CHAPTER 3

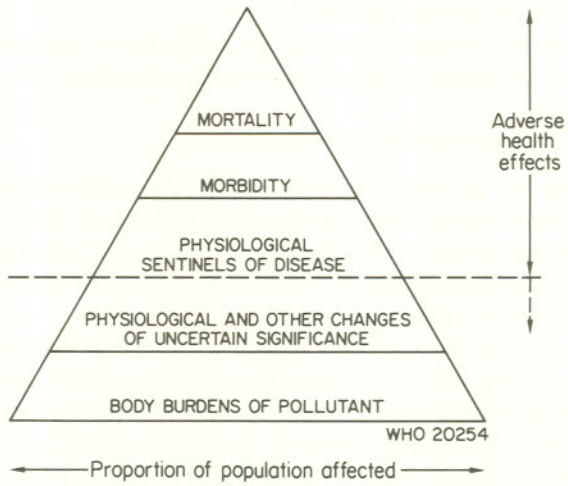
Identifying and Estimating Risks

3.1 THE SELECTION OF TECHNIQUES

The estimation of risk is concerned with collection information on:

(1) The nature and extent of the *source;*
(2) The chain of events, *pathways* and processes that connect the cause to the effects; and
(3) The relationship between the characteristics of the impact (dose) and the types of response *(effects).*

Although response and effects are often equated or used synonymously, the World Health Organization makes a distinction between the two (WHO, 1972). An *effect* is a 'biological reaction itself, while a *response* refers to the relative number of exposed people who react with a specific effect' (Friberg, 1976). This concept is illustrated in Figure 3.1. According to some scientists,



Based on a diagram in United States Congress Document No. 92 241, 1972

Source: Munn, Phillips and Sanderson, 1977

Figure 3.1 Schematic representation of the WHO concepts of effect and response (WHO, 1972)

the WHO distinction is useful and should be extended to other kinds of receptors such as vegetation (Munn, Phillips and Sanderson, 1977).

In practice, risk assessment often begins by looking at one part of the problem, usually the source or the effect, rather than considering the system as a whole at the outset. This is a pragmatic response to the different ways in which risks are discovered. Some risks are identified initially as a known or suspected (such as a carcinogen) source, and the problem is to discover where and how the effects are distributed. Other risks emerge because their effects become apparent (such as a localized high incidence of a disease) and the priority risk assessment task is to determine the cause or source of the problem.

Once a risk is suspected, it is important to bring together as much available information as is possible before designing the ways in which additional data are to be collected. The estimation of risk is an expensive process and initial errors in the design of monitoring or screening programmes are costly in money, delays and inadequate information.

Some of the initial questions that should be asked before any assessment methods are selected are:

(1) Is the problem an analysis starting from a suspected (or known) *cause* or *effect?*
(2) How is the problem distributed geographically? (highly localized, regional, national, associated with urban areas, in lowlands, etc?)
(3) How much time available is there to assess the risk? (fast or slow causes, acute or chronic effects?)
(4) Where do the effects lie? (within which demographic populations or which parts of the environment?)
(5) Is the target (receptor) normal or particularly susceptible? (healthy adults or sensitive groups; unstressed ecosystem or already highly polluted?)
(6) Are the effects reversible or irreversible?

By specifying the problems in terms of questions like these, the decision maker can arrive at some understanding of the magnitude of the task, where to start, and how much time is available. His answers will help him to select the appropriate monitoring or testing techniques as well as provide him with a rudimentary model of what is going on.

It is important also to be able to specify whether the risks arise principally out of a *technological system;* or through *environmental processes,* or through *human biology and behaviour.* This is because the appropriate measurement techniques differ according to where in the sequence of cause and effect you wish to measure. Many risks, of course, involve technical processes and human behaviour as well as environmental processes.

For example, lead pollution from an industrial lead smelter may be caused initially from poor system design and equipment malfunction and here techniques appropriate to technical systems would be appropriate (e.g. fault tree analysis, components testing). Once the lead leaves the chimney stack, it is

subject to environmental processes of transportation and diffusion through the air, settling to the ground, being taken up by plants and animals and movement through the soil and water. Quite different models and measurement techniques from those applicable within the smelter, would be used. How much of the lead concentrated in vegetables or drinking water eventually reaches individual people depends on another set of human biology and behavioural variables, such as how much contaminated food they consume. Thus models and techniques to measure human behaviour are relevant at this point in the sequence.

The selection of techniques thus requires initial decisions to be made about:

(1) The main methods — monitoring, testing, and modelling.
(2) The focus of concern — technical, environmental, or human behaviour subsystems.

Table 3.1 Examples of modelling, monitoring and testing techniques for estimating environmental risks

	Environmental Risk System		
Approaches	Technical	Environment	Human Biology and Behaviour
Modelling	Fuel cycles Event tree analysis Fault tree analysis	Ecosystems models Physical transport models Food chain models Tectonic models Hydrologic and atmospheric models	Genetic models Demographic models Metabolic pathways Epidemiological models Behavioural models
Monitoring and Surveillance	System reliability Pollutant emissions Component quality Design quality	Weather watch Seismic movements Air quality Forest and crop surveys	Mortality statistics Notifiable diseases Clinical records Drug use reporting Epidemiological surveys
Screening and Testing	Materials testing Product quality Destructive tests for failure	Screening tests for persistence transformation toxicity Recovery rates	Mutagenicity (Ames test) Acute toxicity LD_{50} screening test Response mechanisms Physical check-ups

Table 3.1 gives examples of available techniques according to the main approaches and subsystems. The main approaches discussed here are environmental monitoring and health surveillance, testing and screening, and modelling.

3.2 ENVIRONMENTAL MONITORING AND HEALTH SURVEILLANCE

Environmental monitoring is defined as 'the process of repetitive observing, for defined purposes, of one or more elements or indicators of the environment according to prearranged schedules in space and time, and using comparable methodologies for environmental sensing and data collection' (Munn, 1973).

Environmental monitoring is undertaken to determine space and time patterns of environmental elements or indicators, and to estimate their variabilities, for the purpose of:

(1) Understanding environmental processes:
(2) Providing early warnings of environmental threats (natural as well as man-induced);
(3) Assisting in the optimization of the use of renewable and non-renewable resources;
(4) Assisting in the regulatory process, providing data that may be used in the courts or elsewhere to demonstrate cases of non-compliance with environmental standards.

In the case of (2) above, the early warnings may be given by simple extrapolations of upward trends, or preferably from predictions obtained from models that have been already validated with independent sets of data.

Within individual countries, there are many kinds of monitoring programmes which are often uncoordinated with each other and with similar programmes in adjacent countries. In particular, the instrument and sampling techniques may be so disparate the inter-comparisons may be impossible. Yet for environmental risk assessment, the need for reliable data is essential.

There are several multi-national monitoring systems that have been established for particular reasons. Examples include the OECD and EEC long-range transport of air pollution programmes and the Lake Erie International Field Year on the Great Lakes. In most cases, these monitoring systems have been established to determine the existence and/or extent of an environmental problem that has been perceived in general terms only.

Some components of the national and regional programmes, are connected globally through the United Nations Environment Programme in the Global Environmental Monitoring System (GEMS). This is a coordinated programme for gathering data to be used in environmental management (including early warning systems) rather than for enforcement of existing environmental standards. The data will also be valuable in some cases in the development of legislation or international conventions leading to the establishment of environmental controls.

Monitoring and sampling programmes are also used to detect the presence of harmful substances. For example, the discovery of small quantities of carcinogens in the drinking water of several cities in the United States was made during a routine sampling programme to determine water quality.

