SECTION II. TECHNIQUES AND METHODS

Introduction

This section provides a detailed breakdown of:

i. Typologies (defined in Chapter 3) developed under the general methodology of human health risk assessment.
ii. Methodologies developed or being developed for ecological risk assessment, in general generated from the human health approach.
iii. Methods developed specifically for industrial risk scenarios which may incorporate elements of either, or both, human health or ecological risk assessment.

The objective is to provide the reader with a sound knowledge of the principles of each method, the stages or steps involved in each, examples of their use and the problems associated with each. It is essentially a resumé of the environmental risk assessment methods in use or being developed. This section deals with the technical details of the process, some of which are complex in nature and require a certain degree of scientific and technical understanding from the reader. However, the text is, again, pitched at a level to be understood by a reasonably wide audience. It is not targeted towards experts in the respective fields of risk assessment.

Chapter 5 details the risk assessment methods which have been developed to protect human health from damage by physical, chemical and biological agents in the environment. The chapter centres around chemical risks which reflects the current degree of knowledge, experience and concern about such agents in the environment and the potential for human exposure. The typologies based on end-point are dealt with in detail for the risk assessment of chemicals such as neurotoxic risk assessment. Risk assessment techniques used to protect humans from ionising radiation, pathogens and genetically modified organisms are also described.

Chapter 6 looks at the risk assessment techniques in use and under development to protect ecosystems, or the environment, excluding humans. The agents considered include chemicals, pesticides and genetically modified organisms. The method developed for the regulation of new and existing substances is highlighted and also the generation of a methodology for ecological risk assessment from the human health approach, with all the associated problems and difficulties.

Chapter 7 covers the application of environmental risk assessment in industry. The objective is to provide an overview of the variety of techniques used to assess risks to the environment (ecosystems and humans) arising from particular industrial scenarios such as non-routine releases, routine releases, contaminated land and waste disposal.

To illustrate the methods and concepts discussed in each chapter, many practical examples are provided.

This section is targeted towards some government officers, policy makers, regulators and local planners, who do not need to be experts in risk assessment but require a sufficient understanding of the environmental management tool to ensure it is fully utilised. Chapter 7 will prove particularly useful to SMEs as it describes the role that risk assessment is playing in large industrial companies. Its use could provide substantial benefits to SMEs and indeed, in the future, they may be required to use it by legislation. It will also prove interesting to the general public who wish to be informed of the measures

and tools which are used to protect them and the "environment" and, of course, to students of the subject of environmental management.

The information provided is intended as an introduction to environmental risk assessment as an environmental management tool. Part II of this book guides the reader to a vast range of information available on the specific details of each of the methods outlined in this section.

5. HEALTH RISK ASSESSMENT

The methodologies and techniques used in health risk assessment are firmly established. This chapter provides an overview of current health risk assessment methodologies used for physical, chemical and biological agents and will reflect the typologies in Chapter 3. The influence of the NAS model can be seen clearly in chemical and biological risk assessment. Risks to human health from site-specific industrial activities are covered in Chapter 7.

5.1 Physical risks - ionising radiation

Radiation risk assessment methodologies are well developed and, due to the nature of nuclear risks, many international organisations are involved. This book is not concerned with the scientific arguments surrounding the biological effects of radiation, only with its use in radiation risk assessment. Assessments of risk are carried out by the regulatory agencies involved in radiological protection - setting radiological dose limits for instance and site-specific assessments, and by the nuclear industry - compliance with legislation and site-specific decisions. Radiation risk assessment has a longer history than that for other types of risk and the influence of the NAS model - developed for human health risk assessment for chemicals - is less marked. Of the many international bodies involved in radiological protection and radiation risk assessment, the International Commission on Radiological Protection (ICRP), and the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) are influential on a global scale. UNSCEAR does not recommend risk management action but carries out evaluations of doses, effects and risks of radiation, which are then used by bodies such as the ICRP. ICRP has had a major influence on European legislation on radiological protection through the EURATOM directives.

5.1.1 Human health risk assessment for ionising radiation

The risk assessment methodology described here is that used by the ICRP (ICRP, 1990). It is widely accepted and has been shaped by policy and goals laid down by radiological protection principles and enshrined in European legislation. The ICRP has recognised the difficulties in using the term "risk". It specifies that "risk" should only be used as a descriptive term and that "probability" should be used if that is what is meant.

Hazard identification/dose-response relation

Biological damage by radiation can be either deterministic (the severity of effect varies with dose) or stochastic (the severity of effect is not altered by increasing the dose of radiation, only the likelihood of the effect or disease occurring). Radiological risk assessment uses human data to the maximum extent. Data on deterministic effects have come from the side effects of radiotherapy, the effects on early radiologists, from the effects of the nuclear bombs at Nagasaki and Hiroshima and from radiological accidents.

Deterministic effects

Radiation exposure can kill human cells. There is a continuous process of loss and replacement of cells carried out within the body, but radiation can lead to a net reduction in the number of cells available to maintain organ or tissue function. If the decrease is large enough, this may result in a pathological condition such as a loss in tissue function. If the tissue is vital and the damage sufficient, the result will be death. For healthy individuals, the probability of causing harm will be zero at doses up to some hundreds or thousands of millisieverts, depending on the tissue, and will increase steeply to 100 per cent at the threshold of action. Above the thresh-

old, the severity of harm will increase with dose, reflecting the number of cells damaged. Deterministic effects of radiation include sterility, neurological effects and vascular effects such as subcutaneous oedema, or secondary tissue damage such as cataracts.

Stochastic effects

Stochastic effects can be somatic or hereditary. A modified somatic cell may still retain its reproductive capacity and may give rise to a clone of modified cells. The clone may be eliminated or isolated by the body's defence mechanisms. If it is not, after a prolonged and variable delay called a latency period, it may result in the uncontrolled proliferation of modified cells - cancer. The defence mechanisms are not likely to be totally effective even at small doses so they are unlikely to give a threshold in the dose-response relationship. A modified germ cell in the gonads may transmit incorrect hereditary information, thus causing disorders in the offspring.

One of the major difficulties in establishing a dose-response relationship for radiation is the determination of what happens at low doses. This is examined further in Box 5.1. This problem is common to all cancer risk assessments. The ICRP has concluded that, although the simplest relationship between an increment in equivalent dose and the resulting increment in the probability of a defined stochastic effect is a straight line through the origin, different models need to be used to take account of the abilities of different types of radiation to cause damage in cells. In order to project overall cancer risk for an exposed population, from data based on a short time period of radiation exposure, a mathematical extrapolation model is required. The ICRP has selected a multiplicative model in which time distribution of the excess risk follows the same pattern as the time distribution of natural cancers. The use of multiplicative models implies that for the majority of solid cancers there is an increasing risk with time after exposure, following the increase in natural incidence with age.

Detriment is a measure of total harm that would eventually be experienced by an exposed group and its descendants as a result of the group's exposure to radiation (ICRP, 1977). Weighting factors representing the severity of the harm are included in the definition of detriment, as well as probabilistic estimates of occurrence. Detriment is the basis of the assessment of consequences of continued or cumulative exposures, in order to recommend dose limits. Radiation affects different tissue to different degrees. A weighted tissue equivalent dose produces the same degree of detriment irrespective of the tissue or organ involved.

In many risk assessments, especially for chemicals, the only end-point examined is certain specified diseases. Detriment in radiological risk assessment represents a number of end-points, including mortality and morbidity, not only to the individual or society exposed, but to subsequent generations. An aggregated representation of detriment exist which includes the probability of attributable fatal cancer, the weighted probability of severe hereditary effects, the weighted probability of non-fatal cancer, and the relative length of life lost.

Exposure assessment

Because the ICRP is a body for radiological protection, its risk assessments will be used in the recommendation of dose limits. It uses the dose-response relationship and examines three exposure scenarios: public, occupational

Box 5.1 The biological effects of ionising radiation at low doses

The effects of low doses of radiation have had to be estimated by extrapolation from the effects of much larger doses, combined with the knowledge obtained from experiments on laboratory animals and in vitro. Small doses have three possible effects:

- The production of cancer in the irradiated individual.
- The production in his or her offspring of congenital malformations or hereditary disease.
- Possibly, if the irradiated subject is a foetus in utero, some diminution in intellectual capacity.

The first two effects result from mutations in the cellular DNA and they appear in only a small proportion of irradiated people; they are stochastic effects. The last effect is uncertain and at this time, science can neither prove nor disprove an association. Such damage is not the result of mutations and there is no biological reason to assume that no threshold exists. The most valuable evidence relating to hereditary effects is obtained from comparisons of the health of children born to survivors of the atomic bomb explosions in Japan, who were exposed within 2,000 m of the hypocentres of the bombs, and consequently heavily irradiated, with that of the offspring who were more than 2,500 m from the hypocentres (Schull et al., 1981; Neel et al., 1990).

Indicators that have been examined include stillbirths, major congenital defects, cancer under 20 years of age, death under 26 years of age, sex ratio, physical development and specified chromosomal abnormalities. For none of these were there statistically significant differences between the groups. Genetic effects have, however, been observed in all animal species that have been studied experimentally and there is no reason why human tissue should not react qualitatively in the same way. Observations on mice suggest that at low doses, at low dose rates, the doubling dose (the dose required to produce an incidence of mutations equal to the incidence that occurs naturally) is about 1 mSv. (Doll, 1993).

The observations needed to derive a relationship between dose and mortality from cancer, derive from three sources:

- Observations on the survivors of the atomic bomb explosions in Hiroshima and Nagasaki.
- Observations on persons who have been treated by radiotherapy or who have been exposed to a large number of radiographic examinations.
- Observations on people routinely heavily exposed in their occupation.

The first are the most extensive and provide evidence that between about 0.2 and 2 Gray, the mortality from cancer is linearly proportional to dose. At lower doses the evidence is less clear. For leukaemia for instance, the mortality with low dose appears to be less than would be expected if there was a linear relation between dose and response. For other cancers there appears to be a linear relationship.

From these data, four national and international committees have reached very similar conclusions about the effects of exposure to 1 Gray. These are based on a number of assumptions:

- The risk of leukaemia is maximal after 5-10 years and then falls to near zero after 35 years.
- The relative risk of all other cancers rises to a maximum after 10-20 years and then remains constant.
- The relative risk per unit dose observed in the Japanese population applies to other populations where the normal risk of particular cancers are very different (see Table 5.1).

Table 5.1: Estimated lifetime risks of fatal cancer per Gray acute irradiation

Type of cancer	Excess risk per 100 persons			
	UNSCEAR[1]	NRPB[2]	NRC[3]	ICRP[4]
Leukaemia	1.0	1.2	1.8	0.9
Other cancer	9.7	10.6	6.9	8.6
All cancer	10.7	11.8	8.7	9.5

1 United Nations Scientific Committee on the Effects of Atomic Radiation (1988) estimate for population of Japan.

2 National Radiological Protection Board (Muirhead et al., 1993) estimates for population of UK.

3 National Research Council (1990) estimates for population of USA. The National Research Council, unlike the other bodies, gives estimates of risk for 0.1 Gy and uses a linear-quadratic rather than a linear equation for estimating the risk of leukaemia. Use of this equation gives a risk at 1 Gy of 1.8 per 100 persons. The NRC's published estimate of excess risk from all cancers is 0.8 per 100 persons per 0.1 Gy.

4 International Commission on Radiological Protection (1991) estimate for mean of five populations (Japan, China, USA, Puerto Rico, UK).

Source: Doll, 1993

and medical exposure, to recommend appropriate dose limits. The data for exposure assessment come largely from measured doses. Monitoring of radiation levels, particularly occupational doses, has produced much more complete data than for chemical exposures. In estimating public exposures, account is taken of the differing pathways of exposure. The ICRP has examined a range of test exposures based on various exposure scenarios.

Risk characterisation/estimation

Data from the test scenarios are compared with the dose-response evidence to arrive at the probability of a range of effects at those doses.

Table 5.2 shows the attributes of detriment for occupational exposure (ICRP, 1990). From these data, dose limits can be recommended after taking into consideration social, economic and feasibility issues.

UNSCEAR have carried out risk assessments on ionising radiation (UNSCEAR, 1988). They examine radiation levels and doses of radiation received through medical, occupational and general environmental exposure, and the resultant effects. They, like the ICRP, use effective dose equivalents to take into account the different sources of radiation and the biological effectiveness of different types of radiation. They derive risk coefficients, which are the "risk" or probability of a harmful event, per unit dose of radiation. They have calculated both individual risk, which is the probability of an individual suffering a specified harm, and collective risk. For cancer, this may be expressed as the expected number of cancer deaths in a specified population or the number of person years lost because of cancer deaths per unit collective dose.

UNSCEAR does not make any assessment of the expected detriment from exposures, which would be the last stage of a risk assessment because of its terms of reference. Its purpose is to evaluate doses, not to set standards or make value judgements. It believes that all assessments of risk involve assumptions and decisions that are not scientifically based, such as the choice of models and weighting factors.

Table 5.2: Attributes of detriment due to exposure of the working population

Annual effective dose (mSv)	10	20	30	50	50 (1977 data)
Approximate lifetime dose (Sv)	0.5	1.0	1.4	2.4	2.4
Probability of attributable death (%)	1.8	1.6	5.1	8.6	2.9
Weighted contribution from non-fatal cancer (%)[1]	0.4	0.7	1.1	1.7	-
Weighted contribution from hereditary effects (%)[1]	0.4	0.7	1.1	1.7	1.2
Aggregated detriment (%)[2]	2.5	5	7.5	12	
Time lost due to an attributable death given that it occurs (y)	13	13	13	13	10-15
Mean loss of life expectancy at age 18 years (y)	0.2	0.5	0.7	1.1	0.3-0.5

[1] Weighted for severity and loss of lifetime.
[2] The sum of the probability of attributable fatal cancer or equivalent detriment (rounded).

Source: ICRP, 1990

The use of risk assessment for radiation is generally accepted internationally. It has a longer history than risk assessment for other types of risk and the international community involved is relatively small. Concerns in radiological risk assessment are similar to those for chemical risk assessment discussed in the next section. They involve questions over:

- the certainty of the data being used in assessment;
- differing scientific interpretations of the basic data;
- the basis of the choice of mathematical models for use in low dose extrapolation;
- the choice of exposure scenarios used in the risk assessment.

5.1.2 Site-specific radiation risk assessment

The risk assessment procedure for exposure to radiation from a specific source generally follows the NAS model. Radioactive substances are identified at source, their environmental fate, transportation and availability to receptors are then modelled or monitored (exposure assessment). Dose-response information is obtained from epidemiological studies and laboratory experiments, uncertainty and safety (risk) factors are then applied. Risk characterisation is based on the exposure and dose-response assessment data to produce exposure levels such as "effective doses" and "dose constraints". A full risk assessment incorporating exposure assessment and risk characterisation is carried out to determine the need for risk reduction measures. If the public is exposed to levels that present an "unacceptable" risk, risk reduction measures need to be implemented. See Box 5.2.

Risk assessment can be used in a predictive capacity, for example, in the siting of nuclear power plants to determine the risks posed to the public of the development. Risk assessment is also frequently used to estimate the probabilities of possible consequences of accidents at a nuclear power plant. The techniques used to predict potential releases are similar to those used in the process industries and are covered in Chapter 7.

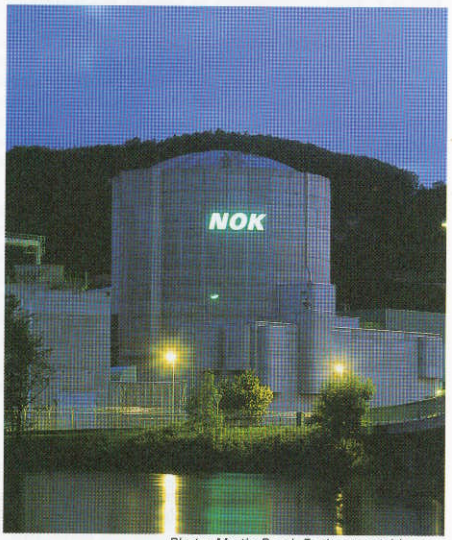


Photo: Martin Bond, Environmental Images

The CEC has produced a methodology for the assessment of the radiological consequences of routine releases of radionuclides to the environment (EC, 1995). The methodology is known as CREAM (Consequences of Releases to the Environment: Assessment Methodology). CREAM consists of a series of inter-linked models, which describe the transfer of radionuclides through the various sectors of the environment, the pathways by which people may be exposed to radiation, and the resulting health detriment. Detailed models

Box 5.2 Risk assessment of cancer occurrences in Seascale from all sources of radiation exposure

A risk assessment was carried out to examine the radiation risks posed to the general population of Seascale (NRPB, 1995). It was carried out for the UK Government in response to concerns about the incidence of childhood cancer in the vicinity of a nuclear reprocessing plant at Sellafield. The sources of exposure examined were: natural radiation; medical practices; fallout from the testing of nuclear weapons in the atmosphere; releases, both planned and accidental, of radioactive materials to sea and to atmosphere from the British Nuclear Fuels plant at Sellafield; the Chernobyl reactor accident in 1986; and routine discharges of radionuclides from a plant near Whitehaven. For each source of exposure, doses were calculated to children and young persons in Seascale between 1945 and 1992.

The procedure adopted for the assessment and the models and the data used were discussed and agreed with independent scientific committees within the UK Government.

All pathways of exposure were considered. Absorbed doses to tissues from external irradiation of the whole body and intakes of radioactive material by ingestion and inhalation were calculated. For calculations of doses from operations at the Sellafield plant, use was made, wherever possible, of results of measurements made in the environment. Where such measurements were not available, discharge data were used, together with mathematical modelling techniques, to estimate dose rates or concentrations of radionuclides in environmental media.

The objective of the study was to calculate the best estimates of doses and risks of radiation-induced cancers in the study population. Best estimates of input data and average values for habit data were used with consideration given to any factors that might lead to a greater dose. The models used to calculate risks were derived from a review of the available human data and were selected to give best estimates of the risk of radiation-induced cancers for the UK population.

The analysis concluded that in the study population of 1,348 children and young persons born in Seascale between 1945 and 1992 and followed to age 24 years or to 1992, whichever was sooner, 0.46 cancers of radiation-induced leukaemia and non-Hodgkin's lymphona would be expected (an individual risk of 1 in 3,000), of which 0.36 would be fatal. Most of the dose and hence risk, is attributable to natural radiation; only about 10 per cent can be attributed to doses that result from all operations at the BNFL plant at Sellafield. The risks of radiation-induced solid cancers were also calculated: 0.22 cases would be expected of which 0.05 would be fatal.

have been developed for the transport of radionuclides in the aquatic and atmospheric compartments. Individual exposures can be compared with the appropriate dose limits or constraints as required by legislatory procedures. Collective doses can be used to estimate health detriment, in the form of the number of possible effects in the population.

5.2 Chemical risks

5.2.1 EC legislation and technical guidance

The procedure for assessing the risks posed by new chemicals in the EU is outlined in Commission Directive 93/67/EEC which lays down the principles for assessment of risks to man and the environment of substances notified in accordance with Council Directive 67/548/EEC. Commission Regulation EC No. 1488/94 outlines the procedure for the evaluation of the risks posed by existing substances. A detailed step-by-step guide to the procedure for the assessment of both new and existing substances is now available in a Technical Guidance Document (TGD) developed by the European Chemicals Bureau (CEC/ECB, 1996). The EU has adopted the NAS framework for the risk assessment of new and existing substances. The assessment of new and existing substances examines human health and ecological risks. EU human health risk assessment addresses eight toxic effects: acute toxicity, irritation, corrosivity, sensitisation, repeated dose toxicity, mutagenicity, carcinogenicity, and toxicity for reproduction. Three human populations are considered: workers, consumers

and humans exposed directly via the environment through inhalation, oral and dermal routes. The principle of the assessment is to compare the concentration of a substance to which a population is exposed with the concentration at which no adverse effects are expected to occur. The risk characterisation involves the calculation of the exposure: effect ratio or a qualitative evaluation of the likelihood that an effect will occur at the given exposure. For each population potentially exposed and each effect, a decision is made on the EU's action in respect of the substance being examined. The conclusions for new substances will either be that the substance is of no concern and no action is required, or that the substance is of concern. In this case the EU may require further information or recommend risk reduction actions.

For existing substances, if the substance is of concern, then further information and/or testing may be required or risk reduction measures proposed, taking into account those already in place. Examples of risk reduction measures include:

i) Providing information to the public regarding the safe and responsible handling of substances or products.
ii) The use of emission permits which set limits.
iii) Marketing restrictions, e.g., limiting production, import volume, or use.
iv) Total ban of a substance or activity (CEC, 1996).

To support the implementation of this legislation a European Chemicals Bureau (ECB) has been established as part of the Joint Research Centre facility in Ispra, Italy. The Centre's activities include the evaluation of chemical substances, involving data collection, priority setting and risk assessment of about 100,000 European Inventory of Existing Commercial Substances (EINECS) and all new chemical substances. The ECB has developed a database for the data collection phase - the International Uniform ChemicaL Information Database (IUCLID), and is developing a risk assessment support tool - the European Union System for Evaluation of Substances (EUSES), which will be used by the competent authorities to evaluate the risks due to new and existing substances.

In addition to the specific items of legislation on new and existing substances, there are many others addressing the risks posed by chemicals, ranging from those on the classification and labelling of chemicals to the proposed directive on air quality. See Box 5.3.

Of course, several international organisations, such as IPCS and OECD, are conducting their own programmes on human health and ecological risk assessment for chemicals. Such organisations often provide advisory expertise to the EU or work together in collaboration with various sections of the European Commission Directorates. Effort is taken to ensure that future work of organisations is complementary. Where such co-ordination is aimed at increasing the number of substances assessed within the objectives of Agenda 21, Chapter 19, it has been recognised that future assessments are carried out on the basis of similar principles. For instance, efforts are being made to harmonise the approach laid down in the OECD Screening Information Dataset (SIDS) manual and that followed by the IPCS in producing their Environmental Health Criteria Document series (McCutcheon, 1996).

In addition to the production of the Environmental Health Criteria documents

Box 5.3 Ambient air pollution and human health

In the urban areas of modern European cities, typical activities including traffic, combustion processes and industrial production generate emissions to air, thus producing elevated concentrations of pollutants. At certain levels, particular pollutants, especially in combination, can cause significant health effects in humans, including respiratory dysfunction, morbidity and mortality. The World Health Organization has produced Air Quality Guidelines for a selection of pollutants based on dose-response toxicity testing with the application of safety factors in order to provide a margin of safety to help protect sensitive members of the population for non-carcinogenic pollutants and based on risk assessment for genotoxic pollutants (WHO, 1995). The recent Council Directive 96/62/EC on Ambient Air Quality Assessment and Management provides the regulatory support for the setting of air quality limit values (AQLV) and alert thresholds for several air pollutants, including those pollutants covered by previous individual Directives.

The AQLV and alert thresholds are designed to protect human health. For certain periods high pollutant emission rates, combined with particular weather conditions, will mean that guideline or limit values will be exceeded. On these occasions the public will be alerted to allow them to take precautionary steps to reduce exposure, this is particularly important for the more sensitive members of the population. Recent monitoring data indicate that for certain pollutants, WHO guideline values are frequently exceeded in many European cities (EEA, 1997).

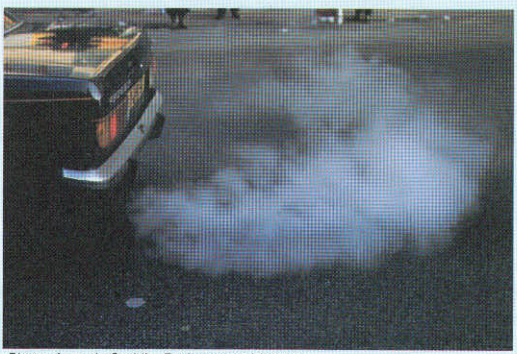


Photo: Amanda Gazidis, Environmental Images

The management of urban air quality utilises the principles of risk assessment effectively, evaluating the dose-response relationship, calculating the exposure concentration from monitoring and modelling studies and then implementing management initiatives to reduce the environmental concentration and therefore to reduce the risk of damage to the public (and ecosystems). There is also mention, in the 1996 Air Quality Directive, of risk assessment being a suitable method in considering further pollutants for air quality management and limit values.

