Evaluating evidence on environmental health risks

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The assessment of adverse health effects from environmental hazards involves integration of evidence from a variety of sources, including experimental studies, both in animals and humans, in vitro studies, and epidemiological research. It requires an understanding of the sources, nature and levels of exposure to which humans may be subjected, the nature of the health outcome or toxic effect and the mechanisms by which this might occur, the relationship between dose and response, and a knowledge of the variability and susceptibility of potentially exposed populations. After outlining the process of risk assessment, this paper gives an overview of the most relevant human study methods used to investigate environment and health effects and discusses issues such as confounding and effect modification, that are important to consider when interpreting the results from such studies. Future challenges are outlined, such as increasing responsibility required by scientists to the sensitive issue of data protection and confidentiality, and also new opportunities, such as the increasing availability of computerized data, the incorporation of molecular epidemiological methods to aid the investigation of mechanistic pathways and gene–environment interactions, and the development and utilization of sophisticated statistical approaches.

Introduction

The assessment of risks of adverse health effects from environmental hazards is an area of increasing public interest and a topic of a large body of research within many different disciplines. It involves both knowledge of the source and nature of the environmental hazard and an understanding of the relationship of the exposure to the disease. The primary aims of environmental research vary but can include:

• the identification of causal relationships between environmental hazards and ill health in general populations and specific subgroups;
• the evaluation of changes in health with environmental changes;
• the provision of evidence for the setting of ‘acceptable’ standards for known environmental contaminants.

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In the broadest sense, the environment can be defined as external conditions influencing the development of people, animals or plants. A distinction is sometimes made between conditions from which individuals may have no or only partial control, for example, exposures encountered at work and to substances in ambient air, and those for which some element of personal choice exists, for example, ‘lifestyle’ factors such as smoking, eating a high fat diet and drinking alcohol. In the public mind, environmentally caused ill-health is often limited to the former situation, particularly hazards such as chemical exposures, and even more so if these are man-made. The research community, however, would generally consider a much broader set of factors, encompassing all that might impact on human health.

Different kinds of research contribute relevant information for assessment of environmental risks. Toxicological studies conducted on experimental animals can rigorously assess the biological response to a range of levels of exposure to a given substance or combination of substances. This has the advantage of specificity but presents a problem when extrapolating to humans, both because of species differences, and because high doses are often used in an experimental setting in animals, whereas typically humans will be exposed to low doses of pollutants. This can be partially overcome through clinical studies employing human volunteers, for example in food challenge studies or chamber studies of air pollutants. However, for ethical reasons these are generally limited to a narrow range of low exposure levels, which cause a mild and reversible health effect. Epidemiological approaches are thus the most widely used methods to test for potentially causal associations between environmental exposures and human health, both in the general population and for specific groups, defined for example, by occupation or location. Epidemiological studies are observational rather than experimental in design and thus have a number of limitations, especially where excess risks of any adverse health effect are small, as will be the case with most environmental exposures encountered today in the developed world. Despite these limitations, a large number of risks that have been recognized so far have been established using these methods. This paper gives an overview of the most common epidemiological methods and their advantages and limitations, and a discussion of the future challenges, for investigating the influence of environment on health. Toxicological methods are not addressed in this chapter, nor are hazards relating to personal choice.

**General considerations**

Assessment of the impact of a potential adverse health effect from an environmental pollutant is dependent on an understanding of several important issues, including:
• the hypothesized health outcome or toxic effect;
• the nature of the exposure;
• the relationship between dose and response;
• the variability and susceptibility of the potentially exposed population, for example regarding sub-groups of the population that might be at especial risk due either to the pattern and distribution of exposures in the population, or to non-environmental factors that might influence the risk of disease.

It is important to have a clear view of these aspects both when designing a study and when interpreting information from the published literature.

**Health outcomes**

There are various ways of classifying a toxic response to a hazard including:

• acute or chronic effects;
• reversible or irreversible effects;
• local or systemic effects;
• immediate or delayed effects.