Research into the source of these contaminants has led to a re-evaluation of current drinking water disinfection practices in the United States, as the source of these contaminants appears to be a by-product of the disinfection practices.

Monitoring of administrative programmes such as the required registration of chemicals, drugs, pesticides and industrial undertakings can also be important. For example, the information submitted to obtain a permit or to register a toxic material can help identify the nature and amount of specific pollutants entering the environment.

Health surveillance is the collation and interpretation of data collected from monitoring programmes and from any other available sources, with a view to detecting changes in the health status of populations (WHO, 1972). Internationally, health surveillance has been most successful in recording the principal causes of death and notifiable diseases such as malaria or smallpox. There is a need however to maintain surveillance programmes that will alert governments and scientists to environmental hazards when symptoms first appear rather than when the patients have died.

One important use of surveillance is for the effects of drugs. In the UK a system of reporting about drug use and effects from doctors to a central agency is a principal means of monitoring and evaluating drugs. The incidence of cancer, and accidents (home, industrial and transportation) are also regularly monitored in many countries.

Monitoring human populations for genetic mutations is of concern in some industrial countries where the chemical environment is rapidly changing and becoming more complex with increasing exposure to chemical mutagens. The counting of spontaneous abortions has been recommended as a simple and practical method for monitoring mutation rates. The spontaneous incidence of abortions is about 1 per cent of all births.

For all these health surveillance programmes, the lack of baseline data on regional and national incidences of health effects, the need for skilled manpower, effective reporting systems and centralized data bank facilities, makes progress slow in all countries. Where detailed health data are already centralized, for example, for financial purposes in national health schemes, they can also be used to help establish the effects of the environment on health.

3.3 TESTING AND SCREENING

If based on accepted, standardized procedures, testing can identify sources of risk (such as defective products or dangerous chemicals) or harmful effects (such as acute toxicity and mutagenicity). Tests can also help to quantify environmental processes such as the persistence of harmful substances in the environment. Testing is often a carefully controlled laboratory procedure. The advantage of field or in-situ testing is that it provides more realistic conditions.

Screening usually involves multiple tests or multiple candidates for evaluation which are 'sifted' by the test procedures into different categories. In numerous instances, tests are carried out for product or drug or food-additive

safety. This is often done with animal test populations, and from this, inference is made of the degree of risk to human populations. In the United States, for example, the Food and Drug Administration screens some 110 petitions for new food additives annually, and the Environmental Protection Agency has been charged under the recent Toxic Substances Control Act (1976) with the formidable task of screening the 30,000 chemical substances now in substantial use and the several hundred new ones being added each year.

It is, in fact, impossible to keep pace with all the products or chemical substances that need to be screened and tested. The United States has fallen behind in this operation and many other countries are even further behind. The cost of tests is another consideration to be added to those of the time they take and the skilled manpower and equipment needed. For example, it was estimated that the basic tests for registering a pesticide in the USA in 1970 cost

Sub system

Table 3.2 Basic Tests Required for Pesticides Registration in the USA (from Blodgett, 1974.

	Tests	Date established	Cost (estimated dollars)	Time implication
1.	Chemical and physical properties (Such as solubility, vapor pressure, flash point)	1947	5,000-15,000	
2.	Degradation studies		A rough estimate of these requirements in their entirety would range between $100,000 and $250,000.	
	Persistence (soil)	1965		6-24 months
	Persistence (water) and sediment)	1970		Less than 1 year
	Photochemical	1970		2-6 months
3.	Mobility studies	1970		Less than 6 months
	Runoff			
	Leaching	1970		Less than 3 months
4.	Residue studies			
	Fish	1970		2-6 months
	Birds	1970		2-6 months
	Mammals	1970		2-6 months
	Lower trophic levels of food chains	1972		6-9 months
5.	Microbiological studies	1970		Less than 3 months

It should be noted that much of the data generated by these tests is utilized in studies of human, fish, and wildlife safety.

between $100,000 and $250,000 and required 1-2 years (Table 3.2). In addition, tests to measure the tolerances of other plants and animals to pesticides cost between $5,000 and $160,000 per test and take between two and 28 months to complete (Table 3.3).

These high cost and time demands of tests have encouraged the development of more rapid and less expensive tests. For example, the current approach in testing effluent is to break-down the effluent into its chemical components and screen each for toxicity, persistence and breakdown or transformation properties. This bank of tests is necessary because it is not sufficient to know just the chemical properties at the point of discharge. Certain chemicals will breakdown or transform *after* they leave the point of discharge into substances which may be more or less harmful than the original chemical. For example, the pesticide myrex, when transmitted through a water environment will break down into kepone, a highly toxic and persistent substance. Thus tests must be run to determine if particular contaminants will break down and if so, into what. Tests must also be run to see if the substance is likely to be captured in sediments and there build-up, or bioaccumulate in various animal and plant forms.

Table 3.3 Tests for Tolerance of Pesticides in USA (from Blodgett, 1974. Reproduced by permission of the MIT Press)

	Tests	Date established	Cost (estimated dollars)	Time implication (months)
1.	Toxicology			
	Acute (rat and non-rodent)	1954	5,000	1
	Subacute (rat and dog)	1954	50,000	6
	Chronic, 2 year (rat and dog)	1954	160,000	28
2.	Reproduction (rat)	1960	35,000	20
3.	Teratogenesis	1970	10,000	2
4.	Mutagenesis	1972	10,000	2
5.	Metabolism			
	Plant	1954	50,000	6
	Animal	Before 1960	25,000	3
6.	Analytical			
	Methodology	1954	100,000	4-6
	Crops, Meat, Milk, Poultry, Eggs	1965 (Poultry)		
7.	Field Residue Data		100,000	12
	Drop, Feed, Meat, Milk, Poultry, Eggs	Before 1960 1965 (Poultry)	100,000	6

The ability to test for the various parameters is uneven. The chemical/physical parameters can be measured rather well. Measuring the biological parameters is much more difficult. Fairly good progress has been made in developing tests for fresh water, and a beginning has been made in the marine area. However, considerable research remains to be done in the developing of suitable biological screening techniques.

In order to streamline the process, rapid screening tests are being developed to screen out those components most likely to be a problem. Those chemicals identified as bad actors as a result of the rapid screening tests, are then put through more detailed tests to develop more refined data on impact levels. These and related procedures are more fully discussed in another SCOPE report (Butler, 1978).

Continuing on the current chemical specific basis, however, is extremely cumbersome because of the sheer number of substances to be considered. Thus, research is also being conducted to see if techniques can be developed to evaluate the effluent as a totality, to make the problem more manageable while still maintaining reasonable safety factors. To make this approach feasible a similar bank of tests for toxicity, transformation and persistence would have to be developed to handle the *total effluent.* This will reduce the regulation task considerably if effluent does not have to be broken down into its components to show that it is harmful.