(see Box 5.4), the work of IPCS includes the Concise International Chemical Assessment Documents (CICAD), and the development and validation of methods, and the Harmonization of Approaches to the Assessment of Risk from Exposure to Chemicals. The OECD Chemicals Programme includes work on the Investigation of the Risks from Existing Chemicals (HPVC) and Risk Reduction Activities. Much of the work carried out by such organisations is at the forefront of risk assessment methodology and practical implementation development, contributing towards the drive for methodology transparency and broad scientific acceptance throughout the regulatory and industrial communities. A technical report published by IRPTC/UNEP and ECETOC provides a comprehensive inventory of critical reviews on chemicals undertaken by international and national organisations (ECETOC/IRPTC, 1996).

5.2.2 Human health risk assessment for chemicals

Most methodologies for human health risk assessment of chemicals are based on the NAS model. A number of methodologies exist

Box 5.4 IPCS Environmental Health Criteria for Methomyl

Sources of human exposure

Methomyl is a carbamate insecticide used on a wide range of crops throughout the world. Crops protected include fruit, vines hops, vegetables, grain and cotton. The main sources of human exposure are during preparation and application of the product and from the ingestion of crop residues in foodstuffs.

Evaluation of human health risks

Methomyl is a carbamate chlorinesterase inhibitor with a well-known mechanism of toxic action. Acute toxic signs in animals are typical of a chlorinesterase inhibitor. The reversibility of acute toxic action is rapid, with survivors showing quick recovery from toxic signs and reversal of chlorinesterase inhibition in the blood and brain. The quick recovery from toxic effects is due to the rapid reversibility of methomyl-inhibited chlorinesterase, which is facilitated by the rapid clearance of the compound from the body.

There was no evidence of carcinogenic potential from three long-term studies in rodents. NOELs were identified in each of the long-term animal studies. These were 5 mg/kg body weight per day in rats, 8.7 mg/kg body weight per day in mice and 3 mg/kg body weight per day in dogs. In the absence of any marked species differences in toxic effects in these studies, the NOEL in the dog of 3 mg/kg body weight per day is recommended for the purpose of human risk estimation.

Conclusion

Considering the qualitative and quantitative characteristics of methomyl toxicity, the Task Group concluded that 0.03 mg/kg body weight per day will probably not cause adverse effects in humans by any route of exposure.

Source: WHO/IPCS, 1996a

due to differences in the toxic mechanisms exerted by different classes of chemical and the toxicological end-point being assessed. The end-point being assessed could be death, or a specific pathological condition relating to exposure to a chemical. When attempting to assess the risks from an immuno-suppressant toxin, specific end-points may be difficult to determine, as may be the role of other agents and stressors on the body. This will lead to risk assessment methodology for immuno-suppressants being different from assessments for irritants for instance.

All human health risk assessments of chemicals include hazard identification, dose-response assessment, exposure assessment and risk estimation/characterisation. If the assessment is site-specific, then a release assessment would be required in the absence of good data on environmental levels or to account for non-routine, accidental releases.

Hazard identification

Hazard identification is defined as "the identification of the adverse effects which a substance has an inherent capacity to cause" (CEC, 1993). This involves consultation of any toxicological and epidemiological data.

The objective of toxicological testing is to identify those substances that could injure humans exposed to them and therefore to reduce injury (Paustenbach, 1989). A fundamental principle in toxicology is that a relation exists between the dose of an agent received and the response produced in the mammalian system (the receptor). The magnitude of the response is a function of the concentration of the agent and the site of action. The principles of toxicity testing are relatively straightforward. Experiments compare observations on two groups of animals. Both are held under the same general conditions, except one group is exposed to the substance under test. The quantity of animals used for such tests is determined by the number of test results needed to give a statistically significant result. Tests are classified according to duration of dose: acute (1-14 days), sub-chronic (1-6 months), chronic (6-24 months), and lifetime

(18-30 months) in rats. Administration routes include oral, dermal, ocular and inhalation. Relevant observations are made such as examination of tissues or body fluids or behaviour according to the end-point being considered such as carcinogenicity, neurotoxicity, sensitisation or irritation.

Epidemiological data are preferential to those from laboratory animals as they are human based. The epidemiological evidence useful in risk assessment is likely to be "environmental" or "risk-factor" epidemiology which try to identify the causes of non-infectious disease. There are huge difficulties in such epidemiology because it is based mainly on observational approaches. In these, biases in the design of the study and confounding factors become hugely important. They represent anything that may cause a study to come up with the wrong answer, to indicate the existence of a causal relationship that does not exist or vice versa. Confounding factors are the hidden variables in the population being studied which can easily generate an association that is real, but not what the epidemiologist thinks it is. For instance, cigarette smoking can confound any study looking at the effects of alcohol on cancer. People who drink tend also to smoke, increasing their cancer risks. Any apparent alcohol-cancer link may be spurious (Taubes, 1995).

Hazard identification can be described as the identification of those substances deemed to be hazardous to health in some concentration or dose, based on their chemical, physical and toxicological properties and environmental fate mechanisms (Paustenbach, 1989). This definition includes the incorporation of information concerning the environmental fate of the substance in the identification of hazards. For instance, if the half-life of a substance in the atmosphere is in terms of seconds, and the toxicological effect takes hours to occur, then it may be possible that the substance would not be identified as a hazard in air.

Dose-response assessment

Dose response assessment is the "estimation of the relationship between dose, or level of exposure to a substance, and the incidence and severity of an effect" (CEC, 1993). In a small number of cases it may be possible to produce a dose-response relationship from epidemiological data. However, for the majority of substances being assessed, particularly carcinogens, an absence of human data prevails. It is then necessary to ascertain dose-response information by evaluating tests performed in laboratory animals. Extrapolation from high to low doses and from laboratory animals to receptor (human or animal) is usually required. Description and justification of the methods of extrapolation used to predict incidence and the characterisation of the statistical and biological uncertainties in the methods used, are a component of dose-response assessment.

Exposure assessment

Exposure assessment is described as the "determination of the emissions, pathways and rates of movement of a substance and its transformation and degradation in order to estimate the concentration/doses to which human populations or environmental compartments are or may be exposed" (CEC, 1993).

Substances can enter the human body via three routes of entry, i.e., the lungs, digestive tract and the skin. In estimating the amount of a substance absorbed through the three routes, certain parameters have to be measured or estimated including the area of exposed skin (for entry via the skin), contaminant concentration

in air (for entry via the lungs), and amount of contaminated medium ingested per day (for entry via the intestinal tract) (Paustenbach, 1989).

Environmental exposure to chemicals can be direct - as a result of emission to the environment (air, land, water) of a substance through industrial manufacture, use or disposal, or indirect - through drinking water or the food chain.

The possible routes of exposure to chemicals and the main factors to be considered are summarised in Figure 5.1 and Figure 5.2.

It is sometimes possible to estimate exposures from direct measurement of the amount of chemical in the environment (environmental monitoring) or the chemical or metabolites in the human body (biological monitoring). Predictive modelling techniques using "fate and transport" and human exposure models may be necessary when this is not available.

Predicted Environmental Concentrations (PECs) for air, land and water can be determined by modelling, taking into account the properties of the substance and possible release scenarios. Exposure estimates for indirect routes can be made by combining the PEC information with consumption data for food and water for the exposed groups. A lot of work has been carried out to attempt to improve exposure assessments. Better characterisation of individual exposure to pollutants or contaminants within a population takes into account not only the mean exposures, but also the variability around mean exposures. It is important to consider the peak concentrations as well as regular/routine concentrations in predictive models in order to protect susceptible groups.

Environmental fate and transport models are important tools in the exposure assessment

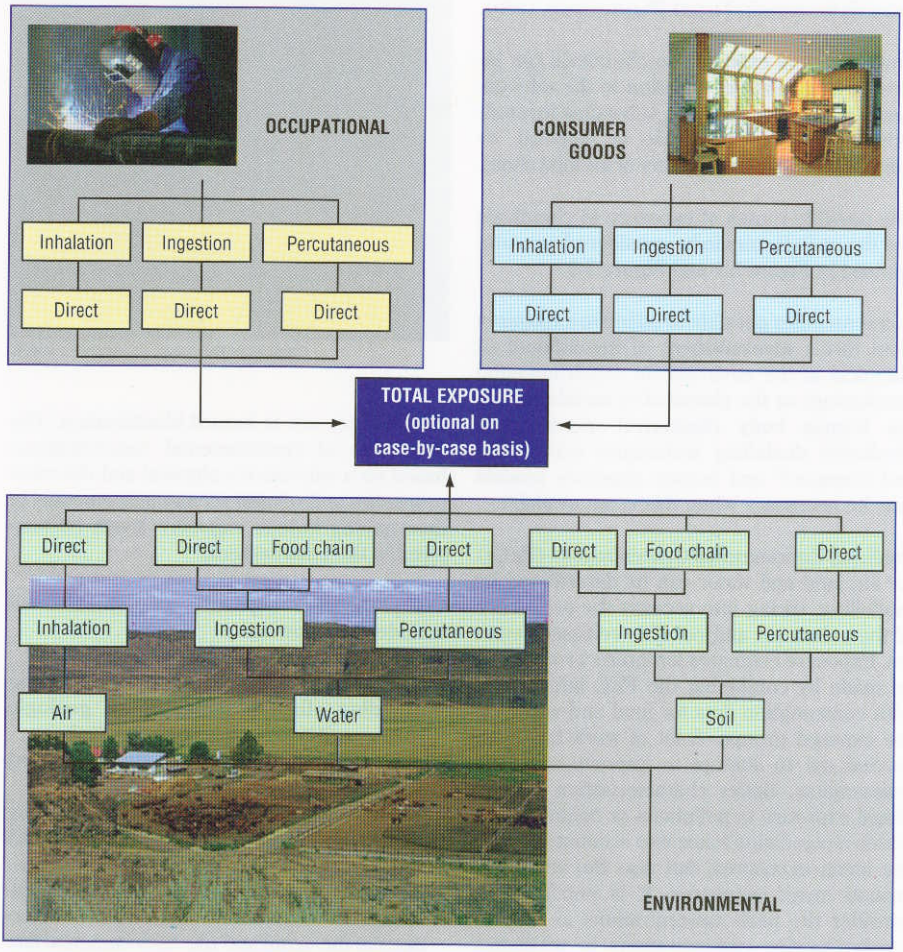
Photo: Chris Westwood, Environmental Images

step, as they are in hazard identification. The prediction of environmental concentrations based on a substance's physical and chemical properties and release scenarios is essential in determining human exposure levels encountered in the environment.

Comprehensive exposure assessments will include the following:

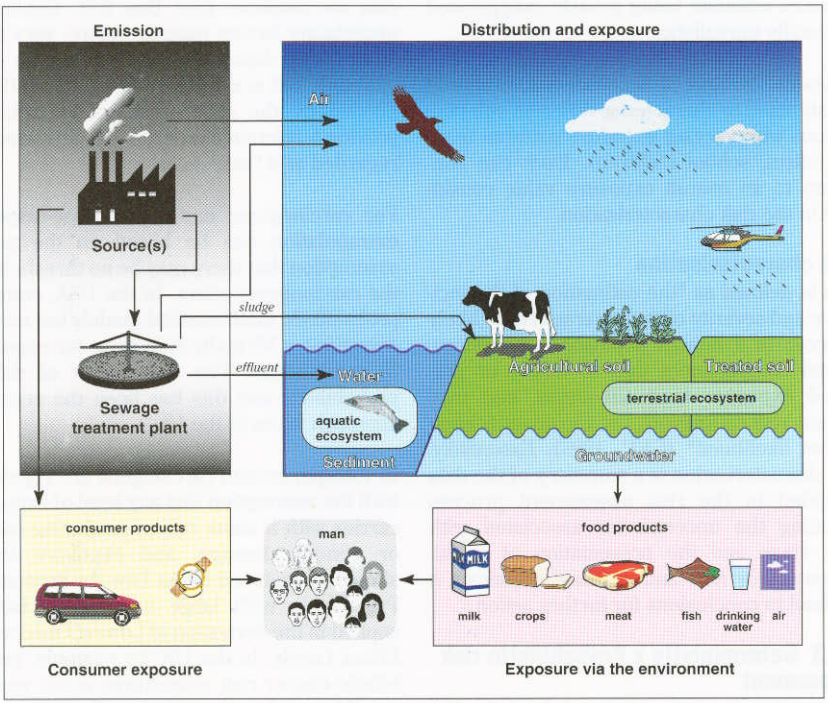
- Characterisation of the physical setting including climate, meteorology, geologic setting, soil type, groundwater hydrology;
- Characterisation of the potentially exposed populations;
- Identification of the exposure pathways by identifying the sources and receiving media and evaluating the fate and transport in release media. This will include an assessment of the physical and chemical characteristics of the agent and the environmental fate parameters and a consideration of factors, such as degradation in the environment, inter-media transfer, possible reactions with other environmental chemicals, etc.

Figure 5.1: Estimation of human exposure – major routes



Source: UK Government and Industry Working Group, 1993

Figure 5.2: A general overview of exposure assessment



Source: van Leeuwen and Hermens, 1995

- Integration of the sources, releases, fate and transport, exposure points and exposure routes into exposure pathways.

It is important to recognise the uncertainties that exist in the assessment of exposure levels. Where possible, exposure levels should be derived on the basis of both measured data and model calculations. The EU TGD suggests that, as a general rule, the "best and most realistic information available should be given preference". Where no monitoring data exist, predictive levels should describe a reasonable worst case situation, covering normal use patterns, including multiple exposures from different sources. This approach can serve as a useful screening tool; for example, if the outcome of the risk characterisation, using worst case default values, is that the substance is not of concern, the assessment for the specified population/effect can be stopped. If the outcome is that the substance is of concern, the exposure assessment needs to be refined and more realistic values used. Over-reliance on

worst case default values can lead to the exposure estimate being grossly exaggerated and totally unrealistic.

If possible, a range of exposure values should be estimated to characterise different sub-populations with varying exposure patterns (e.g., the infirm, school children). Each can contribute to an overall exposure value considered in the risk characterisation.

Risk characterisation

This is defined as "the estimation of the incidence and severity of the adverse effects likely to occur in a human population or environmental compartment due to actual or predicted exposure to a substance, and may include 'risk estimation', i.e., the quantification of that likelihood" (CEC, 1993). Essentially, risk characterisation is a summary of the data compiled in the risk assessment process including the uncertainties associated with each stage and the presentation of a risk estimate. For new and existing chemicals it will lead to conclusions for further action.

5.2.3 Deterministic v Probabilistic risk assessment

For most non-carcinogenic end-points, the effect is deterministic. It is accepted that a threshold exists below which no toxic effects are expected. It is possible to determine, through toxicity tests, a No Observable Effect Level (NOEL) - the highest dose which has no effect or if this is unavailable, a Lowest Observed Effect Level (LOEL) - the lowest dose to produce a toxic effect. These figures then need to be extrapolated from animals to humans which involves the application of safety or uncertainty factors. These factors are also applied to account for variations in sensitivity among humans, for extrapolation from a LOEL to a NOEL and for estimating a safe chronic dose when only acute or sub-chronic data are available (See Box 5.5). Cumulative uncertainty factors may, therefore, vary from 10 to 10,000 depending on the available data, although 100 is a typical value. The NOEL is divided by the appropriate safety factor to provide a reference dose for humans which is regarded as a "safe" dose.

For carcinogenic end-points, dose-response extrapolation can be based on the policy assumption that there may be no threshold for the carcinogenic effect. In the USA, complex probabilistic mathematical models are used to determine a Virtually Safe Dose for exposure to carcinogens. The assessment of risk is probabilistic and this has been the common approach taken in the US.

In Europe, certain carcinogens are regulated with the assumption that any level of exposure carries with it some risk of producing cancer or genetic damage, and exposure levels should be reduced to As Low As Reasonably Practicable with large uncertainty factors applied in the derivation of Lowest Observable Effect Levels. In the UK for example, probabilistic cancer risk assessment is not carried out (Maynard et al., 1995; Lovell, 1996).

Box 5.5 Chloroform - Implications of reliance on low dose extrapolations

Chloroform causes cancer in mice if given in large doses directly into their stomachs. Using these toxicological data, the US EPA concluded that drinking water should contain less than 0.004 ppm of chloroform (a by-product of water chlorination). A question must be raised as to the relevance of these high dose animal studies to humans. Later studies showed that no cancer or liver toxicity was evident in mice exposed to chloroform at concentrations of 1,800 ppm in water. The US EPA is now moving away from the traditional reliance on high dose acute studies and is attempting to incorporate biological models in risk assessment.

Box 5.6 Lead - Neurotoxic effects on children at low doses. An example of the difficulties in determining the significance of effects

The health impacts of lead are well known and include anaemia, encephalopathy, possible reproductive effects, and a range of effects on cognitive function. Attention has been focused in recent years at the effects on cognitive CNS deficit in children. These are the subtle effects on children's intellectual development and behaviour. The research area is fraught with difficulties as is all environmental epidemiology. Confounding factors such as social class, diet and other environmental exposures have confused the studies as have in-built biases in studies, but it is generally accepted that an association exists between lead body burden and IQ in children.

A major difficulty in risk assessment is, what do these very small reductions in IQ mean and how significant are they? The 1992 WHO/IPCS Task Group on Effects of Inorganic Lead concluded that blood levels in young children generally below 25 ug/dl are associated with a reduction in IQ scores (WHO/IPCS, 1995). The size of the apparent IQ effect as assessed at 4 years of age and above is between 1 and 5 IQ points for each 10 ug/dl increment in blood lead. This conclusion is based on a large number of retrospective studies prospective epidemiological studies and is similar to the findings of many international groups (US Centre for Disease Control, 1991; Health Canada, 1994; Edwards-Bert et al., 1992). There is no evidence to date of a threshold for these effects on IQ. The risk assessor has to decide what end-point they wish to use. Are they going to consider a 1 point loss of IQ as significant? Ultimately this is a problem for risk evaluators and managers.

One difficulty arising in toxicity testing is the determination of the possible synergistic and antagonistic effects created by mixtures of substances in the environment. Toxicity equivalence factors are being developed for chemicals in some complex mixtures. The individual chemicals are usually assumed to have additive toxicities, based on their relative potencies.

5.2.4 Neurotoxic risk assessment

Neurotoxic effects can be either acute (related to disruption of neural homoeostasis) or chronic (caused by nerve damage) (Fan et al., 1995). Both types of effects can be mediated either centrally (brain or spinal chord) or peripherally (in the autonomic or voluntary nerves). Although acute doses of neurotoxic chemicals can be important environmentally - methyl mercury poisoning in Japan is an example of this - levels of contaminants in the environment are usually very low. This highlights the problems of the application of toxicity assessment to environmental risk assessment.

Neurotoxicity tests used to determine hazard identification and dose-response which are commonly carried out include observational (rating of spontaneous motor activity, rearing, social contact); behavioural (disruption of trained activities); neurophysiological (measurement of nerve conduction velocity, etc.); physiological (muscle strength, reflexes, etc.) and biochemical (changes in levels of hormones or other biological markers). However, much controversy surrounds the interpretation of the results of such tests as it is difficult to determine what is a significant adverse effect. For example, when does a change in behaviour pattern become significant? The calculation of uncertainty factors is even more difficult. See Box 5.6 and Box 5.7

At this stage, more development work needs to be carried out to produce guidelines for neurotoxicity testing and the use of the data in environmental risk assessments.

Box 5.7 Pesticides - How to derive a no effect level

High doses of acetylechlorinesterase (AchE) inhibitors, such as organophosphate or carbamate pesticides, can be fatal or impair neurological function but low chronic doses have been shown to cause reversible AchE inhibition, detectable only in blood samples, with no other symptoms. The question is where should the NOEL line be drawn to allow incorporation into risk assessment?

A list of examples of neurotoxic agents is presented in Table 5.3.

Table 5.3: Sampler of Neurotoxic Agents[a]

Physical Agents
- Anoxia

Pesticides
- Organochlorines
  - DDT
  - Dieldrin and aldrin
  - Chlordecone (Kepone)
- Pyrethroids[b]
  - Fenvalerate
  - Pyrethrum
- Carbamates[c]
  - Carbaryl
  - Aldicarb
- Organophosphates
  - Reversible only
    - Parathion
    - Malathion
  - Irreversible and reversible
    - Leptophos
    - EPN
    - Chlorpyrifos

Nerve Gases
- Organophosphorus esters

Solvents and Industrial Intermediates
- Acrylamide
- Hexane
- Methanol
- Ethanol
- Tri-ortho-cresyl phosphate (TOCP)

Pharmaceuticals
- Glutamate
- Hexachlorophene
- Isoniazid
- Malonitrile
- Pyrithiamine
- Vinca alkaloids
- Nicotine
- Lathyrogens

"Life-Style" Chemicals
- Opioids
- Solvents
- Cocaine
- Marijuana
- Alcohol

Metals
- Lead
- Thallium
- Mercury
- Manganese

Organometals
- Gold thioglucose
- Triethyltin
- Methylmercury
- Tetraethyllead

Miscellaneous
- Carbon monoxide
- Acetylpyridine
- Azide
- Carbon disulphide
- Cyanide

[a] Although far from exhaustive, this list gives some indication of the variety of substances that can damage the nervous system.
[b] Although pyrethroids are insecticidal by virtue of their neurotoxicity, they rarely cause neurological effects in mammals because they rarely reach the mammalian nervous system. Only reversible neurotoxic effects have been observed.
[c] Neurotoxic effects usually due to inhibition of acetylcholinesterase, and therefore reversible. Carbaryl may cause ataxia, but that syndrome is poorly characterised and seems to be reversible as well.

Source: Francis, 1994

5.2.5 Immunotoxic risk assessment

The risk assessment of immunotoxic chemicals is a relatively new area. Concern over the possible effects of certain chemicals on the immune system has grown due to the general awareness that chemical and biological agents can alter immune responses as seen in acquired immune deficiency syndrome (AIDS); the fact that pesticides are known to modulate immune system response; distinct vulnerability of sectors of the population, e.g., the elderly to immuno-suppression; and the possible development of hypersensitivity.

The immune system is a complex network of lymphoid organs and cells in circulating blood and interstitial tissue spaces that interact to generate the immune response. Because of the complexity of this system, several tests are required to assess the effects of a chemical on different components of the system. In the context of hazard identification, dose-response assessment and exposure assessment, few tests have been validated.

Dose-response relationships for immunological responses differ to those of other non-cancer end-points. This is because high doses may induce tolerance, whereas low doses may encourage sensitisation. Few epidemiological examples of chemically induced immunosuppression are available and in those few examples, the biological significance is unclear. Although a significant change in any immune function can be considered deleterious, a change in function does not necessarily mean that a disease will result. Suppression of an immune parameter may not damage health as immuno-compromised people will function normally in the absence of infectious agents (WHO/IPCS, 1996b). This evidence suggests that a case exists for the existence of a threshold dose for immuno-toxicants as individuals may

have sufficient redundancy/reserve in their immune system to counteract varying levels of imposed immuno-suppressive agents.

To date, very little information is available on the immunotoxicity of chemicals. Work is currently under way in the US to provide test guidelines and data requirements for immunotoxicity testing (US EPA, 1991). The US National Toxicological Programme has developed a two-tier approach to assess the immunotoxicity of chemicals. Tier one consists of relatively simple screening tests on end-points such as immunopathology. If these tests produce a positive result, more comprehensive tests are carried out in tier two (Luster et al., 1988). Efforts are particularly being concentrated on the inclusion of immunotoxicity assessment in the risk assessment of pesticides. Sensitisation is a toxic effect included as a component of the risk assessment principles laid down in Commission Directive 93/67/EEC, but general immunotoxic effects are not specified. Work has recently been completed by the International Programme on Chemical Safety on the Principles and Methods for Assessing Direct Immunotoxicity Associated with Exposure to Chemicals (WHO/IPCS,1996b).

According to the Royal Society (1992), the strategy that is evolving is to enhance the observations made in sub-acute and chronic toxicity studies (for example, by histopathology of organs in the immune system) in order to identify any adverse effects on the immune system. If adverse effects are seen in these standard studies, then specific observations on components of the immune system may help to provide an understanding of the mechanism of production of them. In sensitisation testing, the greater understanding of cellular mechanisms of contact allergy has allowed the development of improved, more quantitative methods of identifying both contact and respiratory allergens.

A list of environmental chemicals with the capacity to cause immunotoxic effects is presented in Table 5.4.

