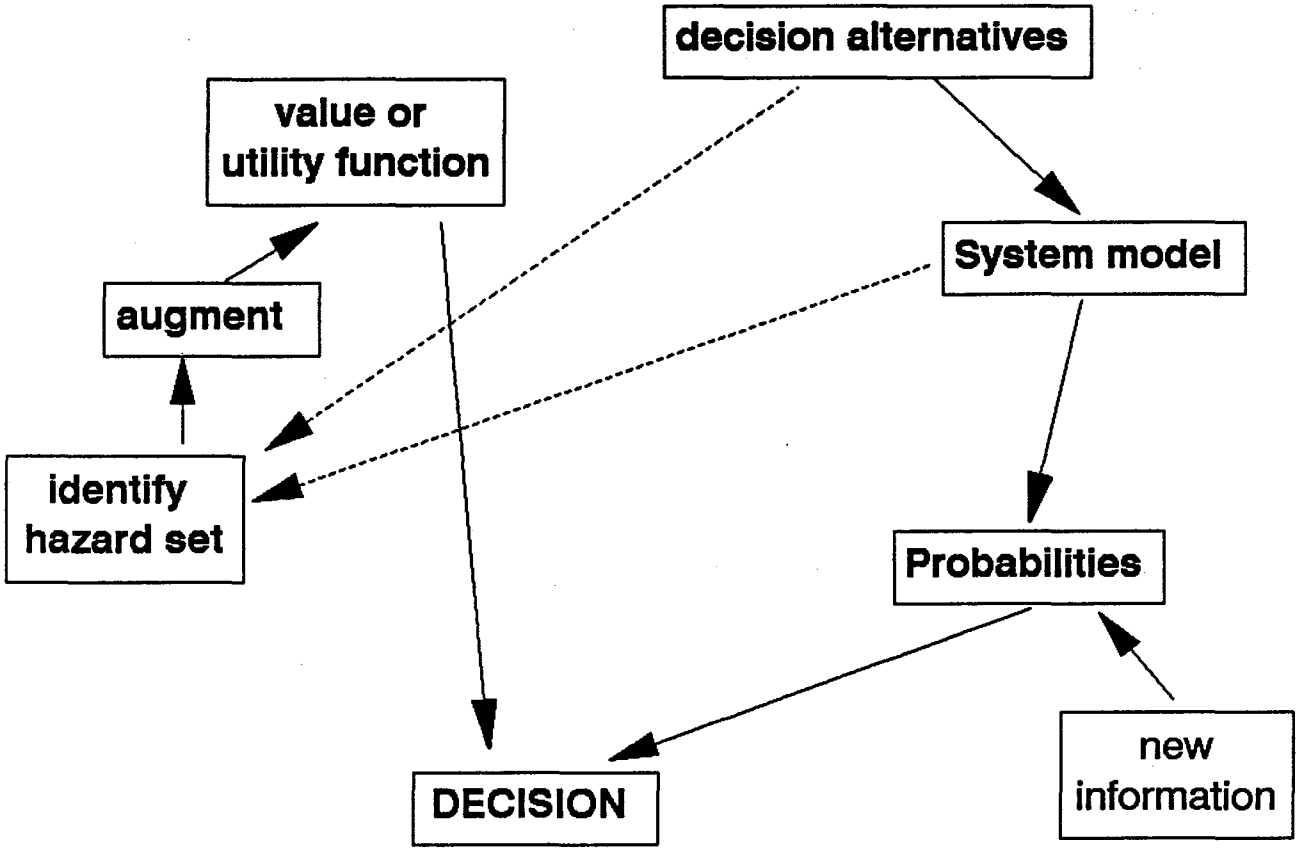
a) Identify the hazard set, $\{c\}$; that is the relevant set of *undesirable* outcomes possible.

b) If appropriate, augment this by the set of *all* outcomes, since any decision will balance good vs. bad outcomes.

An example might be a decision as to the development of some new technology, for which we might wish to weigh risk of adverse consequences against possible benefits.

Other outcomes may be introduced by a particular decision alternative being considered. [For example, in the Livingston train wreck, there might be other effects of excavating, or of erecting a containment barrier]

c) Determine the relevant value or utility function.



1. REPRESENTATION OF THE RISK ANALYTIC DECISION PROCESS

d) Identify the set of decision alternatives.

e) Develop a system model which relates the decision d to a probability distribution over the outcome set. Usually this has involved:

- ★ Develop a deterministic model, using best guess values for parameters and system variables.
- ★ Determine which are the variables for which uncertainties are most crucial.
- ★ Use available information to develop probability distributions for these variables, often followed by a Monte Carlo simulation.

f) Estimate the value of obtaining new information, and balance that against the cost of doing so.

g) If new information is estimated to be worth getting, do so, and use it to revise the earlier probability distributions. Otherwise, use the probability distributions together with the value or utility function to arrive at a decision.

4.5. Risk analyses reported in the literature generally address the hazard identification, modeling, and sometimes the probabilities. The analysis quoted above of the Livingston train accident was done by a noted decision analyst.

5. The decisions that might be involved.

In the following section, we begin considering issues involved in the modeling processes. Since our modeling effort is to be designed as part of decision analysis, it is well to have some list of categories of decisions that we might be dealing with.