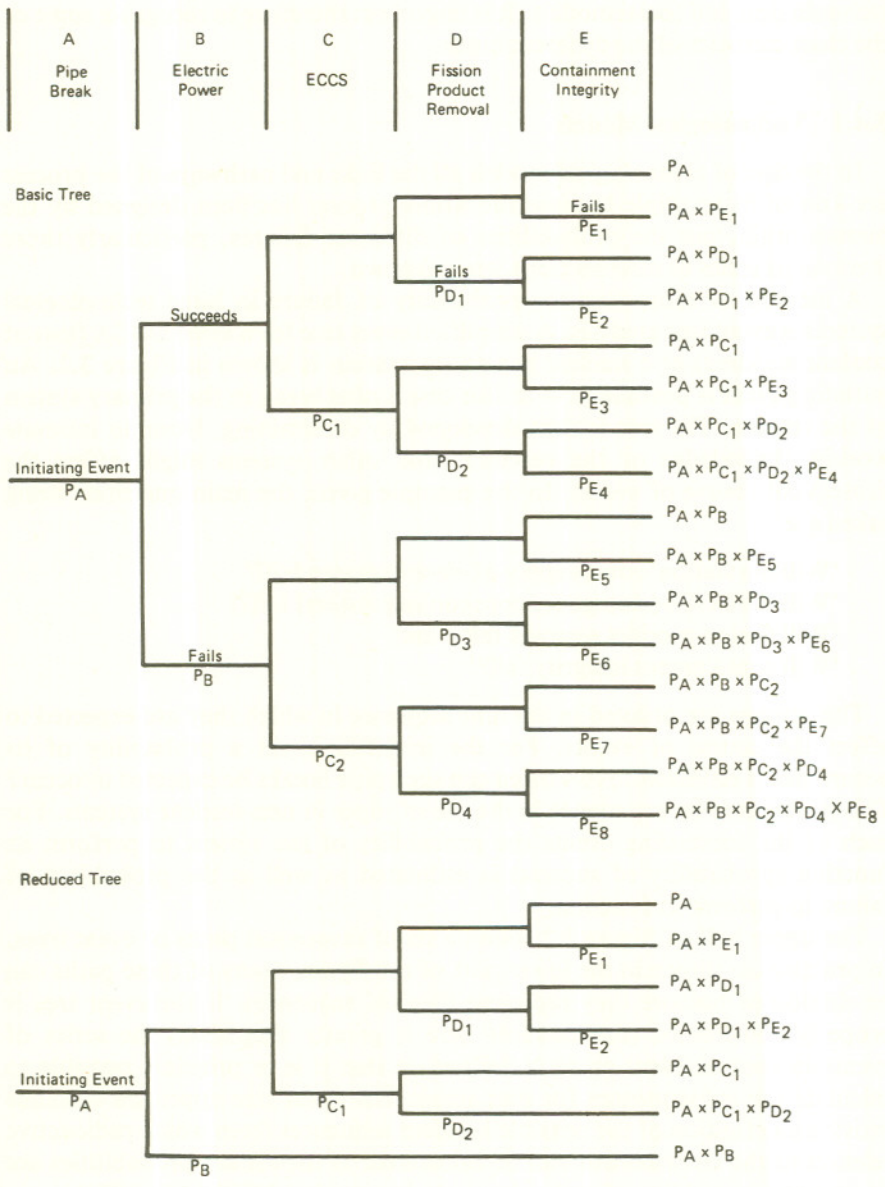
3.4 MODELLING

All the risks that are of concern in this report arise as part of a process. To understand the risk and to manage it effectively models of the total process are developed. Such models have, or should have, both environmental and social components, because the degree of risk depends both upon what happens in the environment and what social processes are at work.

Highly successful models have been developed for some environmental processes, especially in such areas as meteorology (atmospheric diffusion models), hydrology (run-off and flood forecasting models), and plant growth (biomass production models). Capability to model the social systems and how they interact with environment is much less well developed. Thus the more important the social component or human behavioural component becomes in an environmental risk, the less likely we are to be able to model the total process satisfactorily.

Models are developed in every branch of science that bears upon environmental risk questions. The many possible models may be grouped, however, into those that are primarily technological in orientation, those that are primarily concerned to describe environmental processes and those that are concerned with human systems whether social, biological, behavioural and so on.

The focus in this report is on the environmental models, but the environmental models are often connected at one point or more to



Note - Since the probability of failure, P, is generally less than 0.1, the probability of success (1-P) is always close to 1. Thus, the probability associated with the upper (success) branches in the tree is assumed to be 1.

Figure 3.2 Simplified Event Trees for a Large LOCA

Source: US AEC, p.89 (Reproduced by permission of the U.S. Atomic Energy Commission.)

technological and social models. It is important therefore to recognize some of the characteristics of the different kinds.

3.4.1 Technological Models

In the case of technological models all the links and pathways of the process are known in principle because the whole process has been designed by the human brain, but the probabilities of different failures, particularly those involving a series of malfunctions, are unknown.

A method of estimating the probability of failure in large technological systems is event tree analysis. A simplified event tree for a large LOCA (loss of coolant accident) at a nuclear generating station is shown in Figure 3.2. An initiating event is postulated — in this case a pipe break in the primary system of the reactor. The tree is then developed by determining, from an intimate working knowledge of the reactor, what other systems might affect the subsequent course of events. In the example given, the main questions being asked are

'Will the station's own supply of electric power fail?'
'Will the ECCS (emergency core cooling system) fail?'
'Will fission product removal fail?' and
'Will containment integrity fail?'

The systems are ordered in the time sequence in which they are expected to affect the course of events. For the initiating event a probability of its occurrence is estimated. How often can such pipe breaks be expected to occur? This is obtained by experience in the use of pipes in non-nuclear systems. For each of the succeeding events the probability of the system to perform its function (probability of success) is estimated as well as the probability of failure to perform its function.

The upper half of Figure 3.2 shows a set of theoretical paths or event trees. In practice, as shown in the lower part of the figure, many of these paths can be eliminated because they represent illogical sequences. If the event tree is properly constructed (the example here is greatly simplified) the series of events in each accident chain is defined so that it is in principle possible to calculate the consequences for that series. The event tree approach can thus provide a definition of the possible accident sequences from which radioactive releases to the environment can be calculated. If the failure probabilities are known, the probability of each release can be calculated (US AEC, 1974).

3.4.2 Links to Environmental and Behavioural Models

At this point the model of a complex technological system connects with the atmospheric diffusion model. If radioactive fission products are released how far and how fast will they spread? This leads in turn to questions about effect on human populations for which models of the response of the human

biological system to radioactivity are needed as well as models of human behaviour in the face of warnings about radioactivity in a power station accident. These behavioural elements may have substantial effect on the amount of damage to health that is caused, but are more difficult to model in a reliable fashion.

An important source of difficulty in modelling the human behavioural component is that it is self-aware. Accident sequences in nuclear power plants, and the process of biomagnification of toxic substances in food chains all proceed in an automatic fashion which can be modelled in probabilistic terms. The quality of self-awareness in human systems enables the behaviour and functioning of the system to change in response to how people think the process is working or should work. In other words, we now need to know what perceptions of the system are held by the participants in it, since that will change the outcome.

3.4.3 A Perception-Behaviour Model

A model for environmental perception and behaviour is shown in Figure 3.3. It represents one way of organizing the components of a general model (Whyte, 1977). The variables are arranged according to:

(1) The distance from a decision point at the man-environment interface, and
(2) The scale at which decisions are taken from individual or household level to the organization or government level where decisions are taken by a few on behalf of many.

Thus as one moves from right to left across the diagram the variables more directly impinge upon the output variables for a specific intervention (but they may not necessarily be more influential). And as one moves from the bottom to the top of the figure the variables become more relevant to collective rather than individual decision-making, though they are not exclusive to either. Thus a progression can be traced from individual and group characteristics through intervening variables such as values and personality, to decision and choices affecting the environment.

Linking the individual and social variables are four interdependent processes which together act as the main organizing force in the system. These are the 'perception processes' which link all the components. In this model they are considered as four process elements on the pragmatic grounds of what are measurably different components of perception at the field level. Thus, *categorization* and *judgement* are grouped together in the model because they are often measured together although conceptually they are different parts of the perception process.

