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RISK AND RISK MANAGEMENT

A thesis
presented to the University of Ottawa
in partial fulfillment of the requirements
for the degree of Master in Health Administration

by Daniel Krewski

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UNIVERSITÉ D'OTTAWA
UNIVERSITY OF OTTAWA

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ABSTRACT

Recent years have witnessed a surge in both public and professional interest in the assessment and management of health and environmental risks. This focus of attention has culminated in the birth of a new discipline of risk science, and the corresponding systematic study of risk and risk management. The objective of this thesis is to review and extend the existing body of knowledge on risk assessment and risk management in a formal way, so as to synthesize a structural framework within which problems involving health and environmental risks may be analyzed.

In particular, a conceptual model of risk assessment and risk management is proposed to describe the risk problematic. The first step in this model is the identification of a well defined hazard which poses a threat to human health or the environment. Once a specific hazard is identified, the next step is to obtain an estimate of the risk presented, taking into account both the severity of the adverse effects induced and the likelihood of their occurrence. With health risks, which is the primary focus of this research, hazard identification and risk estimation rely largely on toxicological studies conducted in the laboratory and epidemiological studies of human populations. These data are subjected to careful statistical analysis in order to establish acceptable criteria for human exposure to environmental hazards.

Once the scientific enterprises of hazard identification and risk estimation are complete, alternative strategies for risk management need to be developed and evaluated. In addition to various regulatory options for risk management, risk managers are increasingly looking for nonregulatory solutions of an economic, advisory or technological nature. To choose among the available options, general program evaluation methodologies such as cost-effectiveness and cost-benefit analysis may be applied. While this has been widely done in the evaluation of certain health care programs, the transfer of this technology to evaluating environmental risk management strategies remains to be accomplished. To promote this, a general framework for the economic evaluation of environmental risk management programs is proposed.

While program evaluation techniques are useful as an aid to decision making, the actual decision concerning the most appropriate course of action to follow depends on a host of factors, including a balancing of risks against benefits in some cases. Consideration may also be given to the public's perception of risk, which may not always correspond to the actual risk determined by objective analysis. The technical feasibility of each proposed course of action should be demonstrated, including the ability to enforce any regulations that may be contemplated. Economic effects are often important in evaluating alternatives, both in terms of program-related costs and the impact on productive output. Socio-political factors involving equity considerations and repercussions at the international level should not be overlooked.

Implementation of the selected risk management strategy will usually require some commitment of resources, and should be accompanied by attempts to communicate the nature of the chosen control mechanism to all affected parties. Because of the clear need for effective communication of information on risks, models and methods for risk communication are considered in some detail. Once the control mechanism is in place, continued monitoring is recommended.

While other models of risk assessment and risk management have been developed, a careful comparative analysis shows that they involve essentially the same components as those described above. In addition to demonstrating the validity of our model in this comparative fashion, an empirical validation is also attempted by means of three detailed case studies of acidic deposition, saccharin and formaldehyde. This analysis indicates that the proposed model encompasses most of the practical aspects of risk assessment and risk management, and offers considerable insight into the key elements of risk management decisions involving environmental health hazards.

ACKNOWLEDGEMENTS

During the course of my work with the Health Protection Branch of Health & Welfare Canada since 1972, I have become increasingly interested and involved with the manner in which decisions involving environmental hazards are made. This motivated my registration in the MHA Program at the University of Ottawa in 1984 at the subsequent preparation of this thesis. Although written only recently, the thesis draws upon my experience with the Health Protection Branch and with other organizations both in Canada and abroad concerned with similar problems. At the same time, the perspectives expressed in this thesis remain my own and are not intended to represent those of the Branch or any other agency.

My first debt is to Dr. Pran Manga, who agreed to serve as my thesis supervisor, and who has provided much helpful guidance and advice during the course of this work. I am also grateful to Health & Welfare Canada, and to Dr. E. Somers in particular, for supporting the conduct of this project. I would also like to express my general appreciation to colleagues with whom I have worked in the past, including Patricia Birkwood, David Clayson, William Leiss, and George Torrance, as they have helped considerably to shape my thinking on risk assessment and risk management. Finally, I am grateful to Wendy Killeen for her excellent work in preparing the typescript and to Terry Chernis for her thorough bibliographic assistance.

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1. INTRODUCTION

We're not sure we understand the concept of risk, but, the less risk, the better the concept.

Bundestag
Federal Republic of Germany

1.1 THE EVOLUTION OF RISK ASSESSMENT

In the broad sense many different kinds of risks are readily discerned. Social risks such as violent crime and economic risks such as recession are familiar concerns within modern industralized societies. In recent years, however, considerable attention has focused on environmental health risks, particularly those attributable to toxic chemicals present in the environment. In order to better appreciate the current status of environmental risk assessment as an emerging discipline, it will be helpful to briefly trace its origins in science.

Mankind has always been exposed to a wide variety of deleterious agents present in the environment including those found in air, food, drinking water, and the workplace. Although some of these agents were recognized many hundreds of years ago, the development of the scientific method has resulted in enormous advances within the present century in the development of techniques for the identification of environmental health hazards. Within the past two decades, Minimata disease was diagnosed in Japan and attributed to the consumption of fish contaminated with methylmercury (WHO, 1976); PBB contaminated feed has resulted in the death of livestock in Michigan (Fries, 1985); and

food additives such as saccharin and nitrite have been suspected of possessing cancer causing properties (Arnold et al., 1983; NAS, 1981). Although considerate information is available on the potential health effects of these substances, it has been estimated that fewer than 10% of chemicals in use today have adequate data on which to base a proper risk assessment (NAS, 1984).

Another major force in the evolution of risk assessment as a distinct science has been the application of probabilistic risk assessment techniques in the safety evaluation of major technological developments. In simplified terms, some form of fault-tree or event-tree analysis is used to estimate the probability of system failure by considering the reliability of each component of the overall system. Although probabilistic risk assessment was applied in the development of manned space flight during the 1960's, the technique came to public attention when applied in a pioneering study of the safety of nuclear reactors (Rasmussen, 1975). This analysis suggested that the probability of a disastrous accident in the nuclear power industry such as a core meltdown resulting in a massive release of fissionable material would be extremely low, on the order of one in a million or one in a billion. Such analyses have subsequently been undertaken in connection with a number of other risks associated with high technology, including the safety of liquified natural gas terminals and the transportation of hazardous materials by land or sea with generally reassuring results.

While few would doubt that our ability to identify and quantify risk factors present in the environment has improved greatly in recent years, it is clear that the character of risk has also changed. With

life expectancy steadily increasing since the turn of the century, emphasis has shifted to risks which are better described as possible rather than probable. This improved ability to discern previously unsuspected risks with increasing frequency has resulted in an unprecedented public preoccupation with environmental health risks. Concerns over newly discovered hazards are now frequently found in media reports as well as in the scientific literature. In some cases, sensational reports seem to have served to heighten public anxiety as to the effectiveness of government programs dealing with the control of risk. As a result, certain consumer associations and other special interest groups are now demanding greater access to information on risks as well as the installation of formal procedures designed to permit broader public participation in the risk assessment process (Kane, 1980).

Obviously, the role of government in controlling food-related and environmental health hazards is not easy. Even some of the most trivial activities of daily life are potentially hazardous to some extent (Wilson, 1979), while many are associated with significant benefits (Crouch & Wilson, 1981). Thus, it would appear appropriate that government evaluate each newly identified risk in light of any associated benefits in order to develop realistic options for the control of environmental risks (Lave, 1981). This is not, however, easy as risk and benefit usually involve disparate units for measurement.

At present, risk analysis is becoming rapidly accepted as an established interdisciplinary field. Indeed, with the recognition of the fact that risk is an essential part of life often counterbalanced by concommitant benefits, the need for a rational and realistic approach to risk analysis becomes apparent. Thus, modern risk analysts have proposed systematic approaches to environmental risk assessment (Whyte & Burton, 1980; Lave, 1982), ranging from the identification of a potential hazard to an assessment of the risks involved in the regulatory response.

1.2 COMPONENTS OF RISK

A review of the literature on environmental risk assessment reveals that the term risk means different things to different authors. For example, Rowe (1977) defines risk as:

> ... the potential for realization of unwanted **negative consequences** of an event.

Rosenbluth (1980), on the other hand, offers the following definition:

> The risk from an activity is the **chance** that loss or injury will be brought about by the activity in question.

These definitions involve two ideas. The first relates to the hazard associated with a particular event while the second is concerned with the probability of occurrence of the particular hazard. O'Riordan (1979) combines both components into a single definition:

Risk...is regarded both as a **hazardous outcome** and a **probability of occurrence.**

Despite the apparent discord among the above definitions, all involve at least tacitly the notions of hazard and probability. Okrent (1980) cites a simple example which clarifies the meaning of these two ideas. He considers two people crossing the ocean, one in a rowboat and the other in an ocean liner. The hazard (death by drowning) is the same in both cases although the probability of drowning is clearly different.

It is now widely accepted that the risk posed by an environmental toxicant depends both on the type of hazard present and the probability of its occurrence (Figure 1.1). Severe hazards such as chronic debilitating diseases are clearly of greater significance than acute illnesses of minor consequence. Similarly, the frequent occurrence of a particular adverse health effect may be viewed with greater concern than the occasional observation of another effect of comparable severity.

With health risks, hazard may be described in terms of the adverse health effects induced. Such effects are characterized by factors such as their nature, severity, time of onset, and degree of reversibility. The probability of occurrence of a given effect depends on the potency of the toxicant, the susceptibility of the exposed individual, and the level of exposure. The level of exposure in turn depends on the

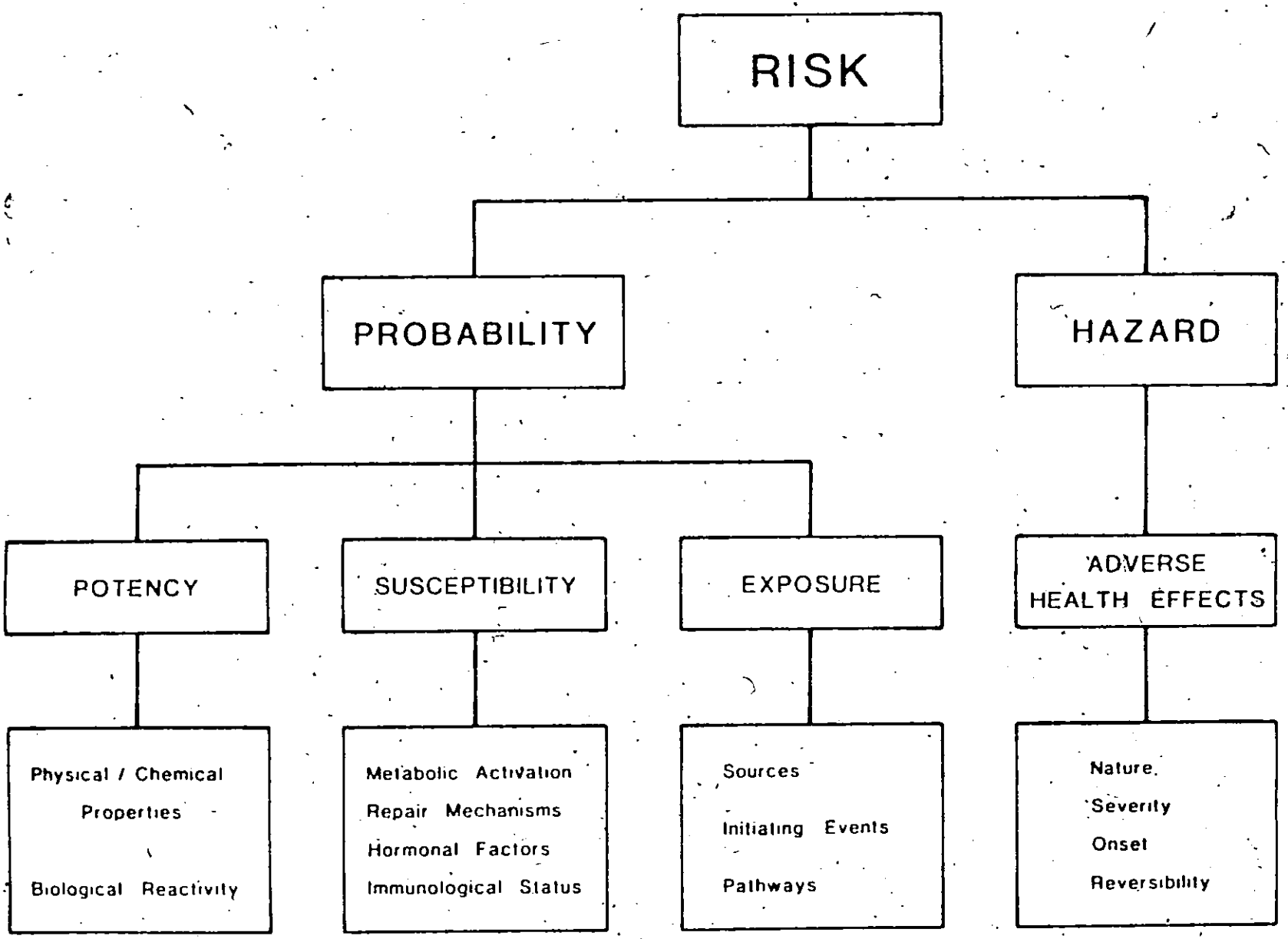


Figure 1:1 Components of Risk

sources from which the toxicant may be released into the environment, the initiating events responsible for its release, and the pathways by which the toxicant may reach human populations. Potency is largely determined by the physical and chemical properties of the toxicant, including its biological reactivity with macromolecules present in mammalian cells. Susceptibility may be influenced by a variety of factors, such as metabolic processes and repair mechanisms.