5.1. Choice of technologies in some industry, based on environmental risk.

5.2. Government regulations which restrict the use or release of suspect chemicals or technologies;

5.3. Choice of technologies and of techniques to reduce or to mitigate existing risks (examples are: cleanup of hazardous chemical sites; repair and maintenance of a railroad over which hazardous materials will be shipped; maintenance schedule for nuclear plants);

5.4. Selection of sites for potentially hazardous activities.

5.5. Another type of decision is encountered if we are considering ecological effects, and that is, the degree to which an attempt will be made to restore a local ecosystem to its pre-exposure condition, once the contaminant has been cleaned up or removed. Changes that may have occurred, for example, include

- loss of species from the ecosystem,
- change in relative abundance or of age structure of the species,
- alteration of the habitat.

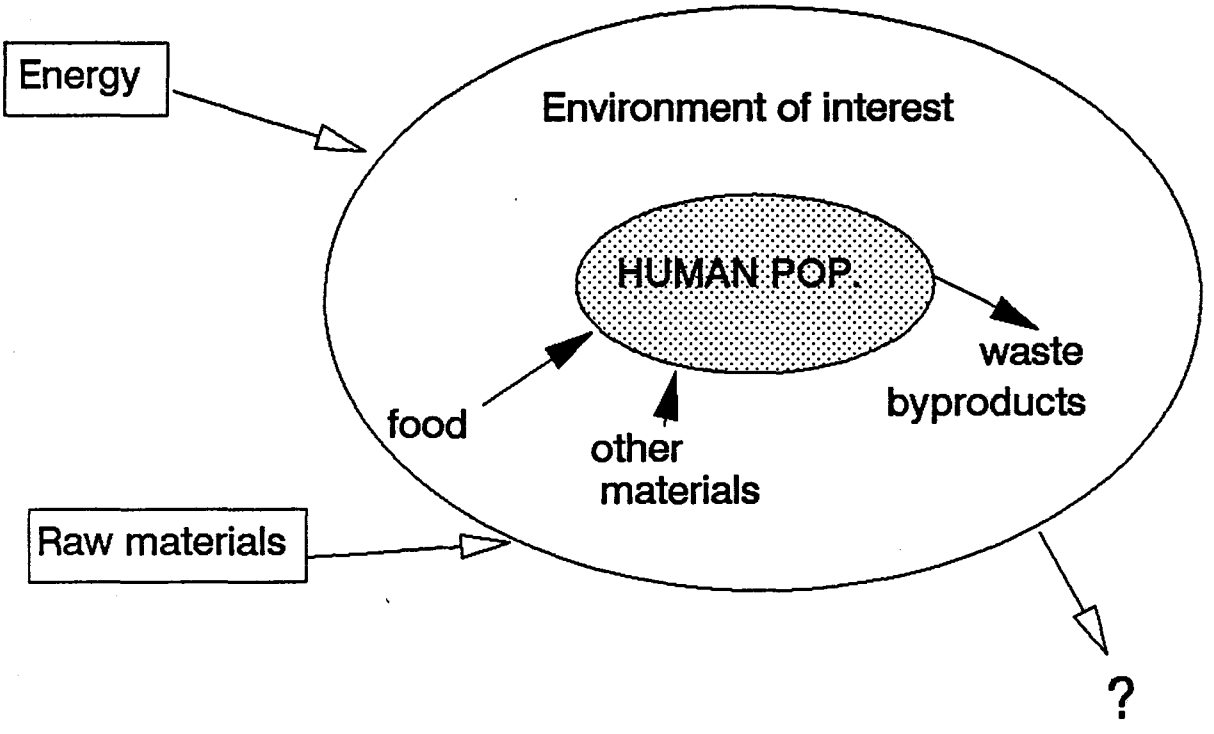
In some cases, the system will recover spontaneously if the damage has not gone too far. For example, a material which reduces egg shell thickness may make it impossible for a species of bird to breed. Once the material is removed from the environment, the population may recover, providing the condition has not gone on too long.

One problem that may be encountered here, is that even if a decision is taken to restore the ecosystem to its original condition, we may not know what that was.

6. Modeling the System dynamics

6.1. First, what do we mean by the word, "environment". In system modeling terminology, one of the first activities is to separate the system from its environment. We may then model the system structure, and the system dynamics; the system may exchange matter and energy with its environment, and the rate at which it does so may be part of the model. By definition, the dynamics of processes within the environment are not included. If the dynamics of processes within the "environment" are specifically modeled, then it becomes part of the system.

We are obviously using the term, environment, in a different way. In fact, we are referring to *our* environment, or more precisely, the environment of the human population (Figure 2). Note, however, that even the question of defining the system vs. the environment itself becomes a value-laden question.



2. THE HUMAN ENVIRONMENT AND ITS ENVIRONMENT

The important state variables that describe the human population describe a complex space. It may include food, health, and physical comfort, as well as more abstract considerations such as the well being of future generations and esthetic considerations, which relates to characteristics of the immediate environment of the human population. In this anthropocentric focus, protection of such things as rare species enters only to the extent that it makes people feel good.

In a similar way, we might focus a study or risk analysis on some other organism, or on some ecological system of interest. In that case, we would still need to define the environment of *that* population or ecological system.