The other three major divisions of perception used here are: sensory perception (e.g. sight, smell); attitudes; and communication and information flow. In the field, these processes (either separately or together) can be

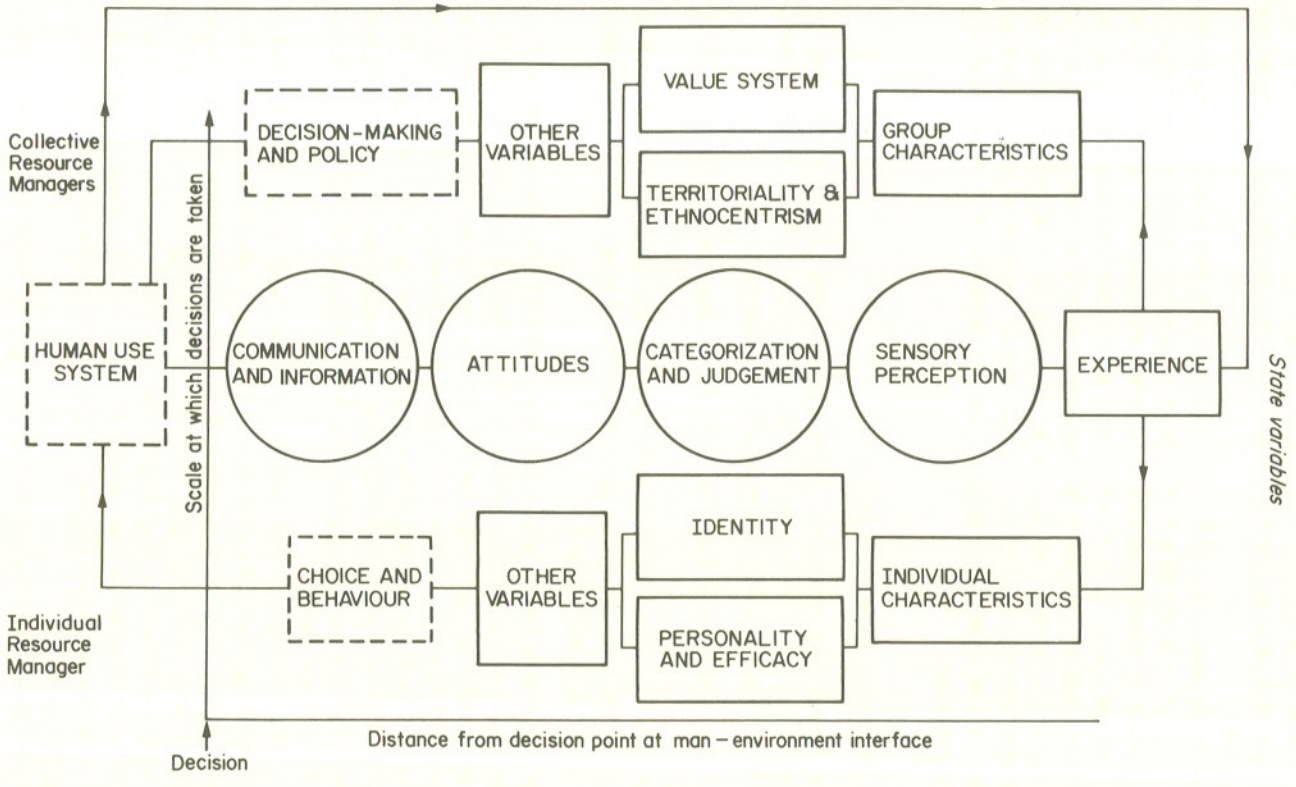


Figure 3.3 Simplified model of environmental perception
Source: Whyte, 1977

investigated as links between any sub-set of variables relevant to the study.

Figure 3.3 is a simple heuristic device to help organize the research planning task. It is not a model of how the system actually functions and it cannot serve as a substitute for specific hypothesis development and conceptual modelling within each research project. No constraints are intended on the boundaries of a specific study or on the operations and connections between variables. Specific environmental risk problems can therefore be examined in terms of their own 'critical paths' through the model.

3.5 ENVIRONMENTAL MODELS

A wide range of environmental models have been developed. The main purpose of environmental models in this context is to study the variables and linking processes between sources or causes of environmental risks and their consequences or effects. Much has been written elsewhere about the development of such models. This section illustrates environmental modelling by three types of example:

(1) Non-quantitative models and probabilistic models,
(2) Global models of biogeochemical cycles, and
(3) Models of persistent substances in food chains.

3.5.1 Non-quantitative Models and Probabilistic Models

The power and value of developing comprehensive models to examine events lies in their ability to portray in an understandable form complex systems and interrelationships. Their weakness is in the simplifying assumptions that are used in some or all of the component parts (Holdgate and White, 1977). Often these simplifying assumptions are made when processes (physical, biological or social) are represented in the form of equations. There is thus good reason to develop as a first step a non-quantitative model which maps out all the possible sources and pathways whether or not they can be quantified. Numerical representations of parts of the system can be developed in various ways as a second stage (including graphical mapping, empirically fitting algebraic equations to represent relationships between phenomena, and the use of high speed computers to solve complex systems of non-linear partial differential equations or stochastic relations representing such phenomena).

Figure 3.4 shows a non-quantitative model of the pathways by which DDT can reach the human body in a poor agricultural area. The pathways include contamination of DDT containers reused for storage of food and water and contamination of food when it is sold after it has been stored with DDT in village shops. Study of the behaviour of people using DDT or even those living in villages where it is used are as necessary inputs to the development of this kind of model (however unquantified) as is knowledge of the natural environmental and food chain processes. Indeed, comprehensiveness is a main objective of these models to ensure that all possible pathways by which risks