Potency, susceptibility, and exposure as described above are properties of the toxicant, the host organism, and the intervening environment respectively. Differences in the potency of chemical substances may be assessed by exposing the same species to these substances, and noting the variation in the effects induced (Gold et al., 1984). Differences in species susceptibility may be demonstrated by exposing different species to the same substance (Haseman & Hoel, 1979). Intra-species differences in susceptibility also exist as reflected in the variation in response among individuals of that species. The effect of exposure may be gauged by observing the effects induced at different doses, and constructing a dose-response curve describing how the probability of these effects occurring varies with dose (Krewski and Van Ryzin, 1981).

The preceding discussion indicates that risk is a two-dimensional concept, depending on both the nature of the health effect in question

and the probability of its occurrence. Thus, larger risks may accrue either as a result of the induction of a more severe effect or due to a greater frequency of occurrence. It is also possible that more than one health effect may be associated with a single toxicant, or that the effects induced may vary with the level of exposure. If these different effects may be expressed in the same units, then the risk may be characterized by a frequency distribution of adverse outcomes giving the probability of observing an outcome of a given magnitude or greater.

A quantitative measure of risk which embodies the dual notions of hazard and probability has been discussed by Kaplan and Garrick (1981). These authors note that the main elements of risk analysis may be identified as responses to the following questions.

What can happen or what events may occur?

What is the probability of this happening?

What are the consequences of such an occurrence, or what is the hazard involved?

Suppose that after careful consideration of the first question, we identify n different mutually exclusive events $E_1,\ldots,E_n$ which may occur. We then go on to determine that event E_i will occur with probability p_i and is associated with a hazard or loss H_i

($i = 1,\ldots,n$), with $H_1,\ldots, H_n$ measured in commensurate units. (Although it may not be possible to identify in advance all of the possible events which may occur in complex situations, we will assume for simplicity that this can be done. Thus $p_1+\ldots+p_n = 1$). After arranging the n events in order of increasing hazard $H_1 < \cdots < H_n$, we calculate the cumulative probability $P_i = p_i+\ldots+p_n$, which specifies the probability of the occurrence of an event for which the hazard is as great or greater than H_i.

Table 1.1 Basic Elements of Quantitative Risk Analysis

Event	Probability	Hazard[1]	Cumulative Probability
E_1	p_1	H_1	$P_1 = p_1 + \ldots + p_n = 1$
.	.	.	.
.	.	.	.
.	.	.	.
E_i	p_i	H_i	$P_i = p_i + \ldots + p_n$
.	.	.	.
.	.	.	.
.	.	.	.
E_n	p_n	H_n	$p_n = p_n$

[1]Arranged in increasing order ($H_1 < \ldots < H_n$)

As an example, let us consider the risk associated with the transportation of a hazardous material by road in tankers from say Toronto to Montreal. The trucks will pass through several urban areas as well as other more sparsely populated areas. In a single trip, there may be an accident involving spillage of the material being

transported resulting in property damage or personal injury. This is a specific event. The hazard associated with the event will depend on the location of the accident, the degree of spillage, and local weather conditions, which will affect the rate at which the material is disseminated throughout the environment.

After categorizing all possible events by the magnitude of the dollar value of the property loss, we might arrive at a risk analysis of the following form.

Property Loss ($)	Probability	Cumulative Probability
100,000	0.005	0.005
50,000	0.015	0.020
10,000	0.030	0.050
0	0.950	1.000

In this hypothetical example, there is a 95% chance of no property loss at all and only a 2% chance of a property loss of $50,000 or greater. If such transportation accidents should result in loss of life, our risk analysis might be expressed as follows.

Number of Deaths	Probability	Cumulative Probability
0	0.990	1.000
1	0.006	0.010
2	0.003	0.004
3	0.001	0.001

While the probability of a particular event occurring in the preceding example might be determined by historical precedent, there are often no historical data available in situations involving the introduction of new technological developments. In the absence of adequate data, however, we might appeal to a panel of experts to construct possible scenarios and evaluate the likelihood of their occurrence subjectively. Alternatively, it may be possible to use engineering techniques to objectively determine the reliability of a particular technology, and hence the likelihood of its failure.

Because of the amount of information on risk contained in Table 1.1, it may be useful for some purposes to consider single measure of risk which summarizes in some useful way the detailed listing of possible events, their probabilities of occurrence and their hazards. In statistical decision theory, for example, such a summary measure of risk R has been defined as the average loss or hazard given by

$$R = p_1 H_1 + \ldots + p_n H_n$$

(Ferguson 1967). In the transportation scenario discussed above, for example, the expected property loss per trip would be $1550.

An important special case of the above analysis occurs when only one particular undesirable event may occur. For example, suppose that a particular lesion may be induced following exposure to a particular environmental contaminant with probability p. Associating losses 0 and 1 units with the absence or presence of the lesion respectively, the average loss is given by

$$R = [(1 - p).0 + p.1] = p$$

Thus, in simple situations such as this the concept of average risk is effectively equivalent to the probability of occurrence. Under these conditions, the terms risk and probability are often used interchangeably. In general, however, it is important to recognize that risk is a function both of the nature of the effects induced and of the probability of their occurrence.

While the expected loss may be an appropriate measure of risk in many cases, other summary measures may also be used, subject to the constraint that increasing either hazard or the probability of occurrence will increase the risk. An extreme case would be to ignore

the probability of occurrence and take the risk R to be the maximum hazard H (Campbell, 1980). This would reflect the intent to consider only information on the nature of the loss and not on the likelihood of its occurrence. An example of this latter philosophy is embodied in the Delaney amendment in the United States (Coulston, 1979), which states that chemicals with any carcinogenic potential whatsoever may not be added to food no matter how small the probability of cancer induction. Here, the hazard in question is regarded as being of sufficiently great concern that no chance of occurrence, no matter how small, would be considered acceptable.

The difficulty with all such scalar measures of risk is that considerable information is lost in the summarization process. For example, it is possible for two different risk management strategies to have divergent risk curves and yet have the same average risk. In the situation illustrated in Figure 1.2, for example, it is not clear whether Strategy A, which results in a greater occurrence of events with small losses, is preferable to Strategy B, which results in a greater occurrence of more hazardous events.

1.3 OVERVIEW

The rational management of health and environmental risks ideally requires a well defined structured approach in order that risk may be dealt with in a complete and equitable fashion. In chapter 2, formal

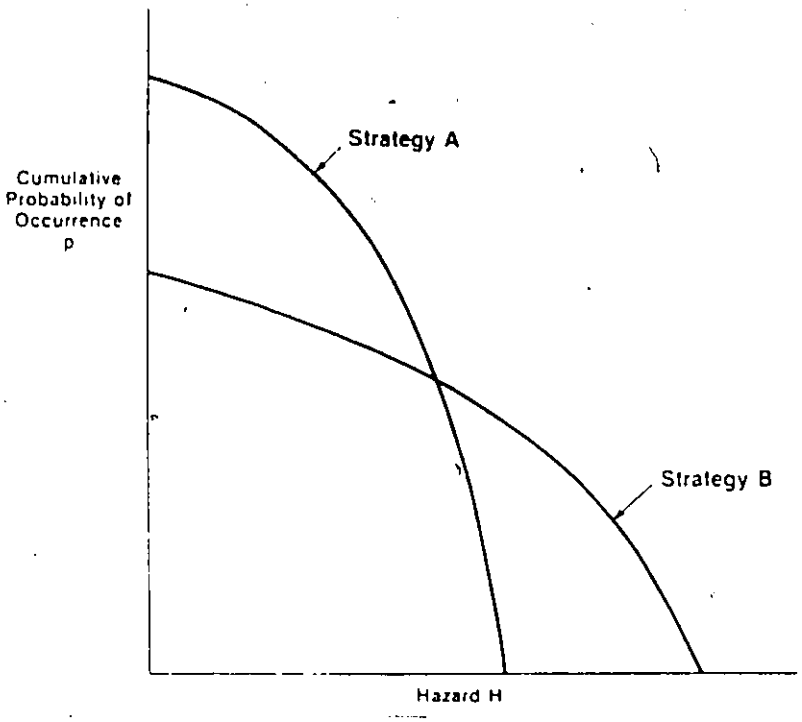


Figure 1.2 Risk Curves for Two Different Risk Management Strategies

models of the risk assessment and risk management process which have been proposed in the literature in recent years are reviewed. Although these models were developed primarily with reference to chemical risks, they are also applicable to other health hazards including those of a radiological or physical nature. Common elements amongst these models are identified, and the potential impact of these approaches on practical decision making is examined.

The first step in the risk assessment and risk management process as described in chapter 2 is the recognition that a well defined hazard to human health exists. In chapter 3, epidemiological and toxicological approaches to health hazard identification are discussed. While epidemiological studies of human populations have the advantage of providing information on adverse health effects directly in man, it is often difficult to identify specific hazards due to the complexity of the human environment and the limited sensitivity of such studies to detect small effects. Thus, toxicological studies using nonhuman laboratory tests systems are often relied upon to identify potential human health hazards. In the case of toxic chemicals, additional information can also be derived by examining the chemical structure and biological reactivity of the compound under study.

Once a health hazard has been identified, it is often useful to determine the magnitude of the associated risk (chapter 4). Risk estimation also relies on toxicological and epidemiological data sources, subject to a more quantitative analysis in order to determine the risk posed by the hazard of interest. Risk estimation based on toxicological data may invoke the application of safety factors to

establish acceptable levels of human exposure to environmental toxicants or dose response models to characterize the relationship between the probability of an adverse health effect occurring and the level of exposure. Similar techniques are applicable with epidemiological studies, although with observations directly on humans, safety factors which allow for inter-species variation in susceptibility are not required. It should be noted that while a good epidemiological data base represents the ideal starting point for risk estimation, direct observation on humans often suffer from limited sensitivity due to low exposures as well as multiple exposure to extraneous hazards. Further, epidemiological data will not be available for anthropogenic hazards which have not yet been introduced into the human environment. For these reasons, toxicological and epidemiological data generally provide complementary sources of information on which estimates of human health risk may be based.

The control of risks to health, safety and the environment may be achieved in a variety of different ways. In the past, regulation has been widely used as a risk management tool with varying success. More recently, nonregulatory options for risk management have received considerable attention, including those based on economic, advisory or technological solutions. In chapter 5, we review and evaluate the

spectrum of control options available for health risk management, and consider possible criteria which may be used to select the most appropriate option for application in specific circumstances.

Of the four main options for risk management discussed in chapter 5, regulation remains the most widely used. In chapter 6, Canadian regulatory statutes pertaining to health and safety currently available as a means of controlling risk are reviewed. The division of legislative powers between the Federal Parliament and Provincial Legislatures based on the Constitution Acts of 1867 and 1982 is considered, including areas where uncertainties over overlapping jurisdiction remain. At present, federal authority for the control of health hazards is vested in ten major statutes, ranging from well-known mature pieces of legislation such as the Food and Drugs Act to more recent statutes such as the Transportation of Dangerous Goods Act passed in 1980. The use of these statutes as a basis for federal regulation is examined in detail, along with concomitant legislation available at the provincial level.

If more than one viable risk management option is identified, it is necessary to select the most appropriate strategy to deal with the problem at hand. Risk management programs designed to improve human health or the environment can be evaluated within the same basic framework used for health care programs. In chapter 7, a general framework for the evaluation of risk management programs is presented.

Consideration is given to the costs of the program itself as well as costs to industry and the public. On the benefit side, improvements to human health are considered along with economic and aesthetic gains. Techniques for measuring both costs and benefits are discussed. Several commonly encountered forms of program evaluation are shown to arise as special cases, including cost-effectiveness analysis, cost-utility analysis, cost-benefit analysis and risk-benefit analysis.

While risk estimation attempts to provide objective scientific ratings of risk, it may not accurately reflect how the public views these same risks. In chapter 8, we discuss the health and environmental risks which are perceived as being of concern by the public, and attempt to elucidate those factors which tend to result in overperception or underperception of risk. Consideration is also given to de minimis levels of risk which may be generally viewed as being essentially negligible and hence accepted by individuals or society as a whole.

An important part of the risk assessment process is the communication of information on risks. Data on risk should be expressed in understandable terms and effective channels of communication established between individuals and institutions involved in the assessment of risk. In chapter 10, models which have been used to describe the process of risk communication are reviewed, along with

risk communication techniques currently in use. The importance of good risk communication practices as a component of effective risk management is indicated.

Chapter 2 through 9 describes in detail a formal framework within which risk assessment and risk management may be described. To evaluate the applicability of this framework, there is a need to apply it to actual health risk management problems. This is done by means of three case studies presented in chapters 10, 11 and 12. These case studies all involve chemical hazards, which have been emphasized throughout this work.

Acid deposition is now recognized as a serious environmental problem facing large segments of Eastern North America and Western Europe. Although the effects on our lakes, forests and wildlife stocks are well documented, the potential for adverse effects on human health is less well established, particularly as a result of the long-distance transport of airborne pollutants. In chapter 10, we review existing epidemiological evidence on the health effects of acid rain, including both respiratory and other disorders. This information is used in conjunction with corresponding data on human exposure to review risk management strategies for the control of acid deposition in Canada.

The second of the three case studies involves the food additive saccharin (chapter 11). This substance was first synthesized in 1879 and eventually became popular as a non-caloric sweetener. While the toxicological data clearly indicates that saccharin can induce urinary

bladder tumours in laboratory rats, the epidemiological studies at best suggest an equivocal relationship between the consumption of saccharin and bladder cancer. Risk-benefit evaluations show few, if any, documentable benefits from the use of saccharin and much genuine uncertainty concerning the level of risk to the human population. Although saccharin was banned for use as a direct food additive in 1977, it continues to be sold in pharmacies for those who perceive a need for its use.

The last of the three case studies involves formaldehyde. This substance, which is ubiquitous in the environment due to release from both natural and man-made sources, has long been recognized to be a sensory irritant. More recently, on the basis of results obtained in animal species, it has been suggested that formaldehyde may also be carcinogenic to man. In chapter 12, the available data on the sources, levels and health effects of exposure to formaldehyde are examined. The regulation in Canada of the manufacture and use of formaldehyde-containing products such as UFFI is considered and the impact of the limitations of the available data on regulatory decisions is assessed.