5.2.6 Developmental toxicity risk assessment

Developmental toxicity can be defined as toxicity that adversely effects offspring through maternal exposure to toxic agents prior to conception, through exposure *in utero,* or through exposure during the period from birth to sexual maturity. Adverse developmental effects include structural abnormalities, growth alteration, functional defects, and death (Fan et al., 1995). Hazard identification and dose-response tests are typically carried out on female test animals such as rodents and rabbits. Doses are administered at various stages of pregnancy to determine at what stage of development the foetus is most vulnerable. It is generally accepted that a threshold exists for developmental toxicants. Particularly dangerous developmental toxins are those which have a threshold for maternal toxicity well above that for foetal toxicity.

Box 5.8 Risk assessment for allergenic substances

Work has recently been carried out by the Swedish Environmental Protection Agency as part of the 'Risk Assessment-Health-Environment' Programme on the causes of the increasing incidence of asthma in industrialised countries (Swedish EPA, 1996). Many risk factors have been identified and the problem is complicated by many individuals being more prone to sensitisation due to their genetic make-up.

The programme produced a comparative ranking system for risk factors for allergies/asthma in specific exposure scenarios. The relative risk factors show that the combination of fur-bearing animals and damp/passive smoking gives the greatest risk of allergic childhood asthma.

Major difficulties exist in the extrapolation of test results for developmental toxicity from animals to humans. It is impossible to extrapolate between species with respect to the occurrence of structural malformations and it is extremely difficult to model effects in an animal which are of concern in a child such as mental retardation and motor and sensory deficits.

The classic epidemiological example of exposure to a developmental toxicant is the thalidomide epidemic. This case graphically illustrates the problems associated with cross-species extrapolation as no structural malformations were evident in rodent studies of thalidomide.

A list of known developmental toxicants is provided in Table 5.5.

Table 5.4: Examples of compounds that are immunotoxic for humans or rodents

Chemical	Immune toxicity	
	Rodent	Human
2,3,7,8-tetrachlorodibenzo-p-dioxin	+	+
Polychlorinated biphenyls	+	+
Polybrominated biphenyls	+	+
Hexachlorobenzene	+	unknown
Lead	+	unknown
Cadmium	+	unknown
Methyl mercury compounds	+	unknown
7,12-dimethylbenz[a]anthracene	+	unknown
Benzo[a]pyrene	+	unknown
Di-n-octyltindichloride	+	unknown
Di-n-butyltindichloride	+	unknown
Benzidine	+	+
Nitrogendioxide and Ozone	+	+
Benzene, toluene and xylene	+	+
Asbestos	+	+
Dimethylnitrosamine	+	unknown
Diethylstilboestrol	+	+
Vanadium	+	+

Source: WHO/IPCS, 1986

5.2.7 Reproductive toxicity risk assessment

Reproductive toxicity can be defined as toxicity that adversely affects any aspect of male or female reproductive function (Fan et al., 1995). The effects may be observed as changes in reproductive cells or organs, in endocrine functions or in behaviour. Endocrine disruptors are covered in more detail in 5.2.8. Hazard identification and dose-response tests used to determine the effects of potential reproductive toxicants include the multi-generation test and the one-generation three-segment assay. The multi-generation test is particularly applicable to long-term exposure scenarios, e.g., pesticides and food additives. The compound is administered typically to three generations of rats and the following end-points are assessed: fertility index; gestation index; sex ratio; weaning index; and growth index. The one-generation three-segment assay determines general fertility and reproductive performance, developmental toxicity and pre- and post-natal toxicity.

Once the hazard has been identified and a dose-response curve obtained, it is possible to model exposure scenarios and estimate human exposure levels in the environment. The risk can then be characterised. Because of the nature of the effects of developmental and reproductive toxicity, there is a tendency for risk assessors to apply stringent safety factors in the estimation of a "safe" dose. Factors of up to 1,000 may be considered necessary to protect the developing foetus. Some reproductive toxins are genotoxic and therefore are assumed to have no threshold below which can be considered a safe dose. Risk quantification techniques may then parallel those for carcinogenicity. A major difficulty exists in that no internationally accepted pro-

Table 5.5: Current status of chemicals suspected of causing human malformations

Known to cause human birth defects	Strongly suspected of causing human birth defects	Probably do not cause human birth defects
Rubella – 1930s	Quinine	LSD
Radiation – 1930s	Amphetamines	Sulphonamides
Toxoplasmosis – 1950s	Hypoglycemics	Adrenocortical steroids
Aminopterin – 1952	Insulin	Antihistamines (meclizine)
Androgens – 1959	Tranquilizers	Bendectin
Thalidomide – 1961	Cocaine[a]	
Methylmercury– 1960s	Aspirin	
Warfarin – 1960s	Marijuana[a]	
PCBs – 1968	Cadmium	
Smoking – 1970s	Dioxin (TCDD)	
Alcohol – 1973	Barbiturates[a]	
DES – 1974	Narcotics[a]	
Diphenylhydantoin (Dilantin) – 1970s		
Methadone – 1980s		
Valproic acid – 1981		
Acutane – 1983		
Vitamin A derivatives – Ongoing		
Lead – Ongoing		

[a] Almost all psychoactive drugs cause behavioural deficits in animals and are suspected of causing these in humans.

Source: Francis, 1994

tocols exist for toxicology and testing for toxic agents.

5.2.8 Risk assessment of endocrine disruptors

Endocrine disruptors became an environmental issue in the early 1990s. Recent evidence suggests that certain chemicals are able to behave like oestrogens upsetting the hormonal balance of the mother and causing irreversible structural and behavioural changes in the embryo. Although this is a new area of research, it has been suggested that the possible effects of endocrine disruptors go further, pollutants can disrupt any hormone system especially the male hormone testosterone, the metabolism-regulating hormone thyroxin, or hormones involved in regulating pregnancy such as progesterone and luteinising hormone.

Research has been undertaken by the Danish, UK and German Governments. Significant research is also being undertaken by the US Government. The conclusions of all the research have been similar. Despite the huge uncertainties and the difficulties that exist in linking environmental pollutants and effects, a strong case remains to be answered. See Box 5.9.

Specific testing protocols are required to detect the endocrine effects.

The US EPA has developed test protocols and the OECD is producing similar protocols. The new protocols will increase the cost of testing and increase the numbers of animals required. The US EPA ecotoxicological tests are run over three generations of laboratory animals and new parameters are measured such as the age of vaginal opening in females.

The mechanism by which endocrine disruptors operate is still largely unknown. There are

many possible mechanisms, the simplest of which is likely to be through binding onto specific receptors inside cells. Hormone receptors are situated on nuclear or other membranes and act as molecular switches controlling the transcription of genetic material and through this the metabolic activity of the cell. Endocrine disruptors can either switch hormone receptors on or off through binding to the operational site of the receptor. The relationship between dose and response for each mechanism is likely to be different and so a thorough understanding of the mechanism of action is necessary before the effects of endocrine disruptors can be predicted. Risk assessment needs to take into account the possible additive effects of compounds and influences during critical development windows.

5.2.9 Carcinogenic risk assessment

The risk assessment of carcinogenic chemicals differs from the assessment of non-carcinogenic chemicals as a policy assumption is made that carcinogens have no threshold dose. Exposure to any level of a carcinogen can result in adverse effects on humans. This is supported by evidence suggesting that irreversible self-replicating lesions may result from a mutation in a single somatic cell following the administration of a single dose (Munro and Krewski, 1981). Opponents to this view argue that it may be possible for the human body to undergo metabolic detoxification on exposure to low doses. For regulatory purposes, however, it may be necessary to protect all individuals, this includes those of whose effective threshold level may be zero for a particular carcinogen. Because of the uncertainty involved in the application of the threshold concept to carcinogens, complex mathematical models have been developed to model low dose response. These mathematical models may be used in

Box 5.9 Phthalates, plasticisters and oestrogenic effects

Phthalates, widely used as inks and adhesives and associated with plasticisers, produce oestrogenic effects in test animals. Phthalates are ubiquitous in the environment and are commonly reported in fresh waters and sediments as a result of industrial discharges. Forty per cent of phthalate compounds are said to have oestrogenic properties. Two compounds, butyl benzyl phthalate and dibutyl phthalate, have displayed oestrogenic effects in cell assays. They have long been recognised as reproductive toxicants in animals although the mechanisms are not fully understood. The effect on human beings is far less clear. The WHO Environmental Health Criteria document on diethylhexyl phthalate (WHO/IPCS, 1992) examined it's reproductive toxicity, embryotoxicity and teratogenicity on laboratory animals but the limited data for humans meant that the implications for human reproductive health could not be determined.

Some phthalates are used in food contact applications, in packaging for instance, which can lead to significant contamination of food. Research carried out by the UK Ministry of Agriculture, Fisheries and Food (MAFF) found levels of phthalate plasticisters in baby formula milk, which exceeded EC precautionary limits. MAFF calculated that a baby's average intake of all phthalates, if the products were used as the sole source of nutrition, would be 0.10-0.13 mg/kg body weight/day over the first six months of the baby's life. This would diminish as milk is no longer the sole source of the baby's nutrition. This is above the precautionary limit of 0.05 mg/kg/day. Tolerable daily intakes (TDI) have also been set by the EC Scientific Committee on Food and none of these were exceeded. Oestrogenic effects have not been taken into account in setting the TDIs due to the lack of information and uncertainty associated with the effects. This research caused a huge outcry in the UK media but little attention was paid to the fact that the estimates of the baby's intake were based on the total phthalates concentration. The limits are based on individual phthalates and so the two are not comparable. It is known that not all phthalates are oestrogenic and that the effects of different phthalates may not be additive. If only the phthalates with proven oestrogenic effects are considered, infants receive a dose 4-17 times lower than the dose said to cause minimal effects in rodents (MAFF, 1996).

combination with epidemiological data and animal bioassay data for risk estimation.

The use of such models is fraught with problems (see Box 5.10) and many EU states prefer to use uncertainty factors when dealing with carcinogens. Genotoxic chemicals are carcinogens, which exert mutagenic effects at a cellular level, affecting the base sequence in DNA. The critical factor is whether the mutation occurs in a germ cell or in a somatic cell. Only if it occurs in a germ cell will the mutation affect the human gene pool and have a chance of being part of the genetic material of the next generation. It is generally accepted that no threshold exists for genotoxic chemicals as only one mutation from a single exposure can cause a genetic defect.

Hazard identification and dose-response tests need to be chronic in nature because of the possible long latency period before cancer effects are evident. Typical tests involve four groups of animals (mice and rats), three are given a fraction of the estimated maximum tolerable dose, and one is a control group. Both males and females are tested. Completion of the tests and report writing can take up to three years. The results are then extrapolated to provide information for human toxicity. This process is fraught with uncertainties and may require the application of safety factors, which may vary considerably in size. Estimating the low dose-response involves relating the probability of a specific response at very low doses. Several cancer models have been developed. Stochastic models are based on the premise that a positive response is the result of one or more biological events. These include the one-hit models based on the concept that a response will occur after the target has been hit by a single biologically effective unit of dose, the multi-hit model which is a simple extension of the one-hit model, the multi-stage models based on the occurrence of a number of random biological events, and the logit and Weibul models.

From the information obtained from bioassays, modelling and epidemiology, it is possible to determine a quantitative estimate of risk expressed as the number of additional cases of cancer; the percentage increase in cancer incidence; the number of additional cancer deaths; the percentage increase in cancer mortality in a population or the loss of life expectancy in the population.

5.3 Biological risks

Biological risks can be separated into those risks associated with biological agents of concern to public health, such as pathogenic strains of bacteria, which are of particular concern as food-borne hazards, and the introduction of genetically engineered organisms into the environment or the food chain. The field of risk assessment of biological agents is relatively novel but, as with chemical risk assessment, it has become a major management tool. The World Health Organization has adopted risk assessment as the main way to scientifically justify food safety standards (FAO/WHO, 1995). However, significant problems exist when applying quantitative risk

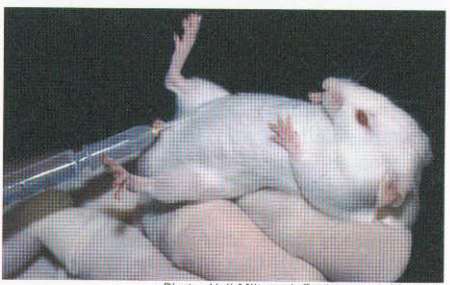


Photo: Ueli Miltpond, Environmental Images

assessment techniques to microbial hazards, such as the difficulties in obtaining dose-response data and elaborating appropriate dose-response relationships in humans (Christiansen, 1996).

5.3.1 Food safety risk assessment

Box 5.10 Controversial risk assessment - dioxins. Which cancer model to use?

Health Risk Assessment methodologies are the most developed and widely accepted. This does not mean that their application produces universally accepted risk assessments. The US EPA risk assessment of dioxins has proved hugely controversial and unlike many controversial assessments, the arguments centre on the intricacies of the scientific procedure. The US EPA risk assessment concluded that there was no threshold of action for dioxin.

A panel of outside scientists (the 39-member panel, organised by the US EPA's Science Advisory Board) attacked the assessment and accused it of "blurring science and policy". A member of the Panel stated "It is hard to determine which conclusions are based on data and which are policy-driven interpretations of data." The main issue the Panel took up was that nothing in the scientific data, amassed by the US EPA, supported the conclusion that dioxin had no threshold. The US EPA chose to use a cancer model - the single dose-response model, which leads to this conclusion, and ignored other mechanisms. This is an example of where policy decisions on choice of model, for instance, determines the result of the assessment. The US EPA now requires that biological mechanisms be taken into account in risk assessments.

The responsibility for setting standards, recommendations and guidelines on food safety on an international scale rests with the Codex Alimentarius Commission (CAC), set up by the Joint Food and Agricultural Organisation of the United Nations (FAO) and the World Health Organization (WHO) Food Standards Programme.

Biological agents (hazards) of concern to public health include pathogenic strains of bacteria, viruses, helminths, protazoa, algae, and certain toxic products that they may produce. Currently, the presence of pathogenic bacteria in foods presents the most significant problem internationally.

The CAC looks at biological and chemical risk in food and have described the basis of their risk assessment methods (CAC, 1993, FAO/WHO, 1995). It is derived from the NAS model and has four components involved in the assessment of biological agents which are:

1. **Hazard identification:** The identification of known or potential health effects associated with a particular agent. Bacterial agents known to cause food-borne diseases have been identified by using epidemiological and other data to link the organism and its source to illness. However, because data are far from comprehensive, a number of bacterial pathogens still need to be identified.

2. **Hazard characterisation:** The qualitative and/or quantitative evaluation of the nature of the adverse effects associated with biological agents which may be present in food. For many food-borne pathogenic bacteria, dose-response data are limited or non-existent. Information on which to base dose-response estimates is difficult to obtain and may be inaccurate because of a whole host of reasons. These include factors such as highly variable host susceptibility to pathogenic bacteria, and antagonism from other bacteria in foods or the digestive system which may influence pathogenicity.

3. **Exposure assessment:** The qualitative and/or quantitative evaluation of the degree of intake likely to occur. The exposure assessment provides an estimate of either the number of

pathogenic bacteria or the level of bacterial toxin in food. This presents a difficulty since populations of bacterial pathogens are dynamic and may increase or decrease dramatically in food due to factors such as processing, packaging and storage of food, and post-purchase preparation steps such as defrosting and cooking.

4. **Risk characterisation:** Integration of hazard identification, hazard characterisation and exposure assessment into an estimation of the adverse effects likely to occur in a given population, including attendant uncertainties. Characterisation of the risk posed by pathogenic bacteria is currently a qualitative process, combining the information described in the three previous steps of the risk assessment process. It has not yet been determined if a quantitative approach is possible and appropriate.

Important factors to include in a bacterial hazard risk assessment are those resulting from methods used to grow, process, store and prepare food for consumption. These can vary greatly depending on cultural, economic and geographical differences.

The qualitative approach depends heavily on experience with a specific food, a knowledge of ecology of bacterial pathogens, epidemiological data, and expert judgement regarding hazards associated with the manner in which the food is produced, processed, stored, and prepared for consumption.

The difficulties involved in the risk assessment of meat-borne microbial hazards are comprehensively described in the paper by Christiansen (1996).

An assessment of the risks associated with biological hazards producing toxins, resulting in effects ranging from short-term mild symptoms to severe intoxications and possible long-term effects, can be made in a quantitative manner, as for chemicals. This entails the derivation of an Acceptable Daily Intake (ADI) for agents with a threshold dose, estimated from dose-response data (the NOEL) with the application of a safety factor. The risks posed by biological hazards which produce pathological responses that result from viable organisms capable of infecting the host, however, may only be able to be assessed by qualitative risk assessment due to the availability of data and the fact that many uncertainties are associated with how and when an organism may express pathological potential.

Risk assessment is regarded by the CAC as an essential tool in its role of producing guidelines, standards and recommendations to protect human health from food-borne hazards. Current procedures and processes do not permit a comparison of relative risk or comparative risk between food safety hazards, e.g., chemical and biological. Codex recognises that risk assessment methodologies need to be developed to address this problem, as it may mean that alternatives are selected which increase the overall health risk associated with food. Such an example is the use of superchlorinated wash water to reduce pathogenic bacterial hazards which creates a chemical hazard from chloramines.

5.3.2 Risk assessment of genetically modified organisms

The procedures for carrying out health and environmental risk assessments for the deliberate release and contained use of genetically modified organisms are set out in two EC Directives (CEC, 1990a,b). This legislation provides a framework for EU States to build their own individual programmes. The

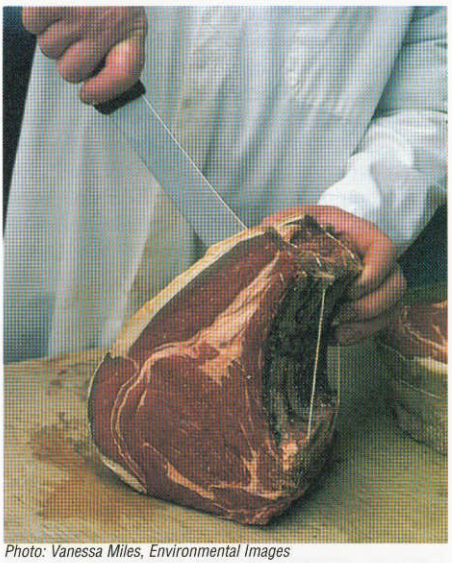


Photo: Vanessa Miles, Environmental Images

methodology developed from the Directives in the UK for health and environmental risks is given in Chapter 6 although emphasis is placed on environmental risks. It is acknowledged that many uncertainties prevail in the risk assessment of GMOs due to the open interpretation of the EC Directives and the primitive stage of development of the methodology.

5.3.3 Developing fields

Biological risk assessment is also being developed in the veterinary field to examine the animal health risks posed by the movement of animals across national borders and in site-specific problems such as pathogens from land-fill sites.Just as the CAC has a vital role in food safety risk assessment, the Office International des Epizooties is now charged by the World Trade Organisation with a key standard setting role. They have produced a number of documents on "risk analysis in the transmission of animals in international trade" and outline the "development of quantitative animal health risk analysis" (OIE, 1993, 1995).

The US EPA has done a considerable amount of work on the risks to humans of pathogens from sewage sludge (US EPA, 1986; Scarpino et al., 1989). They have also produced a computer model for such pathogen risk assessment (US EPA, 1989).

6. ECOLOGICAL RISK ASSESSMENT

Ecological Risk Assessment (EcoRA) involves the assessment of the risks posed by the presence of substances released to the environment by man, in theory, on all living organisms in the variety of ecosystems which make up the environment. EcoRAs tend to focus on the risks from chemicals and Genetically Modified Organisms (GMOs), some address physical risks such as temperature rises caused by cooling water releases from industry.

EcoRA methodology has been developed from that already established for human health. The general principles are widely agreed upon but the application of the process still provokes considerable argument. The Health Risk Assessment (HRA) approach lends itself well in many respects to EcoRA but, due to the complex nature of the potential target(s) or receptor(s), several problems have presented themselves to practitioners. HRA is concerned with individuals and morbidity and mortality, EcoRA is concerned with populations and communities and the effects of substances on mortality and fecundity. EcoRA has to deal with a multitude of organisms, all with varying sensitivities to chemicals and various groups have distinct exposure scenarios, such as free swimmers and sediment dwellers. Because of the difficulty in obtaining toxicity data on all organisms in an ecosystem, the recognised practice is to test selected representatives of major taxonomic groups and use these as surrogates for the whole system. This method is questionable as it may not protect the most sensitive species exposed in the environment. Failure to identify the effects of an agent on a potential receptor can result in widespread damage to organisms and ecosystems. A typical example is the use of anti-fouling paints containing tributyltin and the resulting damaging effect on oysters and dog whelks.

This chapter outlines the EcoRA methodologies developed in the EU for chemical substances, plant protection products and genetically modified organisms. Site-specific examples of EcoRA such as contaminated land and waste disposal sites are provided in Chapter 7.

6.1 The risk assessment process for chemicals

The EU ecological risk assessment methodology is outlined in Directive 93/67/EEC (OJ No. L227) which lays down the principles for assessment of risks to man and the environment of substances notified in accordance with 67/548/EEC (new substances) (OJ No. 196). Article 5 of the Directive outlines the procedures to be followed to assess the risks posed by substances to the environment and a Technical Guidance Document (TGD), produced by the European Chemicals Bureau, provides the detailed step by step procedure for both new and existing substances (CEC/ECB, 1996a). The term ecological risk assessment used in this book is equivalent to the term environmental risk assessment in the TGD.

The EcoRA process for new and existing substances follows the same four steps used in

Photo: Leslie Garland, Environmental Images

Box 6.1 Tributyltin, oysters and dog whelks

The molluscicidal properties of tributyltin compounds were first discovered in the 1950s and 1960s. They were initially used in an attempt to destroy fresh water snail species, in Africa, that are vectors of the disease schistosomiasis. This lead to their use in anti-fouling paints for boats. Bio-fouling of boats and ships can have a very significant effect on the performance of a vessel, reducing speed by up to 10 knots, giving an increase in fuel consumption of up to 20 per cent. The slow release of tributyltin compounds, particularly TBTO, into the water surrounding the hull of the vessel deters molluscs from attaching themselves to the hull, due to the toxic nature of the compound.

However, in the 1980s it was discovered that Pacific Oysters (Crassoterea gigas) in several locations were showing signs of growth abnormalities including shell thickening and many gastropods were suffering from imposex (the formation of male sex organs in females). Oysters and gastropods with these symptoms were invariably found to be living in the vicinity of harbours and yachting marinas and had a high tin content in their tissues. It was subsequently established that anti-fouling paints containing tributyltin compounds were responsible for the effects - not particularly surprising considering the toxicity of TBT compounds to molluscs! The discovery of these damaging environmental effects and the subsequent effects on the oyster industry, has resulted in the adoption of stringent restrictions on the use of TBT-based anti-fouling paints and some countries have banned it completely. The major problems are that TBT compounds are toxic to certain species at very low concentrations (0.01 µg/litre - bivalve larvae (WHO/IPCS, 1990a), they have a low solubility in water, they adsorb readily onto particles and concentrate in sediments, and are therefore readily re-introduced to the water column in harbours and estuaries due to the disturbance of the sediment by vessels.

HRA. They are: i) the identification of the hazard, followed by ii) the estimation of a Predicted No Effect Concentration (PNEC) (effects assessment), iii) the estimation of a Predicted Environmental Concentration (PEC) (exposure assessment) and finally iv) the characterisation of the risk.

An overall framework for ecological risk assessment is illustrated in Figures 6.1 and 6.2. The aquatic and terrestrial ecosystems and the atmosphere are considered in the risk assessment (the three primary compartments of the ecosystems). Micro-organisms in sewage treatment systems and secondary poisoning of top predators via the food chain are also examined. As most chemicals end up in the aquatic environment, EcoRA of these systems is much more advanced than for terrestrial systems. There is more information on the toxicity of chemicals to a wider variety of organisms and on the fate of chemicals in aquatic systems.

6.1.1 Hazard identification

Hazard identification and dose-response assessment are combined in a step called effects assessment in the TGD. It aims to identify the effects of concern, to review the classification of existing substances and to propose a classification for new substances.

6.1.2 Effects assessment

Effects assessment will identify the hazard based on its physico-chemical properties, ecotoxicity and intended use such as a pesticide, detergent or industrial chemical.