An acute effect is generally caused by a high single exposure (occurring in seconds to hours) through accidental or gross over-exposure. The adverse condition develops quickly (within minutes or days) with obvious signs and symptoms, as was the case with chloracne, for example, following exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) as a consequence of the Seveso accident in northern Italy in July 1976. Unless the effect is fatal, damage is often reversible. Chronic effects usually occur after lower exposures repeated over months or years. The adverse condition may develop slowly over months or years with incremental damage and is often irreversible; an example in an occupational setting is lung fibrosis secondary to inhalation of dusts contaminated with silicone dioxide (silicosis). Local effects are those which occur at the site of first contact between the organism and pollutant whereas systemic effects occur only after a substance has been absorbed and distributed within the body. Immediate effects are those which develop shortly after exposure, whereas delayed effects develop some considerable time afterwards.

Early changes as a result of low-level exposure to an environmental pollutant will often remain undetected. Human cancer, for example, may take many years to become clinically apparent following an initial environmental exposure as part of a multistage process in carcinogenesis. Damage may be arrested if the exposure is removed, as happens after quitting
tobacco smoking where eventually risks approach that of non-smokers, especially at younger ages; however with other conditions, for example following asbestos exposure, there may be progressive deterioration.

In environmental epidemiology, concern usually centres on chronic effects from low-level exposures. Although at the individual level the excess relative risks associated with such exposures may be low, they may nonetheless be associated with a substantial burden of disease in the population, that is attributable risk is high. This is because of the ubiquity of exposure to many low-level pollutants, such that large numbers of people, or even whole populations, are exposed (toxicants in outdoor air would be an example). According to Rose’s ‘a large number of people exposed to a small risk may generate many more cases than a small number of people exposed to a high risk’.

**The nature of exposure**

Much of the information regarding the kinetics and metabolism of a substance, i.e. the uptake, distribution, accumulation, excretion and biotransformation, is determined through animal and in vitro studies. An understanding of these and their relevance to humans is essential in assessing the totality of evidence on the potential risk of an environmental pollutant on human health.

Knowledge of the toxicokinetics of a substance can also be very useful in determining the most appropriate measure of exposure to an environmental contaminant that should be used when assessing potential human risk. Human exposure represents a dynamic process from the source of the substance in question, through intake, uptake and transport to a critical organ. A lack of adequate exposure data has been reported to be the major limiting factor in preventing the identification of causal associations. Critical issues include:

- the type of the assessment method used. For example, has direct measurement been carried out using personal monitoring or biomarkers or have indirect methods been used, such as exposure modelling or the use of monitoring and time-activity data?
- the characteristic patterns of exposure over time. For example, for how long does exposure take place, how is it distributed (continuously or intermittently) and are there any critical time windows of exposure which are relevant to the health effect of interest?
- the metric used to represent exposure. Does this represent the relevant exposure patterns? For example is cumulative exposure assessed when some measure of short-term higher intermittent exposures would be more appropriate?
• inclusion of all key sources of exposure \textit{via} all possible routes and all media to give an aggregate exposure level. There is a tendency for many studies to investigate adverse health effects related to a restricted, though often substantial, potential source of exposure and to ignore other sources. For example, very few studies of occupational groups attempt to assess exposure from non-occupational sources. Figure 1 illustrates several of the potential sources, media and exposure routes through which exposure to environmental pesticides could occur.

The relationship between dose and response

The notion that there may be a causal relationship between an environmental exposure and a health effect is strengthened if a clear dose–response relationship can be shown. If the data are suitable, mathematical models can be developed to describe the dose–response relationship. In interpreting and using these models, a distinction is often drawn between situations in which the critical toxic effect is considered to have a threshold, \textit{i.e.} a level below which no adverse health effect would be expected to occur, \textit{e.g.} copper in water, or is considered to have no threshold, \textit{i.e.} it is assumed that as little as one molecule could theoretically cause an effect, \textit{e.g.} ionizing radiation or inorganic arsenic. The former is usually derived from consideration of known metabolic and mechanistic information and empirical observations in animals and humans of an absence of effect below certain doses (though statistical power to detect such effects at low doses is often limited). The latter has been traditionally applied to mutagenic and genotoxic substances. Although in practice studies may indicate an apparent threshold, the uncertainty owing to the inability to detect effects at very low levels of exposure both in animal and human studies limits the use of these models in assessing risk\textsuperscript{7}.