We conclude with a summary of the process of risk assessment and risk management as formulated here (chapter 13). The features of the corresponding model used to describe this process are discussed, and the application of the model to the case studies of acid rain,

saccharin and formaldehyde considered. It is argued that the proposed model of risk assessment and risk management offers considerable insight into the risk problematic, and that the cases selected for study demonstrate that the model encompasses the practical aspects of risk assessment and risk management. Finally, future perspectives on risk and risk management are provided.

2. RISK ASSESSMENT AND RISK MANAGEMENT

2.1 INTRODUCTION

The selection of environmental hazards for government attention has often been handled in an ad hoc fashion in the past, usually in a reactive manner rather than as part of a carefully planned strategy. Issues which attract the public's interest have often been the focus of societal resources at the expense of more serious but less popular problems.

Despite the pressures on regulatory agencies to respond to external initiatives and shifting priorities, several factors indicate the need for a more pragmatic approach to the management of health risks. These include the desirability of balancing risks and benefits across society in an acceptable way, and the trend towards more openness in decision making using consistent and explicit decision criteria. The need for an orderly and systematic approach to risk assessment and risk management is further supported by the existence of resource constraints, which prevent maximum control of all risks. Such an approach can lead to greater objectivity and completeness, consistency of decisions, and public accountability.

A number of formal models for risk assessment and risk management have been proposed in recent years. These models are of great value in

clarifying the main elements of risk assessment and risk management, an have served to establish a well defined framework within which risk may be addressed.

In section 2.2 of this chapter, the different models which have been proposed in the literature are discussed. These models are then compared and a number of common elements identified (section 2.3). A brief discussion of the key components of the models and their role in understanding and improving the overall process of risk assessment and risk management is given in section 2.4.

2.2 MODELS FOR RISK ASSESSMENT AND RISK MANAGEMENT

As discussed in chapter 1, the risk posed by a particular agent depends on both the nature of the hazard presented and the probability of its occurrence. Health hazards may be characterized in terms of the health consequences or adverse effects induced. These effects may be more or less serious depending on such factors as their nature, severity, and degree of reversibility. The probability or chance of a given effect occurring depends on the potency of the agent, the susceptibility of the host, and the level of exposure.

2.2.1 Scientific Committee on Problems of the Environment

The Scientific Committee on Problems of the Environment (SCOPE) and its parent body, the International Council of Scientific Unions, are concerned primarily with scientific problems relating to the environment. Recognizing that environmental science requires an interdisciplinary approach, the Committee involves not only specialists in the physical and biological sciences, but experts in economics, law, community medicine, environmental psychology, as well as a wide range of other disciplines.

The first formal model of the risk assessment and management process appears to have been formulated by SCOPE (Kates, 1978; Whyte & Burton, 1980). A three stage risk assessment process consisting of risk identification, risk estimation and risk evaluation was proposed. The first step involves simply recognizing that a hazard exists. A quantitative estimate of the magnitude of the associated risk is then prepared by scientifically determining its characteristics. This is followed by judgments about the significance and acceptability of risk probabilities and consequences. Following risk assessment, some decision regarding whether or not to intervene takes place. This last stage may be termed risk management.

In the remainder of this chapter, we review a number of different frameworks within which risks may be characterized, evaluated and

controlled in a logical manner. These include models for risk assessment and risk management proposed by national and international bodies such as the Scientific Committee on Problems of the Environment, the United States National Research Council, the Royal Society in the United Kingdom, the Interdepartmental Committee on Toxic Chemicals in Canada, and the World Health Organization. After describing each of these models in detail, their similarities and differences are analyzed. We conclude with a discussion of the potential impact of these models on practical decision making.

2.2.2 U.S. National Research Council

The National Research Council was established by the United States National Academy of Sciences in 1916 to advise the federal government within the broad community of science and technology. Upon the initiative of the United States Congress, the National Research Council conducted a study to strengthen the reliability and objectivity of scientific assessment that forms the basis for federal regulatory policies pertaining to carcinogens and other public health hazards. This study was conducted under contract with the U.S. Food and Drug Administration within the Department of Health and Human Services.

The NRC (1983a) model consists of two stages: risk assessment and risk management (Figure 2.1). Risk assessment refers to the use of a

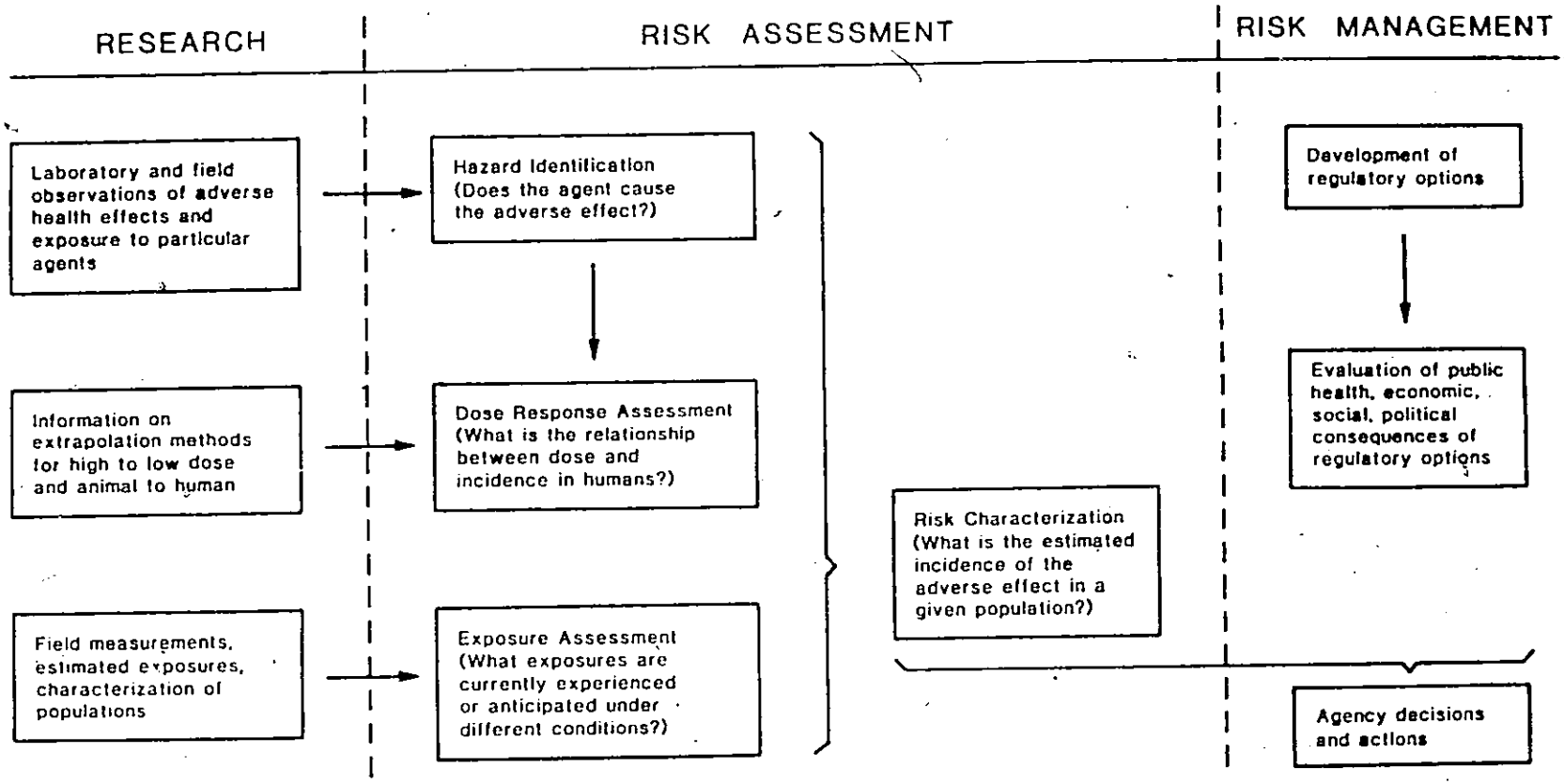


Figure 2.1 United States National Research Council/Environmental Protection Agency Model of Risk Assessment and Risk Management

factual base to define the health effects of exposure of individuals or populations to hazardous materials or situations. Risk management is the process of evaluating regulatory options and selecting among them.

Risk assessment is subdivided into four components: hazard identification, dose response assessment, exposure assessment, and risk characterization. Hazard identification is the determination of a cause-effect relationship between a particular chemical and a decline in health status using epidemiological studies of human populations, animal bioassay data, mutagenicity tests, and examination of molecular structure. Dose response assessment involves examination of the relation of magnitude of exposure and probability of occurrence of the health effects in question. Exposure assessment is the study of the extent of human exposure before or after application of regulatory controls. Risk characterization includes hazard identification, dose response assessment and exposure assessment, and involves a description of the nature and magnitude of human risk, including attendant uncertainty.

At the risk management stage, alternative regulatory options are developed and evaluated. Selection of a particular regulatory option involves consideration of the public health, economic, social, and political consequences of implementation. Other factors of significance include the technical feasibility of the proposed solution, desired level of control, ability to enforce regulations,

uncertainty in scientific data and the corresponding inferential bridges used to fill gaps in knowledge, and the public's perception and level of information. The implementation of a specific course of action should be accompanied by the communication of information concerning the basis of the decision to affected parties.

The NRC model was subsequently adopted by the United States Environmental Protection Agency (1984) with no significant structural or definitional changes. The United States Department of Health and Human Services (DHHS) (DHSS, 1985) has expanded the NRC model to include consideration of nonregulatory options for risk management. These include advisory options as well as risk reduction through technological means. DHHS also recommends expansion of research so as to reduce the uncertainties associated with scientific knowledge.

2.2.3 The Royal Society

In 1978, the Royal Society in the United Kingdom established a Study Group to consider a number of risk related problems, including both engineering and biological risks, and to address questions involving risk perception and risk management. The final report of this Group, which included a number of subgroups comprised of psychologists, statisticians, physicists, biochemists, biologists,

epidemiologists, doctors, engineers and economists, was published in 1983.

The Royal Society model (Royal Society Study Group, 1983) is also composed of two stages: risk assessment and risk management. The former is further subdivided into risk estimation and risk evaluation.

Risk assessment is the general term used by the Royal Society to describe the study of decisions having uncertain consequences. Risk estimation refers to identification and estimation of the probability and magnitude of the consequences of a hazardous event. Risk evaluation is the complex process of determining the significance or value of the identified hazards and estimated risks to those concerned with or affected by the decision. Embedded within this stage are the interrelated processes of developing alternative courses of action and decision analysis. These components take into consideration public awareness and perception, acceptability of risk, and the analysis of risks, costs, and benefits. These latter factors include consideration of the level of justifiable risk, economic and technical feasibility, and resource requirements.

Based on the evaluation of risk, risk management refers to the making of decisions concerning risks and their subsequent implementation. Decision making itself involves consultations between industry, government, the public, and other special interest groups

affected by the decision. Implementation of a decision, along with its monitoring, evaluation and revision are considered an integral part of the process.

2.2.4 Interdepartmental Committee on Toxic Chemicals

In Canada, the four federal government departments having major responsibility for the regulation of toxic substances are represented on the Interdepartmental Committee on Toxic Chemicals. (These are Agriculture Canada, Environment Canada, Fisheries & Oceans Canada, and Health & Welfare Canada.) The model developed by the Interdepartmental Working Group on Risk-Benefit Analysis (1984) for consideration by the ICTC represents an elaboration of the SCOPE model. The version of this model shown in Figure 2.2 includes minor modifications incorporated by Krewski (1986).

The first step in the process is hazard identification which is based on case reports, epidemiological studies of human populations, and toxicological experiments conducted in the laboratory. Another possible approach for the identification of chemical risks is to compare the molecular structure and biological activity of the substance under study with that of known toxicants.