The estimation of the PNEC is primarily made on the basis of results from monospecies laboratory tests or, in some cases, from model ecosystem tests. The available ecotoxicity data are used to derive a No Observed Effect Concentration (NOEC), or a Lowest Observed Effect Concentration (LOEC). The test species used are selected to represent the sensitivities of different taxonomic groups in each environmental compartment. The vast majority of ecotoxicity data available are for aquatic species. These data, therefore, provide the base-set for the effects assessment. For aquatic effects assessment, ecotoxicity data

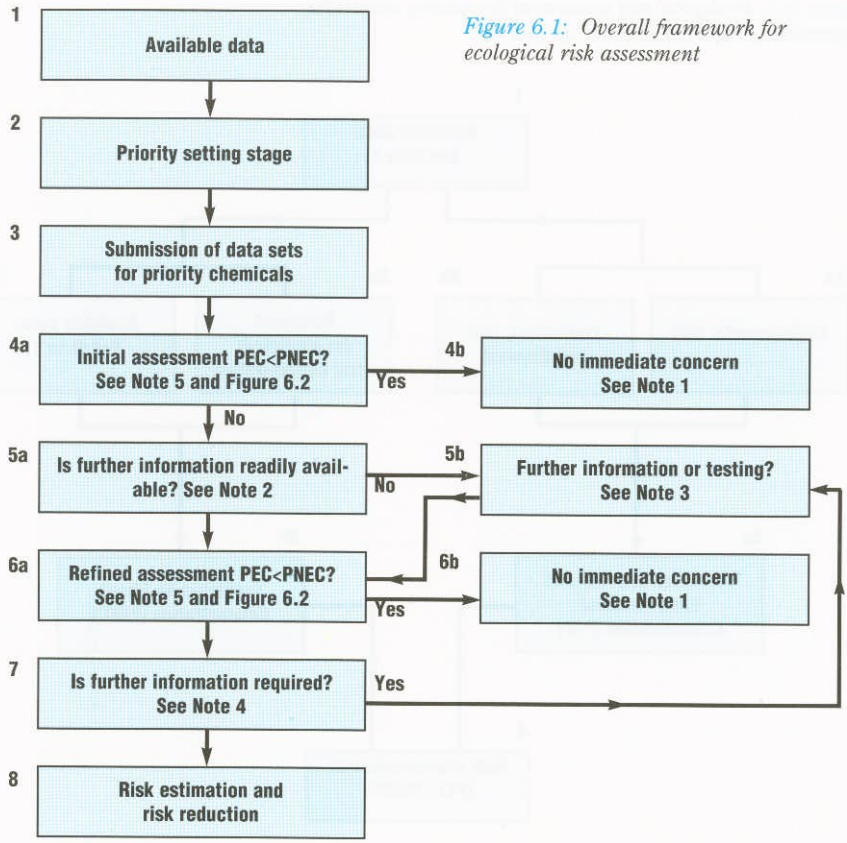
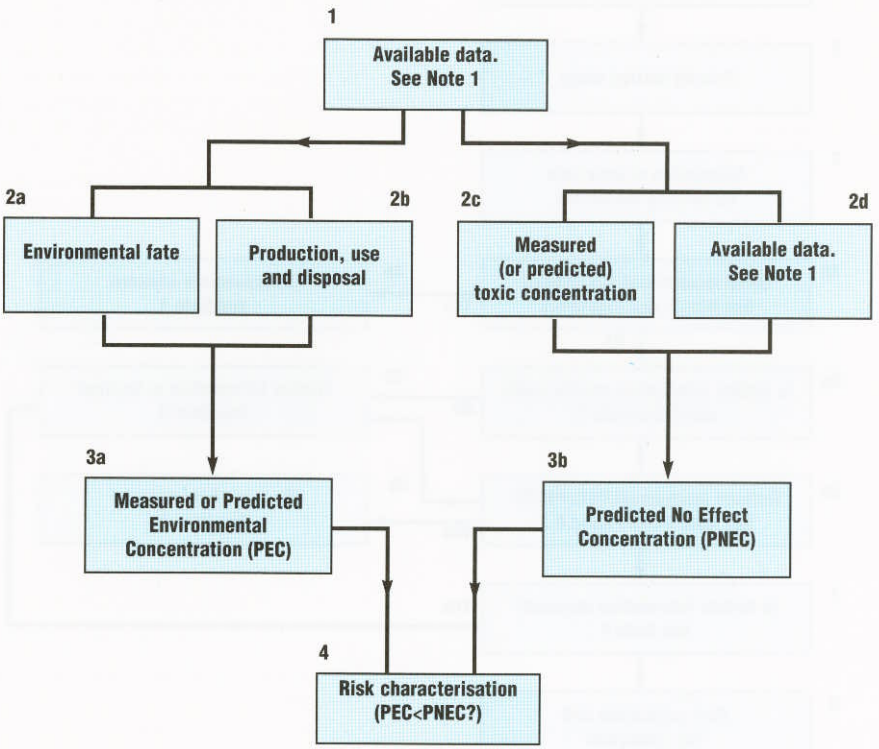


Figure 6.1: Overall framework for ecological risk assessment

1. Further information on substances within this box, e.g., increased production, change of use, regulatory action elsewhere, problems with related compounds, etc. should lead to re-introduction into the scheme at Box 5a.
2. Any information relevant to the assessment may be include here, including SAR or analogue data, site specific data, measured environmental levels, effects form field studies, exposure or modelling data, etc. If no further information is available proceed to Box 5b.
3. Further information on exposure and the pattern of exposure, and/or the need for further testing should be related to improving the assessment.
4. When Box 7 is reached the assessor may ask for further information on the substance or may decide to propose risk estimation and risk reduction steps. At this stage, the notifier should be given the opportunity to submit relevant additional information.
5. As well as PEC/PNEC comparison, which is concerned mainly with aquatic effects, direct toxic effects in other compartments and indirect effects (e.g., food chain effects and atmospheric effects) should also be considered using as appropriate the PEC/PNEC ratio in that compartment.

Source: UK Government and Industry Working Group, 1993

Figure 6.2: Ecological risk assessment for existing chemicals. Expansion of Figure 6.1, Boxes 4a and 6a



Notes:
1. Data as required by 7th Amendment to the Dangerous Substances Directive (92/32/EEC) or other measured/predicted data.
2a. Fate data should include environmental distribution, bioaccumulation, bioavailability, degradation pathways and/or final resting place, e.g., sediments or soils.
2b. Use pattern data: number of sites; dispersed or restricted use; wastage rates in use; measured concentrations; physical form; volume of material in each use, etc.
2c. Toxic concentration from laboratory test result, SAR (structure activity relationship) or from field data.
2d. The assessment factor depends on the amount and relevance of the available data (i.e., toxic effect of concern/ecosystem of concern). See Section 4 for details.
3a. Predicted concentration in various media, particularly water, soil, sediment and the food chain, supported with measured values where possible.
3b. This is the experimentally determined or predicted toxicity data divided by the appropriate assessment factor. See Section 4 for details.

Source: UK Government and Industry Working Group, 1993

are required on representatives of fish species, daphnia and algae. Considering the risk assessment is concerned with protecting ecosystems, this strategy assumes that ecosystem sensitivity depends on the most sensitive species and protecting ecosystem structure protects community function. Assessment (safety) factors are applied to the toxicity value to enable extrapolation from laboratory experiments to the field, acute to chronic effects and for inter and intra species variations. The size of the assessment factor varies according to the number and type of data available and the likely duration of exposure. The assessment factors to be applied to toxicity data, proposed by the TGD, for the aquatic compartment are provided in Table 6.1.

Table 6.1: Assessment factors to derive a PNEC

	Assessment factor
At least one short-term L(E)C50 from each of three trophic levels of the base-set (fish, Daphnia and algae)	1,000
One long-term NOEC (either fish or Daphnia)	100
Two long-term NOECs from species representing two trophic levels (fish and/or Daphnia and/or algae)	50
Long-term NOECs from at least three species (normally fish, Daphnia and algae) representing three trophic levels	10
Field data or model ecosystems	Reviewed on a case by case basis

Source: CEC, 1996

Ideally the PNEC should be derived from chronic toxicity tests relating to survival, growth and reproduction; however, data on chronic toxicity tests is scarce which necessitates the need to extrapolate acute data. The estimation of the PNEC varies according to the type of test, test organism, exposure regime and end-point measured. Initial acute orientated tests based on a few different species will tend to give higher PNECs with wide ranges of confidence limits but as more tests are carried out on a greater diversity of organisms, chronic exposure regimes and specific end-points, the PNEC will decrease and the confidence limits will narrow. A true No Effect Concentration can eventually, in theory, be obtained.

Because of the lack of data and test systems for effects assessment for sediment, the atmosphere, and terrestrial compartments, alternative methods have been adopted such as the equilibrium partitioning method to derive a PNEC. The various methods are described in detail in the TGD.

6.1.3 Exposure assessment

Exposure assessment and the estimation of a PEC are crucially important in the risk assessment process. Although a substance may be discovered to be toxic to an organism in the testing environment, concentrations may never reach such a level in the environment.

The PEC is calculated on both local and regional spatial scales from monitoring data where available, or by using realistic worst-case scenarios. If this information is not available, estimates are made from exposure models. For existing substances, monitoring data should be available. For "new" substances, predictive modelling techniques have to be used.

Exposure assessment is a complex task. It should, in principle, consider all the stages of the life cycle of the substance from production through process, transport and use, to disposal. Information is required on the release rates of all possible emission sources

(point, line, diffuse, continuous and intermittent) and the physico-chemical properties, including partition coefficients and biotic and abiotic degradation rates (and products) of the substance, in order to determine the environmental transportation and fate mechanisms operating and potential exposure pathways. Local relevant emission and distribution routes are shown in Figure 6.3 (CEC/ECB, 1996a).

The PEC is calculated for each environmental compartment using the information available on release quantities and subsequent degradation processes in the "standard" environment (default values are set for environmental characteristics such as density of air, etc.). Site-specific information is used when available and appropriate.

For the surface water compartment, the local PEC is obtained by calculating the local concentration in surface water and adding this figure to the regional concentration in surface water (considered a background concentration for the local scale). The local concentration in surface water is derived from a calculation involving the concentration of the chemical in the source effluent, the concentration of suspended matter in the river, the solids-water partitioning coefficient of suspended matter and the dilution factor. For the local scenario it is assumed that complete mixing of the effluent in the surface water is a representative exposure situation for the aquatic ecosystem. Because of the short distance between the point of discharge and the point of complete mixing (obviously varies from river to river) volatilisation, degradation and sedimentation can generally be ignored. However, they obviously play an important part in the more complex process of calculating a regional PEC. Details of all such calculations are provided in the TGD. Possible fate processes for a substance in surface water are depicted in Figure 6.4.

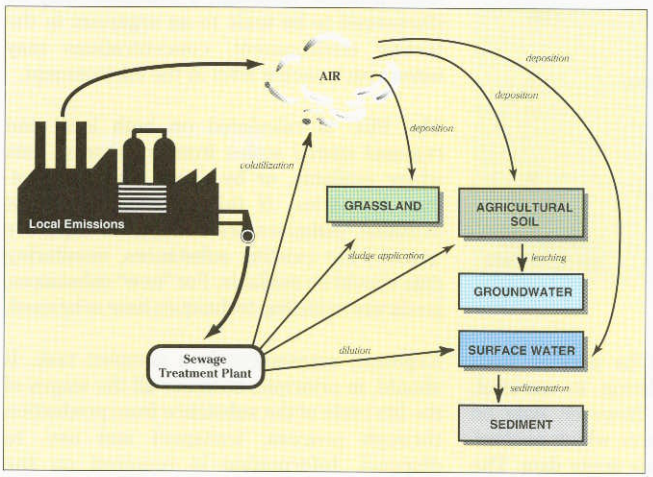


Figure 6.3: Local relevant emission and distribution routes

Source: CEC/ECB, 1996a

6.1.4 Risk characterisation

Risk Characterisation involves the calculation of a quotient - the PEC/PNEC ratio. If the PEC/PNEC is less than 1, the substance of concern is considered to present no risk to the environment and there is no need for further testing or risk reduction measures. If the ratio cannot be reduced to below 1 by the gathering of further information and further testing, risk reduction measures are necessary. The procedure is illustrated in Figure 6.5.

Other similar schemes have been developed:

1. OECD Provisional Guidance for Initial Hazard Assessment of High Production Volume Chemicals with full Screening Information Data Set; Initial Assessment of Aquatic Effects and Initial Assessment of Environmental Exposure.
2. US Ecological Risk Assessment Schemes under the Toxic Substances Control Act (TSCA).
3. Netherlands Uniform System for the Evaluation of Substances (USES) - General Chemicals and Pesticides.

6.1.5 Problems

Although the quotient method using simple toxicity tests provides valuable information for the assessment of ecological risk to single species or a limited range, comprehensive EcoRA of chemical substances requires examination of higher levels such as population and ecosystem. Several difficulties are evident, however.

The difficulties are a function of the essential differences between the receptor in human risk assessment (an individual with a well recognised and established life cycle) and the receptor in ecological risk assessment (millions of different species). EcoRA has the problem of incorporating a vast range of influential factors and criteria into the process such

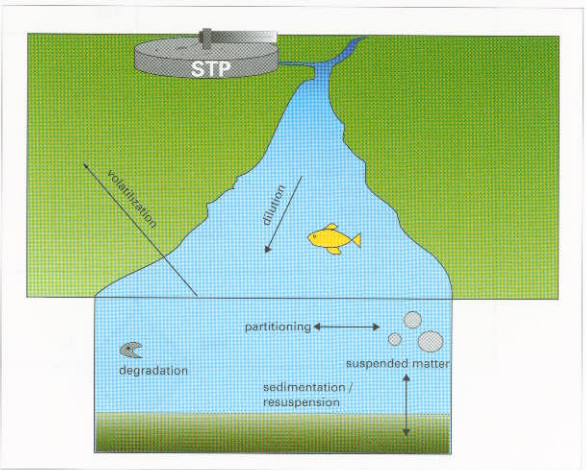
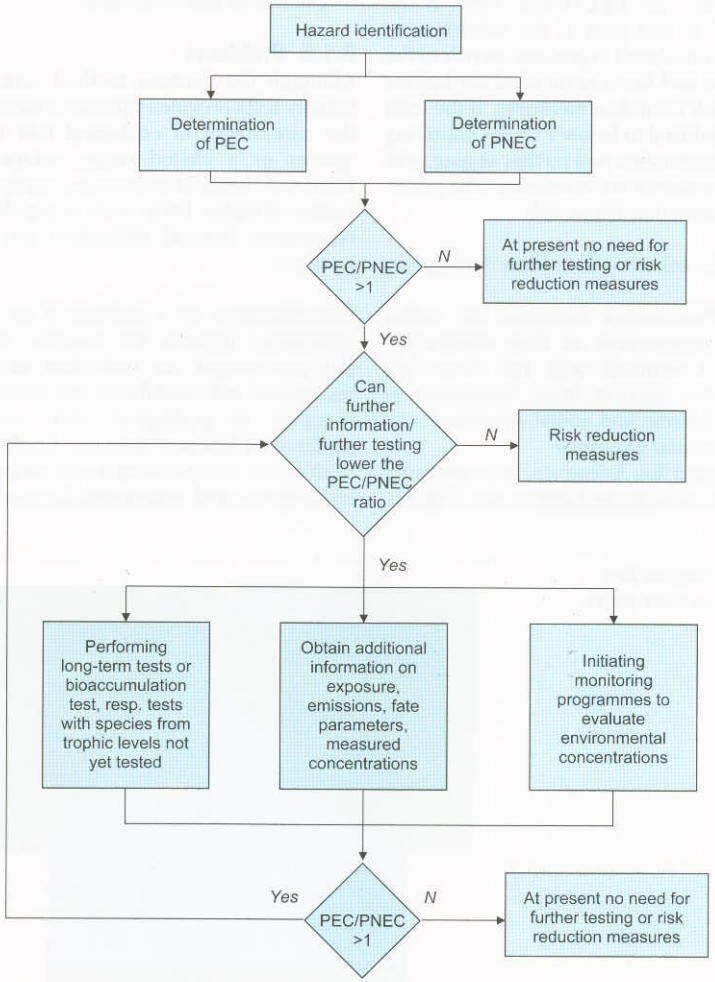


Figure 6.4: Possible fate processes in surface water

Source: CEC, 1996

Figure 6.5: General procedure for environmental risk assessment of new and existing substances



Source: CEC, 1996

as taxonomic diversity, toxicological end-points, spatial scales, temporal scales, and complexity of exposure.

The major difficulty encountered in EcoRA at the ecosystem level is the selection of significant end-points. To date, end-points at the lower levels of organisation have been used, such as at the organism or sub-organism level to provide an indication of ecosystem effect. At higher levels of organisation (population and ecosystem), the end-points are more complex and difficult to interpret but are more ecologically significant when addressing ecosystem effect.

The following high level end-points are suggested by Falco and Moraski (1989):

Ecosystem Structure End-Points: Abundance and biomass of communities/species richness.
Ecosystem Function End-points: Primary productivity/material and nutrient cycling.
Population-level End-points: Abundance, distribution, age structure, gene make-up of exposed populations.

These end-points can be measured and are sensitive to low levels of pollutant stress, but their measurement is considerably more time consuming and expensive than acute individual species end-point tests.

Other difficulties encountered in ecological risk assessment are:

- The selection of indicative species, typically sensitive or endangered species and physiologic end-points.
- The selection of ecosystem media and incorporating the interaction of pollutants within these media.
- The selection of field laboratory, mesocosm or microcosm tests.
- The selection of fate, transport and exposure models.
- The incorporation of resilience and recovery factors of the ecosystem.

A further problem is the lack of understanding regarding the mode of action of chemicals. Specific modes of action are only known for a few groups of compounds and very little is known about the relationship between mode of action and mortality, i.e., what do organisms die of in toxicity tests?

The problems associated with EcoRA are comprehensively covered in several texts including Suter (1993) and van Leeuwen and Hermans (1995), and development work is constantly being published in various journals.

6.1.6 Developing methodologies

EcoRA is a developing field. Organisations such as US EPA and US NRC are producing methodological approaches to ecological risk assessment developed from the NAS health risk assessment methodology. In Europe, organisations such as SETAC-Europe and ECETOC are involved in the development of methods and applications of EcoRA. EU legislation on chemicals has provided the impetus for such development programmes.

The US Committee on Risk Assessment and Management (CRAM), a sub-branch of the NRC, defined ecological risk assessment in 1989 as "the characterisation of the adverse ecological effects of environmental exposure to hazards imposed by human activities" (NRC, 1993). CRAM proposed the use of the human health risk assessment model developed by the NAS with modifications to address the influence of legal and regulatory considerations on the initial stages of ecological risk assessment.

Hazard identification was redefined by CRAM to be the "determination of whether a particular hazardous agent is associated with health or ecological effects of sufficient importance to warrant further scientific study or immediate management action". The NAS dose-response relationship was redefined as "the determination of the relationship between the magnitude of exposure and the probability of occurrence of the effect in question" and renamed Exposure Response Assessment. The responses include both direct effects of exposure and the much broader indirect effects, such as secondary poisoning of predators, and the effects of harvesting on fish community structure. The revised framework is summarised in Figure 6.6 (CRAM perspective) and has been criticised (Barnthouse, 1994).

The US EPA published a Framework for Ecological Risk Assessment, which is similar to CRAM. It describes Ecological Risk Assessment as a process for evaluating the likelihood that adverse ecological effects have occurred, are occurring, or will occur as a result of exposure to one or more stressors.

Implicit in this definition is that i) environmental stressors have the inherent ability to cause one or more adverse effects, and ii) the stressor co-occurs with or contacts an ecological component (i.e., organisms, populations, communities, or ecosystems) long enough and at a sufficient intensity to elicit the identified adverse effects.

The framework is composed of three phases (Figure 6.7) (Norton et al., 1992).

The first phase is defined as Problem Formulation, a planning and scoping process that links the regulatory or management goal to the risk assessment. The end product is a conceptual model that identifies the environmental values to be protected (the assessment end-points), the data needed and the analysis to be used.

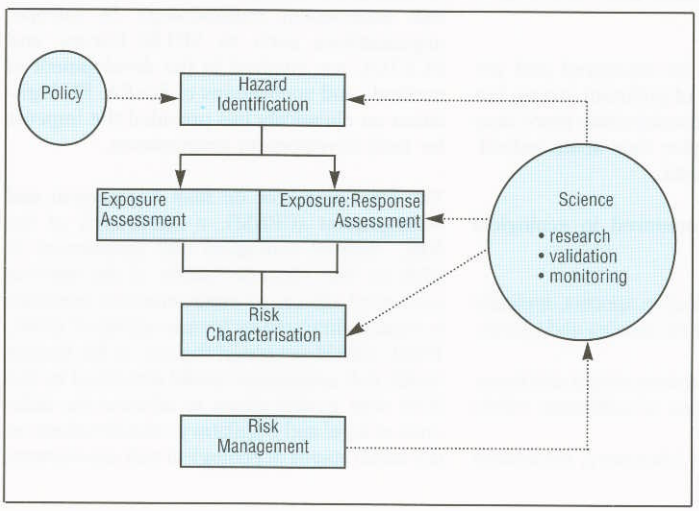
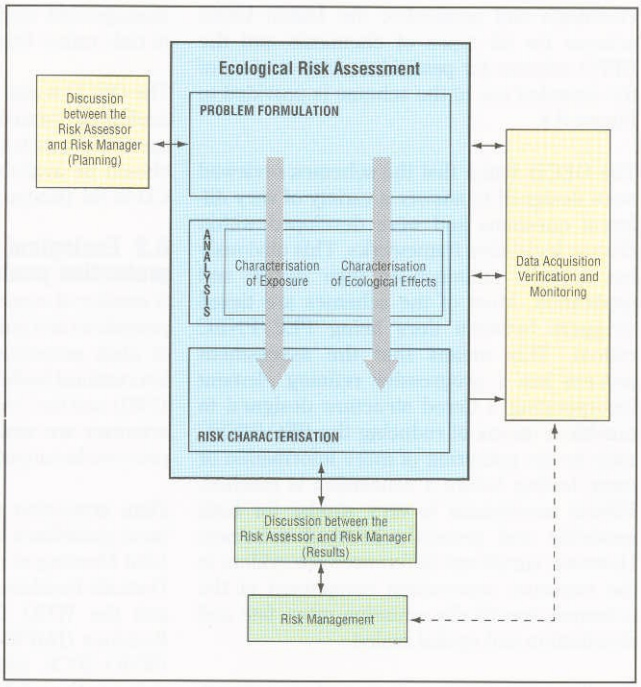


Figure 6.6: The CRAM integrated human health/ ecological risk assessment framework

Source: Barnthouse, 1994

Figure 6.7: Framework for ecological risk assessment



Source: Norton et al., 1992

The second phase is Analysis, which consists of the technical evaluation of the data on the potential effects and exposure of the stressor. This is essentially the characterisation of exposure and the characterisation of effects.

The third phase is Risk Characterisation which integrates exposure and ecological effects information to evaluate the likelihood of adverse ecological effects associated with exposure to a stressor. It will include a summary of any assumptions used, and uncertainties involved in the analysis.

Guidelines are currently being developed by the EPA, based on the Framework which also take into account some of the specific problems of ecological risk assessment outlined above.

Work has been carried out by the OECD on the comparison of 13 ecological hazard/risk assessment schemes developed and used by international organisations and regulators in several nations (OECD, 1995). The schemes analysed include the EU schemes for new and existing substances; the OECD SIDS scheme for prioritisation of high production volume chemicals; the US EPA scheme for general

chemicals and pesticides; the Dutch USES scheme for all types of chemicals and the EPPO scheme for pesticides. An overview of the intended use of the scheme is provided in Figure 6.8.

The OECD stated that the schemes reviewed were designed to answer a variety of very different questions and were developed within diverse legislative frameworks. This obviously has a heavy influence on their design and application. Most of the schemes are tiered (triggers between tiers being PEC/PNEC ratios). This means that the assessment process has a progressive refining element incorporating a tiered structure designed to provide a means of reducing the PEC/PNEC ratio by the gathering of more information or more testing before a conclusion is reached. Effects assessment is very similar for both pesticide and general chemical schemes. However, significant differences are evident in the exposure assessment component of the schemes, specifically emission rates, fate and distribution and spatial scales.

For the exposure assessment of new chemicals, predictive modelling is relied on as obviously no monitoring data exist. All the schemes recommended that priority be given to data from tests performed to Good Laboratory Practice, all extrapolate data from simple to complex situations, using worst-case assumptions to deal with uncertainty and provide scope for expert judgement to be applied. Differences exist in cut-offs, risk criteria, and application factors that reflect differences in the political, economic and ecological circumstances in the various nations in which the schemes were developed and also technical differences of opinion. The OECD suggests that local differences and considerations could be incorporated into the risk management decision based on the measure of risk, rather than in the measure of risk.