![Fig. 1 Factors to consider in an aggregate exposure assessment of a pesticide.](image)
Risk assessment methodology

In carrying out a risk assessment for an environmental hazard the totality of evidence is evaluated. The formal process of risk assessment shown in Figure 2 is now accepted internationally and incorporates:

- knowledge of the source of exposure to a substance, methods of release and transport of the substance and an assessment of exposure to human populations of concern;
- hazard identification, *i.e.* identification from animal and human studies of the potential adverse health effects and hazard characterization, in particular determination of the dose–response relationship;
- integration of hazard identification and characterization and human exposure assessment to give a risk characterization which evaluates the nature and severity of adverse effects at given levels of exposure.

The results of a risk assessment inform the setting of environmental standards in water, air, food and soil. The methods used for deciding the values to be used in standard setting depend on whether a substance is thought to have a threshold or is a non-threshold substance. With a threshold substance, many countries set standards by selecting a pivotal or key study (often an animal study) to identify the most sensitive endpoint—generally the health effect that occurs at the lowest dose level. From this, the No Observed Adverse Effect Level (NOAEL) is obtained, the highest dose level at which

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**Fig. 2** Essential elements of assessing risk.
Evaluating evidence on environmental health risks

no adverse health effect is observed. This level is then adjusted by dividing by ‘uncertainty’ factors to account for interspecies and intraspecies differences. Additional uncertainty factors are also sometimes added, for example if it is necessary to extrapolate from a study that gives only a lowest observed adverse effect level to obtain a NOAEL, if the exposure route differs between the study and the typical human exposure situation, or if the severity of the effect is deemed to justify additional protection. The magnitude of each adjustment factor ranges from 1 to 10. In practice the first two are usually sufficient, and the figure of 10 is used, so that typically the permitted concentration of a chemical is one-hundredth that of the highest concentration associated with no observed adverse effect in the key animal study. Alternative approaches used by a few countries, notably the USA, include the use of benchmark doses and probabilistic modelling (including Bayesian).

There are three approaches taken for non-threshold substances: banning or non-approval of substances to which humans would be deliberately exposed, e.g. a water treatment chemical; the use of probabilistic methods to extrapolate from animal studies to the low levels of exposure experienced environmentally by humans to set a level at which risk is regarded as very low, e.g. 1 in 100,000 excess incidence of cancer over a lifetime of exposure; setting of an exposure level which is as low as technically achievable or reasonably practicable, e.g. the setting of a maximum exposure limit for occupational settings.

There are several comprehensive textbooks which explain in detail the scientific methodology employed to carry out both animal and human studies and interpret the results from them8–15 and it is not within the remit of this paper to repeat this. However, as the following papers in this volume summarize and evaluate the evidence on various environmental issues, particularly from studies on humans, a brief overview follows of the most relevant human study methods.

**Human study methods**

Studies of humans can be categorized as (i) experimental or (ii) observational, with several different types of study within these two categories as illustrated in Figure 3.

Essential elements of an experimental study include the control of intervention assignment by the investigator, comparison of interventions with standard or control treatment and, usually, some form of random allocation to different interventions. Of the experimental designs, formal clinical trials which in most cases involve patients, i.e. sick individuals, are the least used for environmental issues.

Field trials involve individuals without any particular disease, i.e. who are not patients, and are recruited from the general population. An example
of a field trial is testing the effectiveness of large doses of vitamin C in
prevention of the common cold. Community intervention trials differ
from clinical and field trials in that the interventions are implemented
for whole communities, for example, administering fluoride to a community
water supply. Challenge tests or controlled human exposure studies, such
as air pollution chamber studies and food challenge tests can potentially
provide good evidence between exposure and effect as exposures take place
in a controlled setting, and potential confounders can be systematically
identified and controlled for. However, for ethical reasons, these studies are
limited to studies of mild and reversible health effects, and usually low levels
of exposure. In addition, a laboratory setting may not always simulate
the real world.

The majority of human studies of environmental influences on health
are carried out using observational study designs. The three most common,
in which observations are recorded on individuals, are cross-sectional,
cohort and case-control studies.

Cross-sectional studies describe the frequency of the disease of interest
in a population at a particular period of time and the variation between
subgroups defined in terms of personal characteristics, time, place, and
where available, relevant exposures. They represent a ‘snapshot’ of a
population and are useful for generating hypotheses about the aetiology
of a disease. They are particularly useful when dealing with health data
that are continuously distributed in the population, such as blood pressure
or serum cholesterol. For categorical health outcomes, the cross-sectional
study is in fact similar to the case-control study (see below) except that
sampling is based on prevalent rather than incident cases\textsuperscript{15}. Their drawback
is that information on exposure and effect are collected simultaneously,
thus making it more difficult to attribute causation.