The next step is to obtain an estimate of the magnitude of the risk in question. This involves the statistical analysis of

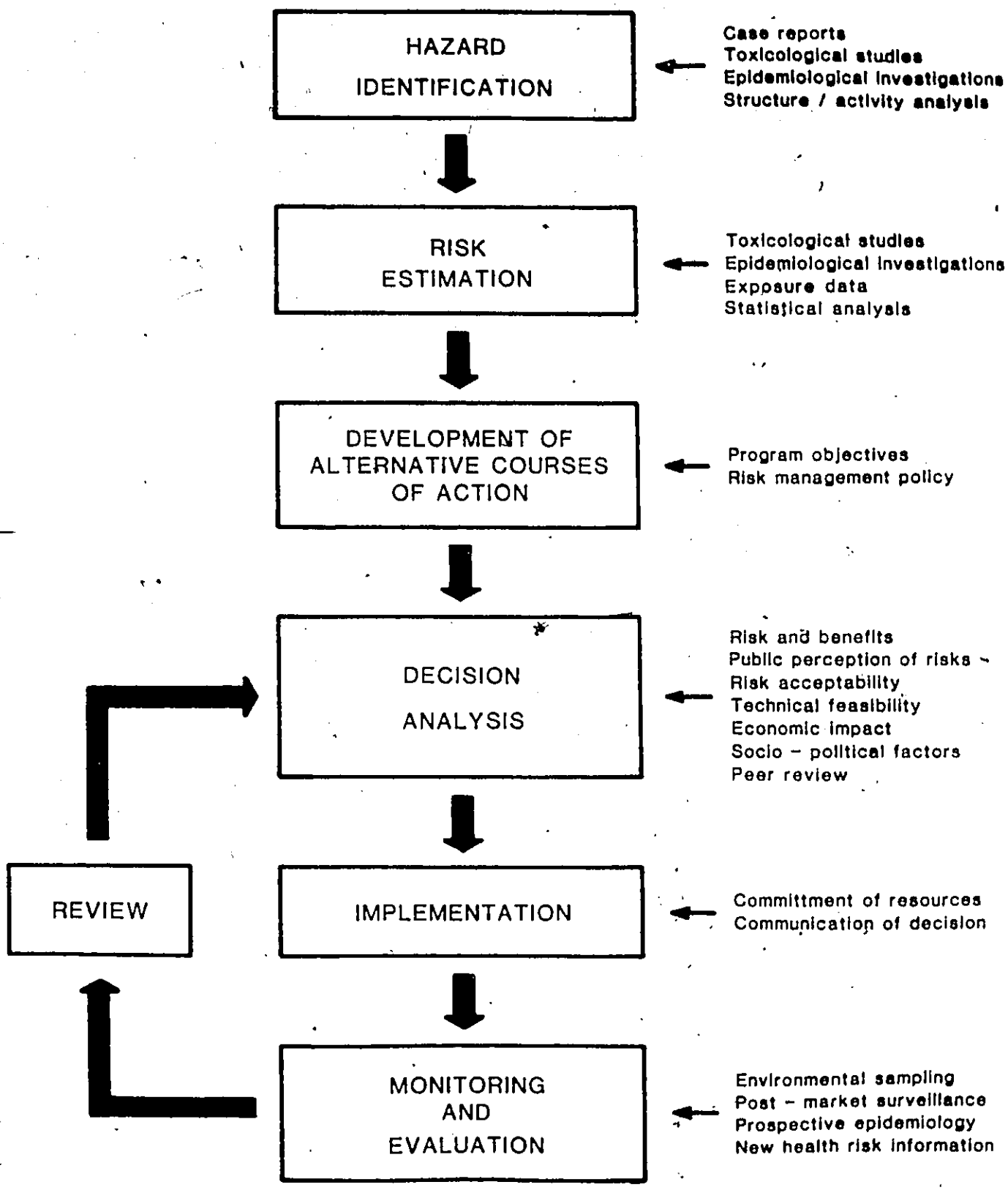


Figure 2.2 Interdepartmental Committee on Toxic Chemicals Model of Risk Assessment and Risk Management

epidemiological and toxicological data to determine the level of risk associated with specific hazards and to establish acceptable criteria for exposure to environmental hazards. This process is subject to considerable uncertainty and may require strong assumptions, as in the conversion of animal results to the human situation.

The first step towards selecting a strategy for dealing with a given environmental risk is the development of a number of alternative courses of action. Available options can range from advisory to economic to strict regulatory control. In order to ensure a consistent approach to risk management, the set of options selected for further evaluation should be compatible with existing environmental health program objectives and remain cognizant of any overall risk management policy guidelines.

The decision as to the most appropriate course of action depends on a host of factors, including a balancing of health risks against health benefits in some cases. Consideration may also be given to the public's perception of risk, which may not always correspond to the actual risk determined by objective analysis. The technical feasibility of each proposed course of action should be demonstrated, including the ability to enforce any proposed regulations. Economic effects are often important in evaluating alternatives, both in terms of program-related costs and the impact on productive output.

Socio-political factors involving equity considerations and repercussions at the international level should not be overlooked.

Implementation of the selected risk management strategy will usually require some commitment of resources and should be accompanied by attempts to communicate the nature of the chosen control mechanism to all affected parties. Once the control mechanism is in place, continued monitoring is recommended. Continual evaluation and review of new health risk information may suggest modifications to the risk management strategy currently in place.

Leiss (1985) has modified the original ICTC model in two ways. First, Leiss introduces a separate pathway for consideration of benefits parallel to hazard identification and risk assessment (benefit identification and net benefit assessment). Second, Leiss recommends incorporation of new consultative and communicative procedures in the risk management process. Specifically, it is suggested that risk and benefit assessments be available as public documents for examination by all interested parties; that standard procedures be developed to notify interested parties about the availability of public documents and about the results of decision making; and that regulatory decisions be accompanied by an explanation of their inherent reasoning.

The refinements to the ICTC model proposed by Krewski (1986) included the role of peer review. This can provide objective

criticism, expose new ideas and findings, maintain the quality of scientific information, and increase public confidence (DHHS, 1985). Covello & Merkhofer (1984) advocate peer review so as to ensure that the best data are being utilized, that any assumptions made are reasonable, that critical calculations are free from error, and that uncertainty is adequately recognized and taken into account.

2.2.5 World Health Organization

The World Health Organization (WHO) is a specialized agency of the United Nations with primary responsibility for international public health matters. The WHO is involved in a wide variety of health related activities through cooperation with its some 160 Member States in order to achieve its broad objective of health for all by the year 2000.

The WHO (1985) model is a four stage process consisting of hazard identification, risk estimation, risk evaluation, and risk management (Figure 2.3). The process as a whole is influenced by a number of participating bodies including scientists, industry, special interest groups, public, the media, and politicians.

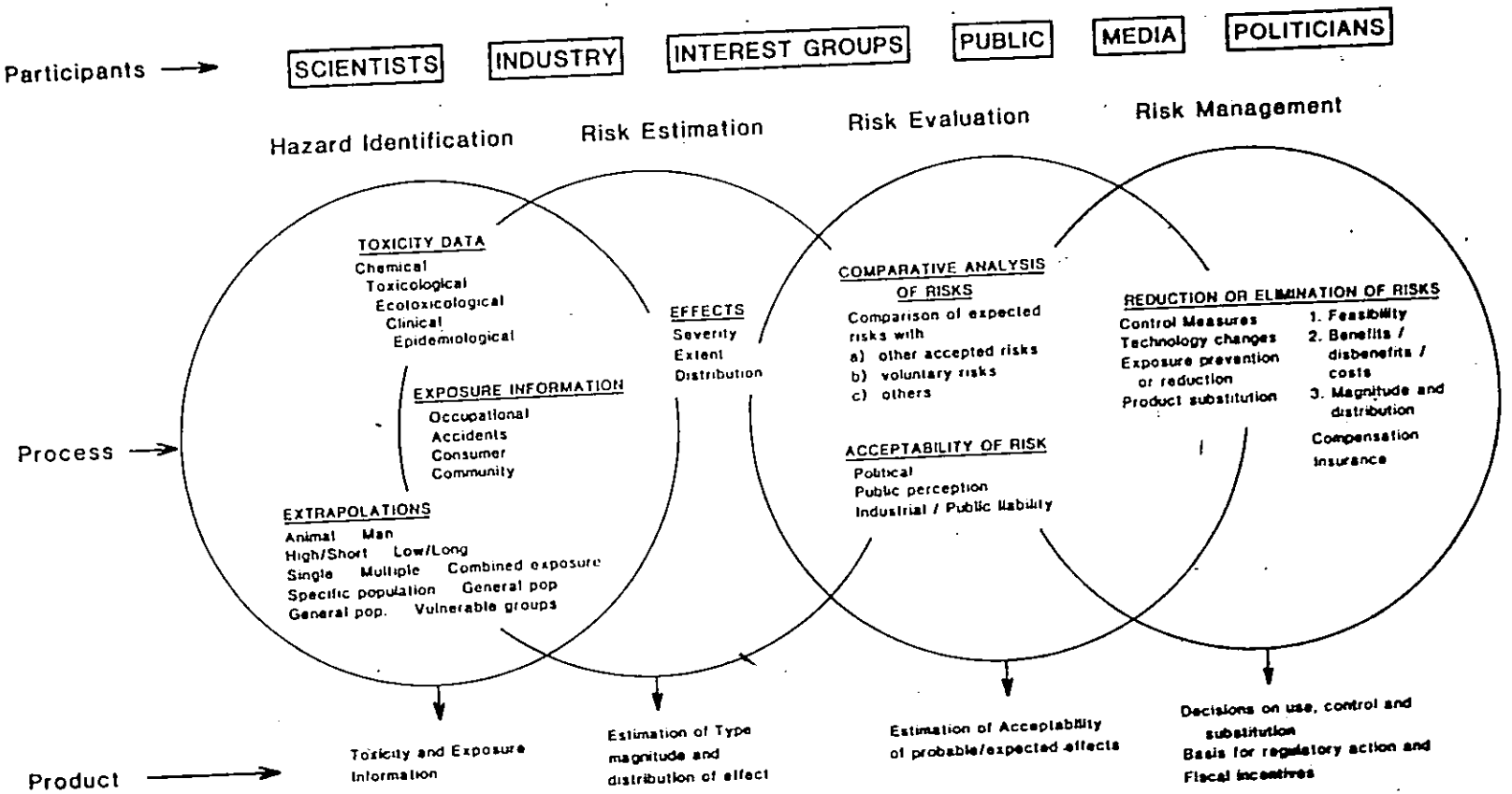


Figure 2.3 World Health Organization Model of Risk Assessment and Risk Management

Hazard identification requires the collection of chemical, toxicological, ecotoxicological, clinical and epidemiological data. Toxicity and exposure information are obtained at this first stage.

Risk estimation characterizes the extent of harm and the probability of its occurrence. This stage utilizes the information gained in hazard identification to predict the severity, extent, and distribution of the increased incidence of disease, disability, or defects caused by exposure to a given hazard.

The risk evaluation stage involves comparative analysis between the risk in question and accepted risks, voluntary risks, and other risks. Consideration is also given to political factors, public perception, and industrial and public liability.

The risk management stage consists of decision making with the aim of reducing or eliminating the risk in question. Decision making must take into account cultural, socio-economic, and political factors, and the type and nature of the risk in question. The possibility of reducing or eliminating the risk through control measures, technology changes, prevention or reduction of exposure, and product substitution must be considered in terms of feasibility, costs/benefit and magnitude and distribution. The decisions resulting from such an analysis form the basis for regulatory action.

2.2.6 Other Models

Various individuals have also proposed models for risk assessment and risk management. Baram's (1982) framework consists of six steps: hazard identification, risk measurement, risk management options selection, economic and technical feasibility analysis, ordering of risk management initiatives, and deployment of risk management options.

Lave's (1982) model contains eight stages: hazard identification, risk assessment, identification of regulatory alternatives, decision analysis, regulatory decision, legal or political challenge, implementation, and monitoring. This model closely resembles that adopted by the ICTC.

Rodricks & Tardiff (1984) consider only two broad stages: risk assessment and risk management. The former is subdivided into three phases: hazard identification and evaluation, dose response evaluation, and identification of conditions of exposure. The latter stage also consists of three phases: examination of alternative courses of action, decision analysis, and implementation.

Shrader-Frechette's (1985) framework includes three stages: risk identification, risk estimation, and risk evaluation, which closely parallel those in the SCOPE Model.

2.3 COMPARISON OF MODELS

The models for risk assessment and risk management presented in section 2.2 follow the general framework devised by SCOPE, although the degree of similarity and level of detail presented in each of the models varies (Figure 2.4). In addition to delineating the steps comprising the risk assessment and risk management process, each model distinguishes between the scientific and extra-scientific components of the overall process.

With the exception of the Royal Society model, each of these models explicitly designates hazard identification as the initial step. The NRC/EPA model emphasizes the use of scientific research as the basic tool for identifying risks. Several models more correctly refer to the initial phase as hazard identification rather than risk identification since the probability of the occurrence of an adverse effect is generally not calculated at this stage. In current usage, the term hazard is used to describe the nature of the adverse effect, whereas risk involves both the hazard and the probability of its occurrence (Kaplan & Garrick, 1981; Krewski et al., 1982).

There also appears to be general agreement that hazard identification should be followed by risk estimation. (The Royal Society includes both hazard identification and risk estimation in

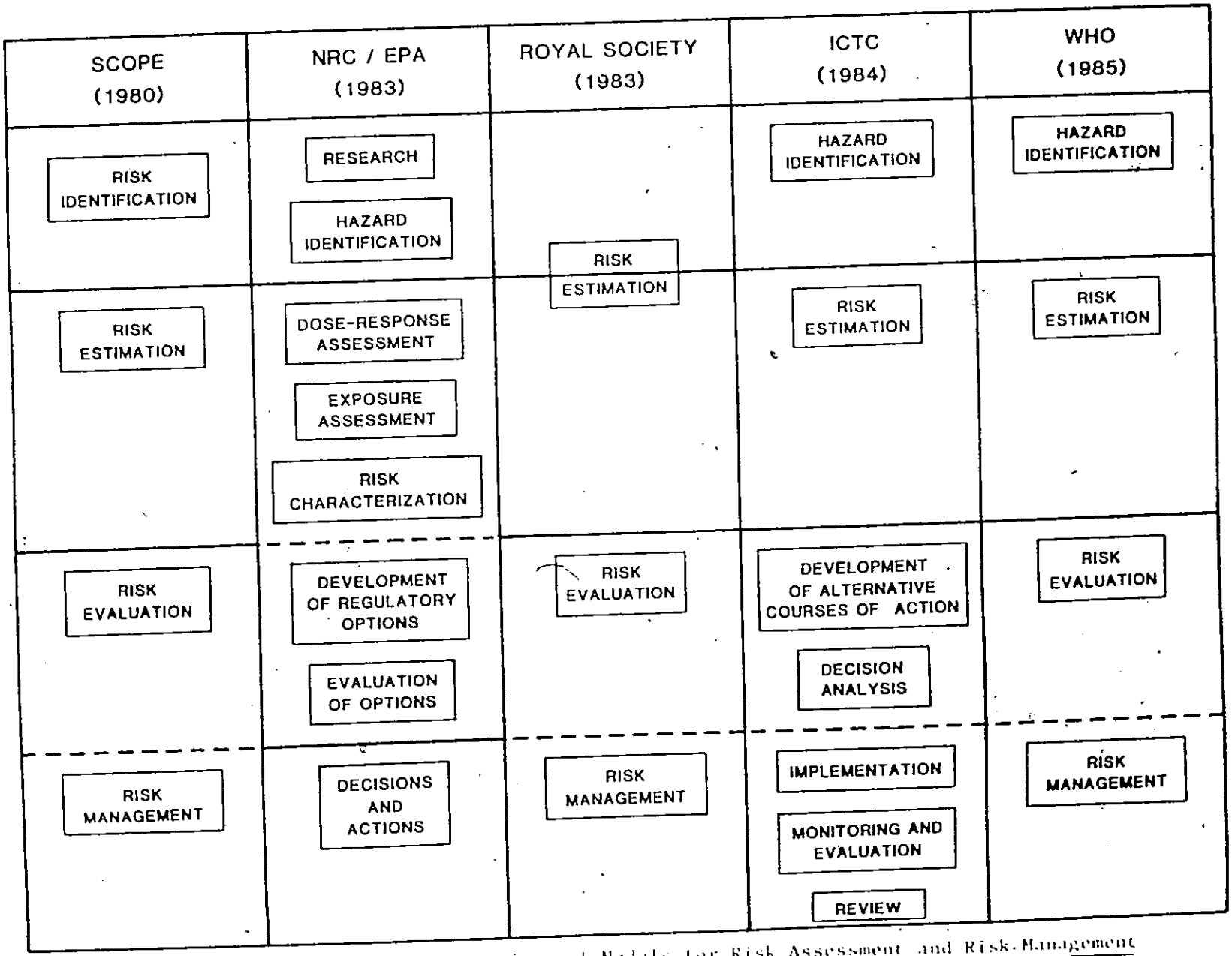


Figure 2.4 A Comparison of Models for Risk Assessment and Risk Management

their definition of risk estimation, but does not clearly identify these as distinct sequential steps.) In the NRC/EPA framework, the term risk characterization is effectively equivalent to risk estimation.