Six relevant processes need to be modeled (see Figure 3),

- generation of the toxic substances
- introduction to the environmental system
- movement and degradation
- exposure - contact with the target organisms or biological systems
- absorption and distribution into the organism, "toxicokinetics"
- toxicology or "ecotoxicology"

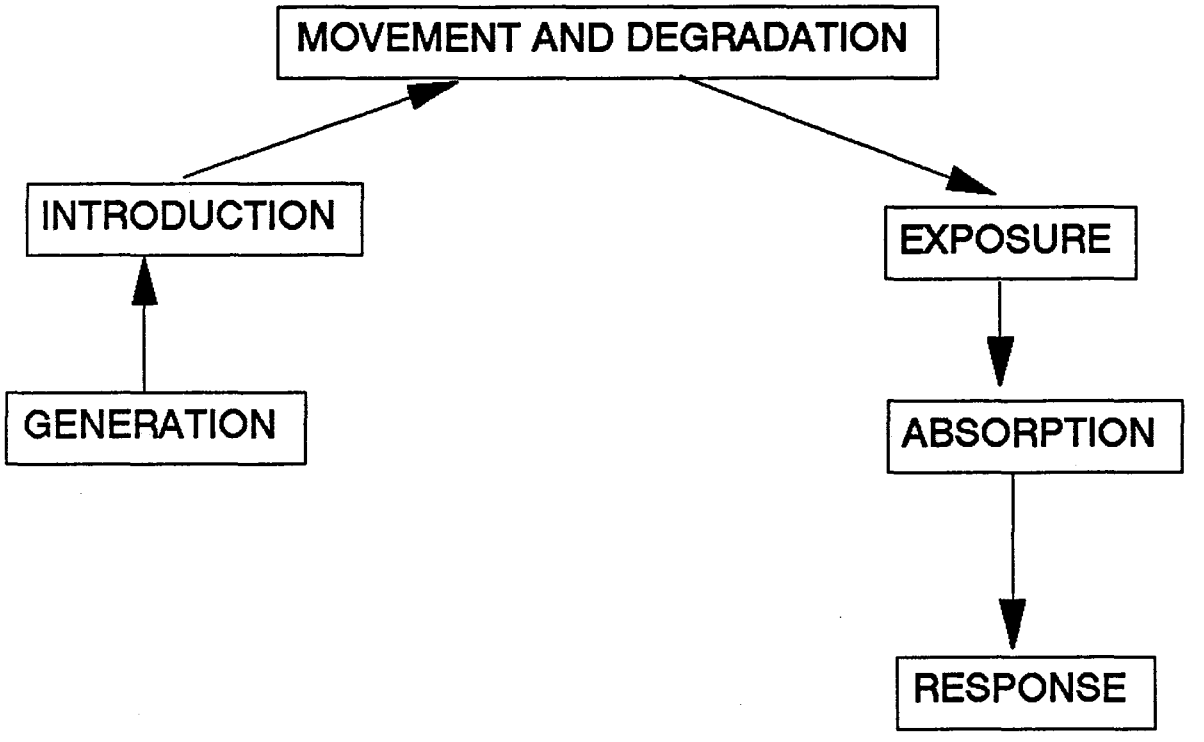
The first three take place entirely within the environment. The next two represent the interaction of the system (human population or other system) with the environment, while the last process occurs entirely within the system under study.

6.2. Generation and introduction into the environmental system.

One way of classifying the routes by which all this stuff can get into our environment is as follows:

6.2.1. Intentional introduction for some useful purpose; the hazardous effects are unintentional side effects. Some examples are:

- Pesticides
- lead in paint becomes a hazardous part of children's environment
- asbestos in pipe insulation



3. Sequence of processes to be modeled

- radon emitting bricks
- plasticizers, which may get leached from plastic food films or containers.
- food additives

6.2.2. Neglectful release -

- By-products and waste from manufacturing and industrial use.
- Consumer level use of fuels
- Waste solvents from both consumer and industrial cleaning use.

6.2.3. "Accidents"

- Accidental chemical spills.
- Illegal dumping
- Tritium releases from nuclear plants

6.2.4. Waste disposal and treatment

- Leaching from landfills and dumps. Note that hazardous materials my be formed and released even from municipal waste landfills.
- Incineration products.

Depending upon the specific application, the modeling problem may include

- Estimation of the level and distribution of the substance already in the environment; examples are lead, asbestos, mercury, DDT

- Estimation of rate of release. This may require modeling the activity or pathway of release. Some examples:

  In the case of a manufacturing by-product, we might need to model the manufacturing activity itself, with special reference to the *efficiency* of the process (see, for example, Conway et al., 1982). Depending on the nature of the decision problem, this might also require estimating the amount of manufacturing activity that will take place, given alternative governmental policies.

  In the case of accidental spills, we would need to estimate the probabilities of various types of accidents. A considerable literature exists on the *fault tree*

analysis and *event tree* analysis, much of it concerned with estimation of accident probabilities with the nuclear power industry.

6.3. Environmental Fate.

The term, *environmental fate* refers to transport, chemical alteration and physical alteration, once a substance gets into the environment. The processes involve:

6.3.1. Physical transport

- Through aqueous environments.

  Soluble materials. Substances which are soluble are simply carried along with the flow of water (Burns, 1983). The job is then the hydrodynamic modeling of water currents. Somewhat different hydrodynamic models have been developed for rivers, for estuaries and for ponds/lakes and oceans (Samiullah, 1990).

  Particulates. Modeling of the transport of particulate materials is somewhat more complicated, since the stuff may want to settle out and get picked up by water turbulence.

  Adsorbed substance. Some organic compounds have absolutely no consideration for the system modeler. They may be adsorbed onto soil or other particles. While adsorbed, movement may follow the mechanisms of particulate transport. However, they may also be in equilibrium with a dissolved phase, which moves directly with the water flow.