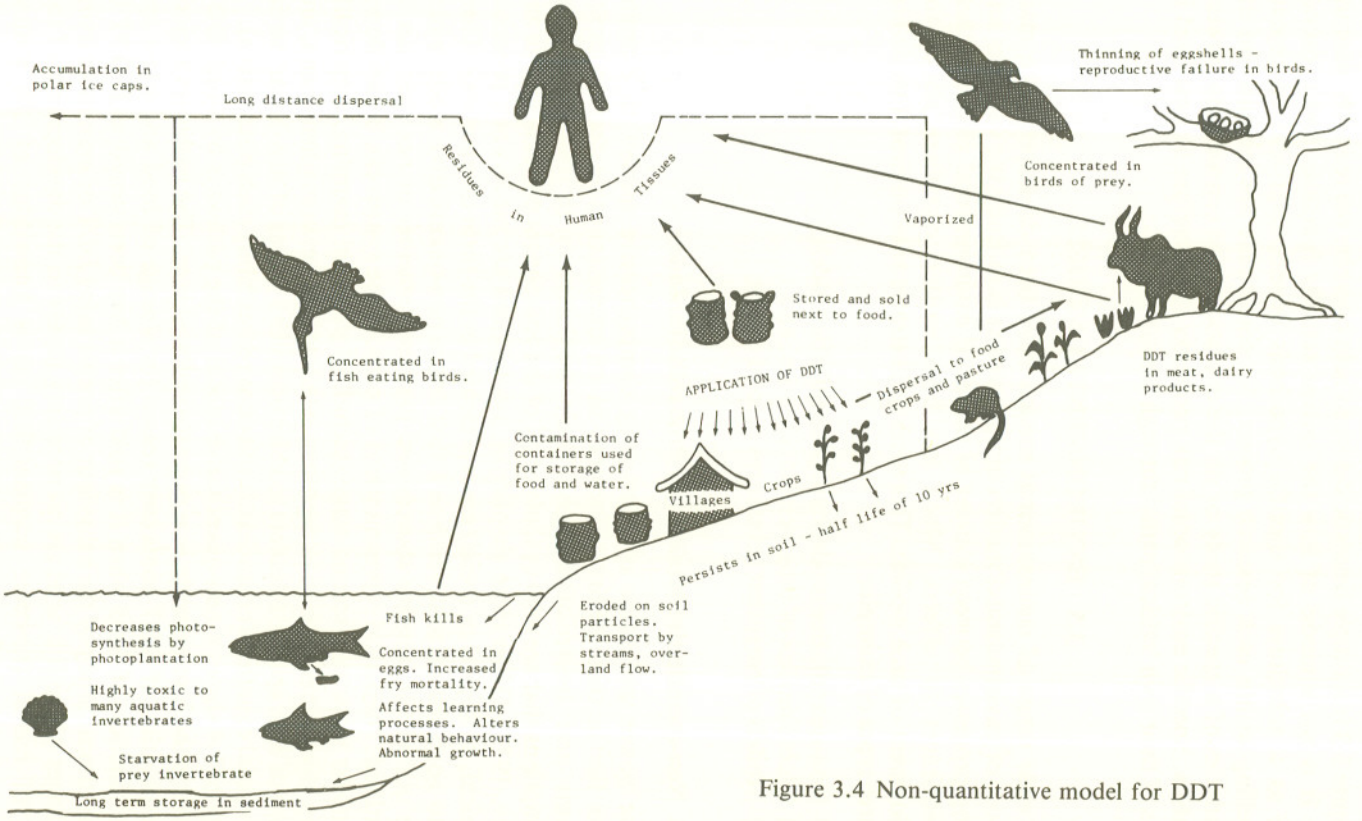


Figure 3.4 Non-quantitative model for DDT

are transmitted are included in the first picture so that the decision-maker is provided with an overall view of the magnitude and complexity of the problem he has to deal with. Later, simplifying assumptions will be introduced to enable processes to be quantified and parts of the model may even have to be omitted.

In many physical and a few biological applications, quantitative models are available that permit estimation of the *frequency distribution* of hourly, daily or monthly values of some environmental state or condition. For example, the frequency distribution of hourly concentrations of sulphur dioxide at a point in the vicinity of a chimney may be estimated, even though prediction for a *specific hour* could be wildly in error. From such calculation, the probability of any given concentration being exceeded can be specified.

3.5.2 Global Models of Biogeochemical Cycles

Some of the largest environmental modelling exercises now being attempted are in the area of biogeochemical cycles of nitrogen, phosphorus, sulphur and carbon. Understanding the volume of flows and the various pathways is essential to knowing how the cycles have been or might be changed by human action and what management actions might be possible or required. For example, the model of the global nitrogen cycle (Figure 3.5) underlies estimates of present and future possible contributions of N_2O to the depletion of the ozone layer. Similarly the development of knowledge of the sulphur cycle (see Figure 3.6) underlies the acid rain problem described in more detail in Chapter 6.

When a particular environmental problem arises, it may be necessary to combine parts of existing models into one that serves the purpose in question. For example the ozone layer depletion problem requires an understanding of atmospheric diffusion (how fast will chlorofluoromethanes and nitrous oxide diffuse up to the stratosphere?); it also requires a chemical interaction model that states the rate of interaction with ozone: thirdly, there is need to know the relation of ozone depletion to the amount of increase in ultraviolet-B radiation; finally a biological model is needed of the effect of increases in ultraviolet-B upon target organisms at the surface. Because of the uncertainty of estimates, models of this sort are continually being revised and updated as more observations become available.

3.5.3 Persistent Substances in Food Chains

Within the grand design of global cycles it is often necessary to examine small components to trace the entry and pathway of toxic substances. Food chains are a good example of this type of modelling.

In any ecosystem the energy captured by green plants becomes available in a stepwise fashion to animals and microorganisms by flowing through a food chain. There is an inevitable loss of available energy at each of the steps in the chain (Figure 3.7).

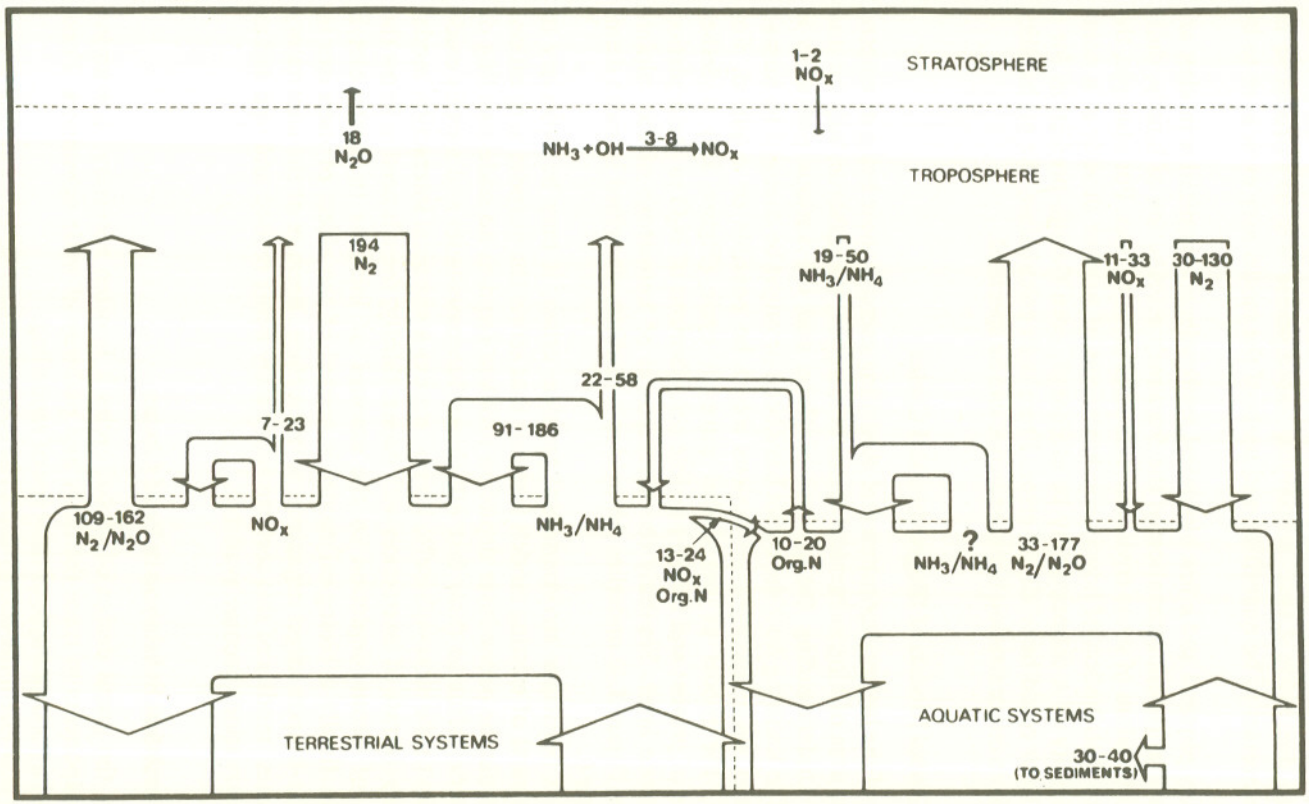


Figure 3.5 The global nitrogen cycle
The rates are given as Tg N yr^{-1}. The flows of N_2/N_2O are residuals obtained when balancing the terrestrial and aquatic systems (Soderlund and Svensson, 1976)