All models cite the use of toxicological and epidemiological data as the primary sources of information for health hazard identification and risk estimation. In the case of chemical hazards, structure/activity analysis may also be used.

As with the original SCOPE model, all models then proceed to some form of risk evaluation. At this stage, scientific method is subsumed by public policy considerations. The subsequent models are generally described in more detail than is the SCOPE model, and, with the exception of the Royal Society model, differentiate between alternative courses of action and the decision analysis tools used to choose amongst them. Alternative options may be of an advisory, economic or regulatory nature, and many factors need to be considered in selecting a preferred management strategy. These include the use of formal economic tools for program evaluation (Torrance & Krewski, 1986), tempered by the public's perception of the risk involved as well as prevailing socio-political factors. Public risk perception and the notion of acceptable risk are important considerations in that decisions made by regulatory authorities ultimately reflect public understanding of risk, perception of its relative importance, and

expectations for risk management (DHSS, 1985). Although not highlighted as distinct components, similar considerations are included within the Royal Society's risk management phase.

The final risk management stage involves the implementation of the selected control strategy. Each of the models, except those of the NRC/EPA and WHO, stresses the need for risk monitoring and follow-up, so as to modify the risk management strategy currently in place if this is considered inappropriate. All models except the SCOPE model also stress the importance of communication at the implementation stage so that affected parties are properly informed as to both the risks and risk management strategy adopted. The Royal Society model in particular emphasizes the importance of providing affected parties with an opportunity to interact in the decision-making process in an informed manner.

Each of the models, except those of the NRC/EPA and WHO, stresses the need for risk monitoring and follow-up, so as to modify the adopted risk management strategy as appropriate. The risk assessment/risk management process is thus generally considered to be a set of interactional rather than sequential steps because decisions must be reconsidered in light of relevant new information (Davis, 1983; Leiss, 1985).

One point which remains obscure is the separation between risk assessment and risk management. The original SCOPE model seems to consider risk assessment as encompassing both the scientific enterprises of hazard identification and risk estimation as well as the more politicized function of risk evaluation. In this model, the term risk management is reserved for the final implementation and follow-up stage. This point of view is also adopted in the Royal Society, ICTC and WHO models. The NRC/EPA, on the other hand, define risk assessment as consisting only of hazard identification and risk estimation.

Taking into account differences in terminology, all models essentially agree on the division of the scientific and social aspects of the risk assessment and risk management process. Hazard identification and risk estimation are clearly in the scientific realm, whereas risk evaluation and risk management fall within the domain of social decision making. Thus, the responsibility for risk analysis rests largely with the scientific community, whereas those responsible for the establishment and implementation of risk management decisions play the leading role in extrascientific matters (Ruckleshaus, 1983).

Although there is general agreement that the risk assessment/risk management framework can be divided into scientific and policy concerns, Davis (1983) maintains that both science and values play a role in risk assessment. Several of the assumptions and approximations employed in risk assessment have political implications; consequently

it is unrealistic to assume that risk assessment can be fully immune to biases resulting from specific interests or viewpoints (Covello & Merkhofer, 1984).

During the risk assessment process, analysis may overlook hazards, deem them unimportant, or ignore them because they are difficult to assess. Decisions are often influenced by policy judgments due to gaps in scientific information. Assumptions must frequently be used in order to fill in gaps in the underlying scientific data base, and to allow for timely decision making. In order to maintain scientific credibility, the inferences and judgments used in such assumptions should be supported by scientific data and the assumptions and their likely impact on the risk assessment outcome be identified and communicated to the public (DHHS, 1985).

The NRC (1983a) has developed inference guidelines to estimate human risk levels in situations where the data is inadequate for direct calculation from human observations. For example, the assumption that risk is directly proportional to the level of exposure is often made in extrapolating laboratory carcinogenesis data obtained at high doses down to the low dose region of interest (Office of Science and Technology Policy, 1985). Such guidelines are explicit statements which pragmatically select one of several inference options for a particular risk assessment situation. The NRC also notes that, despite their scientific orientation, policy considerations virtually always

influence the selection of an inference option. The nature of the inference options for any risk is a mixture of scientific fact and consensus, informed scientific judgment, and policy determinations.

2.4 SUMMARY AND CONCLUSIONS

In this chapter, we have reviewed the major models of the process of risk assessment and risk management. All of these models reflect the basic elements of the original SCOPE model (risk identification, risk estimation, risk evaluation and risk management) as originally described by Kates (1978) and Whyte & Burton (1980). Subsequent models more correctly refer to the initial step as hazard rather than risk identification, reflecting the fact that risk estimation requires a quantitative rather than qualitative description of adverse health effects.

Although all models involve scientific and public policy considerations, the only model which equates these two dimensions with those of risk assessment and risk management is that of the NRC/EPA. In the other models, social evaluation is included in the risk assessment phase, leaving only the implementation of the chosen control strategies to the risk management phase.

The existence of uncertainty in risk estimation cannot be overlooked. Scientific data may be incomplete or plagued with

uncertainty regarding estimates of risk. While quantitative analyses of risk can improve understanding, its limitations must be recognized (Covello & Merkhofer, 1984). Of particular concern is the uncertainty inherent in the assumptions made during the extrapolation of animal test data to the human situation and the determination of current and future exposure levels.

Public involvement in the risk assessment/risk management process is necessary in order to facilitate understanding of the public's perception of risks, and the nature of the values which they place on different health and environmental consequences (Covello et al., 1986). Awareness of the public's views better ensures that they are incorporated into the decision making process, and that the policies which result are socially acceptable. As a result, public support of and compliance with risk management decisions should increase, as should public confidence in agencies responsible for risk assessment and risk management (Starr, 1985; Covello et al., 1986).

It should be recognized that risk management decisions can legitimately differ among jurisdictions even in the presence of a common scientific database. This may occur as a result of varying economic or socio-political conditions, as well as differences in culture or perspective. Effective risk management thus requires an optimal combination of risk analysis, technical feasibility, human intervention and political support (Starr, 1985).

The application of the models of risk assessment and risk management discussed here to practical decision making situations should facilitate identification and clarification of the many important considerations in the complex process of risk assessment and risk management. This is particularly true of some of the more recent models which describe the component steps in detail, including the development of a range of viable risk management options and the criteria and tools to be applied in choosing among these options. Other considerations which may otherwise be overlooked include the need for continual monitoring and review as well as communication of information on risks and risk decisions to all affected parties. Utilization of such models implies an analytical approach to risk assessment and management, and provides a systematic framework within which flexibility may be introduced on a case by case basis.

3. HAZARD IDENTIFICATION

Epidemiology asks the right question and answers it poorly: toxicology asks the wrong question and answers it well.

Jack Semiatycki
Institute Armand Frappier

3.1 INTRODUCTION

There are four main approaches to the identification of human health risks: direct observation of adverse health effects in man; epidemiological studies of human populations; toxicological experimentation using animal models for man; and indirect analytical tests conducted in the laboratory. The risks inherent in driving an automobile, for example, are readily apparent through direct observation although formal epidemiological methods are required to organize the available data and quantify the risks involved. At the beginning of the current era of space exploration, there were no human data available on which to base judgments of risk. Thus, animals were sent into space to obtain some information on potential human health risks. Indirect testing is of most use where animal experiments are difficult or costly to conduct as in assessing carcinogenic potential of environmental chemicals. In this case, a range of short-term tests, including those based on microbial mutagenicity, may provide indications as to whether a particular agent is likely to induce cancer.

In this chapter, we review epidemiological and toxicological methods for hazard identification. Epidemiological studies can range from simple clinical observations of adverse health outcomes in clusters of patients to descriptive studies of mortality and morbidity rates to more analytical studies designed to address specific hypotheses concerning cause and effect relationships. These studies have the advantage of providing information on health hazards directly in man, but can be difficult to conduct due to the complexity of the human environment and the limited sensitivity of some study designs, particularly in detecting small effects.

For these reasons, toxicological experiments conducted in a well controlled laboratory environment are widely employed as a means of individually identifying human health hazards. Highly sensitive tests are now available to evaluate a wide variety of deleterious effects. These include an evaluation of acute and chronic toxicity in animal species, studies of the metabolism of chemical substances, short-term tests for genetic damage, and long-term tests for carcinogenic effects. More recently, new tests for the assessment of behavioural abnormalities and immunosuppression have also come into use. Taken as a whole, all of this information can be used to make useful inferences about the likely effects of toxic substances in humans.

3.2 EPIDEMIOLOGICAL INVESTIGATIONS

Epidemiology is the study of the distribution and determinants of disease frequency in man (MacMahon & Pugh, 1970). The simplest form of epidemiological investigation is the observation of unusual clusters of cases of a rare disease in population subgroups exposed to a common risk factor. While useful in establishing epidemiological hypothesis worthy of further study, such observations require corroboration using more rigorous carefully planned investigations. The observation of three cases of a rare form of liver cancer known as angiosarcoma in workers involved in the manufacture of polyvinyl chloride (PVC), for example, was confirmed in subsequent epidemiological studies to be related to their exposure to vinyl chloride monomer (Purchase et al., 1985). Similar effects have also been observed in animals (Maltoni et al., 1981). The reporting in the media of an apparent excess in the number of cancer deaths in a particular residential area in Montreal, on the other hand, was subsequently shown to be comparable to or less than what would be predicted on the basis of cancer death rates for the province of Quebec as a whole (Spitzer, 1982).

3.2.1 Descriptive Epidemiology

Descriptive epidemiologic methods may be used to provide statistics on patterns of morbidity and mortality in the population at large (Health and Welfare Canada, 1980, 1984a). Longitudinal analysis

of these data can provide useful information on trends in disease occurrence. In Canada, for example, the overall cancer mortality rate in men increased by 32% between 1981 and 1983 (Wigle et al., 1986). Excluding lung cancer, however, cancer mortality actually decreased over this period in both men and women. Extrapolation of current trends suggests that lung cancer will displace breast cancer as the leading case of cancer mortality in women by 1990.

2.2 Ecologic Studies

Ecologic or correlative epidemiological studies involve comparisons of exposure to a specified agent with the occurrence of a particular disease within groups such as communities or geographic areas (Kanarek, 1981). An association between exposure and disease is then held to indicate a possible causal effect. Cairns (1975), for example, noted an apparent correlation between per capita consumption of meat in different countries and cancer of the large intestine in women (Figure 3.1). A similar type of relationship has been noted between breast cancer mortality in women and consumption of fat (Carroll, 1975). Ecologic studies cannot control adequately for possible confounding factors such as occupation, diet and lifestyle and their results serve primarily to suggest the need for more rigorous epidemiological investigation. In the former case, for example, the observed differences in cancer rates could conceivably be due to low

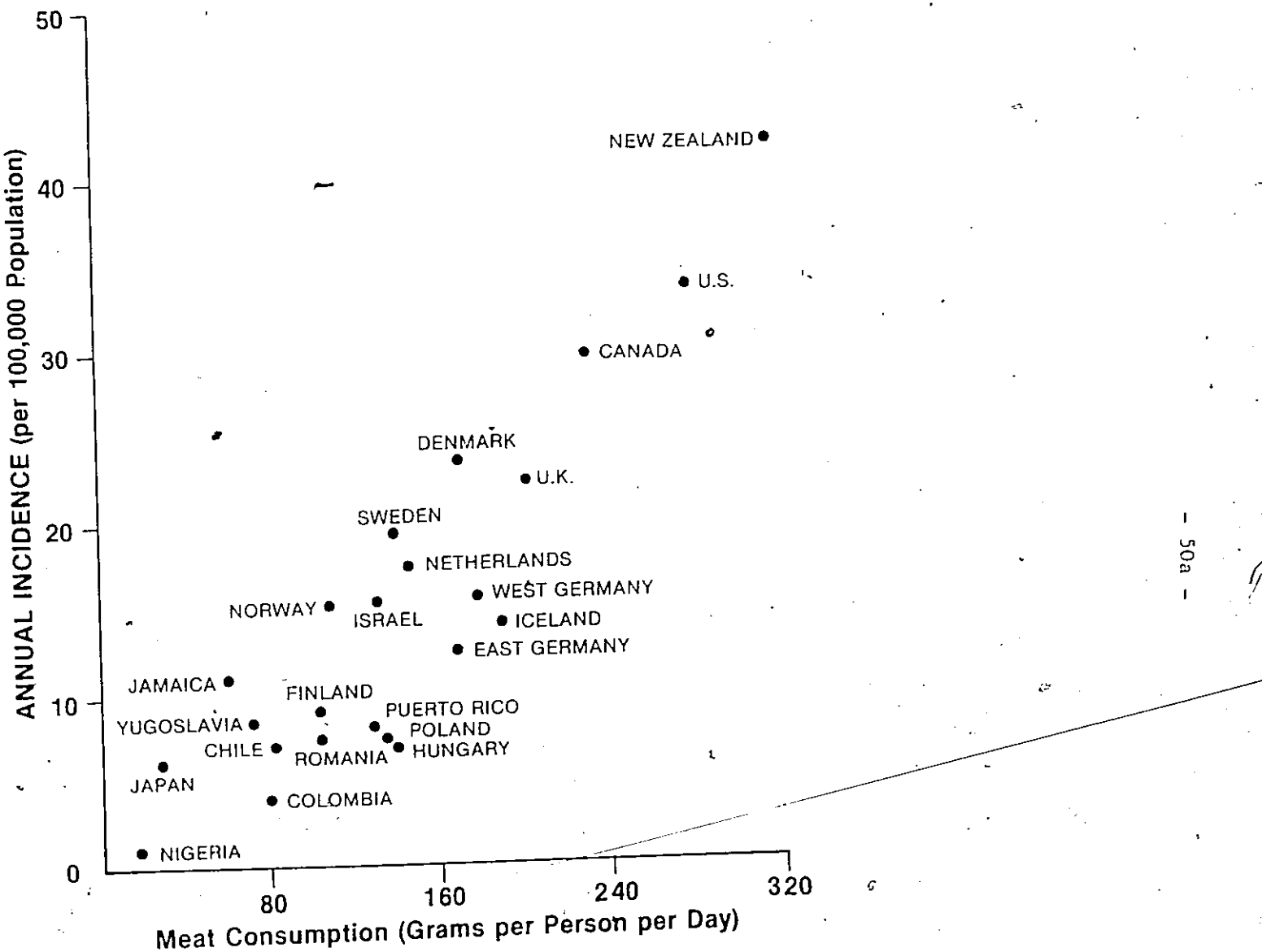


Figure 3.1. Association of Intestinal Cancer in Women and Per Capita Meat Consumption

cereal consumption since this tends to be associated with high meat consumption.