- The soil environment

  For purposes of discussing transport, the soil environment can be divided into three zones (see Figure 4, from Bonazountas, 1983).

  - the land surface (watershed dynamics)
  - the region which is saturated with water, and is called either the groundwater zone or the saturated zone.
  - the region in between these which is called the unsaturated zone or the soil zone.

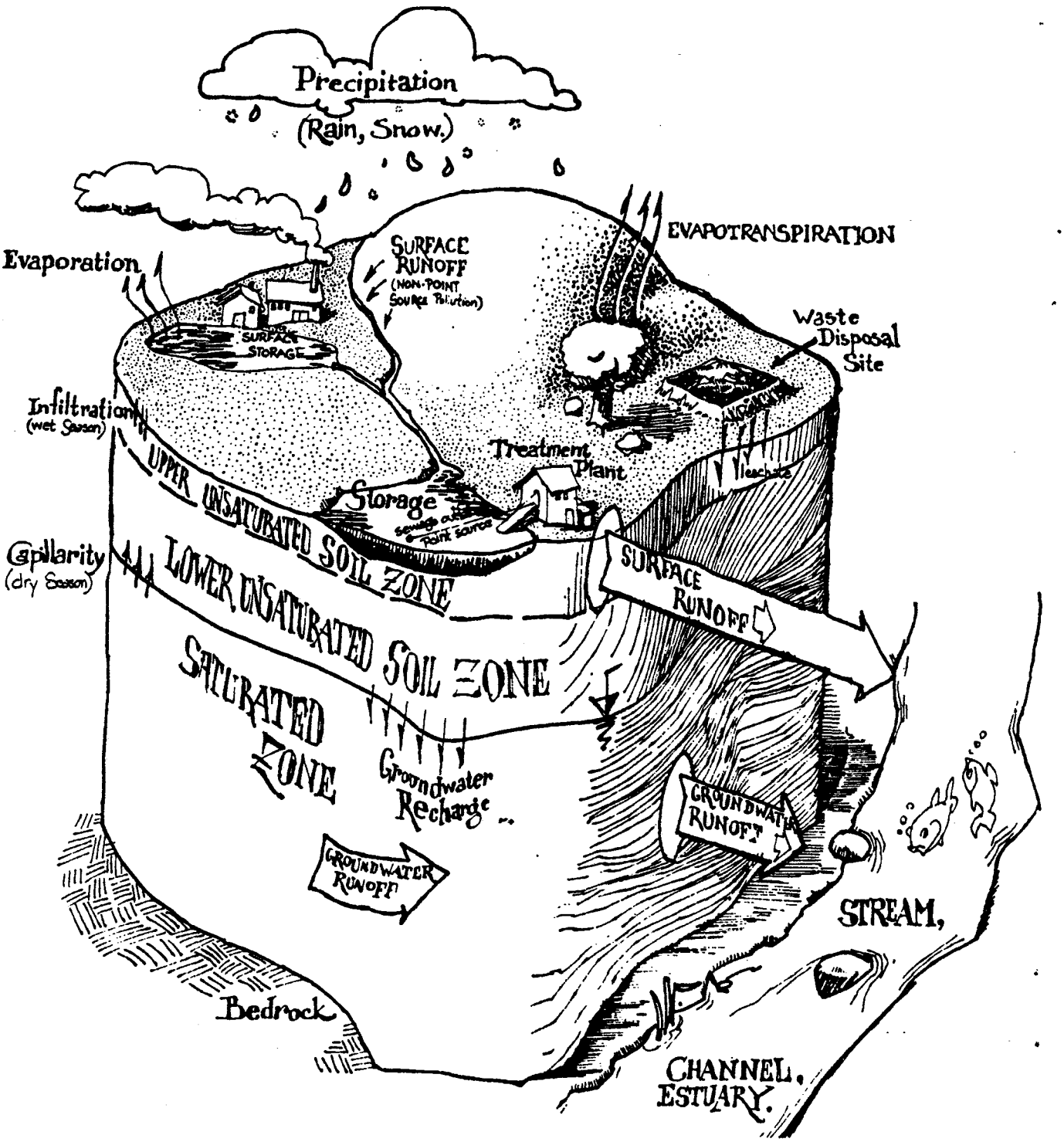


Figure 4. Schematic representation of the soil compartment (from Bonazountas, 1983).

The mechanisms for transport, and therefore the models, differ for the three zones.

Modeling transport on the land surface and in the unsaturated soil zone is complicated by its dependence on exogenous factors, such as rainfall and temperature.

Modeling of the unsaturated soil zone is especially complicated, since the material is moving and partitioning between three phases: soil particles, water and air. The movement is therefore governed by movement of a dissolved phase with the water or by diffusion in the water phase, diffusion in the gas phase, and movement along the water-air and water-solid interface.

In the saturated zone, the modeling is simplified by the absence of an air phase.

Finally, in some cases, as with accidental spills, or leaking gasoline storage tanks, we may have a layer of free liquid organic liquid riding on top of the ground water. Movement of such a layer will depend upon its density and viscosity.