The OECD is also developing an inventory of all ecological hazard/risk assessment schemes operating in each member state. The information should be available as a database on disc or CD-ROM (Koepp, 1996).

6.2 Ecological risk assessment of plant protection products

A number of countries have developed their own procedures and guidelines for the risk assessment of plant protection products, as have certain international bodies, such as FAO (1989), OECD (1981) and the Council of Europe (1992). These schemes are similar to the risk assessment process developed for chemicals. See Box 6.2.

Plant protection product residue risk assessment guidelines have been developed by the Joint Meeting of the FAO Panel of Experts on Pesticide Residues in Food and the Environment and the WHO Expert Group on Pesticide Residues (JMPR), (CAC, 1993) and the IPCS (WHO/IPCS, 1990b). The guidelines aim to reduce the risks to human health from exposure to pesticide residues in food. The JMPR makes recommendations to the Codex Committee on Pesticide Residues (CCPR - a subsidiary body of the Codex Alimentarius Commission) concerning the Average Daily Intake and the Maximum Residue Limits for specific pesticides and pesticide/food combinations to ensure adequate food safety.

In order to provide a general, consistent and explicit decision-making approach suitable for adoption by all individual European regulatory authorities with regard to EC Directive 91/414/EEC, a joint panel of the European and Mediterranean Plant Protection Organisation (EPPO) and the Council of Europe has been set up.

Scheme/ Question	OECD-SIDS General chemicals	OECD-Aquatic General Chemicals	EC-New General Chemicals	EC-Existing General Chemicals	US-TSCA General Chemicals	Canada Existing General Chemicals	USES Pesticides and General Chemicals	ECETOC Pesticides and General Chemicals	Sweden Pesticides	US-FIFRA Pesticides	EPPO Pesticides
What type of chemical will be analysed in the scheme?	High Production Volume existing general chemicals	New and existing general chemicals	New general chemicals	Existing general chemicals	New and existing general chemicals	Existing general chemicals	New and existing general chemicals, pesticides and biocides	New and existing general chemicals, pesticides and biocides	Pesticides	Pesticides	Pesticides
To what regulatory decision will the result be applied?	Priority setting for further activity	Priority setting and setting of environmental quality objectives	Notification	Decisions on need for risk reduction	Pre-notification	To designate a toxic under CEPA and for setting priorities	Priority setting and registration (for use by EU Member States)	No details	Registration	Registration	Registration
To which legislation does the scheme apply (e.g. within the EU, the US, OECD-wide?	OECD Council Decision [C(90)163/Final)]	No details	Directive 93/32/EEC Directive 93/67/EEC	EC Regulation 793/93 Regulation 1488/94	US Toxic Substances Control Act (TSCA)	Priority Substances List of the Canadian Environment Protection Act CEPA	EC Regulation 793/93, Directives 67/548/EEC, 91/414/EEC, 93/67/EEC and two Ads from the Netherlands	7° Amendment of EU Directive 67/548/EEC in 1992	Swedish Ordinance (1985/836) on Pesticides	US Federal Insecticide, Fungicide and Rodenticide Act (FIFRA)	To influence implementation of EU 91/414/EEC
What is the scheme intended to assess (e.g. risk to individuals, etc.)?	Risk to individuals, populations and ecosystems, mainly ecosystems	Risk to individuals, populations and ecosystems, mainly ecosystems	Risk to communities and ecosystems	Risk to communities and ecosystems	Risk to individuals, populations and ecosystems	Risk to population, community and ecosystem	Risk to populations and ecosystems	To protect populations, communities and ecosystems	Unacceptability of chemicals	Risk to species and populations	Risk to individual, population, community and ecosystem
Intended for initial or comprehensive risk assessment?	Initial	Initial, refined and comprehensive	Comprehensive for all three compartments and secondary poisoning	Comprehensive for all three compartments and secondary poisoning	Initial, refined and comprehensive	Comprehensive	Initial and refined; NOT comprehensive	Initial, refined and comprehensive	Initial screening	Initial, refined and comprehensive	Initial, refined and comprehensive
Predictive or retrospective?	Predictive and retrospective	Predictive	Predictive	Predictive and retrospective	Predictive (mostly)	Retrospective and predictive	Predictive and retrospective	Predictive and retrospective	Predictive and retrospective	Predictive and retrospective	Predictive and retrospective

Figure 6.8: Overview of intended use of the schemes

Source: OECD, 1995

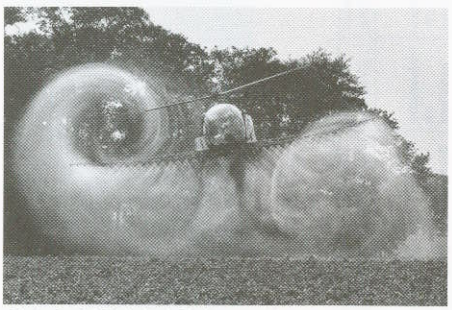


Photo: Patrick Sutherland, Environmental Images

The objective of the EPPO scheme is to provide a framework for the evaluation of risks, leading to the approval and setting of conditions for use (OEPP/EPPO, 1993). The scheme provides preliminary guidance to aid the identification of the aspects of environmental concern that require detailed assessment in any particular case, followed by the nine assessment sub-schemes:

1. Soil
2. Ground water
3. Surface water
4. Aquatic organisms
5. Soil macroflora
6. Earthworms
7. Arthropod natural enemies
8. Honeybees
9. Terrestrial vertebrates

The final section seeks to provide principles for the integration of the results obtained for each of the environmental aspects covered.

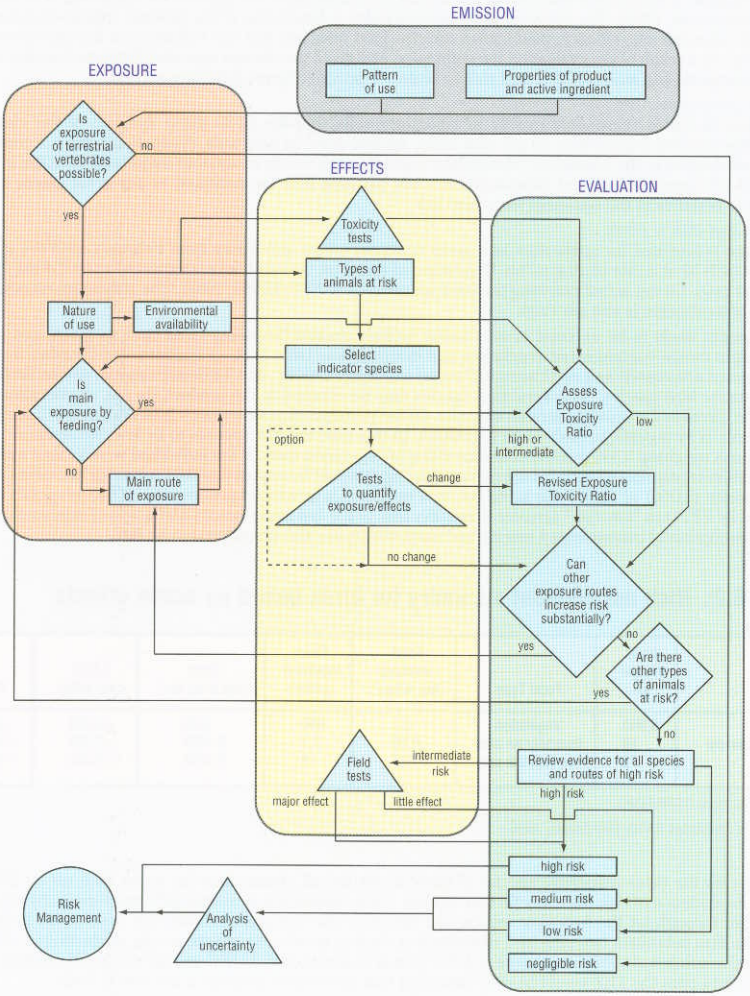
Each sub-scheme involves the identification of the potential adverse effects and an estimation of the likelihood of their occurrence by regarding factors such as the pattern of use of the product, the toxicity and environmental fate of the active ingredient, and species likely to be at risk. This format is based on the general approach developed by Greig-Smith (1991). The sub-schemes are divided into two parts. The first is a sequence of questions based on decision points with two alternative options, commencing with basic preliminary data through progressively more detailed aspects leading to the final categorisation. The second part consists of a set of explanatory notes, which provide further information about suitable test methods, issues connected with interpretation, and the need for advice from experts. A flow diagram for each sub-scheme illustrates the four-stage process of risk assessment, defined in this case by EPPO as emission (pattern of use, properties of product), exposure, effects and evaluation (characterisation).

The scheme ends by placing products into categories of "high", "medium", "low", or "negligible" environmental risks, pre-empting the risk management process. This risk classification is based on the quotient method, in this case dose/toxicity.

To illustrate the process, a flow diagram of the sub-scheme for terrestrial vertebrates is shown in Figure 6.9.

The overall scheme provides a comprehensive procedure for the risk assessment of plant protection products. However, one weakness exists in that it fails to address the effects on populations and the ecosystem as a holistic unit, although the conclusions drawn from risk assessments directed at protecting the individual can be used to indicate the proportion of animals in a population that are likely to be affected. Additional problems are encountered in multiple exposure scenarios where

Figure 6.9: Simplified diagram of the sub-scheme for evaluation of the risk of a plant protection product for terrestrial vertebrates



Source: OEPP/EPPO, 1993

Box 6.2 Example of ecological risk assessment of crop protection products

Metosulam - Herbicide

A full evaluation of Metosulam was carried out as part of an application for approval for marketing (UK Pesticides Safety Directorate, 1996). The approval procedure includes a description of the product (physio-chemical properties), its intended use, efficacy, mammalian toxicity, food residues, fate and behaviour in the environment and ecotoxicity. As a result of the evaluation the compound was given provisional approval. Extracts from the sections on environmental fate and exposure and risk to birds are provided in the following sections.

Fate and Behaviour

Metosulam degrades reasonably rapidly in laboratory soils (half-life 11-30 days) and two of the identifiable metabolites reached greater than 10 per cent of the applied dose. In field soils, dissipation occurs at comparable rates to dissipation in the laboratory. Metosulam degrades very slowly in aqueous conditions (half-life in natural waters 69-135 days). The parent compound does not partition into the sediment to any great extent and non-extractable residues in the sediment were very low.

Exposure and Risk to Birds

Metosulam is intended for application as a spray to winter wheat and barley from February until the middle of May up to GS 32 at a rate of 10 g ai/ha. Deposition of the ai on cereal shoots is expected to be 2.1 mg/kg, with as a worst case, 1.0 mg ai/m2 reaching the bare soil surface (OEPP/EPPO, 1993). The maximum recommended number of treatments is one per crop.

Birds thought likely to be exposed to metosulam include grazing/browsing birds such as geese and pigeons, which might forage contaminated vegetation, and insectivorous birds such as sparrows and thrushes feeding on contaminated arthropods and earthworms. Data on exposure, toxicity, and TERs are summarised in Table 6.1 for representative bird species. As several bird species may be exposed to the product, its risk classification should reflect the threat to the most vulnerable example. Of the principal exposure routes listed in Table 6.1, the greatest risk to birds is by ingesting residues on vegetation. In this case the risk appears to be low. For example, an individual goose weighing 3,300 g could consume 990 g of vegetation daily. If the individual were to feed exclusively on vegetation contaminated with 2.4 mg metosulam/kg, it would be exposed to 0.63 mg ai/kg bw. Given an acute lethal toxicity (LD50) of >2,000 mg ai/kg bw, this results in a toxicity/exposure ratio (TER) of >3,175 which is classified by EPPO as "low risk" (greater than 100). Due to the low avian toxicity and levels of exposure, the risks from consumption of contaminated insects and earthworms are also considered to be low.

Table 6.2: Risk assessment summary for birds based on acute effects

Species	Body Weight (g)	Food Type	Food residues (mg ai/kg)*	Food ingestion (g/day)	Dose (mg ai/kg bw)	LD50 (mg ai/kg)	TER
Goose	3,300	Vegetation	2.1	990	0.63	>2,000	>3,175
Hedge sparrow	19	Invertebrate prey	0.29	7.1	0.1084	>2,000	>10,000
Thrush	75	Invertebrate prey	0.027	18	0.0065	>2,000	>10,000

* calculated from OEPP/EPPO.

Source: UK Pesticides Safety Directorate, 1996

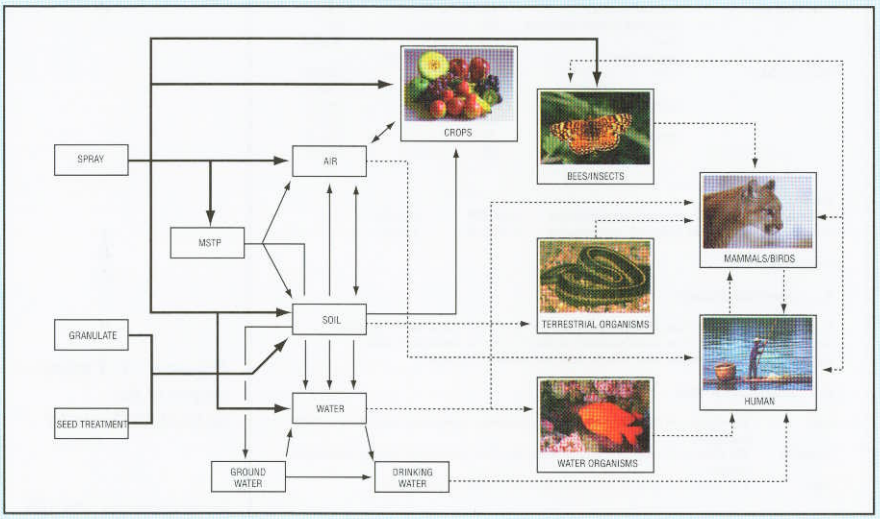
The TERs for the three respective species of birds at risk are all greater than the upper limit set by EPPO and there is no evidence of risk from any further studies. Birds should not be continually and repeatedly exposed to metosulam throughout the breeding season and therefore the compound should not pose a risk to breeding birds. The firm submitted two pilot bird reproduction studies; no adverse effects on egg production or other non-reproductive sub-lethal effects were seen at 1,000 ppm ai. Based on this NOEC and the predicted residues on vegetation of 2.14 ppm the TER would be 467, indicating that metosulam presents a low risk to birds.

Note: ai = active ingredient, bw = body weight.

Box 6.3 Application of the Evaluation System for Pesticides (ESPE) for agricultural pesticides

The ESPE has been developed as a component of the Dutch USES which is a substance evaluation scheme designed in response to EU legislation with the intention of assessing the risks of substances before they can be used or marketed in the Netherlands. The ESPE exposure scheme is presented in Figure 6.10. Three stages, emission, distribution and effect assessment can clearly be seen. The objective of the system is to provide a Predicted Environmental Concentration (PEC) which can then be compared with the No Effect Concentration (NEC). This ratio indicates the likelihood of adverse effects occurring in the organism of concern. For this example the SLOOT.BOX model is used for calculating the concentration in surface water by drift of the pesticide after application. The model takes into account the physico-chemical properties of the substance, the application regime and the removal of the substance by advection, hydrolysis, volatilisation, biodegradation, sedimentation and resuspension. The results of an evaluation for Fenvalerate using the SLOOT.BOX model are presented in Figure 6.11.

Figure 6.10: Exposure scheme for agricultural pesticides



Source: Linders and Luttik, 1995

Continued over

the combined effects of products can be considered to be at least addictive and possibly synergistic (Thompson, 1996).

The National Institute for Public Health and Environmental Protection (RIVM) in the Netherlands has developed an Evaluation System for Pesticides (ESPE) as part of the USES (Uniform System for the Evaluation of Substances). The ESPE is a tool for rapid, quantitative assessments of the general risks of pesticides, both agricultural and non-agricultural. The first tier of the assessment provides an opportunity to estimate the risk. In the second tier, the estimation of the concentration of pesticides in the environment

Box 6.3 continued

Date: 20-4-1994 Time: 6:21

EVALUATION OF SURFACE WATER BEHAVIOUR

Substance:	fenvalerate			
Characteristics:	molecular weight		419.9	[g/mol]
	vapour pressure		0.00002	[Pa]
	solubility		0.016	[mg/l]
	Henrycoefficient		2.16E-07	[-]
	T1/2-biodegr.water/sed		67	[d]
	ads.coef.soil study		150	[dm3/kg]
	frac.orgC-soil		0.6	[%]
	oct.water-part.coef.		25118.86	[-]
Data on dose:	number of doses		14	[-]
	time period between doses		10	[d]
	dose		0.06	[kg/ha]
	application:		Brussels sprout, etc.	
Toxicity data:	fish	LC50	0.64	[mg/l]
		NOEC	0.06	[mg/l]
	daphnids	LC50	0.03	[mg/l]
		NOEC	0.005	[mg/l]
	algae	EC50	50	[mg/l]
		NOEC	10	[mg/l]

Results:

estimation of long-term exposure concentration	0.001899	[mg/l]
estimation of short-term exposure concentration	0.001618	[mg/l]

Conclusions:

\# short-term exposure

fish	The chance on acute mortality or effects must be considered negligible.
daphnids	The chance on acute mortality or effects must be considered small.
algae	The chance on acute effects must be considered negligible.

\# long-term exposure

fish	The chance on long-term effects (most sensitive parameter) must be considered negligible.
daphnids	The chance on long-term effects (most sensitive parameter) must be considered present.
algae	The chance on growth inhibition must be considered negligible.

Figure 6.11: Typical output of the SLOOT.BOX model

Source: Linders and Luttik, 1995

and the possible effects has to be refined, e.g., using additional testing. The third tier deals with an in-depth analysis of the local situation possibly at risk (Linders and Luttik, 1995). A quantitative comparison of the results of the exposure assessment and the effects assessment is made, to produce a hazard quotient (as for PEC/PNEC for chemicals). See Box 6.3.

6.3 Genetically modified organisms

A risk assessment is required by EC Directive 90/220/EEC on 'The Deliberate Release into the Environment of Genetically Modified Organisms' before consent is approved on the marketing or deliberate release of Genetically Modified Organisms (GMOs). In response to the resultant UK legislation (Part VI of the

Environmental Protection Act, 1990), guidance issued by the UK Department of the Environment and the statutory advisory committee, ACRE (the Advisory Committee on Releases to the Environment) sets out the requirement for risk assessment (DOE/ACRE, 1992). Most EU States have developed methodologies for the risk assessment of GMOs and have organisations conducting research to refine the methodologies used to protect man and the environment (details can be found on the Belgian Biosafety Server on the Internet - see information sources). The Austrian Federal Environment Agency has produced revised criteria for the assessment of releases of GMOs into the environment (Gaugitsch and Torgersen, 1995). This is based on a strategy of minimum data requirements and differentiating assessments into genetically modified micro-organisms, plants and animals.

There has been much criticism of the current methodologies used for the assessment of GMOs, particularly of the EC Directive guidelines. Concerns include the fact that our current knowledge does not provide us with the means to predict the ecological long-term effects of releasing organisms into the environment (von Schomberg, 1996) and the inconsistency of definitions between EU states leading to different criteria for approval (Levidow et al., 1996). Work is currently under way by the European Commission to address this problem of a lack of harmonisation (Chapter 2, Box 2.3).

At an international level, UNEP has been influential in the establishment of an international agreement on biodiversity, the Convention on Biological Diversity, which is signed by over 150 nations. This body is now working towards the creation of a Biosafety Protocol to be enacted in 1999. The work includes the evaluation of the existing policies and guidelines of the OECD, the FAO and the International Technical Guidance on Safety in Biotechnology being finalised by UNEP.

In the UK guidance document, risk assessment consists of seven steps (risk management is also integrated within the procedure).

Step 1 is the identification of the characteristics of the GMO which constitute the hazard. The following considerations are considered to be the most important:

(i) Capacity to survive, establish and disseminate.
(ii) Potential for gene transfer.
(iii) Products of expression of inserted sequences.
(iv) Phenotypic and genotypic stability.
(v) Pathogenicity to other organisms.
(vi) Potential for other effects.

Step 2 examines the characteristics of the receiving environment and identifies which of the hazards of the GMO are likely to be realised. For each of the hazards identified in Step 1, it is necessary to consider whether the receiving environment will cause or allow the hazard to be realised and to consider whether

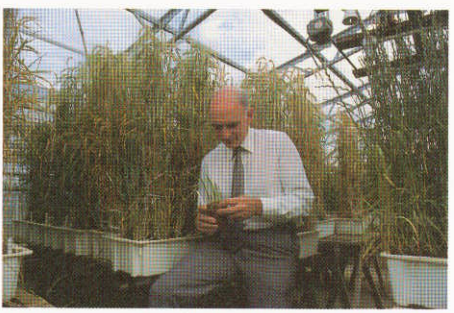


Photo: Pete Addis, Environmental Images

different ecosystems will be affected by the introduction of the GMO into the environment.

Step 3 estimates the potential harmful effects, and their magnitude, from each of the identified hazards of the GMO in the particular receiving environment. The magnitude of environmental harm is expressed qualitatively in terms of severe, moderate, low or negligible harm.

Step 4 assesses the probability or frequency with which the potential harmful effects identified in Step 3 might be realised. Considerations include the proportion of released GMOs that might be involved in the realisation of a particular hazard as well as the number of times that hazard might be realised over a particular period. An example is the assessment of the hazard survival capacity of the GMOs as some will die on release but some will also survive.

Step 5 evaluates the risk of each of the hazards identified in Step 1 being realised (i.e., the risk caused by the presence of that hazard in the environment). This combines the information on the potential harmful effects (Step 3) with the probability of the potential harmful effects being realised (Step 4) to produce a qualitative estimate of the risk of damage. It is clear that it is necessary to consider each GMO on a case-by-case basis, taking into account any previous experience.

Step 6 is concerned with risk management and intends to prevent or minimise any significant environmental risks by the application of appropriate control measures. The DOE guidance suggests that if the risks of a hazard are judged to be high or medium, the risks should be re-assessed to ascertain whether the application of additional management techniques could reduce the level of risk.

Step 7 evaluates the overall risks to the environment of the release of a GMO, combining the effects of the risk from each hazard to make a value judgement.

7. THE APPLICATION OF ENVIRONMENTAL RISK ASSESSMENT IN INDUSTRY

Environmental Risk Assessment (ERA) is currently being used in industry in Europe. The extent of its use depends upon the industry sector and the size of the business. Legislation is one of the major reasons companies will use ERA. In "major hazards" industries covered by the 'Seveso Directive', ERA is commonplace.

If the industry involves processes subject to Integrated Pollution Control (IPC - UK) authorisation or the proposed EU Integrated Pollution and Prevention Control, ERA can be a useful assessment tool. In the process industries, new and existing chemicals now need to be risk assessed using techniques specified by the European Commission.

Some companies have extended their risk management strategies (particularly for public safety) beyond the requirements of national regulations. These companies tend to be large multi-nationals who have the capital available for investment in new environmental initiatives with the potential to influence and drive future legislation and reduce their own long-term costs, boost company image and protect shareholders. In some cases, significant risk management programmes may have preceded legislation. Generally, small and medium sized companies tend to limit the extent of their environmental activities to compliance with legislation because of investment constraints.

This chapter outlines the major ERA methods used in European Industry. It examines ERA used for non-routine industrial releases, routine industrial releases, transportation risks, financial risks, the risks posed by chemicals and products, risk minimisation and reduction decision-making, and specific industrial applications such as contaminated land and land-use planning. As most risk assessment activity in industry addresses non-routine

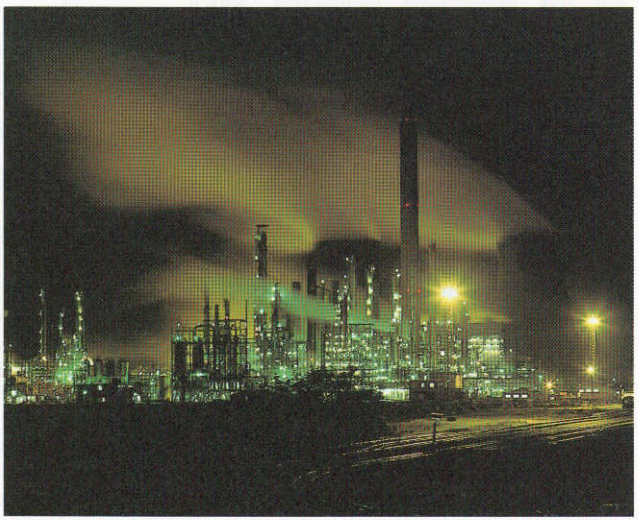


Photo: Martin Bond, Environmental Images

accidental releases and product/chemical risk assessments, these are the major focus of the chapter. Little work is carried out by industry itself on ERA of routine releases other than emission modelling for licences, as this is not yet a legislative requirement. Routine releases are restricted and controlled by regulators applying concepts such as Integrated Pollution Prevention and Control, Best Practicable Environmental Option and Best Available Technology (Not Entailing Excessive Costs).