Cohort (or prospective) studies follow over time a group of people,
the cohort, with particular characteristics in common, including levels of
exposure, to observe the development of disease. The rate at which the
disease develops in the exposed people in the cohort is compared with

\textbf{Fig. 3 Epidemiological study designs.}
the rate in the non-exposed or in a standard group such as the national population; this comparison of rates is known as the *relative risk* of the disease for a particular exposure. In case-control studies, individuals with a given disease (the cases) are compared with a group of individuals without the disease (the controls). Information on past exposure to possible risk factors is then obtained for both cases and controls and compared, giving the *odds ratio* (an estimate of the relative risk) of disease associated with a particular exposure.15

Given the large numbers of people potentially exposed to many environmental pollutants and the likelihood that for many pollutants the excess risk of disease at the level of the individual is small, then in order to detect any excess risk, many thousands, hundreds of thousands or even millions of people may need to be studied, over a prolonged period, if the cohort approach is adopted. This is especially true for rare diseases such as congenital anomalies, childhood and many adult cancers that may be thought to be of particular interest with regard to the possible health effects of environmental pollutants. An alternative is to use the case-control approach which is much more efficient as only cases and a relatively small number of controls need to be assembled and studied. However, because of the retrospective nature of the exposure assessment, and possible selection effects, such studies are prone to bias that can seriously distort risk estimates.

Where individual-level follow-up studies are infeasible, and case-control studies are considered too expensive or impracticable, group-level (or ecological) studies are often undertaken. Ecological studies are generally thought to be of weaker design than individual-level studies because inferences made at the group level may not pertain at individual level, the so-called *ecological fallacy*16. However, such concerns notwithstanding, ecological studies have played a major role in the investigation of aetiological associations of public health importance, such as the relationship of type and amount of dietary fat to heart disease.17 Also, it is not always understood that in many common study designs, people are grouped for the purposes of exposure assessment—such studies are in fact ecological in nature. Examples include the use of job exposure matrices for occupational studies18 and studies of health effects associated with differing measures of social class19. Because of the difficulty in studying low-level risks from environmental exposures in large numbers of people, much of our knowledge concerning risk of chemicals in humans is in fact derived from occupational studies. Features of occupational and environmental epidemiological studies are compared in Table 1.

In the context of environmental epidemiology, two ecological designs are commonly employed, as indicated in Figure 3—those that group people in time, and those that group in space. Temporal studies are exemplified by studies on the health effects of outdoor air pollutants. In these studies, time-varying data on concentrations of air pollutants are available from
one or a small number of monitoring stations at city level. It is assumed that exposure to a range of outdoor pollutants of whole city populations can be characterized by such data; patterns of daily mortality or morbidity (such as hospital admissions for asthma) are then compared for the city in relation to daily fluctuations in air pollution. This is a powerful study design since the daily mortality and exposure to outdoor air pollution for many thousands or millions of people can readily be captured using routine data sources, and problems of confounding by individual characteristics such as smoking are minimized, as effectively people act as their own controls. Such studies have been mostly consistent in finding higher rates of mortality associated with days with higher levels of air pollutants (allowing for appropriate lag periods), especially particulate matter and sulphur dioxide. These effects have been reported at much lower concentration levels of pollutants than those traditionally associated with air pollution episodes, such as the London smog of 1952.

The second type of ecological study involves using location as a proxy for exposure. Often, proximity (to a factory or polluting industry) is used as the marker of exposure; examples include the incidence of certain cancers near municipal solid waste incinerators, and the occurrence of congenital anomalies and other birth effects near landfill sites in Great Britain. In other instances, some kind of exposure modelling is done to define areas considered to have high levels of exposure. Semi-ecologic designs offer an attractive means of reducing the possible biases that may affect ecological studies. In these studies, data on the exposure of interest (such as air pollution) is measured at the ecological level, but other data, including major confounders (see below), are collected at individual level, e.g. for a representative sample of individuals in the study regions.