- The atmospheric environment

Transport in the atmosphere will depend on whether we are dealing with a particulate material or with a gas. The transport principally depends upon a mixture of diffusion processes and wind currents. In a recent book (Samiullah, 1990), the author lists 19 different air pollution models published between 1972 and 1986. The author asserts, however, that one type of model, the Gaussian plume model, has been for the most part used by EPA in setting regulations for air pollution. This type of model makes a number of simplifying assumptions. In particular, it represents movement in terms of *average* wind speed and neglects ordinary diffusion in the direction of the wind, but does include a term for wind turbulence. The result, is that we get a plume which is described by a spreading family of Gaussian curves, as it travels from its source (see Figure 5, from Samiullah, 1990).

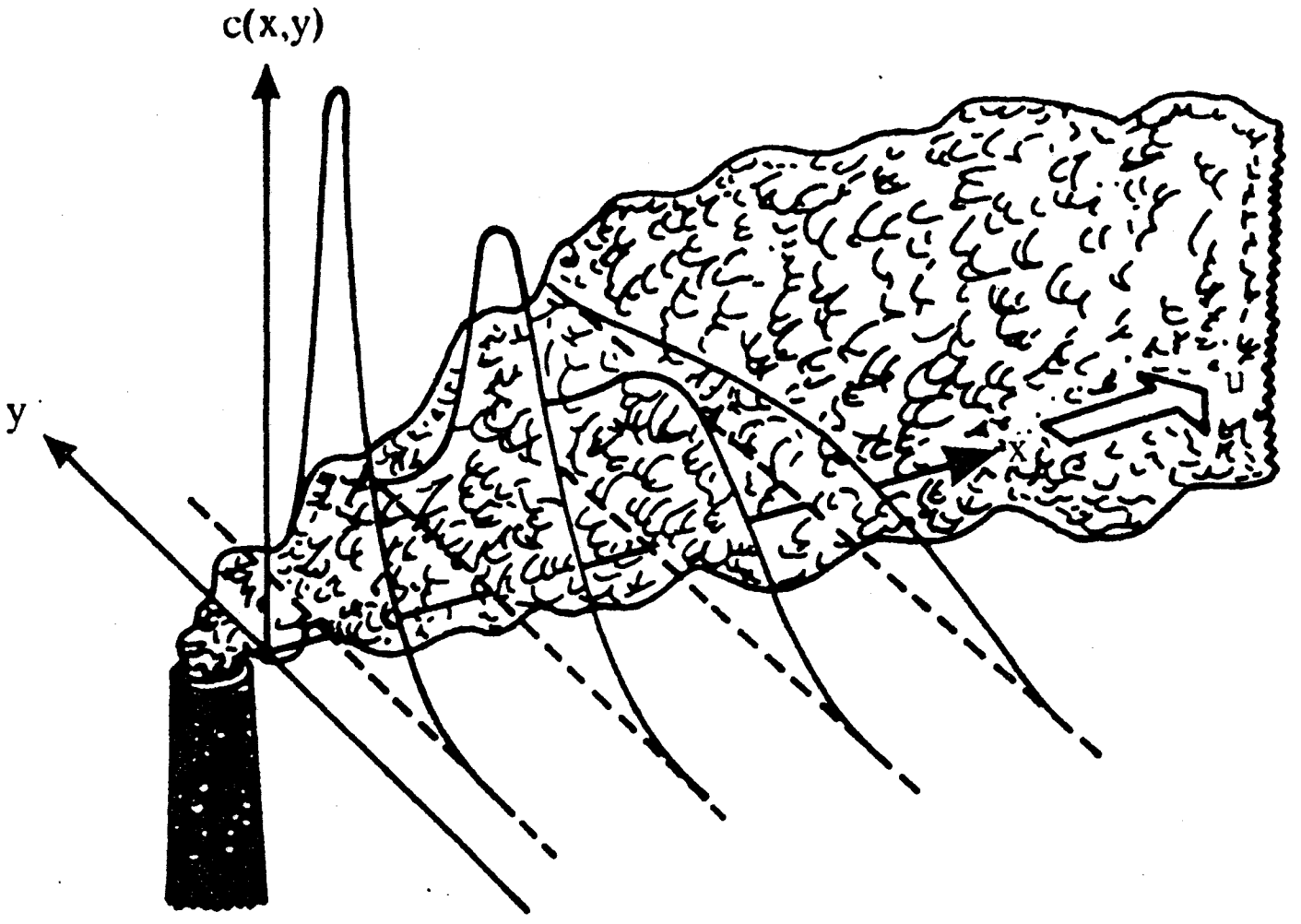


Figure 5. The Gaussian Plume (from Budiansky, 1980).

- Movement through biological organisms and food chains.

  The movement of toxic materials through biological pathways has been appreciated for some time with respect to some specific materials, such as DDT and mercury. Its more general importance is only now being understood. Entry into the food chain begins with passive uptake by either terrestrial or aquatic plants. For terrestrial plants, uptake from the soil would be through the roots, and from the atmosphere by the leaves through gas exchange. These uptakes of xenobiotics would be by passive diffusion and would depend upon the physical-chemical process of partitioning between phases. The same might be true of uptake by terrestrial invertebrates, by terrestrial or aquatic microorganisms (which have a huge surface area to mass ratio) or by the gills of fish. In many cases, because of solubility and physical binding, the biological organism may build up concentrations in excess of that in the environment, a process referred to as *bioaccumulation.*

  Having entered into a biological organism, the xenobiotic may then be further accumulated through the food chain. This may have serious effects on organisms higher up in the chain, including humans.

- Exchange between soil, aquatic, atmospheric and biological phases.