Source: Holdgate and White, 1977, p.40. (Reproduced by permission of SCOPE)

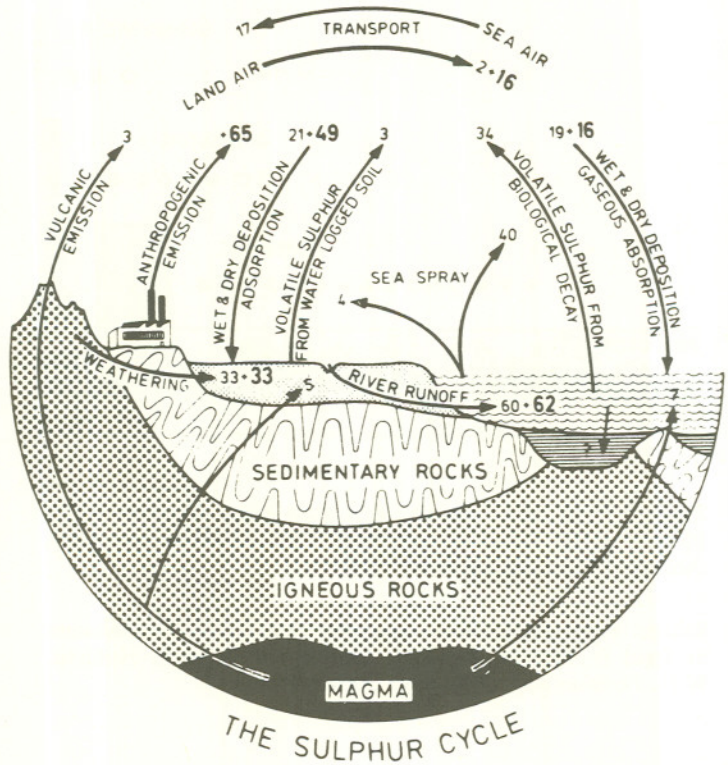


Figure 3.6 The first global sulphur cycle
Based on a preindustrial balance of the soil compartment. Fluxes are given in Tg (millions of tonnes) of sulphur per year. The man-made parts of the fluxes are indicated by plus signs (Hallberg, 1976)

Source: Holdgate and White, 1977, p.47. (Reproduced by permission of SCOPE)

Two consequences of the ecological principle have significance in assessing the risk to the environment of 'abnormal' levels of substances, and of 'abnormal' substances resulting from man's activities:

(1) Organisms at one level may be killed, breaking one link of the chain and thus disrupting the whole system.
(2) A substance which is persistent (not metabolised or biodegraded) may reach increasingly high concentrations as it is passed through the various levels of the chain. This process is referred to as biomagnification.

Persistent substances which have received attention recently include:

(1) Chlorinated hydrocarbon insecticides, e.g. DDT, dieldrin,
(2) Polychlorinated biphenyls (PCBs),
(3) Heavy metals, e.g. mercury, lead,
(4) Radioactive materials, e.g. Strontium-90, Iodine 131.

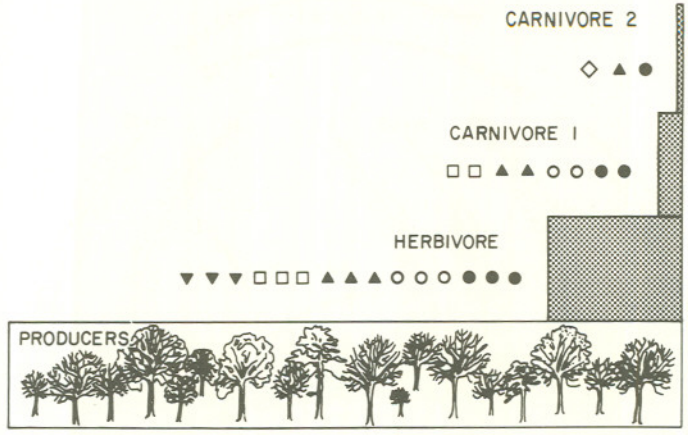
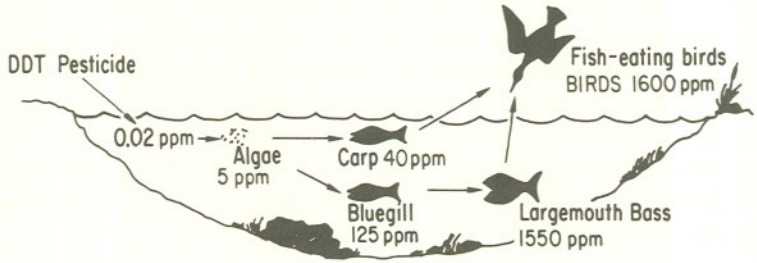


Figure 3.7 An intact natural ecosystem (e.g. a forest) where 10-20% of the energy at each level is passed to the next level. (Modified from Ehrlich *et al*, 1977)

The first three are discussed briefly here as examples of food chain models that have been developed.

DDT With the information that DDT could be found in carnivorous birds at concentrations a million times greater than those in the abiotic environment (e.g. Woodwell, Craig and Johnson, 1977), it was clear that this particular compound did obey the laws which could have been predicted at the time of its first extensive use in 1948. The relevant properties of this man-made compound in this context are:

(1) It is chemically stable, very few organisms can degrade it. Its half-life (time for 50% to be lost from the original site of application) has been estimated at 10-15 years.
(2) It is readily dispersed in water and in air in the form of suspension and by codistillation with water into air.
(3) It is barely soluble in water, but soluble in lipids, which are components of all biological systems. Thus it will preferentially associate with living over non-living material.
(4) It has become ubiquitous.
(5) In addition to being bioaccumulated by individual groups of organisms, it is biomagnified in food chains. The aquatic food chain shown in Figure 3.8 exemplifies this.



Buildup of chemicals along a representative food chain.

Figure 3.8 DDT in Clear Lake California (Modified from Treshow, 1976)

(6) It is a current constituent of the animal and human body (Wasserman, Tomatis and Wasserman, 1975).
(7) It is transferred from mother to foetus through the placenta and to the infant through breast milk.
(8) It activates or inactivates liver microsamal enzymes and thus interferes with normal metabolic pathways. This fact explains the majority of its biological effects.
(9) It is toxic not only to target organisms (the insect pests) but to a wide spectrum of living organisms.
(10) Some insects can become resistant to the toxic effect so that other compounds of higher toxicity are required to control the pests.

DDT was deliberately introduced for controlling insects and insect borne disease (O'Brien, 1967) and for future insect control so here the message is clear: there is an element of choice. The other persistent substances to be discussed here have been released as a result of man's activities, but usually as waste products.

Polychlorinated biphenyls (PCBs) PCBs share many of the properties of DDT. They are chemically inert, extremely stable, have low solubility in water, a high affinity for lipids and accumulate in living organisms from which they are excreted only very slowly. They have become widely distributed through the biosphere e.g. in the blubber of seals and porpoises on the East Coast of Scotland and Eastern Canada. Thus they are prime candidates for food chain accumulation and biomagnification and are now, like DDT, found in animal and human tissues around the world. Well documented effects of PCBs on human health include the rice-oil disaster in Japan (Yusho disease) which affected more than 1,000 people who consumed food contaminated by PCBs which had leaked into cooking oil from a heat exchanger (Kuratsune *et al.*, 1972). PCBs are also carcinogenic and are known to change the activity of the liver and to interfere with normal metabolic pathways.