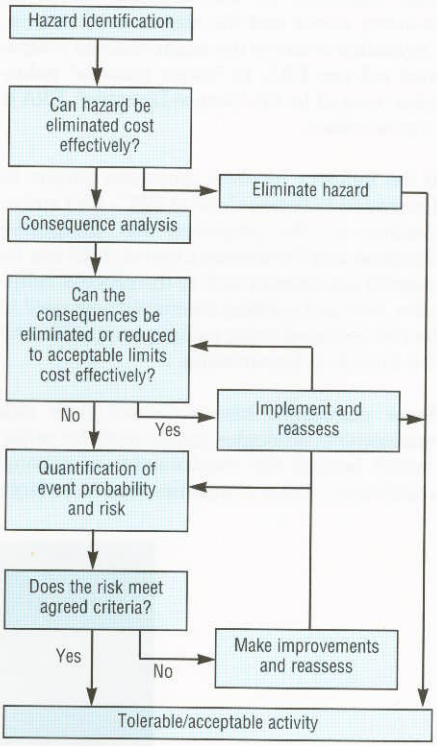
Brief case-studies of organisations willing to provide information on the use of ERA are given in the chapter as are examples of risk assessment techniques used in contaminated land, waste disposal site assessment, transportation and land-use planning.

7.1 Site-specific ERA for non-routine releases

Risk assessment techniques developed in the process industries are derived from engineering risk assessment techniques, which examine plant or process engineering risks. Quantitative Risk Assessment (QRA) has been used as a reliability and safety decision-making tool in the nuclear energy and the aircraft and aerospace industries for many years. Interest in the use of QRA techniques for assessing the safety of process plants has grown considerably in Europe (Pitblado and Turney, 1996). This is partly a response to European Legislation such as the 'Seveso Directive' (EEC, 1982).

In many cases, QRA addressed plant or equipment reliability and safety in respect to employees. The consequences of major accidents from hazardous processes and their potential impact on public health and the environment have been incorporated into techniques that focus on plant safety and reliability.

Figure 7.1: Procedure for the application of risk assessment



Source: Pitblado and Turney, 1996

These techniques are very similar to those used in an Environmental Impact Assessment carried out at the planning stage of an industrial plant. Techniques to assess the risks to human health from non-routine industrial releases are far more advanced than those addressing ecological risks. Several companies have developed their own methodology for risk assessment, either quantitative or

qualitative, or have adapted existing methodologies to suit their own needs and requirements. Some risk assessment methods address human health risk, others attempt to address ecological risk. A limited number of companies have developed fully integrated techniques looking at all risks at all stages of production and use.

The principles of ERA for non-routine accidental releases are to identify the hazard and the release scenario, to analyse the effects or consequences and, if necessary, to provide a quantitative estimation of the event probability and compare it with agreed criteria. This will lead to risk acceptance or the implementation of risk reduction measures that reduce the likelihood of the event or reduce the consequences to a satisfactory level. This basic procedure is illustrated in Figure 7.1.

Site-specific risk assessment for non-routine releases consists of several components:

7.1.1 Hazard identification/release assessment

As discussed in Chapter 4, hazard identification is, in practice, rarely an isolated step in ERA. In the ERA of plant, the same techniques will be used to identify hazards and assess the likelihood and extent of releases.

Hazard identification and release assessment are most effective when carried out at the conceptual and design stage in the development of a plant or process. However, it is important to emphasise that hazard identification and release assessment are important processes throughout the life cycle of the plant, particularly if modifications are made.

The result of the hazard identification and release assessment stage of the ERA can be a quantitative, probabilistic estimate of the likelihood of a release of a certain quantity of hazardous material. An example of this would be the release of x mg/m^3 of a certain gas, which is likely once every 100 years. Qualitative estimates, such as the release of high concentrations of a certain gas is unlikely, are also common.

Hazard identification methods

Hazard indices are useful in the planning stage. They provide an indication of the potential for a given design of plant to produce a hazardous incident. An example of a hazard index is Dow's Fire and Explosion Index. These tools are useful in the design stage as they require limited data and can prioritise areas of the design which may require more attention.

In the design stage, several hazard identification techniques may be used. Those most frequently used include:

Photo: Dave Ellison, Environmental Images

- Hazop
- What-if
- Knowledge-based Hazop
- Checklists
- Failure mode and effect analysis
- Fault trees
- Event trees
- Task analysis

Hazop is a widely used method. It uses guide words such as "more", "less" and "reverse" which can be applied to process stages to generate deviations from the designer's intentions. An example of a Hazop worksheet is provided in Figure 7.2 (Wells, 1996).

What-if methods are creative, brainstorming examinations of a process or operating procedure, carried out in a small team with a chairman asking questions. The analysis considers the results of unexpected events that would produce an adverse consequence.

Knowledge-based Hazop uses the knowledge gained by the company from previous experience. The guide words are supplemented or partially replaced by both the company's and the team's knowledge supported by specific checklists.

Checklists specify those components of a plant which require safe design, and help to ensure that designers address known hazards. The technique uses data from industry codes, past accidents and expert judgement.

Failure mode and effect analysis is a method for evaluating the ways in which equipment can fail or be incorrectly operated and the effects these failures may have on the plant. The method identifies areas of the design that may need improvement or change.

Fault tree analysis (FTA) is a graphical model that illustrates combinations of failures that will cause one specific failure of interest - the "top event". The model is essentially a logic diagram. The root is the main event and possible causes of the event are traced back to several initiating events. It is a deductive process.

Event tree analysis evaluates the potential for an accident as the result of a general equipment failure or process malfunction, known as an initiating event. Event tree analysis is an inductive process where the analysis begins with an initiating event and develops the possible sequence of events that lead to potential accidents. An example of a logic diagram, which is produced, is provided in Figure 7.3.

Task analysis is used to analyse the human characteristics of systems, operations and procedures to identify likely sources of error. The use of task analysis is generally limited to situations where other techniques, such as Hazop, What-if or FTA, have shown that human errors could lead to high risk.

Hazard identification and release assessment are considered vital components of risk assessment of plants and processes, and are also important in their own right as they draw attention to areas of unacceptable risk such as potential plant mechanical failures, and initiate the risk reduction process through modification of the design or safety system. However, they can also be a source of failure in the system if all the hazards and release points are not identified.

Information sources are extremely important. Knowledge of hazards is acquired through personal experience, consultation, comparison with an identified hazard, engineering codes

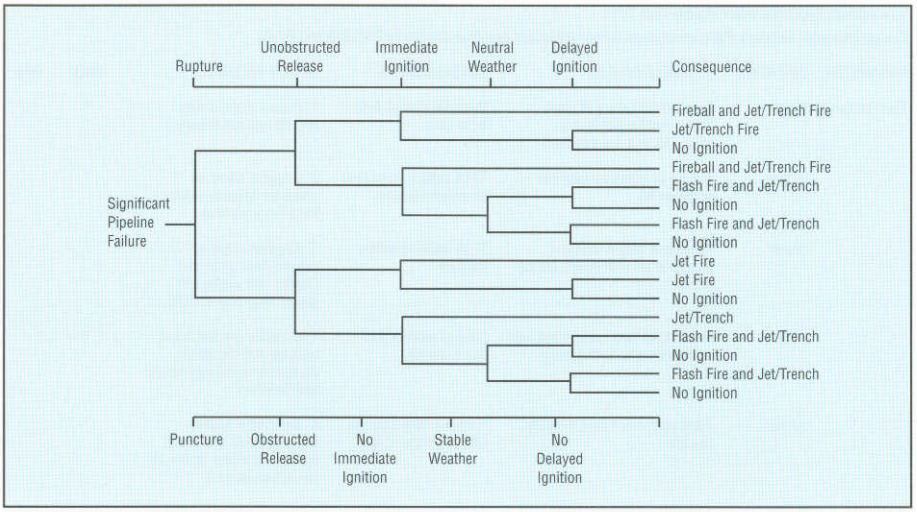
Figure 7.2: Hazop study action report form: water injection upgrade

Line section: Produced water storage tank T710.
Design intention: Receives PW from storage surge facility and provides suction to P715A/B and P716.

PARAMETER	DEVIATION	POSSIBLE CAUSES	CONSEQUENCES	ACTION REQUIRED	PROJ	OPS
Temperature	Higher	Mechanical failure of shell fittings/instruments, e.g., LSLL753 in a fire situation	Possible loss of fluid from tank	1. Ensure instruments and fittings are fireproof.	*	
	Higher	Failure of pressure vacuum valve on N4	T710 suffers from over or underpressure	2. Suggest alarm on pressure vacuum valve for a fire condition.	*	
	Lower	Excessive cold if vessel is empty or on roof space during winter	T710 liable to brittle fracture	3. Suggest check by independent audit that Charpy test values are adequate at minus 10°C.	*	
	Lower	Freezing of tank instrument legs and penetrations in general	Loss of control and trip systems	4. Consider electric trace heating and insulation, preferably non-hygroscopic and fireproof.	*	
	Lower	Tank contents freeze if levels are low	Possible tank damage and blockage of N11	5. Determine lowest freezing point and check design. Consider action as per action point 3.	*	
Pressure	Higher	External adjacent or local pool fire	T710 pressure vent required in a demand situation	6. Ensure pressure/vacuum relief valve sized for fire condition. 7. Ensure pressure side sufficiently reliable.	*	
	Higher	PCV7108 fails closed in service	Total reliance on PVRV7101 to relieve high tank pressure	8. See action 6.	*	
	Lower	PVRV7101 seat freezes in cold weather	Tank suffers pressure damage (vacuum) as level falls	9. Suggest valve selected can without high differential pressure break the effect of ice formation on seat. 10. Suggest consideration of two emergency vent designs to avoid common mode failure scenarios.	*	*
Level	Higher	LAH756 and LSHH754 fail in a demand situation	Tank suffers from hydraulic damage/over stressing and possible loss of containment	11. Consider the use of tank overflow. 12. Determine requirement for trip reliability and compare with specification. 13. Consider frequency of operator checks and evaluate effect of improved vigilance.	*	*
Composition	Impurities	Oil component separates into heavier layer and gas boils off	Heavy materials settle forming residue and anaerobic conditions	14. Possible corrosion. Consider tank lining. If lining used watch for coating over shell base preparations.	*	*

Source: Wells, 1996

Figure 7.3: Pipeline risk assessment method: Event Tree



Source: Carter, 1991

and practices, and accident and "near miss" history. Details of data banks containing safety and reliability data and accident data are provided in the information sources section.

Release assessment methods

The same techniques used in hazard identification are essential in the analysis of how a release will occur and with what frequency. Release assessments will simply attempt to give a measure of the likelihood of a release.

A quantitative estimation of the probability of release can be approached in two ways: the historical approach which uses direct statistical data on plants or systems, or the approach which uses analytical and simulation techniques, breaking the system down into contributing factors and causes.

If the historical data are of high quality, relevant and statistically significant, their use can be advantageous, as the assessment should not omit any important events that could lead to the event. However, the information may be outdated and not include recent process improvements, which may lead to a "conservative" estimate of the probability and the data are very likely to be dominated by older plant.

If the historical data are considered inadequate, synthesis of event probability needs to be carried out. This will calculate the chance of an event (release) occurring. This is primarily achieved through the use of logic diagrams such as those also used in hazard identification: Fault Tree Analysis and Event Tree Analysis. When used to calculate probabilities, FTAs can involve some complex

mathematics (algebraic and Boolean functions), particularly if sub-events appear more than once in the tree. Simple fault trees, however, can be evaluated by multiplication/addition of probabilities at AND/OR gates, if no event appears twice. In the past this may have encouraged the analyst to over-simplify the process to aid calculation but now computer packages are available to solve such complex mathematical problems.

Event Tree Analysis is more straightforward, following the initial causes through to several possible outcomes. Event Trees are evaluated by allocating a probability of occurrence to each outcome, which is conditional only on the occurrence of the precursor event. The probability of each outcome - conditional on the initiating event of the tree occurring - can be obtained by straightforward multiplication of all the branch probabilities leading to that outcome.

As for hazard identification, available data are extremely important in the quantification of event probabilities. Data banks comprising accident data, incident data and reliability and event data are all useful in probability analysis. Obviously the most appropriate data are those relating to the particular plant under assessment. If these are not available it is necessary to use data from other sources on similar plants. Much of the available data may not be suitable which introduces uncertainty into the assessment.

7.1.2 Exposure assessment

Exposure assessment attempts to determine the magnitude of the effects of an undesirable event (identified in the hazard identification and release assessment stages), and the pathways and transport modes of the hazard to the receptor.

Photo: Martin Bond, Environmental Images

This stage requires the use of predictive exposure modelling techniques including discharge (e.g., blow-down from a punctured vessel or line), aerosols (liquid flash, entrainment and rain-out), evaporation (on land and water), dispersion (Gaussian plumes and heavy gas dispersion), thermal radiation (flash fires, jet fires, pool fires, fireballs/BLEVES), and vapour cloud explosions (TNT-based models and fuel-air blast charge models, numerical simulations), and also population mapping models incorporating GIS (Geographical Information System) techniques. Exposure assessment attempts to quantify the potential exposure levels of the hazard at the receptor site.

7.1.3 Consequence assessment

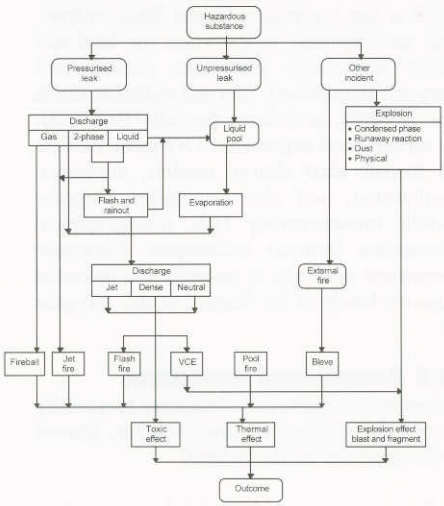
Consequence assessment attempts to quantify the possible damage to the receptor, caused by the exposure to the hazard.

In the context of industrial non-routine releases, this process incorporates the use of so-called vulnerability models including explosion damage (structural and human), fire

damage (structural and human), and toxic injury. Models and data are also available to assess the effects on the environment but, at this stage, information is still limited. It is beyond the scope of this book to explain in detail the various models and techniques used in consequence assessment. A brief description can be found in Pitblado and Turney (1996) and more comprehensive detail can be obtained by referring to the relevant publications in the information sources section.

Figure 7.4 is a logic diagram depicting the stages of risk assessment for non-routine releases from hazard identification to consequence assessment.

Figure 7.4: Logic diagram for consequence analysis



Source: Pitblado and Turney, 1996

Box 7.1 Calculating a risk estimate for human health from a non-routine release

Quantification of risk to human health from the non-routine release from an industrial activity involves the multiplication of the consequence for each damage-causing event with the frequency of that event.

The consequences of a damage-causing event are usually stated as casualty probabilities, i.e., the probability of harm occurring due to exposure to a specified level (or range) of hazard, or alternatively, the degree of exposure required to produce set ranges of casualty probabilities.

The number of people present in the areas covered by each probability band is multiplied by the appropriate casualty probability producing the total number of people predicted to be affected by each event. When combined with the frequency for each event, a risk estimate can be produced. For individual risk at a given location, the casualty probability for that location is multiplied by the frequency of the event to give a risk estimate (Pitblado and Turney, 1996).

7.1.4 Risk estimation

The risk estimation stage consists of integrating the estimation of the probability of release events with the results of the consequence assessment to produce an estimate of the overall risk of an activity. See Box 7.1.

The corporate risk assessment programme for non-routine releases addressing public safety developed at BOC is presented as a case study. (see Box 7.2).

7.1.5 The extent of ecological risk assessment in site-specific ERA for non-routine releases

The use of ERA for ecological risks is in its infancy. It is performed by some environmental leaders, mainly by the large multi-national chemical and petro-chemical companies, but its use in small to medium-sized businesses is

Box 7.2 Case Study - BOC Group plc

The Company

BOC is a global company with operations in 60 countries. The company has a portfolio of four businesses - industrial and special gases, health care, vacuum technology and distribution services. The industrial and special gases business is the most established and important activity of the company (70 per cent products). BOC produces over 20,000 gas mixtures of anything up to 99.99999 per cent purity. These gases are used in a wide variety of applications, such as the production of microchips, freezing food and water treatment. The company has a very proactive attitude towards environmental protection with a vision of a globally environmentally sustainable business in which they take full account of the environmental and economic consequences of their current and future activities (BOC Management Magazine, No. 24). As part of this vision, the company is a signatory to the International Chamber of Commerce Charter for Sustainable Development, which commits the group to 16 principles of environmental practice.

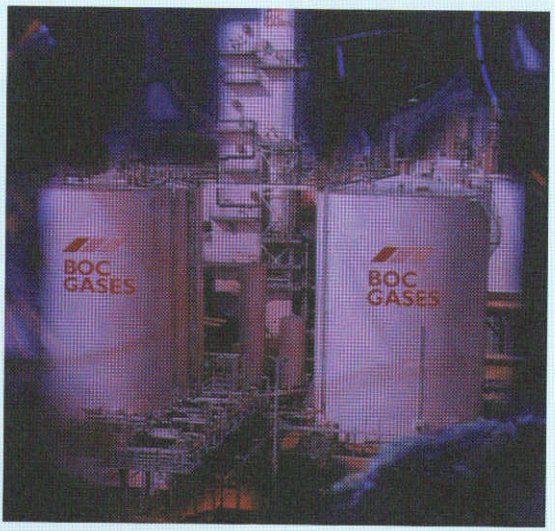


Photo: BOC

Risk Assessment of Non-routine Releases

The BOC Group initiated the Major Hazards Review Programme (MHRP) with the objective of ensuring that large-scale hazards from their operations are properly understood and controlled. The programme focuses on recognising, managing and controlling risks to the public. The main drivers for the implementation of this programme were the catastrophic industrial accidents in Bhopal and Mexico City in 1984. The MHRP is a corporate policy implemented and put into practice by a network of co-ordinators. It is essentially about recognising, acting upon and controlling high consequence risks. The MHRP is a four-step process:

Step 1 Completion of a site activity and materials inventory.

Step 2 If quantities of these materials exceed a specified threshold level, a quantitative hazard assessment is performed which includes public exposure modelling and the generation of hazard ranges.
Databases such as RTECS, TOXLINE and HSDB are used for toxicity information and models such as the Gaussian Dispersion Model and BLEVE for exposure assessment.

Step 3 If the consequences of a release extend off-site and a significant off-site population is affected, a detailed quantitative risk assessment is carried out. The objective of the QRA is to satisfy the Loss Prevention Council of BOC that the risks presented at a site are within the BOC Group guidelines and meet the company criteria (risk levels that are based on the most recent criteria used by industry and the public sector).

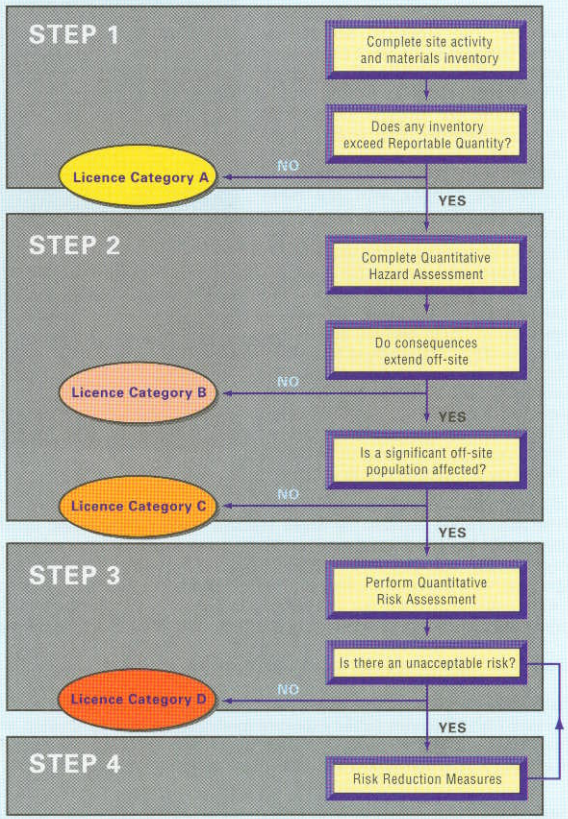
Step 4 If the level of risk is considered unacceptable, risk reduction measures are taken to eliminate, reduce or mitigate the risk such as making engineering changes or changes in plant design or reducing or removing inventories of materials.

Continued over

Box 7.2 continued

Each step involves the issuing of a licence. A licence can be issued for a site at each of the steps in the programme, ranging from Licence A at the first step for a site with no reportable quantities of hazardous materials, to Licence D for a site with reportable quantities of hazardous materials. Where consequences extend off-site, significant off-site population may be affected and adequate plant controls and acceptable risk is demonstrated through QRA. The steps and licensing procedure for the programme are illustrated in Figure 7.5.

Figure 7.5: Flow chart for MHRP and site licensing at BOC



Source: BOC, 1995

Major benefits of the implementation of the MHRP are the introduction of tools such as Hazop, which identifies potential hazards at the plant design stage, better response to new regulatory initiatives to control hazards, and a general reduction in inventory. The MHRP allows the BOC Group to set consistent hazard standards in all the countries in which they operate.

Prioritisation of Risk Reduction Measures

BOC have developed a system which looks at the activities carried out at each BOC site, identifies the environmental impacts and the risks to the environment and the risk management measures in place. An "environmental risk fingerprint" is produced for environmental performance in respect of contaminated land, waste, energy, water, air, and regulatory compliance. The system is essentially one of site/activity prioritisation for risk reduction and management. The quantification of the risk involves combining the environmental impact with the frequency of the events. Environmental impact is derived from a combination of "ecopoints" (obtained from national emissions and targets), costs and decisions of an expert panel. The system produces a comparative score for each site and identifies those areas of normal everyday operations that pose a risk to the environment and consequently those activities that need to be targeted for risk management and reduction.

very limited. In terms of hazard identification, release assessment and exposure assessment, the techniques used for safety related human health assessment, can be readily incorporated into a methodology for assessing the ecological risks. At present, hazards can be identified and the probabilities and magnitudes of possible releases estimated. Models have been and are being developed to predict the environmental fate and transportation of substances in different media, such as the gas dispersion models described above, and surface and ground water models, such as PRAIRIE (UK risk assessment tool for predicting the risks associated with accidental releases of hazardous materials into rivers and estuaries) and VERIS (a similar tool developed by VROM, the Environment Ministry for The Netherlands). However, these models are principally deployed by regulators to predict the possible consequences of pollution incidents and are often deployed post-release. There is currently no legislative requirement for industry to carry out an ecological risk assessment of its activities. One of the major difficulties is the lack of adequate toxicity data, particularly on the effect of very short-term exposures (possibly several minutes to a couple of hours for accidental releases). Possibly, in the future, industry may be able to carry out full ecological risk assessments but it will require clear guidance on acceptability and tolerance criteria, which are not currently available. Industry is in a position, however, to at least identify possible hazards to the environment and take appropriate steps to minimise the risk of a non-routine release of such hazardous substances. One approach is quantitative reliability analysis, which attempts to relate component reliability to the environmental risk posed by accidental or non-routine releases as a result of component failure (Imperial College/HMIP, 1995).