### Confounding and interaction

An important issue in the interpretation of results of an observational study is whether they might be explained by a factor or factors other than that under investigation. This is not usually a problem in an experimental setting (such as a randomized controlled clinical trial) since the process of randomization, provided that the sample size is sufficient,
should ensure that the groups under study are similar with respect to other potentially causative factors. In epidemiological studies, the term *confounding* is used to describe the situation where an association between the factor of interest and the disease outcome is explained by the association of both these factors with another variable, the *confounder*, which itself is either a cause or closely related to a cause of the disease. Age and social class, for example, are commonly regarded as confounders as they are strongly related to disease occurrence and are also related to a wide range of environmental exposures. The effects of confounding variables can be at least partially removed, either by *matching* (in a case-control setting) or by statistical *adjustment* in the analysis. Either way, in the presence of possible confounding, judgments as to causality for the factor of interest have to be tempered by the possibility that the results from a study may be affected by potential confounding effects, unless the association is so large (relative risk > 5 or so) that it cannot readily be ascribed to other factors. In studying the effects of low-level environmental pollutants, risks are likely to be much lower than this, so that unmeasured or inadequately controlled (*residual*) confounding in the observational data should always be considered. This is one of the most important reasons why it is essential to examine all the evidence, from animal and human experimental studies as well as epidemiological studies, when making judgments as to causality.

Another important issue to consider is whether the effect on disease outcome of one factor is modified by levels of another factor, so-called *effect modification* or *interaction*. An example is the effect of cigarette smoking on risk of cardiovascular disease, which is strongly modified by age. At 40 years follow up of the British doctors study\(^4\), below the age of 65, the relative risk of cardiovascular disease among smokers compared with non-smokers was 2.1, whereas it was 1.2 at ages 85 and over. Interaction effects are increasingly thought to be important, and may lead to new ideas about aetiology and mechanisms of disease; however, they are difficult to investigate as much larger sample sizes are required than studies examining only ‘main’ effects. In particular, recent interest has focused on potential *gene–environment interactions* in determining the combined genetic and environmental influences on disease risk (see section on molecular epidemiology, below).

**Challenges and future developments**

**Data availability and quality**

In order to make robust inferences about potential environmental causes of disease, it is imperative to obtain valid data on health outcomes and
environmental exposures. For purpose-designed environmental epidemiological studies such as case-control or cohort studies, evaluation of the health data will often involve a detailed validation exercise and assessment of the quality of the diagnostic information (for example, case note and histology review). In contrast, the quality of the exposure data has been regarded as the ‘Achilles heel’ of such investigations, as often only group data or proxy information on environmental or occupational exposures has been available. Poorly measured exposure data will lead to misclassification which in turn leads to bias toward no effect—the so-called ‘regression dilution’ problem. Recent developments in exposure biomarkers and molecular epidemiology (see below) should in the future lead to improved exposure assessment methods, with increased specificity, and hence improved ability to detect true differences in disease risk.

Such purpose-designed studies are, however, time-consuming and expensive to carry out, and often recourse is made to routine sources of data, especially in geographical studies where location is used as a proxy for exposure, so-called spatial epidemiology. The health data may be available with associated point locations, or as aggregated counts for areas, and will potentially be subject to a number of inaccuracies. For any health event there is always the potential for diagnostic error or misclassification, especially at older ages where diagnostic tests and post-mortem examinations are carried out less frequently than at younger ages. Some events may be captured poorly, if at all, in routine registers (e.g. miscarriages). For others, such as cancers, case registers may be subject to double counting and under-registration as well as diagnostic inaccuracies. Some assessment of the basic quality of the routine data is therefore essential to inform their use in spatial analyses.

For the future, the increasing use and availability of computerization in medical care means that potentially large new databases of morbidity (for example from general practitioner consultations) will become available. The quality of such data will need careful evaluation and no doubt will vary across specialties, practices and over time and space. Nonetheless they promise exciting new opportunities for carrying out environmental epidemiology, which hitherto has been limited with respect to the range of outcome data available from routine sources.

Data protection and confidentiality

The current climate of legislation in the European Union and elsewhere is moving toward greater recognition of the rights of individuals to confidentiality of personal data, including health data, and the need for consent for medical research. It therefore behoves the research community to be sensitive to their responsibilities for the protection of the privacy
and confidentiality of individuals. In purpose-designed individual-level studies, this will involve obtaining proper consent to participate, the right to withdraw consent at any time, and the right of privacy and confidentiality of data obtained on individuals. Secondary use of routine data for epidemiology where the data were originally collected for other purposes (e.g. health care management or delivery) is a more complex issue, because here in many instances it can be argued that the public good (in terms of describing disease patterns, potentially identifying new causes of disease in the population, etc.) outweighs the individual right to privacy of data. In these circumstances, often it will be impossible or impracticable to obtain consent, and in any case data on the entire population are required to avoid possible biases as a result of selection effects. In the UK, recent legislation has made it possible to use such routinely collected data without consent provided that certain conditions and safeguards are met. It is imperative for the future of epidemiological research that such uses of the data are allowed to continue, given that appropriate safeguards are in place.