  Soil → aquatic
  through solubilization

  aquatic and soil → atmospheric
  through volatilization

  atmospheric → soil and aquatic
  through precipitation or sedimentation, or absorption at earth's surface

  exchange with biological organisms, as we have already discussed

  Material in the atmosphere may also be "lost" through transport into the stratosphere.

6.3.2. Destruction of Xenobiotics

Fortunately, there are also processes which result in removal or destruction of xenobiotics -- that is, by which the environment "cleans" itself.

- Chemical reactions:
  photolysis and photo oxidation
  hydrolysis reactions
  oxidation/reduction reactions

- biotic degradation
  microorganisms (terrestrial and aquatic)
  plants (terrestrial and aquatic)

  (animals also have capacity for biological degradation or detoxification of xenobiotics, but the amount degraded in this way might make negligible contribution to environmental reduction)

6.4. Modeling exposure and uptake.

Exposure and uptake are very closely related, and are often confused in the literature. Partly, that's partly because both terms are used in two different ways, as in the following sequence:

environmental exposure → uptake by the organism → distribution through the organism → exposure to sensitive organs and tissues → uptake by the organs and tissues → initiation of response sequence.

The toxic effect does not begin to operate until the substance reaches sensitive tissues or organs. Thus a distinction should be made between exposure and uptake by the entire organism vs. exposure and uptake for the sensitive tissues. The mechanism for uptake by the organism, and distribution to tissues, will depend upon the type of environmental exposure (see Figure 6 from ENVIRON Corp.). The principal pathways for uptake are through inhalation, ingestion and dermal absorption.

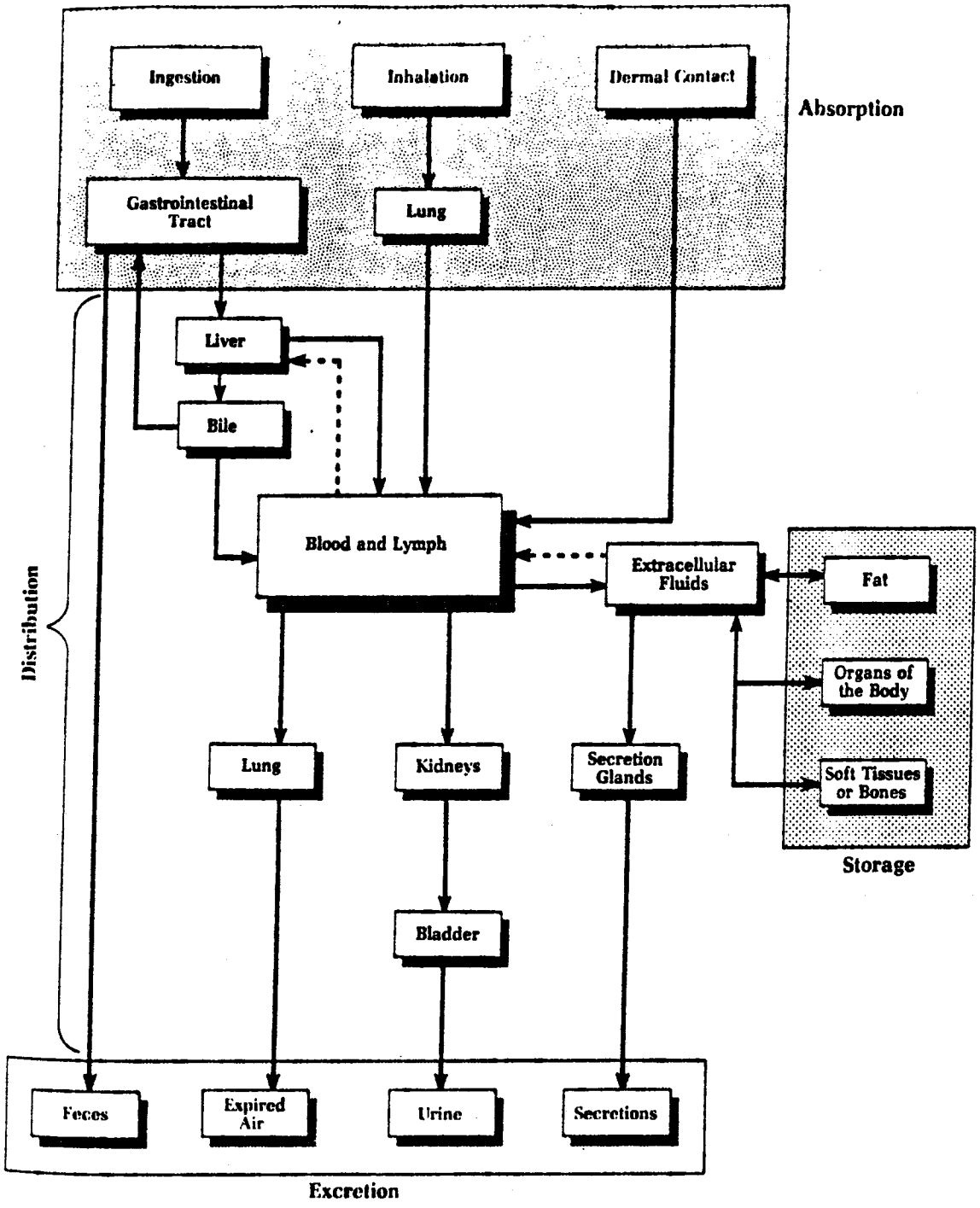


Figure 6. Fate of chemical toxicants in the body (from ENVIRON Corp., 1986)

6.4.1. Inhalation.

We are concerned with anything that can be carried in the air; that is, gases and particulates. Exposure might be expressed in terms of concentration in the air and time.

$$\text{uptake} = C \times \text{time exposed} \times \text{breathing rate.}$$

Note that the level of exposure also depends upon the breathing rate, which also has to be included in any model. Occasional advisories in areas with high air pollution levels have suggested staying indoors, where the air is filtered, and not doing anything which causes you to breath too much.