Sources include transformers, capacitors, plasticisers, solvents, adhesives, sealants, coatings for lumer and concrete, tires and brake linings. The compounds get into the biosphere by atmospheric emissions, dumping into water, and by human contact with paints and plastics. In Canada and the US they are not prohibited in food containers and processing plants. The US Environmental Protection Agency has set standards of 0.01 ppb in water and 0.5-1.0 mg/m^3 for air. Even with complete restriction on further use however, existing residues will persist for long periods of time.

Mercury While mercury has long been recognized as an extremely toxic substance (ReVelle and ReVelle, 1974), attention has more recently been given to the accumulation of mercury in aquatic and terrestrial food chains. Between 1953 and 1960, mercury poisoning (Minamata disease) resulting from consumption of contaminated fish occurred in Japan (Irukayama, 1966) and in the early 1960s Sweden experienced a mercury problem in food. The source for the Japanese illness was industrial mercury waste from a chemical factory, accumulated from sediments and water into fish, which were consumed by humans. In Sweden the source was identified as a mercury fungicide which passed through the food chain from grain to hens to eggs to humans (Löfroth, 1970). The eggs contained 0.029 ppm mercury compared with 0.007 ppm in eggs from other European countries.

For mercury, the chemical form of the metal is all important in recognising the health risk. Organic mercury, especially methyl mercury, is much more soluble and more toxic than inorganic mercury. Methyl mercury has a half-life of 70 days in the human body compared with six days for inorganic mercury. Mercury accumulates in food chains and in addition, biological processes can transform inorganic to methyl mercury (Jensen and Jernelov, 1969). The chemical and biological reactions of mercury in the environment are complex and incompletely understood. Even naturally occurring mercury can be solublized and released into the biosphere as a result of other processes such as acidification of water resulting from acid precipitation.

Detailed studies on mercury in food chains have resulted not only from increased awareness of the potential risks, but also from improved analytical techniques (e.g. Linstedt and Skorfuing, 1972). Studies on fish have shown that mercury is taken up as methyl mercury, either emitted as such, or methylated in sediments by microorganisms. The rate of uptake and the retention time for mercury in fish varies greatly with the species and environmental conditions. It is not yet clear whether the major pathway of mercury to fish is via the water (direct uptake) or from contaminated food (food chain accumulation). Both of these processes can and do occur. However, at the next level of the food chain, when birds prey on fish, or man consumes fish in the diet, food is the major source of mercury intake.

It is tempting to extrapolate from these data and make generalizations for the potential impact of all persistent substances in food chains. Clearly, compounds or elements which exhibit any of the properties listed for DDT

should be regarded as potential risks to the health of humans, biota and the environment and thus worthy of close scrutiny. More precise models can however only be constructed on the basis of hard scientific evidence of which there are basically two types:

(1) *Observations and measurements* of concentrations of the pollutant in field situations which give a snapshot at a moment in time but no information on the dynamics of the system — the rates of uptake, loss, retention time etc. at each level of the food chain, nor the toxic effects on populations. Sampling extended in time can partially clarify this, and is also useful for monitoring the disappearance of the pollutant with time.

(2) *Experimental situations* where populations and organisms are isolated and examined for toxicity, rates of uptake and excretion etc., or where simplified ecosystems are set up in microcosms, and known amounts of the substance (often isotopically labelled tracers) are introduced.

Both types of study have great value; ideally the experimental approach should precede the field studies, but this is clearly not possible for pollutants which have already been allowed to escape into the biosphere.

Our awareness of food chains as risk systems should alert us to the need for careful screening of potential pollutant substances. Experimental work in microcosms (experimental artificial ecosystems) are concerned with this aspect of the problem. But we have not always been wise before the event. Where contamination of food chains has already occurred, or is continuing, e.g. from lake sediments which acted as 'sinks' for PCSs, continued monitoring will improve understanding of the complex biological transfers that we are trying to model.

3.6 ESTABLISHING THE RELATIONSHIP BETWEEN THE DOSE AND THE EFFECT

The last link in the food chain, the last piece of any model of a risk system, is the link that connects the impacting variable to its receptor. The importance of the last link justifies the special attention that it frequently receives in the estimation of risks.

What amount of ultraviolet-B radiation, or what increase in the amount is required to produce a specified additional number of cases of skin cancer? In a drought, what level of moisture deficiency and what period of time is required to reduce crop yields significantly or to produce crop failure? How much ingestion of mercury in fish or in bread made from seed-grains is required to produce mercury poisoning? How much damage will result from a flood that covers the floodplain with a metre's depth of flood water, and will the damage be twice as large if the water rises another metre?

These are all examples of the same kind of relationship although the terminology used to describe them differs. For toxic pollutants in the

environment or in food chains the term dose-response or dose-effect is used. For droughts the phrase more frequently used is impact-response or impact-effect. If one particular indicator of drought is used the term may be moisture deficit-effect or drought index-effect. For floods the relationships may be expressed in stage-damage curves.

Whatever the terminology used there are important questions to be asked about the curve, its slope and the nature of the relationship. They are important because they have considerable bearing on the setting of standards and the adoption of policies to reduce risk.

3.6.1 Dose-Effect Relationships

Threshold relationships are shown in Figure 3.9. The simple case is in curve (1) where there is no risk until a certain level of exposure takes place. The level of exposure is the point where curve (1) leaves the abcissa.

A more complex and more common case is illustrated by curve (2). Here there is some effect at low doses, increasing relatively slowly, until a take-off point is reached at which the effects increase dramatically. Measured over a

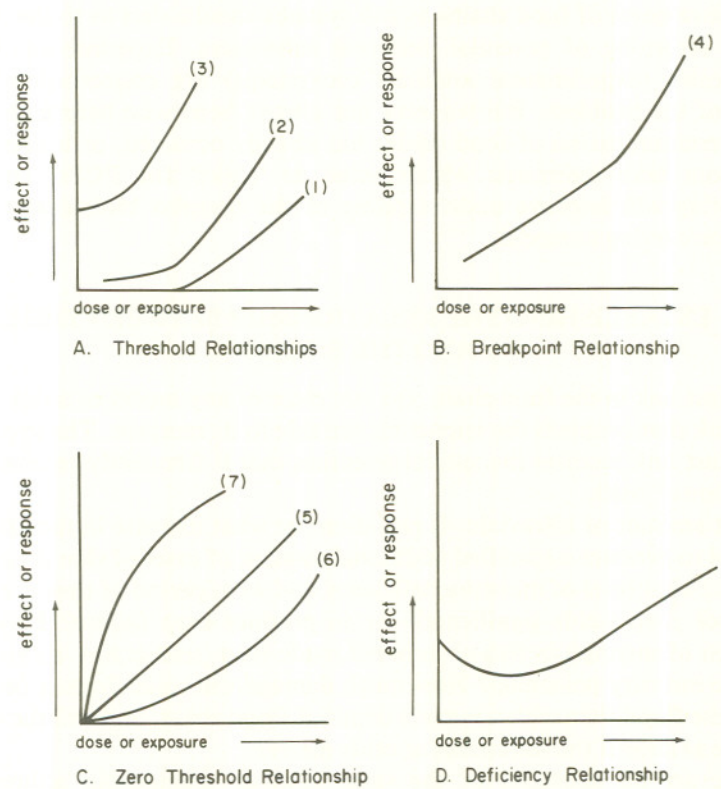


Figure 3.9 Dose-Effect Relationships

whole population this could be a case where a few susceptible people are affected by low exposures but the mass of the population are not affected until a certain threshold or take-off point is reached.