3.2.3 Case-Control Studies

In the most common form of epidemiological study, the case-control study, information on the etiology of the disease under study is obtained retrospectively on both affected and unaffected individuals (Cantor, 1981). The sampling of diseased subjects coupled with a scheme for collecting exposure information retrospectively makes this design well suited to testing etiologic hypotheses for specific rare diseases. Unfortunately, information on exposures such as prescription drug usage and certain lifestyle habits is often less than adequate, especially when it is necessary to look as far into the past as with cancer. Nonetheless, retrospective epidemiological studies have been able to identify certain hazards, such as vaginal cancer in young women whose mothers were given the synthetic hormone diethylstilbestrol (DES) for pregnancy maintenance (Herbst et al., 1971). These authors interviewed the mothers of 8 cases and 32 controls. Case mothers were more likely to have had prior pregnancy loss or bleeding during the relevant pregnancy: this is natural as these were the indications for use of DES. The main difference was the exposure to DES during the relevant pregnancy: 7 out of 8 case mothers and none of the 32 control mothers were exposed. This difference was, of course, highly statistically significant.

3.2.4 Cohort Studies

In prospective (cohort) epidemiological studies (Blair & Spirtas, 1981), information concerning exposure and possible confounding factors is obtained at the outset in the study population. The population is then monitored over a period of time. The length of follow up required is a function of the cohort size, the background incidence rate of the disease(s) of interest, and the desired relative risk detection limit. Often, historical exposure records (e.g., occupational exposure histories) are used with the strong advantage of reducing the time required to accumulate an adequate number of person-years of observation. The latter is usually based on searches of health outcome files such as vital statistics, cancer registries, and hospital admissions. The direct sampling of subjects with variable exposures coupled with a plan of recording health effects as they occur is appropriate for relatively common disorders such as coronary disease, motor vehicle injury, common infections or death from all causes combined. In studies of cancer, the follow up time required may be several decades unless large populations are recruited. Very large numbers of person-years under observation are required for the study of rare diseases. Cohort studies were successful in demonstrating a doubling in overall mortality due to smoking (Doll & Peto, 1976) and more recently in identifying increased lung cancer mortality among passive smokers (Hirayama, 1981).

3.2.5 Administrative Records

Administrative records offer the potential means to conduct low-cost large-scale epidemiological investigations without the need to collect new data. Much valuable information on the health status of the Canadian population is collected on an ongoing basis in the form of hospital records, health insurance data, disease registries and national statistics on morbidity and mortality compiled by Statistics Canada (Chappel et al., 1985). For example, Bates & Sizto (1983) demonstrated a positive association between hospital admissions and air pollution in southern Ontario.

The use of existing computerized databases for epidemiological purposes often requires that linkages be made between records on the same individual that may reside in several different files. The record linkage technology originally proposed by Newcombe (1967) has evolved over the years to the point where it has been successfully applied in a number of major studies (Howe et al., 1979; Roberts et al., 1980). Studies of this type are currently underway within the Health Protection Branch to investigate possible relationships between cancer mortality and occupational exposure to radiation and certain agricultural chemicals.

3.2.6 Interpretation of Epidemiological Data

Even with sound epidemiological methods and an association of statistical significance, one cannot necessarily assume a cause-effect relationship. In judging whether or not a relationship is causal, the strength of the association, gradient of risk versus exposure, consistency of the association or replication, biological plausibility, time sequences of the variables involved, and specificity, should all be taken into account.

Despite the advantage of providing evidence of health effects directly in human populations, epidemiological studies are subject to certain limitations (Leviton, 1984). These include problems of simultaneous exposure to a variety of hazards, the potential confounding effects of unsuspected hazards present in the environment, adequacy of exposure data, and the relative insensitivity of studies of even moderately large size to detect small effects. A major difficulty with environmental carcinogens is the protracted time scale often required for disease development. Since some degree of human exposure must take place prior to the initiation of any epidemiological investigation, adverse health effects cannot be predicted in advance of introducing a new substance into the human environment. Consequently, epidemiological studies alone are insufficient for a purely preventive regulatory model, and are perhaps more often used to support public health decisions rather than to initiate them.

3.3 TOXICOLOGICAL STUDIES

Although providing only indirect information on human health risks, toxicological studies conducted in the laboratory provide valuable information on the toxic potential of environmental chemicals. The modern toxicologist now has at his disposal a large and expanding armamentarium of tests for adverse health effects (Hayes, 1984; Tegeris, 1984). While traditional endpoints such as general toxicity, teratogenesis and carcinogenesis have been studied for many years, more recent developments include tests for immunosuppression, behavioural abnormalities and genetic damage.

3.3.1 Acute Toxicity

Acute toxicity tests are used to assess the potential of the test agent to induce adverse health effects upon exposure to high doses over a short period of time. While not considered to be of great predictive value for human health effects, acute toxicity studies are useful in identifying clinical manifestations of toxicity as well as in selecting the dose levels to be used in studies of longer duration. Acute toxicity studies may also be used to rank the toxic potential of different agents using indices such as the median lethal dose, defined as that dose resulting in 50% mortality in the exposed population.

3.3.2 Subchronic Toxicity

Subchronic toxicity studies may be used to assess potential toxic effects associated with longer periods of exposure. Experimental animals are typically subjected to repeated doses of the test material for periods of 90 days or longer. Several exposure levels are generally used in an attempt to characterize the shape and nature of the dose-response relationship. Subchronic and reproductive studies provide some indications as to potential human health hazards as well as a guide for the selection of doses to be used in chronic toxicity studies.

3.3.3 Chronic Toxicity and Carcinogenicity

Chronic toxicity studies may be used to detect toxic effects which occur only after prolonged exposure. While most toxic effects will be manifest in subchronic studies, certain irreversible progressive effects such as cancer may only occur following extended periods of exposure. Despite the problem of interspecies extrapolation, the chronic animal bioassay remains an important toxicological tool for assessing potential human health risks due to long-term low-level exposure to environmental contaminants.

There are generally three reasons for conducting a carcinogenicity bioassay. First, the experiment may be part of a screening program for

the detection of potential carcinogens. Second, the experiment may enable estimation of risks at low levels of exposure. Finally, the experiment may allow verification of scientific hypotheses about the mechanisms of carcinogenesis. The specific purpose of the experiment has to be kept firmly in mind when considering questions of statistical design and analysis. Since a particular experiment may have more than one objective, moreover, the actual procedures used may represent a compromise that is not best for any one purpose, but is reasonably good for all.

Recent guidelines on experimental design for carcinogenicity bioassays generally recommend three or more dose levels, with at least 50 animals of each sex assigned to each dose. The high dose is generally taken to be the maximum tolerated dose (MTD), which is defined as the dose that is sufficiently great to elicit minimal signs of toxicity, yet not high enough to reduce survival from causes other than tumor occurrence. Statistical procedures for the analysis of carcinogenicity studies have also been the subject of much recent discussion, with extensive recommendations having been made by the International Agency for Research on Cancer (Gart et al., 1986).

When the purpose of the experiment is to discriminate between rival hypotheses of carcinogenic mechanisms, no general guidelines can be given, since the best design will depend heavily on the particular hypothesis being tested. Specific aspects of the carcinogenic process

that could be studied include in utero vs. post weaning exposure, initiation/promotion phenomena, reversibility of lesions, and the effects of mixed exposures. All of these situations require special experimental protocols (Bickis & Krewski 1985). Two generation studies, for example, may be employed in cases where the carcinogen is suspected of exerting its effects as a result of in utero exposure. In order to fully distinguish between prenatal and postnatal effects, several dosing regimens encompassing these two stages of development are required.

3.3.4 Metabolism and Pharmacokinetics

Metabolic and pharmacokinetic studies may be used to determine the absorption, distribution and elimination of the test compound and to determine the rates at which these processes occur. This information is essential in order to develop adequate protocols for subchronic and chronic toxicity studies (Withey 1978). Metabolic and pharmacokinetic data are also invaluable in the interpretation of mutagenicity and other toxic tests and in the comparison of results in a different species.

3.3.5 Genetic Toxicology

Much progress has been made in recent years in the development of short-term tests for genetic alterations using bacteria, fungi, plants,

insects, cultured mammalian cells and small mammals (NRC, 1983b); ILSI, 1984a). These tests are of interest not only because of their ability to identify gene and chromosomal mutations, but also as predictors of carcinogenicity. They also offer economic advantages in terms of time and cost and may in future reduce the need to use laboratory animals for testing purposes.

Of the many available short-term tests, the Ames Salmonella/microsome assay (Ames et al., 1973ab, Maron & Ames, 1983) is the single most widely used and most thoroughly validated test system. The assay uses mutant strains of bacteria which, deficient in their ability to synthesize the essential animo acid histidine, can undergo cell division only in a medium containing sufficient histidine for growth. The mutagenic potential of the test substance is evaluated by adding it to the medium in which the bacteria are grown, and observing the number of colonies formed by bacteria undergoing reverse mutation to histidine independence.

3.3.6 Systematic Approaches to Toxicological Testing

Systematic approaches to toxicological testing have been proposed to deal with the vast array of tests in an orderly fashion. The Food Safety Council (1982), for example, has proposed a decision-tree approach in which the most widely used tests are carried out in a sequential fashion. Weisburger & Williams (1981) proposed a formal

decision point approach designed to divide chemical carcinogens into two broad categories according to whether their mechanism of action involves genotoxic effects or epigenetic effects such as hormonal imbalance, immunosuppression, chronic tissue injury and promotion of previously altered cells. However, it is probably premature to rely solely on genotoxicity testing as a means of distinguishing between high and low risk animal carcinogens (ICPEMC, 1982). A more general categorization scheme has been proposed by Squire (1981), in which information on the nature of the neoplastic events induced in long-term toxicological tests is considered along with evidence of genotoxicity obtained in short term tests. This procedure utilizes perhaps the most relevant toxicological data to construct five different categories of animal carcinogens, thereby providing a possible basis for prioritizing regulatory concerns.

3.4 STRUCTURE/ACTIVITY ANALYSIS

Attempts have been made to see if the chemical structure of the molecule itself may serve as a predictor of toxic or carcinogenic effects. Such predictions are often based on particular active groups, stereochemistry, or electrochemical properties. Some such attributes that have been proposed to establish relative potencies are the number of aromatic rings in polycyclic hydrocarbons, or the number of chlorine atoms in chlorinated hydrocarbons.

The success of such classification rules is fairly good, but is far from perfect, giving misclassification rates of between 1 and 20% for certain classes of compounds (Jurs et al., 1983). This is to be expected when similar molecules such as 2-AAF (2-acetylaminofluorene) and 4-AAF exhibit respective evidence in favour of and against carcinogenicity (Office of Technology Assessment, 1981).

3.5 WEIGHT OF EVIDENCE

In order to reach the best possible decision in labelling a particular substance as hazardous or otherwise, all of the available toxicological and epidemiological data should be carefully evaluated. Consideration should be given to the quality of data, the biological relevance of the health parameters monitored, the consistency of the results between studies, and the magnitude of the effects induced. The overall strength of the data implicating a given material as hazardous may then be assessed using a weight of evidence approach.

This concept has been widely applied in the case of carcinogenic effects. In particular, both the International Agency for Research on Cancer (IARC, 1982a) and the U.S. Environmental Protection Agency (EPA, 1986) have established criteria by which evidence of carcinogenicity in either animals or humans may be categorized as limited, sufficient or inadequate. A similar approach has been suggested by Doll (1985) for reconciling negative epidemiological results with positive toxicological findings.

3.5 SUMMARY AND CONCLUSIONS

Epidemiological and toxicological studies currently provide much of the information used in the identification of human health hazards. Epidemiological studies of human populations have the unique advantage of providing evidence of adverse effects directly in humans. Prospective studies may be employed to evaluate the occurrence of a variety of possible health effects in exposed and unexposed cohorts followed over a period of time. When a specific health endpoint has been identified, retrospective studies may be employed to compare past exposures to affected and unaffected individuals although with certain environmental variables, accurate historical data may be difficult to obtain. Both types of studies can, however, be of restricted sensitivity when exposure is low and may be subject to unsuspected confounding risk factors. Epidemiological studies also necessarily antecede human exposure, and are thus inconsistent with a preventive regulatory philosophy.