6.4.2. Dermal absorption.

Dermal absorption comes primarily from soils and dusts adhering to the skin. Exposure might be expressed in terms of concentration in the soil, amount and time of skin area exposed, capacity of the soil to adhere to the skin, and then rate of passage from the soil through the skin.

$$\text{intake} = C \times \text{amount of skin exposed} \times \text{adhesion} \times \text{time} \times \text{rate of absorption}$$

This is a favorite pathway of exposure for children, and those whose work bring them into contact with soil, including people who work with cleanup of hazardous waste sites.

6.4.3. Ingestion.

Here, we are of course primarily concerned with the amount taken in with food and water. Exposure is in terms of concentration in the substance to be ingested, and in terms of the amount ingested per time.

$$\text{uptake} = \text{conc. in food} \times \text{amount ingested}$$

Ingestion of soil is also an important pathway, especially for children, and some studies have dealt with trying to estimate how much (e.g., Thompson and

Burmaster, 1991). Laborers who are in contact with soil may also inadvertently ingest soil which adheres, for example, to hands and face.

Ingestion of old paint is a notorious mechanism for ingestion of lead by children.

Note that in modeling exposure, we are confronted with the necessity to take account of the behavior of the target population.

6.5. Distribution in the body; "toxicokinetics".

Regardless of the level of exposure, it is the dose that reaches the body's tissues that dictates the toxicity. The mechanism of absorption will depend upon the exposure pathway and is well summarized by the accompanying diagram from a publication of ENVIRON Corporation, 1986.

The study and modeling of uptake, distribution and elimination of xenobiotics through the body is closely parallel to the study of pharmacokinetics with an obvious complicating factor: in pharmacokinetics, the exposure pathway is in practice controlled and well defined.

6.6. Toxicology and the dose/response relation.

Once the xenobiotic dose is inside the organism, there is then the problem of determining the relation between dose and toxic response. Of course, we have to define what toxic response we are talking about. Some toxicants have more than one. Toxic effects are commonly divided into

- Acute effects, traceable to a single exposure or a few exposures and observable within a few days after exposure. These include, in particular (Hodgson and Levi, 1987), interference with the nervous system and with oxidative phosphorylation.

- Chronic toxicity refers to longer term effects. Types of chronic toxicity discussed by Hodgson and Levi are:

carcinogenesis
mutagenesis - hereditary changes in the genetic information stored in DNA
teratogenesis - malformation of developing fetuses
organ toxicity - to liver, kidney or to the lungs
behavioral effects

In vivo experimental work in studying chronic toxicity is generally divided into sub-chronic exposure, which may involve exposure for a few weeks to a few months, and chronic exposure, which usually involves exposure over a substantial part of the life span of the test animal, and sometimes over more than one generation.

6.7. Mathematical Modeling of the dose/response relation.

There are several approaches to modeling the relation between toxicant exposure and response. They differ largely in the type of information and observations they use.

6.7.1. Epidemiological Studies.

Epidemiological studies provide the primary source of data based on the direct exposure of human beings. Such studies are based on estimates of exposure and on medical records documenting disease incidence. The main sources of such data are:

- areas with high pollution levels.
- occupational exposures.
- accidents.

Epidemiological models are for the most part statistical regression models, relating exposure to disease incidence or death.

Problems with epidemiological studies include

- Absence of well defined control group.
- inaccurate measures of exposure.
- confounding with multiple exposures to same chemical, to other chemicals, and with other health effects.

6.7.2. Animal Bioassay

Animal studies can of course overcome most of the problems listed for epidemiological studies. Within the range of the experimental data, one may describe the dose-response relation in terms of a suitable regression equation – possibly non-linear. Unfortunately, such studies have their own problems, largely related to violation of a basic injunction of statistical inference:

THOUGH SHALT NOT EXTRAPOLATE BEYOND
THE RANGE OF THY DATA.

The violations are of two types:

★ Extrapolation to low doses.

For application to human health, we are often interested in quantifying responses which may occur in very low numbers in the population. Many EPA regulations are geared to levels and effects that occur in the range of 1 in every 1000 to 1 in every 1,000,000 exposed persons. It is simply not feasible to conduct experiments for a sufficient length of time and in sufficient numbers to give meaningful statistical data at such levels. The answer of course is to use much higher levels of toxicant, which produce much greater frequencies of effects. We then extrapolate to low doses. Of course, we have no choice. A decision has to be made, and we have to do the best we can. Traditionally, the extrapolation used has been linear, passing through the origin – that is, zero effect at zero concentration. This will be in error if the mechanism itself is nonlinear, or worse yet, if different mechanisms operate at different levels. For example, there is a large repertoire of biochemical toxification and detoxification mechanisms (Hodgson and Levi, 1987) which may operate so as to prevent any toxic effect from being exhibited until a certain threshold is reached.