**Molecular epidemiology**

Advances in molecular epidemiology offer the opportunity to combine the scientific disciplines of epidemiology and molecular toxicology to investigate the interactions between genetic and environmental factors in the cause of disease. Many studies have demonstrated that exposure to relatively high levels of substances, such as carcinogens, does not affect all individuals equally, supporting the theory that genetic factors may influence an individual’s susceptibility and resistance to disease. Future research will need to incorporate measurement of susceptibility to aid the investigation of mechanistic pathways and to detect gene–environment interactions.

**Dealing with uncertainties**

Uncertainty and variability permeate all aspects of assessment of risks from environmental pollutants. Barnett and O’Hagan in discussing the setting of environmental standards draw attention to several sources of uncertainty inherent in the investigation of a pollutant–effect relationship. These include an imperfect understanding of the mechanisms by which the pollutant influences the effect, variation in the way individuals react to a given level of a pollutant, variation in the levels of exposure received by individuals, imprecise exposure assessment methods, and other causes of the disease of interest. Barnett and O’Hagan suggest that the quantification of uncertainty and variation should be addressed
using statistical inference procedures and the use of probabilistic modelling. In particular, these methods facilitate the incorporation of sensitivity analyses to examine the robustness of models to input assumptions and to identify those parameters to which the output is most sensitive. In addition, Bayesian modelling has been successfully used to analyse risks when available data have been inadequate for use in the classical statistical approaches to risk assessment and when there is the desire to incorporate the opinions of experts. The use of modelling approaches to the assessment of risk has been viewed with some scepticism by some governments, particularly in Europe. Effort is needed by scientists to ensure that adequate testing of the robustness of results from modelling is carried out and that they are able to interpret and communicate these results to non-specialists.

The role of systematic review and meta-analysis

Systematic review and meta-analysis methods are well developed in the area of clinical trials, and involve the collation of the literature on a particular area of interest, assessment of the extent and quality of the studies found and provision of a compilation of the results, often including quantitative estimation of risk estimates from the combined studies. These methods facilitate transparency and reproducibility of the methodology and results, and ease of updating. They can identify gaps in the knowledge base and areas for future research. Quantitative meta-analyses can give greater statistical power than single studies and provide a framework for investigation of possible sources of heterogeneity between studies32. In spite of controversy over the opportunity for bias and other sources of heterogeneity compared with clinical trials, these techniques are being increasingly used in epidemiological research, and a number of guidelines have been produced recently on the topic33,34. To date there has been very little use either of systematic review methodology or of meta-analysis techniques in toxicology or in environmental epidemiology.

Within the past 10 years, there has also been a growing interest in cross-design synthesis, i.e. the quantitative combination of results from different study designs35. In particular, the potential for the use of meta-analysis techniques to combine data from animal studies with that of human studies is beginning to be explored, which will have an important impact on the development of risk assessments and the setting of environmental standards.

Conclusion

Integration of the evidence available from a variety of sources for the assessment of adverse health effects from environmental hazards is a complex
process. The methods used in the individual scientific components contributing to the totality of information are, themselves, also often complex. This paper has given an overview of the commonest methods used for human studies and discussed issues important for interpretation of results. A difficult challenge will continue to be that of communication, both of complex methodology and the interpretation of results, and a need to acknowledge and address the concerns of the public whom the scientific community and regulatory authorities are aiming to protect. Risk communication is itself a developing scientific discipline and involves interaction, both co-operative and contending, between a large number of scientific, social and political institutions, media organizations, the public and the decision-makers. The problems widely encountered are founded in a lack of trust and confidence in risk information provided by ‘experts’ and in the processes by which decisions are made. A cultural shift is required by all bodies concerned to develop appropriate ways of establishing more open and transparent methodologies for assessing risks and to increase the contribution of the public to the development of risk management and risk reduction options.

References