Many of these problems can be overcome through the use of toxicological experiments conducted in the laboratory. Here, exposures can be carefully controlled and biases eliminated through the use of sound experimental design. Similarly, sensitivity may be increased through the use of high doses. However, these advantages are achieved at the price of obtaining only indirect evidence of potential hazards in surrogate test systems.

Toxicological tests have been devised to evaluate many different health effects ranging from acute lethality to reproductive and carcinogenic effects. Specialized tests designed to assess immunological and behavioural abnormalities are also available. While most tests are conducted using surrogate animal species, many new procedures designed to detect damage to genetic material have been developed recently for use with primitive microorganisms and cultured mammallian cell lines.

While both toxicological and epidemiological studies clearly have a vital role in human health risk assessment, difficulties can arise when the two approaches are in discordance. This is particularly true when the toxicological data base suggests a potential health hazard yet the available human data is largely negative. While prudence would suggest that the epidemiological studies may simply be insensitive, the possibility that man is genuinely resistant to the effects observed in animals cannot be wholly discounted, particularly in the presence of an extensive series of sound human studies revealing no effects. In general, a weight of evidence approach may be used to evaluate all of the available data and reach a balanced assessment of the potential health risks to humans.

4. RISK ESTIMATION

All things are poisons, for there is nothing without poisonous qualities. It is only the dose which makes the poison.

Paracelcus (1493-1541)

4.1 INTRODUCTION

As discussed in chapter 2, the process of risk assessment and risk management has been the subject of systematic study in recent years. Despite some minor differences in detail, most current frameworks for this process distinguish between hazard identification or the qualitative description of toxic phenomena, and risk estimation in which a more quantitative evaluation of adverse effects is conducted. In this chapter, we consider possible approaches to the estimation of human risks based on both toxicological and epidemiological data.

In section 4.2, we discuss the extrapolation of toxicological data for purposes of human risk estimation. This includes the application of safety factors and mathematical models to experimental data in order to establish human safety standards. The assumptions underlying these procedures are enunciated, and the need to consider all of the available biological data in order to select the most suitable statistical methods for handling risk is emphasized.

4.2 RISK ESTIMATION BASED ON TOXICOLOGICAL DATA

Centuries ago, Paracelsus (1493-1541) noted that "All things are poisons, for there is nothing without poisonous qualities. It is only the dose which makes a thing a poison." This simple observation has two important implications. First, overexposure to almost any substance can pose serious health risks. Second, the level of risk depends on the level of exposure. The fundamental problem of toxicology is thus to determine safe levels of exposure to toxicants present in the environment. In this section, we examine the available procedures for establishing acceptable levels of human exposure to environmental toxicants based on the results of toxicological experiments.

For purposes of risk estimation, toxic processes may be divided into those that are stochastic and those that are nonstochastic in nature. Stochastic processes such as carcinogenesis result from the random occurrence of one or more specific biological events in individuals in a population. The consequence of this is that the response of a particular individual under specified conditions of exposure cannot be predicted with certainty. Nonstochastic events such as enzyme inhibition, on the other hand, are more uniform in nature and occur predictably following exposure to a certain critical level of the toxicant under study. Different approaches to risk assessment are usually employed for these two categories of response.

4.2.1 Safety Factors and Thresholds

The traditional approach to setting acceptable levels of human exposure to environmental toxicants has been to apply a suitable safety or uncertainty factor to that dose level at which no adverse effects were observed in toxicological studies. With contaminants present in food, for example, the no-observed effect level (NOEL) in toxicological tests is divided by the safety factor (SF) to arrive at an acceptable daily intake (ADI = NOEL/SF) for humans (Committee on Food Protection, 1980).

A similar approach may be used to establish threshold limit values (TLV's) for workplace exposures to substances in ambient air (Royal Society Study Group, 1983). The TLV is set so that nearly all workers will experience no adverse effects following repeated daily exposure, and is often supported by observations on occupationally exposed populations.

The safety factor approach to risk estimation seems to have been formalized first by Lehman and Fitzhugh (1954). In a detailed review of this procedure, Dourson & Stara (1983) note that a 100-fold safety factor has often been applied for general toxic effects other than cancer. This allows for a 10-fold variation in susceptibility between animal and man as well as a 10-fold variation in individual susceptibility within the human population. In the absence of a

no-effect level, it has been proposed that the safety factor might be applied to the lowest effect level with an additional factor of 5 or so incorporated so as to adjust for the absence of a no-effect level. For example, a safety factor of 5000 has been proposed for certain kinds of carcinogenic effects (Weil, 1972; Truhaut, 1980), with the additional factor of 10 included as protection against the severity of the response.

In applying the safety factor technique, the underlying assumptions should be clearly understood. The use of safety factors in arriving at acceptable human exposure levels would appear to rest at least tacitly on the assumption of the existence of a threshold dose below which no adverse effects will occur. However, it is precisely because the threshold concept may not be universally applicable to carcinogenesis that the regulation of carcinogens is regarded as a unique issue in risk estimation.

In mathematical terms, the absence of a threshold precludes the possibility that a sufficiently low level of exposure will be free of any attendant degree of hazard. Biological arguments in favour of the no-threshold concept for carcinogenesis are generally based on the fact that irreversible self-replicating lesions may result from a mutation in a single somatic cell, often following the administration of only a single dose. Arguments against this position draw on the existence of metabolic detoxification, DNA repair, immunological surveillance and

other mechanisms that may operate to nullify effects at low doses. Even admitting their existence, thresholds are likely to vary among individuals. The determination of a population threshold thus presents the difficult statistical problem of determining the minimum of the individual thresholds, a minimum which may well be effectively zero in some cases (Brown, 1976). For example, with fifty animals on test, there is a better than even chance of observing no effects with a compound for which the population risk is actually as high as 1% (Cornfield et al., 1978).

Less generally recognized is the fact that the application of a standard safety factor does not take into account the slope of the dose-response curve for the particular response of interest (Cornfield et al., 1980). Clearly, a moderate safety factor may provide an adequate margin of safety if the dose-response relationship is relatively steep but may not be sufficiently conservative if the dose-response curve is relatively shallow. Conversely, the universal application of a very large safety factor will result in tolerances that will often be unduly low.

Despite these limitations, safety factors are widely used as a means of risk estimation based on toxicological data. Provided that the toxic phenomena under study demonstrates a threshold below which no adverse effects will occur, the safety factor approach seeks to set the

ADI below this threshold. However, other methods of risk estimation are required for toxic effects for which a threshold may not exist.

4.2.2 Mathematical Models and Virtual Safety

In the absence of a population threshold, any level of exposure will induce some risk. In this event, the ideal of zero risk can only be achieved if exposure is completely eliminated. Although absolute safety can only be guaranteed in the absence of exposure, sufficiently low levels of exposure may lead to small but acceptable risks.

In practice this involves the application of mathematical modeling techniques to bioassay data in an attempt to determine a virtually safe dose or VSD corresponding to some suitably low level of risk (Figure 4.1). Although originally introduced by Mantel and Bryan in 1961, this procedure has only received widespread attention as a regulatory tool within the last decade. Formally, the VSD is defined by that dose d_0 for which

$$\pi = P(d_0) - P(0),$$

where P(d) denotes the probability of a response at dose d and π is the acceptable risk level.

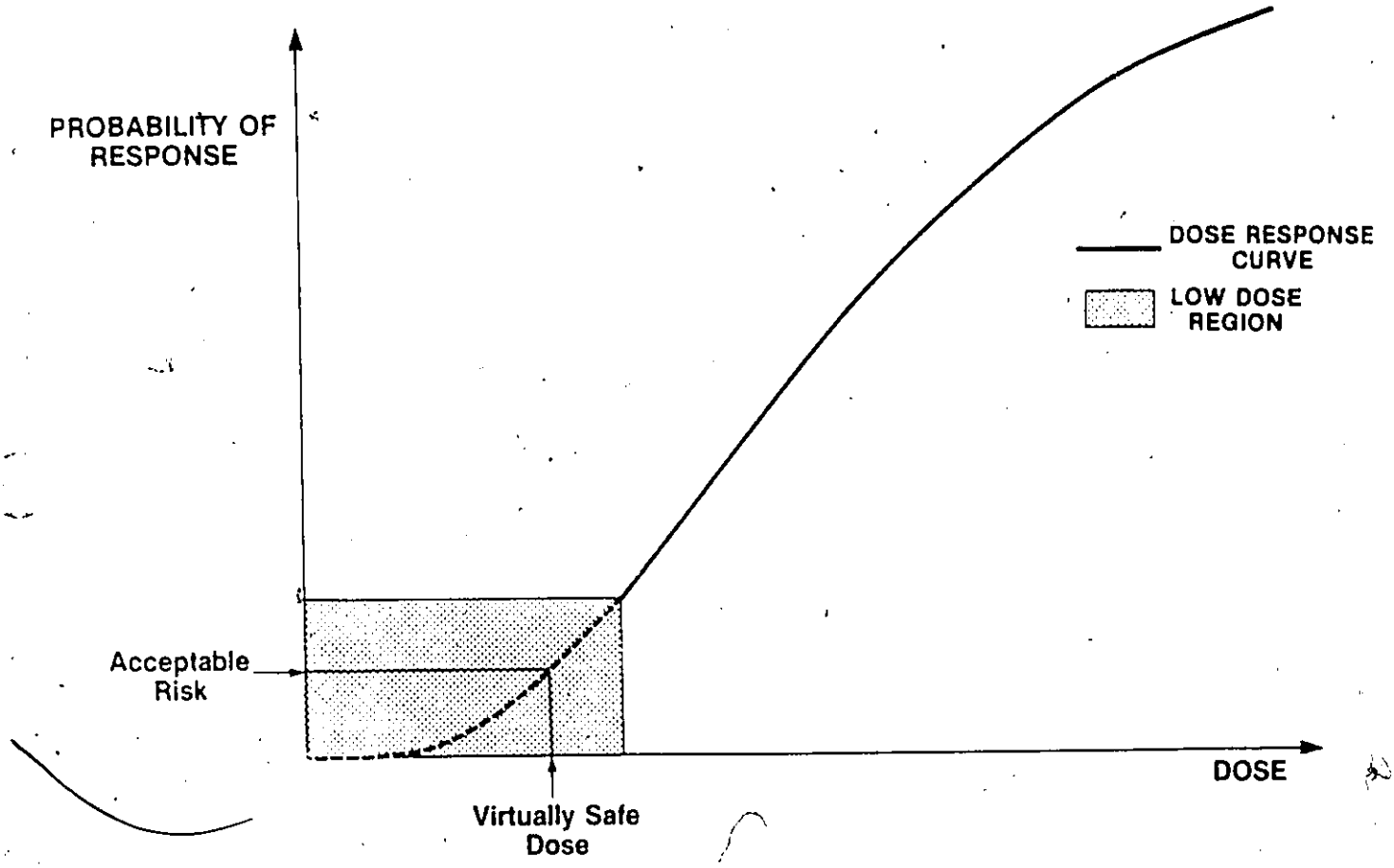


Figure 4.1 Definition of a Virtually Safe Dose

The shape of the dose-response curve in the low dose region can have an appreciable effect on the VSD (Figure 4.2). Dose-response curves approaching zero at a faster than linear or supralinear rate are theoretically possible under certain constructs, such as in a heterogeneous population composed of a high proportion of susceptible individuals (Hoel, 1985). From the practical point of view, however, such a disproportionate increase in risk with increasing dose seems somewhat questionable. Many toxicologists currently seem to feel that dose-response relationships for carcinogenic agents will be at worst linear in the low dose region. For compounds for which spontaneously occurring lesions can be modeled as arising from an effective background dose, it can be shown quite generally that low dose linearity will in fact obtain (Crump et al., 1976). Other observations consistent with such behavior for compounds acting through direct interaction with genetic material include the linearity of many mutagenicity data sets at subtoxic doses and the linear relation between the formation of DNA adducts and low doses of several alkylating agents (Hoel et al., 1983). For compounds that are thought to produce tumors as a result of secondary effects induced by metabolic overload at high doses, on the other hand, even sublinear or threshold effects cannot be ruled out (Clayson et al., 1983). Because of the statistical difficulties in determining thresholds, however, most mathematical models have dispensed with the threshold concept (Brown, 1976).