Both these curves may clearly be linked to policy options. In the case of curve (1) it may seem necessary to keep the level of exposure for all persons below the threshold level. In curve (2) it may not be practicable to reduce exposure to zero in order to protect the relatively small number of susceptibles affected. In this case the standards may be set at the take-off point or somewhat below it, and additional steps taken in other ways to safeguard or reduce the exposure of susceptibles.

A more complicated case is shown as curve (3) where the effects of exposure are impossible to separate from similar effects which occur without exposure. In other words there are some 'effects' at zero dose and any dose above zero will increase those effects.

A breakpoint relationship is shown in Figure 3.9(b). In curve (4) a marked break in response occurs at some (usually higher) level of dose.

The curves shown in Figure 3.9(c) are all variations of zero threshold relationships. Curve (5) is the classical linear exposure/effect relationships when no theshold exists. Zero risk occurs only at zero exposure. This is the conservative position that is assumed to exist for exposure to ionizing radiation.

Curve (6) is a variant showing lower sensitivity to risk at lower levels, and curve (7) is the reverse showing increasing sensitivity at lower levels of risk exposure.

In practice it is rarely possible to specify the slope of the dose-effect curve with confidence or to state exactly where the threshold level is if it exists. A number of factors account for the lack of precision in dose-effect curves. The vulnerability of individuals varies for day to day and physiological diversity in human populations is such that effects may vary according to the segment of a population exposed. Measuring techniques have their limits of detection and monitoring of levels can only be carried out in a few selected samples or sites. At very low exposures there may be effects which are not easily detectable and observations at the upper end of the curve are difficult to obtain because massive exposures are relatively rare. A great deal has been learned about radiation exposure of high levels from cohort studies of the populations of Hiroshima and Nagasaki. Normally, ethical considerations prohibit the deliberate exposure of human beings to large amounts of hazard. An alternative approach often used is the conducting of tests on animals but extrapolation of animal data to estimate human experience is always imprecise, and unavoidably so.

The principle use of dose-effect curves is thus to predict the consequences of very high and very low exposures which usually cannot be adquately observed or measured.

In Figure 3.10 a generalized exposure-effect curve shows uncertainty of estimates at high and low levels of exposure and the need to extrapolate

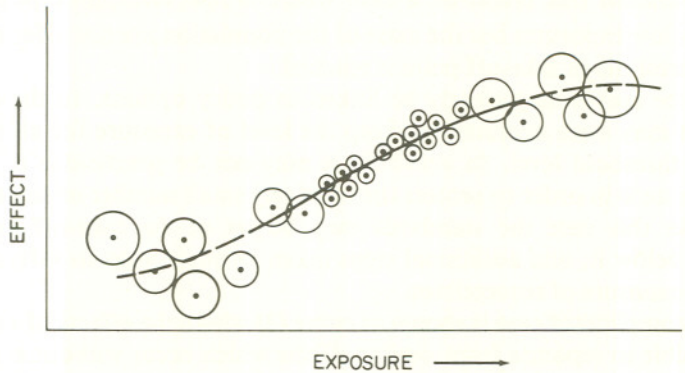


Figure 3.10 Generalized exposure-effect curve, showing uncertainty at high and low exposures: diameter of circles indicates degree of certainty about data points

Source: Lowrance, 1976, p.38. (Reproduced by permission of Wm. Kaufman, Inc.)

upwards and downwards from 'middle range' observations.

As indicated above, the practice in radiation exposures has been to extrapolate to the point of origin — in other words to make a 'no threshold' assumption. In many other areas the conventional approach has been to assume that a threshold level exists and that a small amount of a bad thing is harmless. There is a current trend towards the discovery of harm at lower and lower doses. The threshold concept is on the defensive and many scientists are coming to believe that for many hazardous substances — especially new substances not previously found in the human environment — any exposure is potentially harmful or risky.

There is other evidence however, that some toxic substances are required by the human body in very small quantities for complete health. Deficiencies of some elements, including the toxic heavy metals can in fact be detrimental. In such cases the dose-effect curve would appear as in Figure 3.9(d).

Three different types of tools are used to develop the information needed to construct and interpret dose-response curves: (1) Clinical studies; (2) Epidemiological (population) studies; and (3) animal studies.

Clinical Studies The most reliable subject for determining health effects on man is man himself. For obvious reasons, the use of man as the subject of experiment (clinical studies of harmful impacts) must necessarily be extremely limited and subject to extremely stringent control and safety procedures. There are areas where effects of low exposures can be examined in a carefully controlled clinical study. For example, the US Environmental Protection Agency is conducting a series of controlled human exposure studies to examine the effect of being exposed to low concentration of sulphates.

Epidemiological Studies A second tool which directly provides data on human effects are epidemiology or population studies. These studies focus on following a specific group of individuals over a period of time to see if patterns linking cause and effect can be discerned. The conclusions drawn are necessarily based much more on inference than in the controlled clinical studies where one knows to what the subject has been exposed. There are two types of population studies that are being developed, *retrospective* and *prospective. Retrospective* population studies essentially start from an observed effect and attempt to trace back to find what might be the possible cause or causes of the effect. For example, such a study might focus on a geographical area that has a high cancer incidence (high cancer cluster). The study would be designed to detect similar patterns and life style, conditions, or environmental factors, which could have contributed to the observed effect. *Prospective* population studies, on the other hand focus on following populations which have a known exposure to a particular substance, for example, a carcinogen, in order to determine what the possible effects may be to that exposure. Figure 3.11 portrays how such studies fit into a proposed US EPA research Plan to develop data needed to determine Environmental Exposure/Cancer Relationships.

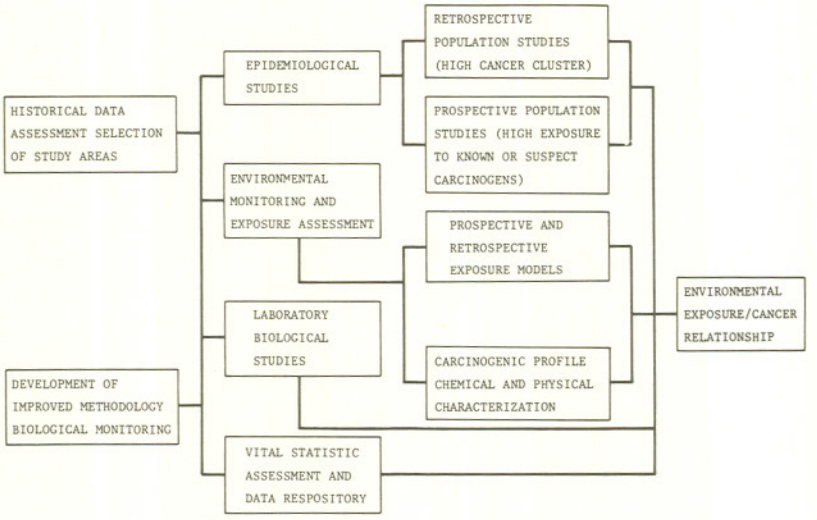


Figure 3.11 Studies being undertaken to establish the dose-effect relationship for environmentally caused human cancers
(US Environmental Protection Agency, 1974)

Animal Studies Most of the information on dose-response comes from animal studies. Tests are run on various animals to determine acute, chronic and genetic effects. Selection of the specific types of animals depends upon the effect to be examined; the target organ that is likely to be affected, and the

extrapolation of that response to man; and on the sampling, reproducibility, and timing requirements of the experiment. Extrapolation of animal data to man is the major problem for using this tool and considerable research is needed to narrow the error bar associated with such extrapolations.