Photo: Dave Ellison, Environmental Images

7.2 Site-specific ERA for routine releases

In the form of an entire process, legislation does not require industry to assess the risks posed by their emissions from routine operation of industrial plant. They are required to comply with emission and effluent discharge standards based on principles such as BATNEEC or BAT and to meet Environmental Quality Standards (EQS) or Environmental Assessment Levels (EAL). EQS/EAL may be based on toxicity or ecotoxicity testing of representative species and the application of extrapolation factors or risk criteria incorporating guidelines from recognised bodies, such as the World Health Organization. The main priority of industry, subject to such regulatory control, is compliance with the emission

or discharge limits set by the regulator. The consequences of non-compliance are significant. Regulatory authorities have the power to serve an enforcement notice if authorisations are breached. If a serious risk to the environment is posed, a prohibition notice will be served resulting in possible closure of the facility. Continued non-compliance or major environment-threatening incidents result in prosecution, fines and potentially the imprisonment of the responsible person. Non-compliance also has additional negative effects, including the expense of authorisation re-application, diversion of management time, demotivation of employees, higher insurance premiums, and consumer boycott (Welford and Gouldson, 1993). With the introduction of Pollutant Release and Transfer Registers (Chemical Release Inventories) and general public access to information on industry environmental performance, it is increasingly important for companies to comply. Management programmes are put in place to ensure that the risk of non-compliance is reduced to a level which meets company criteria or satisfies the regulator.

Large multi-national companies may set their own internal standards for routine emissions which are more stringent than current regulations demand, and some adopt the precautionary principle and try to reduce emissions to the minimum that is technically possible. The case study of BASF shows how a general risk minimisation policy can be applied across the spectrum of a company's activities. It does not incorporate formal probabilistic QRA but risks are controlled and minimised by appropriate design of plant and safety systems and training (see Box 7.3).

7.3 Transportation risk assessment

Significant quantities of hazardous substances are transported by road, rail and pipeline. Mobilising a hazard creates both risk-lessening factors and risk-increasing factors when compared with a stationary hazard scenario. Risks are increased, for instance, by bringing the hazardous substances into close proximity with the general public or by on-board safety systems being less robust than fixed systems.

Photo: Martin Bond, Environmental Images

Box 7.3 Case Study - BASF

The BASF Group has production sites in 39 countries, producing 8,000 different products. The largest single company within the Group is BASF Aktiengesellschaft, which owns the largest single chemical site in the world at Ludwigshafen, Germany where almost 45,000 people work in more than 350 production plants, laboratories, technical centres and offices.

BASF's products include pharmaceuticals, fertilisers, crop protection products, dyestuffs, pigments, basic chemicals, solvents, glues, plasticisers, plastics and fibre products, and oils and gases.

A company of this size has the potential to have an enormous impact on the environment. It is only through the application of an effective environmental management strategy and the implementation of corporate environmental guidelines, values and available finance that BASF has been able to develop such a high profile in the area of environmental protection. A particularly notable corporate guideline is "not to give economic considerations precedence over environmental protection and safety".

BASF AG has a general risk minimisation/prevention and management policy which covers all of its activities. The risks can be considered to be any process or situation involving hazards that could be or are released acutely or chronically to the environment and may have an effect on man or the ecosystem. The risk analysis process may be quantitative or qualitative. Hazop analyses and consequence assessments are carried out on all new plants and modifications of existing plants using the worst case scenario technique as input for environmental fate predictive modelling. In the event of acute, non-routine releases, emergency planning procedures are in place to deal with consequences and effects on the local population. Any such event is subject to a critical analysis which will provide information for risk management decision-making regarding plant and site safety. A transport risk management strategy is also in place.

Photo: BASF, Ludwigshafen

For chronic releases, emissions to all media are monitored to ensure compliance with set standards. The vast majority of waste is recycled internally within the company, treated in the waste water treatment plant or incinerated with energy recovery; as little as possible is sent to landfill.

BASF perform risk assessments on the components of its products to minimise the risks to man and the environment. It is a major company providing information for the EU Programmes on the risk assessment of New and Existing substances and for the German Advisory Committee on Existing Chemicals of European Relevance. The risk assessment methodology used for products and New and Existing substances is that which is laid down in the CEC Technical Guidance Document. BASF also carries out risk assessments for the approval and registration of its plant protection products. The company has an established expertise and facilities for the accruement of data on toxicity and ecotoxicity of chemicals and substances and is carrying out research and development activities to develop new, "environment-friendly" products and processes with a reduced risk of polluting the environment.

Box 7.4 Case Study - Tank Truck Transportation of Chemicals and Petroleum Products

This case study looks at a financial risk assessment carried out by ERM for a confidential client. Four elements and member companies of the Environmental Resources Management (ERM) Group have developed a methodology for addressing human, environmental and business risks on a consistent financial basis. Financial Risk Assessments have been carried out using this methodology for various forms of transportation and distribution systems and for fixed facilities (Meyers and Mudan, 1996).

The Company
A transport company has 2,000 non-pressurised tankers, which are used to ship chemicals and petroleum products across all of North America. Liquid feed stocks, intermediates and products are transported, one-third of which are petroleum products. The methodology used in the assessment considers the fleet as a whole, rather than individual tankers.

Tank Truck Frequency Analysis
A key component of the risk assessment is the determination of the likelihood of accidents and accidental releases. The likelihood of accidental release is a function of the total miles travelled by the tankers, the accident rate per mile, and the probability of the accident resulting in a release. The severity of releases is included by incorporating spill size distribution.

Consequence and Impact Analysis
The consequence of an accident is expressed in terms of the hazard zone. The impact analysis addresses the effects of hazard on people, the environment, property and business. In general, a hazardous material release may pose flammable, explosive and/or toxic and ecotoxic hazards.

Human Impact Calculations
The chemicals and petroleum products in this study are not severely toxic or highly flammable or explosive but tanker truck accidents can result in injuries and fatalities. A database of 12,000 incidents is available on tanker releases. The impact of releases on people was evaluated using data from the Research and Special Programs Administration (RSPA). This database provided a probability distribution of the number of deaths and injuries caused by on-highway releases. Consequence analysis and a review of case histories and reports for the period are used to qualitatively validate the appropriateness of the statistical information.

Environmental Impacts
Spills result in significant clean-up and restoration costs. In order to place all risks on a financial basis, data from actual accidents are used to estimate the total cost of environmental liabilities. The consequences of spills are calculated by determining the area contaminated by each release and applying clean-up cost data. For spills that ignite, clean-up costs are minimised while property damage will increase. RSPA data are used to determine the fraction of spills igniting. For spills that do not ignite, environmental impacts are expected and evaluated.

The clean-up costs are primarily from soil remediation, groundwater clean up, water spill clean up and the incineration of hazardous wastes.

Continued opposite

The individual risk associated with transportation is very low as an individual member of the public will only be exposed for a very short time. Therefore, an assessment will only normally take into account societal risk.

About one-third of shippers and transport companies are currently using risk management in some form, while another third are interested in the use of risk management ('Carriers and Shippers Find Common Ground', Special Supplement to Chemical Week, September 27, 1995).

A number of tools exist specifically to address transportation risk (see information sources section). For instance, Transport Risk Assessment Tool (Transport RISKAT) has been developed to estimate the risks to people arising from potential releases of toxic and

Box 7.4 continued

A range of typical conditions and emergency response factors for a wide range of commodities is used in the analysis.

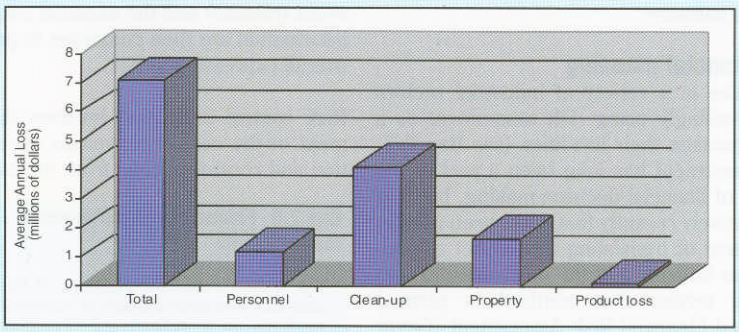
Property Damage Impacts

Historical data are used to determine the distribution of property losses. Consequence modelling is used to estimate the extent of possible damage. Data from the Federal Highway Administration are used to arrive at the cost distribution. This data set contains over 5,000 accidents and nearly 1,000 releases to the environment.

Result of the Transportation Risk Assessment

Figure 7.6 shows the estimated annual average losses of the tank truck fleet. The predicted annual losses are about US$ 7 million per year. The greatest contributor to the estimated losses is environmental clean up which accounts for 60 per cent of the total losses. The results of the case study are currently being used by the transport company to study risk control alternatives, especially those that impact the smaller losses which happen relatively frequently. The company uses a combination of self and external insurance, transferring the risk of high magnitude losses. Insurance coverage is now being reviewed. As a result of this study the transport company is planning to vigorously expand in the hazardous materials transport market.

Figure 7.6: Average annual expected loss profile – tank truck fleet



Source: Meyers and Mahini, 1996

flammable substances during transport by road and rail. On a selected transport route, the risks to both the on- and off-route populations are estimated and the total risk is expressed in terms of "route societal risk". Societal risk is used because of the potential for harming a significant number of people in a single incident. Transport RISKAT enables comparisons to be made between the risks arising from different routes and different modes of transport, or from different sections of the same route. In addition, it can be used to identify risk "hot spots" along a particular route and the lowest option from a number of alternatives (Leeming, Gadd and Riley, 1996).

In general, the transportation of dangerous substances is subject to national and international regulations and control but even when enforced, a residual risk still remains, thus

creating a need for the possible application of QRA, despite the uncertainties involved in the process.

Comprehensive coverage of transport risk assessment methods is available in the American Institute of Chemical Engineers publication 'Guidelines for Chemical Transportation Risk Analysis'. The book describes the general methodology and looks at frequency analysis and accident rates and presents specific case studies for selected modes of transport such as pipelines, rail, road transport, barges and ocean-going vessels. Guidance is also provided on the calculation of risk estimates (individual, societal, etc.) and available software.

7.4 Financial planning

Companies in a variety of industrial sectors are increasingly using risk assessment as a tool to manage their liabilities (see Box 7.5). Risk assessment has long been a part of the process of financial decision-making, but it is only relatively recently that it has been used in the context of translating the impact of accidents (on humans and the environment) into financial terms. One method is termed Integrated Financial Risk Assessment (Geyer and Morris, 1996). It facilitates the comparison of various consequences and incorporates the total cost of incidents to obtain a true financial liability of the undesired event. The approach enables sound financial decisions to be made on the extent of risk reduction measures by ensuring the cost of risk reduction is relative to potential liability costs. Being able to express the impacts of an accident in financial terms also provides opportunities for the transfer of risks to insurance companies.

When determining the costs of an accidental release to the environment it is important to consider all the potential loss areas, such as human fatalities and injuries, environmental damage, regulatory fines and clean-up costs, lost production, asset loss, loss of market share, product boycott and negative company image.

The total process of Integrated Financial Risk Assessment consists of three stages: developing the risk profile, risk control evaluation, and risk finance options.

The first stage involves the steps of the risk assessment methodology described in Chapter 4 with the additional step of the estimation of cost of each predicted accident scenario. The estimated frequency of the event (release) and the financial consequence information are then combined to produce an overall expression of risk.

Risk control evaluation involves the assessment of the cost effectiveness of risk reduction and control options from the assessment

Box 7.5 Financial risk assessment - ERM and four elements

The approach uses the principles of financial risk analysis to measure impacts of hazardous material accidents on a uniform and consistent financial basis. This enables comparison of various consequences and incorporates the total cost of accidents to obtain a true estimate of the financial liability from the undesired events. This financial basis lends itself to the cost-benefit analysis of business ventures, alternative approaches, and potential risk mitigation options. The impacts of accidental releases or spills are measured in terms of acute fatalities and injuries, but also associated environmental and longer-term impacts, property damage, product loss, and business impact. These are combined to arrive at an estimate of the overall financial risk (or risk profile) for the business. Risk control options are then reviewed and may be re-evaluated. For those risks that remain, finance and risk strategies are devised and evaluated. This approach was applied to the transportation risk assessment case study in Box 7.4.

of the impact of risk reduction measures and the estimation of the cost of their implementation. The risk will often need to be reduced to a level that meets the company criteria, that is the maximum level of risk it is prepared to tolerate.

The final stage considers the options available to finance the residual risk once risk reduction/control measures have been implemented. This could be simply the acceptance of the risk and no further action is taken, self insurance or transference of the risk to a third party such as an insurance company or an external contractor in the case of a manufacturing process.

The key benefit of the technique is that it measures all liabilities on a common basis thus enabling the direct comparison of all risk scenarios and facilitating effective decision making.

7.5 Product risk assessment

Many companies conduct risk assessments on their products or components of their products. This is a well established procedure for food, medical, pharmaceutical and chemical products, necessary to reduce the risks to a minimum of harm occurring to an individual through consumption of a particular food or drug or from using a chemical such as a pesticide.

There are established programmes in place under EU legislation to address the risks from new and existing chemicals. Companies involved in such programmes include BASF and GlaxoWellcome.

Due to increasing public concern over the use of toxic substances in products and possible human exposure, manufacturers also address the risks to humans and the environment posed by their products incorporating a life-cycle approach, particularly emphasising the use and disposal phases. Risk reduction programmes and the removal of certain toxic substances from consumer products has also been initiated and controlled by regulators. Typical examples include the reduction in the use of lead in paint and responses to concerns over the presence of phthalate plasticisers in food, particularly baby milk preparations.

In order to minimise potential damage to the environment from its products and activities, Procter and Gamble conducts ecological risk assessments on its products. Information is provided in the case study (Box 7.6).

7.6 Risk minimisation and reduction measures

If the results of an ERA for a given scenario suggest that the risks are too high, a risk management decision needs to be made. This will result in the implementation of some form of risk reduction strategy such as a reduction in inventory of certain materials at a site, the re-design or modification of a particular section of a plant, the re-routing of a tank truck or selection of a different mode of transport.

It may not be necessary to carry out a comprehensive risk assessment. A hazard assessment may be sufficient involving hazard identification and suitable measures to reduce the risk of the substance being released to an absolute minimum through appropriate design and the use of Hazop, strict safety procedures and effective employee training.

An example of a risk minimisation and reduction strategy is provided by GlaxoWellcome (see Box 7.7).

Box 7.6 Procter and Gamble

Procter and Gamble (P&G) is a world-wide company producing consumer goods with on-the-ground operations in 58 countries. Procter and Gamble has 130 manufacturing sites world wide. Its main product categories include laundry and cleaning materials; health and beauty care products; paper products; and food and beverages.

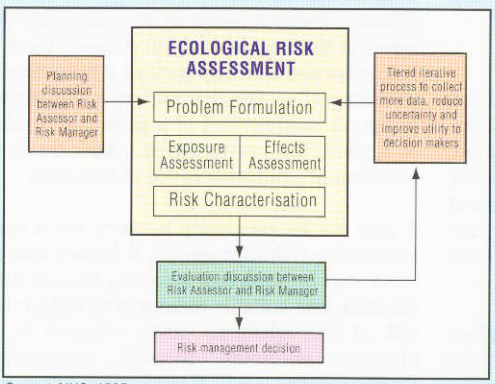
Procter and Gamble's first priority is "to ensure the safety of its products, packages and operations" (Procter and Gamble, 1994). Three science-based management tools are used to accomplish this - Risk assessment, Total Quality Management and Life-Cycle Assessment.

Within Procter and Gamble, a tiered iterative approach to risk assessment is taken, advocated by both the US EPA and the European Commission.

Risk assessment is used in P&G in a number of ways. The one examined in this case study is ecological risk assessment of chemicals and products. In P&G, risk assessment of both human health and ecological risks are examined in an integrated approach.

Procter and Gamble's approach to ecological risk assessment is based on the US EPA framework for ecological risk assessment (US EPA, 1992) as refined by the American Industrial Health Association (AIHC) (AIHC, 1995). Figure 7.7 shows the US EPA framework with AIHC refinements. It can be seen that this framework recognises the importance of discussions between the risk assessor and the manager to ensure that the assessment satisfies the manager's needs. The problem formulation examines the available environmental data that are used to determine goals for risk assessment; to identify needs for appropriate exposure and effects end-points and models; to determine decision-making criteria including the amount of data required, their acceptability and variability; and to recognise and incorporate regulatory, societal or corporate policy issues (Pittinger et al., 1996).

Figure 7.7: A framework for ecological risk assessment



Source: AIHC, 1995

Figure 7.8 shows P&G's approach to effects and exposure analysis in risk assessment (Feitjel and Lally, 1995).

Exposure and effects analysis provides the data required to produce PECs and PNECs which are then used in the risk characterisation process. Exposure assessment examines the spatial and temporal distribution of the product or chemical, including the magnitude, intensity, duration and routes of exposure to species and ecological communities across the geographic range of the product's market. The use of exposure models, such as the Geographical-Referenced Exposure Assessment Tool for European Rivers (GREAT-ER) developed by ECETOC (ECETOC, 1995a) which allows a mathematical analysis of the source and quality of water at any point in the major European river systems, is expected to provide valuable information on water quality and chemical exposure.

Effects assessment is also focused by the geographic range of the product's market. The different approaches to Assessment Factors taken by regulators world wide adds complexity to the process. The effects assessment is a tiered process. It involves the use of quantitative structure-activity relationships at the initial tiers whilst laboratory toxicity tests using sensitive representative species, microcosms and mesocosms are performed in higher tiers. Biological field surveys may be performed for high volume chemicals or where particular regulatory concerns exist.

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Box 7.6 continued

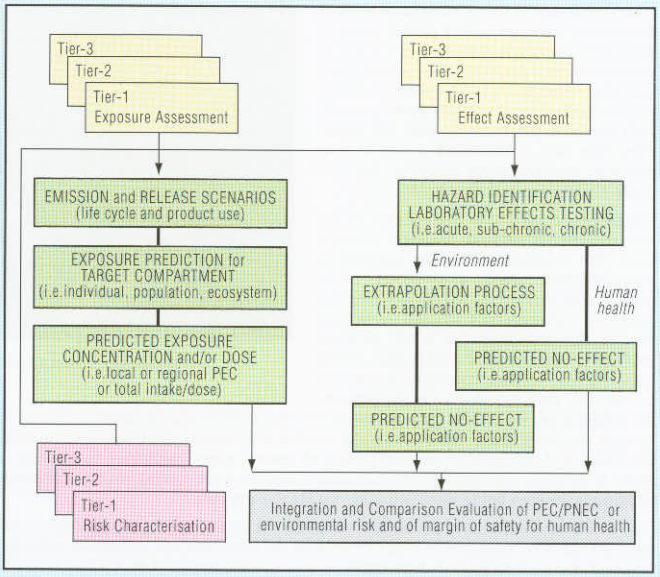


Figure 7.8: Illustration of components of environmental and human exposure and effect assessment

Source: Feitjel and Lally, 1995

Effects assessment is also focused by the geographic range of the product's market. The different approaches to Assessment Factors taken by regulators world wide adds complexity to the process. The effects assessment is a tiered process. It involves the use of quantitative structure-activity relationships at the initial tiers whilst laboratory toxicity tests using sensitive representative species, microcosms and mesocosms are performed in higher tiers. Biological field surveys may be performed for high volume chemicals or where particular regulatory concerns exist.

Procter and Gamble has produced its own toxicity data banks and uses commercially available bibliographic services such as Current Contents. Commercially available exposure software such as that produced by RIVM and the US EPA is used. Basic and applied research is carried out within the company on effects and exposure, and P&G employees actively publish in the scientific press.

Risk characterisation integrates the exposure and effect analysis to characterise the likelihood, significance and conditions of ecological risk in terms of the assessment end-points chosen.

An example of an ecological risk assessment carried out by P&G on a surfactant is given in a paper by Versteeg et al., 1992.

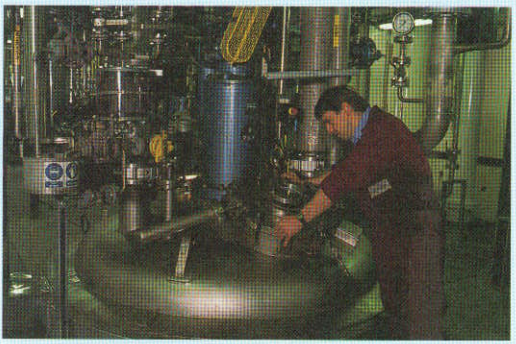
Risk decisions are taken on the basis of the risk characterisation and the social, economic, legal and communication issues. Comparative risk analysis using other products or chemicals as reference points is used to aid decision-making.

Procter and Gamble believes that the value of a risk-based approach is that exposure can be compared with effect, which produces much more realistic and sensitive results than a hazard-based approach. While there are legitimate applications for hazard assessment, risk-based approaches provide the most accurate and realistic means for evaluating the potential for harm to the environment. Risk assessment must be coupled with sound risk management techniques in order to answer the broad societal questions of "How safe is safe?" and "How clean is clean?" The answers to these questions are best determined through discussion with all potential stakeholders in society, and through considerations of costs, feasibility, and social acceptability, as well as safety. The policy-driven science necessary to answer policy makers' questions takes a number of years to be carried out, whilst in the meantime, difficult decisions still need to be taken.

Once risk assessment becomes a regulatory routine, it will be inevitable that the process will be simplified and more generic. This could lead to difficulties from an industrial viewpoint if, in pursuit of simplicity, conservative assumptions are introduced into assessments.

Box 7.7 Case Study - GlaxoWellcome

GlaxoWellcome is the world's largest pharmaceutical company with a global share of the world prescription medicine market of approximately 5 per cent. The company has operating companies in 70 countries (manufacturing and research and development) with products marketed in over 120 countries. The site at Ulverston is one of four primary manufacturing sites in the UK (bulk pharmaceutical chemicals).



Cephalosporin plant, GlaxoWellcome, Ulverston, Cumbria, UK

Operations at Ulverston concentrate on the manufacture of several products derived from the production of the antibiotics cephalosporin and grisiofulvin. The cephalosporin process, which includes the stages of raw materials storage, fermentation of the cephalosporin broth, product extraction and chemical conversion, sterile finishing and solvent recovery, is covered by one IPC authorisation as is the process for grisiofulvin. Other IPC authorisations are required for the incineration plant, the combustion plant and the solvent recovery plant. GlaxoWellcome carries out various environmental initiatives including monitoring of fence-line and ambient air concentrations of several gases, sediment monitoring, and dispersion modelling in order to identify any impacts the company's activities are having on the environment. All new processes and existing processes being modified undergo Hazop analysis.

One of the requirements of the authorisation for the cephalosporin process was to ensure that storage tanks used were adequately contained in order to prevent the unauthorised release of substances to the environment. This involved the surveying of storage tanks on-site and the application of a risk assessment procedure. Information was collected on the provision of containment for each storage tank and was used in a rating system of risk assessment based on five criteria: the nature of the substance, storage tank capacity, secondary containment provided, spillage containment, and bund drainage. Scoring factors were then assigned to each option within the five main risk criteria to rank the degree of risk (see Table 7.1). The total risk score for each tank was obtained

Table 7.1: Scoring factors

RISK FACTOR	RISK FACTOR DESCRIPTION	RANKING FACTOR	SCORING FACTOR
A	Nature of substance	Flammable/toxic Oil Corrosive Other	10 5 3 1
B	Storage tank capacity	Greater than 10,000 1 1,000 to 10,000 1 Less than 1,000 1	10 5 1
C	Secondary containment provided	No bund Bund provided but less than 100% Bund provided to 100%	10 5 1
D	Spillage containment	Spillages pass directly to stone chippings Spillages pass directly to surface drainage system Spillage contained in an interceptor pit Spillage contained in bund	10 5 3 1
E	Bund drainage	Drainage to stone chippings Drainage to surface water drainage system Drainage to process waste effluent system Discharge to interceptor pit	10 5 3 1

Source: Glaxo Operations HMIP Report, 1995

Continued opposite

Box 7.7 continued

by multiplying the scoring factors for the five main criteria. Each tank was then placed in a risk category according to its respective score (see Table 7.2). The objective of the procedure was to prioritise those tanks with containment facilities in need of improvement in order to reduce the risk of release to an acceptable level. Any improvements or upgrades will be made by the application of LATNEEC - balancing the cost of upgrade with the potential environmental harm so that damaging releases are reduced without imposing excessive cost.

Table 7.2: Ranking categories for storage tanks

OVERALL RISK RATING	RANKING SCORE
Very high	50,000-100,000
High	15,000-50,000
Medium	5,000-15,000
Low/Medium	1,000-5,000
Low	<1,000

Source: Glaxo Operations HMIP Report, 1995

Certain GlaxoWellcome products or intermediate products are classified as new substances under the EC Directive EEC/93/67 regarding new substances. This requires a full health and environmental risk assessment to be carried out according to the guidelines described in the EC technical guidance document on the risk assessment of notified new substances. A brief summary of the results of a risk assessment carried out by GlaxoWellcome for a new substance (which cannot be named) is provided here.