★ Interspecies extrapolation

This problem is even more serious. The first type of interspecies extrapolation addresses the differences in size between species, or sometimes

difference in surface area. That's relatively easy. What's difficult is to account for differences in metabolism between animal species. One can hardly do that without consideration of the biological pathways of the different species and of the biological mechanisms of toxification and detoxification. A good case in point is that of dioxin. The situation is summarized by Cohrssen and Covello (1989).

Dioxins are a family of compounds, the most studied of which is 2,3,7,8 tetrachlorodibenzo-p-dioxin (TCDD). It is formed as a by-product in the manufacture of a number of substances, including certain herbicides, wood preservatives, the anti-bacterial agent hexachlorophene, and in the bleaching of wood pulp to make paper. Its also a by-product of house fires.

Animal studies with TCDD have shown it to be one of the most acutely toxic materials known – for certain species. It is most acutely toxic for guinea pigs: 1 mg per kg of body weight is lethal. For hamsters, on the other hand, the LD_{50} is about 5000. So, are humans more similar to guinea pigs or to hamsters?

In vitro studies in cell cultures have indicated that TCDD is a powerful promoter of genetic transformation, but the evidence from animal experiments was, at least up till a few years ago, inconclusive.

Results from two major accidental exposures, resulting from chemical explosions, seemed to indicate some acute human effects, most of which diminished after was terminated, and no long term effects such as increased rates of cancer or increased incidence of birth defects.

6.7.3. Models based on consideration of physiology and biological mechanism.

Such models are based on the attempt to formulate a detailed chain involving distribution of the toxin through the body, and the mechanism of toxic action. Some degree of controversy exists in the literature concerning the use of such models. Detractors assert that there is too much room for uncertainty in our mechanistic knowledge; promoters assert that sources of uncertainty in non-mechanistic models are great enough to justify use of mechanistic models.

Consideration of the relation of chemical and electronic structure of chemical compounds to their physiological action has been a guide to identification of hazardous substances for many decades. Moreover, consideration of mechanisms

and of enzymatic pathways is an important consideration in deciding how relevant a particular animal study is to human toxicity.

6.7.4. Other types of evidence, used especially in preliminary screening tests, include effects on cell and tissue cultures, and effects on microorganisms and invertebrates.

6.7.5. Comment: it is clear that information about existence and degree of toxicity comes from a variety of sources, all of which have some light to shed, none of which is definitive, and therefore all of which should be used. The methodology of Bayesian belief networks (see, for example, Gold et al., 1990; Oliver and Smith, 1990) may have something to offer in such a situation.

6.8. It is clear that each of the processes shown in Figure 3 is subject to substantial uncertainty. The problem is to evaluate the relevant uncertainties, to try to develop adequate models for how the uncertainties combine, and to develop some coherent basis for decision making given the uncertainties.

7. The value function; in what terms do we measure value?

In an area with difficult questions, not the least difficult is the choice of units in which to measure the value function, Some alternatives are:

7.1. Individual lifetime risk.

This was the measure used in the Livingston train spill. If we have a population of people with varying degrees of sensitivity, we might use the mean individual lifetime risk. Note that this is totally insensitive to the number of people exposed. Using this as a criterion, we are unable to distinguish the urgency of a problem that occurs in downtown New York with millions of people exposed and a problem that occurs in a desert area with nobody exposed.

On the other hand, one might argue that a citizen of New Mexico has as much right to protection as a citizen of New York.

7.2. Population risk; number of cases expected to result from exposure.

This is individual risk $\times$ no. people exposed. Such a criterion would require

stricter criteria in high population regions. Taken to an extreme, we could conceivably wind up with population enclaves which are kept clean, while dumping the junk into our hitherto pristine wilderness areas.

7.3. Loss of life expectancy.

This can be applied on an individual or a population level. Instead of counting deaths, however, we count number of years of life expectancy lost. As a basis for prioritizing action, this places higher priority on hazards that affect younger people.

7.4. Averaged utility of life.

Most diseases not only decrease life expectancy, but also diminish the quality of life before death. A number of authors have argued that such considerations should enter into the setting of environmental priorities, and have proposed scales for doing so (see, for example, Keeney and von Winterfeldt, 1991; Mauskopf and French, 1991).

7.5. Problem – we need to make choices. There is no free lunch, and we do not have unlimited resources. For example, if we impose stricter polluting standards on power plants, we run up the cost of electricity and home heating, which might result in increased disease and death among the poor. We might, of course, consider energy subsidies to the needy as part of the cost of cleaner air. BUT, somebody has to pay.

8. Concluding remarks

I have tried to make several points.

8.1. The risks resulting from contamination of our environment with toxic chemicals are substantial and wide-spread. It is not too extreme to say that they pose at least some threat to civilization as we know it.

8.2. **The science is varied, challenging and fascinating. There is room for people interested in a wide variety of disciplines, or in working with interdisciplinary problems. The menu might include**

biology
geosciences
engineering
mathematical sciences
social sciences

The menu also includes the combination plate: any combination of the above, but the price goes up, in terms of the effort needed.

8.3. **This is clearly an arena in which physical, biological and management science and mix with politics, which, in our society, is where we debate questions of values and ethics. People who work in this area may choose to restrict themselves to the science, or to also get involved in the politics. Caution: people who combine the science with the politics often fall into the trap of corrupting or distorting the science to serve predetermined political (or economic) ends. The uncertainties and ambiguities involved provide wonderful opportunities to do this. Given this, it is remarkable and heartening that there is a research community committed to approaching these problems as objectively and dispassionately as possible.**

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