Risk Evaluation of New Substances

Substance trade name:	MGH
Generic name:	A Hydroxy cycloalkyl ether
Emission pattern/points of release:	Site limited use
Human health (toxicity):	Human exposure will only occur in the work place, the substance will not enter the public domain.
Effects assessment	
Acute toxicity:	oral LD50 >2,000 mg/kg dermal LD50 >2,000 mg/kg
Irritation:	not classed as skin irritant
Corrosivity:	not applicable
Sensitisation:	negative maximisation
Repeated dose toxicity:	Rat oral 28-day study, NOEL 15 mg/kg/d at 150 mg/kg/d, one death and also marked inflammatory changes in stomach.
Mutagenicity:	Ames negative, IVC negative
Carcinogenicity:	not tested
Toxicity for reproduction:	not tested
Explosivity:	negative
Flammability:	Flammability of solids: negative, self-ignition temperature: 302°C
Oxidising potential:	negative
Environmental exposure:	A site-specific assessment establishes that there are no releases to the aquatic compartment because all process residues and waste streams, including aqueous wastes, are collected and subsequently incinerated. As there are no releases, the substance is considered to be of no immediate concern for the environment.
Environmental effects assessment	
Acute toxicity to fish:	96 hr LC50 6.7 mg/l
Acute toxicity to Daphnia:	48 hr EC50 15 mg/l
Algal growth inhibition:	72 hr EbC 50 2.4 mg/l, 24-72 hrs ErC 50 3.7 mg/l
Aquatic compartment:	NOEL 1.25 mg/l

Conclusions: The substance is of no immediate concern for man and the environment, at the current levels of supply.

On a more general scale, non site-specific risk reduction programmes for chemicals are extremely important as outlined in Chapter 19 of Agenda 21. The ongoing collaborative work of major international organisations such as the EU, OECD, IPCS and FAO is at the core of the quest for comprehensive information on chemicals and the development of effective management strategies for their "safe" use. In Europe, an additional key player is the European Centre for Ecotoxicology and Toxicology of Chemicals (ECETOC), which serves as an important collaborative, linking body between industry and regulators. It is financed by over 50 major industrial companies and was established to "provide a scientific forum in which the extensive specialist expertise of the European Chemical Industry could be harnessed to research, review, assess and publish studies on the ecotoxicology and toxicology of chemicals. The main objective of these activities is to identify, evaluate and minimise any potentially adverse effects on health or the environment which might arise from the manufacture and use of chemicals." (ECETOC, 1996). ECETOC played a leading role in the development of the TGD with the European Commission.

7.7 Risk assessment techniques developed for specific industrial application

In addition to the risk assessment techniques outlined for industrial processing sites, techniques used in certain other industrial activities are also very important. These include activities such as the assessment of contaminated land, the risk assessment of waste disposal by landfill, risk assessment for offshore installations, risk assessment of nuclear energy installations and risk assessment in land-use planning.

7.7.1 Risk assessment of offshore installations

Quantitative Risk Assessment (QRA) is now an established and integral component of safety management strategies and practices for offshore gas and petrochemical installations in the North Sea and all over the world. A comprehensive methodology has developed in response to major accidents on two North Sea platforms in the 1980s - the Norwegian 'Alexander L Kielland' and the UK sector 'Piper Alpha'. The risk assessment is presented as a safety case under national regulation and requires "that all potential major accident hazards be identified, the risk of these hazards evaluated and that suitable measures be implemented to reduce the risk to people to a level regarded as ALARP" (Health and Safety Executive, 1992). Such hazards include hydrocarbon releases, vessel impacts, structure failure, fires, etc. The risk assessment methodology, however, is designed to protect the workforce. It does not need to take into account public safety as the public is not exposed and ecological risks are not considered. Potential environmental impacts are considered though as part of an impact assessment.

The QRA methodology therefore cannot be considered environmental risk assessment, as its scope does not extend beyond the boundaries of the engineering structure. The methodology includes all the stages of onshore QRA but obviously the scenarios for release assessment, event tree modelling and consequence assessment involve different considerations and priorities.

7.7.2 Risk assessment of nuclear installations

Most experiences of the application of quantitative risk assessment techniques for employee

protection and public safety are to be found in the nuclear industry due to the enormous potential of the associated activities to cause a major catastrophe. This industrial sector was the first to develop and use predictive probabilistic QRA as an aid to decision-making in the areas of reliability and safety. The necessity was borne out of the obvious complete lack of information and experience on nuclear installation operability. The techniques are well established and much of the methodology is now used in other industrial sectors such as the process industries, as described in this chapter.

Detailed coverage of the techniques used in the nuclear industry is not possible in this book. The reader is directed to information on this very specialised area of risk assessment in the information sources.

7.7.3 Land-use planning - risk assessment for public safety in the vicinity of hazardous industrial installations

Another application of risk assessment methodology is in the consideration of the risks posed by industrial installations in the planning and development of adjacent areas for residential or commercial use.

The UK Health and Safety Executive (HSE) have developed a methodological tool known as RISKAT to comprehensively assess and quantify the risks and acquire the failure rate data for major toxic and flammable installations. The assessment of risk is then used by the HSE as a basis for advice given to Local Planning Authorities. (See Box 7.8).

The information provided by RISKAT for a given hazardous site can be used in conjunction with risk criteria produced by the HSE, which considers both individual and societal risk, as a basis for formulating advice on planning applications for new developments in the vicinity of an existing major hazard. The risk criteria are described in detail in an HSE Discussion Document (Health and Safety Executive, 1989). An example is that the HSE suggests that, for individual risk, a level below 10^{-6}/yr frequency of receiving at least the specified dangerous dose, as calculated via RISKAT, would not be "significant" for housing for the general public. The HSE has not proposed numerical criteria for the judgement of societal risk, as there are difficulties in judging the significance of an increment to an existing societal risk. For scenarios where a low risk exists for individuals, but a potentially high societal risk exists (e.g., supermarkets), the risk is estimated by calculating the individual risk to a person in the location, judging the significance of the proposal in comparison with a number of houses and applying the rules for housing development risk.

The RISKAT approach principally produces a numerical estimate of the risk, which is then compared with a criteria window of acceptable risk. The HSE is well aware of the importance of risk perception and stresses that the criteria outlined for the formulation of advice on land-use planning in the vicinity of existing major hazards should be regarded as limited to that purpose.

A well-defined and standardised methodology has also been developed in The Netherlands to address the risks associated with the siting of hazardous installations and the development of the surrounding area. The methodology is presented as three reports; methods for the determination of probabilities (the 'Red Book'), methods for the calculation of physical effects (the 'Yellow Book'), and methods for

Box 7.8 RISKAT

Nussey et al. (1993) describes the principles of the tool RISKAT and its application to local planning decision-making. The procedure can be broken down into a number of steps:

- Analysis of the major hazard plant, its control and safety systems, and operational procedures so that a representative number of hypothetical releases with the potential to affect neighbouring populations can be identified.
- For each hypothetical release the chance that such an event will occur in a given time period is determined either from historical failure statistics or by synthesis from basic component failure rate data using well-established techniques such as fault tree analysis.
- For each release case, estimates are made of the rate of release of hazardous material and the duration of the release.
- For toxic, and certain types of flammable release, calculations are made of the atmospheric dispersion of the hazardous material in various weather conditions. For flammable releases, immediate ignition and delayed ignition scenarios are considered.
- These dispersion, explosion and flame calculations enable the spatial and temporal variations in the effects of the hazard to be mapped out.

In summary, RISKAT calculates the chance of a hypothetical individual at a particular location receiving at least a specified criterion dose of the toxic material, a specified dose of thermal radiation or a specified level of over-pressure. The dose calculations can then be converted to probabilities and to provide expressions of both individual and societal risk. Uncertainties and sensitivities associated with the RISKAT procedure are described in full in the paper (Nussey et al., 1993).

the determination of possible damage (the 'Green Book'). The results of the risk assessments carried out provide information for risk reduction measures and zoning policies around hazardous installations.

7.7.4 Contaminated land

Risk assessment is used to prioritise sites and to set action and clean-up criteria for contaminated land. The objective is to ensure that land is made safe for re-use without presenting a long-term environmental liability.

The assessment of contaminated land is a complex business due to the complex nature of soil itself and the myriad of possible contaminants from various industrial and waste disposal processes that can be distributed within it. For this reason and many others, respective to different countries such as land use, legal and administrative systems, land ownership and industrial histories (Visser, 1995), countries have pursued their own policies in regard to the contaminated land problem. Although differences exist, most countries use a criteria-based approach and common, key issues are addressed in each policy formulation as suggested by Visser (1995):

- Which risk level is acceptable or tolerable and which level of human and environmental protection is desirable or reasonably achievable?
- Is it preferable to use generic clean-up criteria or a strictly site-specific approach?
- Should generic soil quality criteria and clean-up criteria be related to intended land use?

Countries implementing this criteria-based approach can be divided into four groups:

Group 1: Using single soil quality criteria to act as a trigger value for action and also a target value for remediation, and remediation targets are based on "multifunctionality", i.e., the land is suitable for any intended use, e.g., Denmark.

Group 2: As for Group 1, but remediation targets are based on intended use, for example, residential housing or a sealed car park, e.g., UK and Sweden.

Group 3: Using separate trigger and target values (target values are generally more stringent than those for Groups 1 and 2) and multifunctional use, e.g., Netherlands.
Group 4: As for Group 3, with remediation based on specific intended use, e.g., France and Belgium.

In addition to the above complexities, the question of the use of generic criteria for all sites as opposed to site-specific assessment has to be addressed. As in all environmental risk assessment, exposure is extremely important. Concentrations of contaminants at one site might pose a risk to a given receptor but not at another because the characteristics of the specific scenario mean that exposure pathways do not exist. Applying general criteria could result in remediation target levels being too stringent and conservative, or too low for adequate protection.

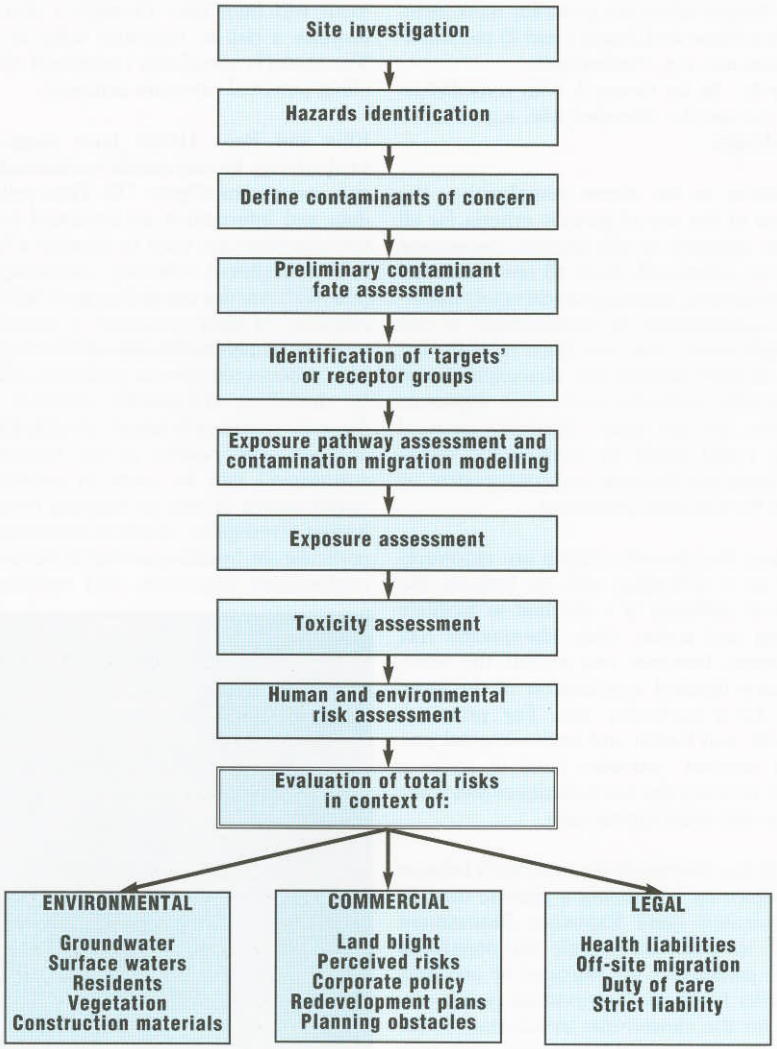
It is clear that generic criteria are extremely useful as a screening tool, to indicate the degree of pollution at a site and to facilitate planning and action. Only site-specific risk assessment, however, can provide the often, necessary detailed examination of the risks posed by a particular site. For practical, economic and health and environmental protection reasons, countries need to strike a balance between the two techniques and apply them as and when appropriate.

The UK has addressed the need for a balance to a degree by developing a generic model - Contaminated Land Exposure Assessment (CLEA) in which site-specific and population specific parameters are replaced by probability density functions representing typical scenarios for the chosen use (residential, recreational or commercial/industrial). Soil type and other relevant parameters can also be specified. A tentative guideline value as an input will then pass through a scenario to produce a risk or exposure value as output. The model is specifically concerned with modelling potential exposure pathways.

Ellis and Rees (1995) have suggested a methodology for site-specific contaminated land risk assessment (Figure 7.9). First, assimilated data and information accumulated from site investigations are used to conduct a baseline risk assessment whereby contaminants of potential concern are determined and the significance of their presence is quantified in terms of human health and environmental risk. This process requires an evaluation of the site by modelling site-specific exposure routes through source-path-target identification and analysis. The results of the baseline risk assessment can be used to establish the requirements (if any) for ongoing monitoring, further investigative works or remediation on a particular site by taking account of human health, contaminant migration, and environmental

Photo: Robert Brook, Environmental Images

Figure 7.9: Key stages in a risk assessment



Source: Ellis and Rees, 1995

impacts. The accuracy of the risk assessment depends upon a thorough understanding of the fate and effects of contaminants under site-specific conditions and use. If remediation is warranted, the risk assessment procedure can be further used to develop site-specific clean-up goals by determining "how clean is clean enough"?

Qualitative risk assessment can be used as a method of prioritising contaminated sites for remediation. This is illustrated by a case study of the development of a risk assessment methodology for British Gas sites in the UK (see Box 7.9).

European co-operation

Two complimentary Europe-wide programmes on contaminated land have been set up under the Environment and Climate Research and Technological Development Programme funded by the European Commission DGXII.

CARACAS (Concerted Action on Risk Assessment for Contaminated Sites in the European Union) is a project carried out by all EU States and Norway and Switzerland. The prime objectives are:

- To identify, compile, assess and review all relevant RTD projects and scientific approaches for risk assessment developed in the Member States of the EU;
- To propose scientific priorities for future RTD Programmes and Projects in the EU and Member States;
- To elaborate guidelines and recommendations for assessing risks from contaminated sites.

The programme "focuses on the co-ordination of research in order to achieve a secured state of scientific knowledge on environmental risks from contaminated sites. The results will support the development of consistent risk assessment methodologies, and will strengthen the collaboration between the EU Member States" (Kasamas, 1996). The programme is sub-divided into seven topic groups including human toxicology, ecological risk assessment, models, and methods for risk assessment.

Box 7.9 British Gas - Contaminated Land Case Study

British Gas is a company with large land-holdings, a number of which are considered to be contaminated because of the history of industrial activities carried out at such sites.

In response to increasingly more stringent environmental standards and the obvious need to reduce the risk of legal non-compliance, British Gas has introduced a programme to prioritise its potentially contaminated sites for remediation and risk management. The programme has been described by Walker et al., 1994.

The first step was the introduction of a contaminated land survey involving the collection of relevant data on each site, such as:

i) Recorded evidence of contamination and incidents of cross-boundary migration of pollutants;
ii) Permeability of the underlying geology;
iii) Proximity to water (rivers and abstraction wells).

On this basis sites were categorised as high, medium or low priority.

Those sites selected as high priority were subjected to a desk study as a second step in the process, in order to confirm the priority ranking. This involved the collection of information such as the location of old process areas and the history of site use. Those sites confirmed as high priority then underwent a boundary survey involving sample taking, trial pits, etc.

This process of qualitative risk assessment/prioritisation of sites, leads to a short list of sites which can then be assessed in a semi-quantitative or quantitative manner in order to determine the risks to man and the environment and the necessary clean-up/remediation criteria.

NICOLE (Network for Industrially COntaminated Land in Europe) is "industry led and will provide a forum for the dissemination and exchange of scientific and technical knowledge and ideas relating to all aspects of industrially contaminated land" (CARACAS, 1996). The network includes industrial companies, research organisations, trade associations, representatives of national and EU research programmes and vendors of remediation advice and processes. NICOLE is sub-divided into four working groups, one of which is contaminant behaviour and risk assessment.

These EU programmes on contaminated land should pool together the collective information from Member States' individual research programmes and assist in the harmonisation of approaches towards risk assessment and management of contaminated land.

7.7.5 Waste management

Concern over the possible human health effects resulting from exposure to hazardous substances disposed to landfill sites, has driven the need for the application of risk assessment to such scenarios. Particularly of concern is the fact that existing hazardous waste sites may not have been designed with sufficiently preventative considerations for human health or the environment in mind.

The requirement, therefore, is to carry out risk assessments on a site-specific basis with the objective of determining the risks to which the human population and the environment are exposed. It is also possible and desirable to include risk assessment in the design process and planning stage of future disposal sites.

Petts and Edulgee (1994), suggest a methodology to assess the risks posed by a hazardous waste disposal site. This includes:

Hazard Identification which involves the identification of i) the chemicals to be accepted and handled on site, ii) the processes on site which may result in releases to the environment, iii) the sources and identification of these releases, and iv) indicators for risk assessment.

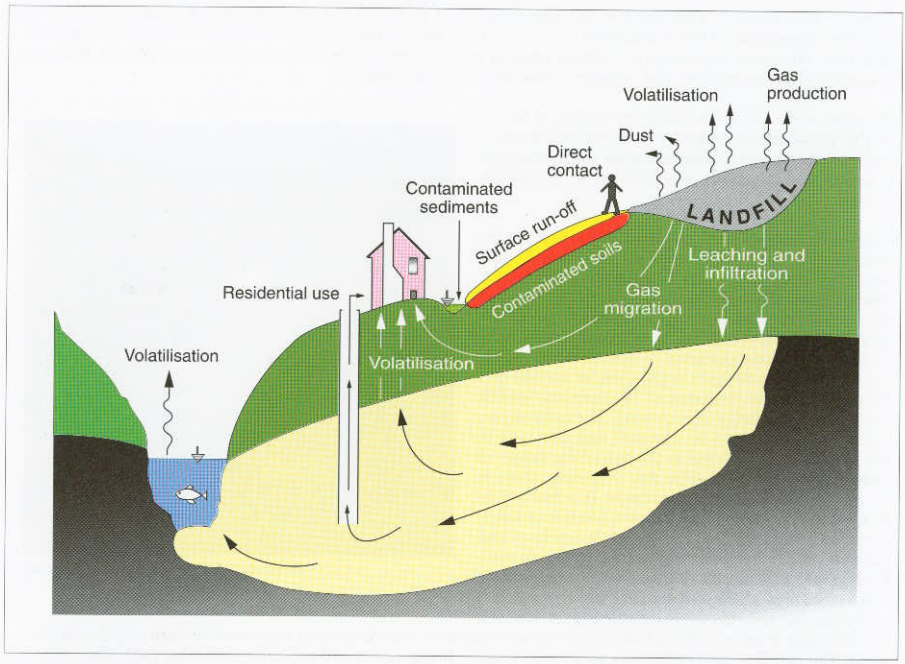
Hazard Analysis which involves i) estimation of emission or discharge rates of the indicator chemicals, ii) characterisation of the general physical features of the site, iii) characterisation of the potentially exposed populations and activity patterns, iv) identification of exposure pathways and modelling of fate and transport processes in released media, and v) calculation of doses in the relevant media, at the receptor locations, and calculation of intake. (Figure 7.10 illustrates a conceptual model of landfill exposure sources and environmental pathways).

Risk Estimation which involves dose-response assessment and the characterisation of the risk.

Risk Evaluation which involves the comparison of the estimated risks associated with a particular site with defined criteria to assess the tolerability, or acceptability of the risk.

Carrying out a full risk assessment of a hazardous waste disposal site is both time consuming and expensive and involves a significant degree of uncertainty, particularly in dose-response assessment and hazard identification, along with the lack of relevant data. However, it remains the most effective tool for the evaluation and management of the risks to human health and the environment arising from waste landfill sites. Box 7.10 is an example of the application of risk assessment to landfill design.

Figure 7.10: Conceptual model of landfill exposure sources and environmental pathways



Source: Petts and Eduljee, 1994

7.8 The relationship between ERA, environmental management systems and life cycle assessment

The application of certain environmental management tools such as management systems, waste minimisation strategies and LCA can be considered as risk management/reduction initiatives. In this context the risk to be managed or reduced may have a more general definition or description. For example the adoption of an effective environmental management system will result in a reduced risk of polluting substances being released to the environment, or a reduced risk of non-compliance with legislation and therefore a reduced risk of prosecution and negative publicity. It is important to recognise the distinction between the use of risk terminology in such a qualitative manner and the use of risk terminology in the specific, usually quantitative process of risk assessment. It is possible that some risk analysts may object to the inclusion of such a general,

Box 7.10 Landfill Design - An Example of Risk Assessment

Hazard assessment. This involves the identification of particular events/occurrences which may have an adverse consequence. Different hazards may arise at the various stages of the life cycle of the site (design, construction, operation, post-closure). Typical hazards include penetration of liner containment system, type and thickness of geomembrane, and collapse of leachate collection system.

Release assessment in which the modes of failure of the containment system will be identified using such information as data on materials and operational practices and assigning probabilities to the failure events.

Photo: Robert Brook, Environmental Images

Exposure assessment can then be carried out by determining the release rate of the leachate and inputting these data into a suitable groundwater model which will calculate the size of the pollution plume and the subsequent contamination concentrations at potential receptors. Theoretically, an assessment of the consequences of the receptor being exposed to the substance could then be carried out. However, for the purposes of containment landfill design this is probably unnecessary. The most important risk that needs to be assessed and managed in the landfill design process is the risk of the failure of containment. If there is little risk of release, there is no need to assess the consequences. It is impossible to achieve zero leakage from a site, but it is possible to reduce the rate of leakage to a minimum level that poses no risk to humans or the environment. In practical terms, this means carrying out a comparative risk assessment of selected containment designs for a particular site and effectively quantifying the risk of a site releasing a volume of leachate which exceeds specified criteria derived to protect humans and the environment. The US EPA have defined an acceptable leakage being >2.2 x 10-6 m3/s/ha (190 l/ha/day). Therefore, if there is a risk of the site exceeding this release rate, risk reduction measures need to be implemented, such as design modification, via the process of cost-benefit analysis.

Source: McKendry, 1995

qualitative and non-scientific interpretation of risk assessment and management in a book on environmental risk assessment, but it is one way in which risk assessment and management is perceived and interpreted and, therefore, merits a brief mention.

It may be possible to incorporate ERA principles into the formal Life Cycle Assessment process within the impact assessment stage of LCA and actually quantify the risks imposed by a product or process on the environment rather than the impact or burden. See Box 7.11.

Box 7.11 ERA and LCA

Although Life Cycle Assessment (LCA) and ERA are not yet integrated, there is likely to be scope for progress in this field in the future;

"The Impact Assessment stage of LCA in conjunction with the CML Problem Orientated Impact Assessment (POIA) methodology perhaps offers the best way of progressing in terms of integration. Within the context of POIA, the individual categories e.g. Global Warming and Ozone Depletion represent risks of environmental impact. Once the impacts associated with a product or process have been fully quantified, then any improvements made represent a reduced risk of environmental damage under the Impact Assessment categories. With the development of suitable metrics this may become possible.

The reason that POIA can be related to risk is simply that the significance of the related categories cannot yet be determined with 100 percent accuracy. There is, therefore, an element of uncertainty that applies when using this methodology. Also, a lower environmental impact associated with a product e.g. by 10 percent does represent a reduced risk of the environment being harmed. The next step in research would be to develop a methodology for applying metrics to correlate risk with POIA methodologies and the valuation step of LCA." (Francis, 1997).