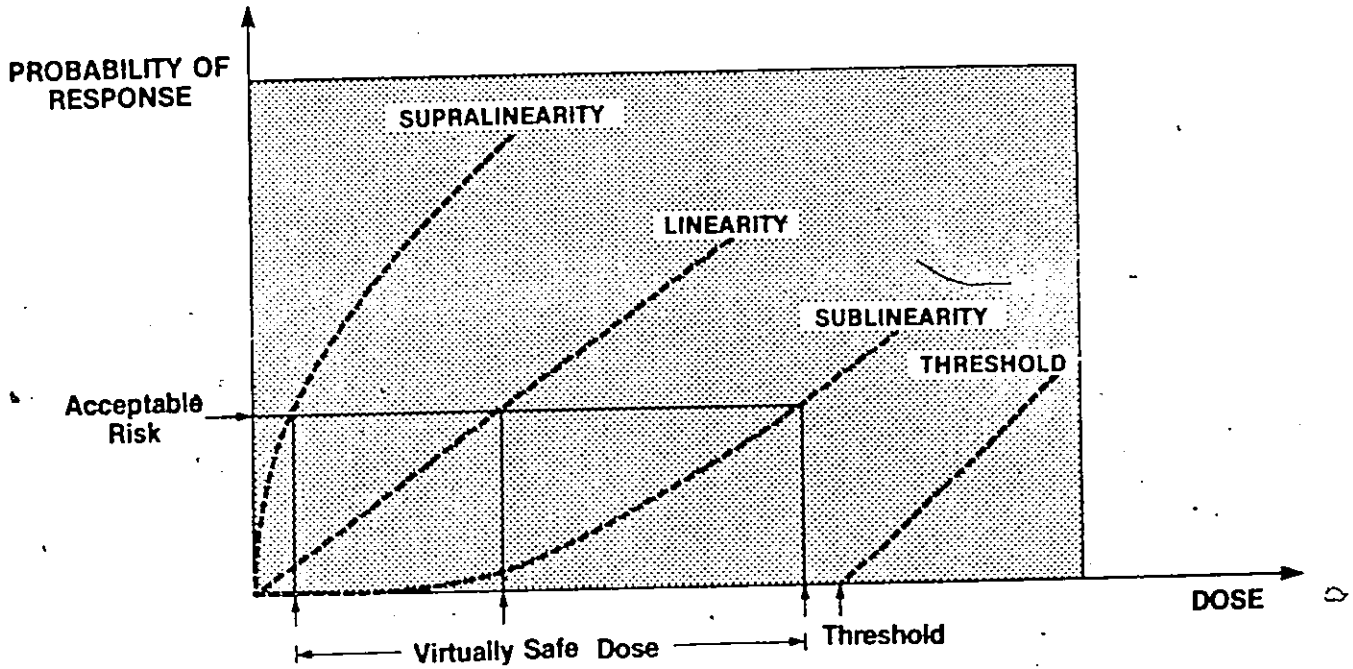


Figure 4.2 Possible Shapes of the Dose Response Curve in the Low Dose Region

The many mathematical models proposed to date vary in their degree of biological sophistication (Munro and Krewski, 1981; Brown and Koziol, 1983). Simple tolerance distribution models such as the probit, logit, and Weibull postulate the existence of a certain distribution of tolerances within the population, with the absence of a population threshold reflected in the fact that the minimum of the individual tolerances is allowed to be zero. Stochastic models such as the multihit or multistage assume that a positive response is the result of the random occurrence of a specified number of hits at the target tissue or the completion of a certain number of stages in the response process. Other approaches visualize the host as being composed of a number of distinct biological compartments with the rate of tumor occurrence being a linear function of dose within the critical compartment (Hartley et al., 1981).

For purposes of estimating the VSD, the linear hypothesis may represent a realistic position with respect to the low dose behavior of some carcinogenic agents. For others displaying sublinearity or even threshold effects, moreover, linear extrapolation will provide an upper limit on the actual risk in the low dose region. This position is supported by the U.S. Office of Science and Technology Policy (1985), who stated that "when data and information are limited, models or procedures which incorporate low-dose linearity are preferred when compatible with the limited information".

Pharmacokinetic models offer considerable promise as a means of improving estimates of risk by relating the response rate not to the level of exposure to the agent of interest, but to the dose of the reactive metabolite reaching the target tissue (Krewski et al., 1987). For example, physiologic pharamcokinetic models envisage the body as being comprised of a number of relevant physiologic compartments, and can be used to describe the uptake, distribution and metabolism of chemical substances once they enter the body. This is of importance since the relevant tissue dose is not always proportional to the external level of exposure.

4.2.3 Measures of Potency

Historically, Twort and Twort (1930, 1933) and Iball (1939) proposed several measures of potency in an attempt to summarize the data obtained from their experimental studies. For example, one of their measures was based on the time at which 25% of the animals would have developed tumours. Irwin and Goodman (1946) subsequently considered other related measures of potency, and noted that the different indices tended to give somewhat similar results. Bryan and Shimkin (1943) suggested the use of the dose required to induce tumours in 50% of the exposed animals, the quantity on which Meselson and Russel's (1977) index is defined.

More recently, Jones et al. (1981) considered the use of the time until 50% of the exposed animals would die from the tumour of interest as a measure of potency. Crouch and Wilson (1981) took the slope parameters in the one-hit model as a measure of potency. Noting that the probability of tumour induction $P(d) \simeq \lambda d$ for low levels of exposure d, Crouch and Wilson also proposed using the value λ as a means of estimating the response rate P at a given dose d. This will be reasonable when the one-hit model, which is essentially linear even at moderate doses, provides an adequate description of the observed dose-response curve, but will be less satisfactory when the dose-response curve is highly nonlinear.

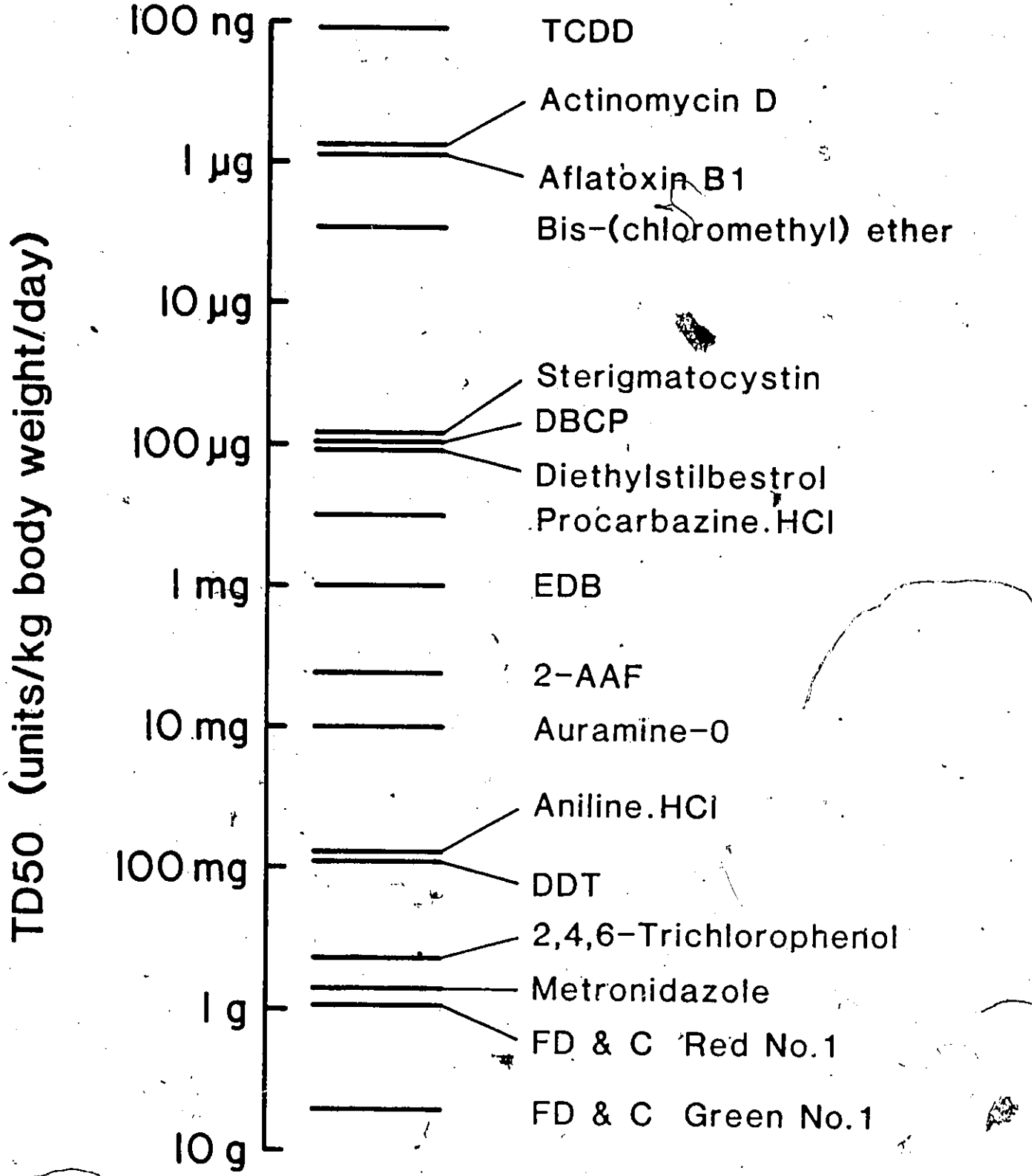
Sawyer et al. (1984) proposed a potency index based on the TD_{50}, or dose estimated to induce tumours in 50% of the exposed subjects. The method provides for the effects of both dose and time, and, like the method of Crouch and Wilson, is based on a simple exponential or one-hit model for the effects of dose. In cases where the time to tumour occurrence may not be directly observable, Sawyer et al. used the time to death with tumour present as a surrogate for the observable failure time (see Peto et al., 1984, for further discussion of this point).

The index proposed by Sawyer et al. (1984) has recently been calculated using an extensive data base of known animal carcinogens compiled by Gold et al. (1984). Expressing the TD_{50} in terms of the daily intake of the compound relative to total body weight, this analysis revealed potencies varying over more than seven orders of magnitude (Figure 4.3). Only a few nanograms of TCDD, for example, were required to induce a 50% tumour occurrence rate during the course of a rodent lifetime, whereas several grams of the food colours FD & C Red No. 1 and FD & C Green No. 1 were required to elicit the same rates of response.

4.2.4 Species Conversion

Extrapolation of risk estimates between species must be done with great caution. Each species has distinct physiological, biochemical and anatomical characteristics that differentiate it from other species. Interspecies extrapolation is understandably more difficult with complex toxicological effects such as teratogenesis, mutagenesis, and carcinogenesis because of our incomplete knowledge with respect to the underlying mechanism of action.

Differences in carcinogenic effects between rats and mice are well documented (Purchase, 1980). Aromatic amines likewise exhibit appreciable differences in carcinogenic potential in a variety of different species (Garner et al., 1984). In contrast, however,



Range of carcinogenic potency in male rats

Figure 4.3 Range of Carcinogenic Potency in Male Rats

nitrosodiethylamine appears to be an effective carcinogen in all 18 species tested (Schmahl et al., 1978).

Interspecies extrapolation is of critical importance when the target species is man. Of the handful of chemicals known to cause cancer in man, the vast majority are also active in one or more animal species. Thus, it seems reasonable to consider chemicals found carcinogenic in animals to be presumptive human carcinogens. Of the 11 known human carcinogens which have been adequately tested in either the rat or the mouse using a comparable route of exposure, the majority appear effective in at least one of the same sites as in man (Munro & Krewski, 1982). Although the degree of correspondence in terms of site-specifity is not perfect, it seems reasonable to conclude that animal tests may serve as a fair predictor of human risk, at least on a qualitative basis.

One area which offers some promise for improvement in the manner in which risk estimates are translated from one species to another is pharmacokinetic modelling. By constructing physiologic models to describe the metabolism of a substance and the concentration the reactive metabolite at the target tissue, it may be possible to obtain better predictions of tissue concentrations in tumours if the appropriate model parameters can be determined (Fiserova-Bergerova, 1983). This approach has recently been employed by Anderson et al.

(1986) to improve the estimates of cancer risk posed by methylene chloride, a substance found in trace quantities in drinking water.

At present, fully satisfactory techniques for quantitative trans-species extrapolation generally do not exist. Until they are developed it is prudent to rely on the assumption that humans will be at least as sensitive to a stochastic effect as the most sensitive experimental species (Clayson, 1985). However, the price to be paid for this assumption is the possible cost of overestimating the risk of some carcinogens in the human population. For example, Booth et al. (1981) have presented evidence that human liver may be considerably less sensitive to the carcinogenic effects of aflatoxin B_1 than rat liver.

4.3 RISK ESTIMATION BASED ON EPIDEMIOLOGICAL DATA

4.3.1 Measures of Disease Frequency

Two commonly used measures of the frequency of disease or death in epidemiological studies are "prevalence" and "incidence". Prevalence is simply the proportion of a particular population which is affected by the disease in question at a specific time. (Note that prevalence does not take into account the time of disease occurrence, only that it is present at that time of observation.) Incidence is the proportion of disease-free individuals developing the disease during a specified

period and is more appropriate for relating occurrence to potential causative risk factors.

Rates are used to express the frequency of occurrence within a specified time frame. The "incidence rate", for example, is simply the incidence of disease occurrence divided by the duration of the period of observation. For example, male and female mortality rates across all age groups in Canada are 836 and 603 deaths per 100,000 population per year. Because the incidence of most diseases varies with both calendar time and age, however, time or age-specific incidence rates are necessary in order to describe the longitudinal development of disease patterns. Such time-dependent rates are often described in terms of the "hazard rate", which is obtained by letting the time interval on which the incidence rate is calculated become arbitrarily small (Kalbfleisch & Prentice, 1980). (This "instantaneous" incidence rate can in fact be shown to be the rate at which prevalence is increasing at a given point in time relative to the proportion of the population unaffected at that time.) The hazard rate for human mortality, for example, has a bathtub like shape due to infant mortality and childhood diseases and the effects of aging.

Although time-dependent rates may be most informative, it is sometimes convenient to employ a single summary measure of disease occurrence encompassing the entire period of interest. One such measure is the "cumulative incidence rate" obtained by simply summing

up the component time or age-specific incidence rates (Breslow & Day, 1980). When comparisons between different populations are made, such cumulative rates should be standardized so as to reflect differences in the age distributions between populations (Fleiss, 1981).

The data obtained from many prospective and retrospective studies can be summarized in a 2x2 table of the following form. Here, the entries in the body of the table represent the number of individuals falling into each of the four possible categories defined in terms of disease and exposure status.

Exposure Status	Disease Status		
	Present	Absent	Total
Exposed	a	b	$m_1 = a+b$
Unexposed	c	d	$m_2 = c+d$
Total	$n_1 = a+c$	$n_2 = b+c$	$n = a+b+c+d$

Note that in the retrospective case-control study, groups of diseased and disease free individuals are first identified and then compared with respect to their historical exposure to the risk factors in question. In the prospective cohort study, on the other hand, the exposed and unexposed groups are first identified and then followed in order to determine their disease status at some future time.

With a single disease state, risk may be conveniently summarized in terms of the probability of disease occurrence. Thus, the risks among exposed and unexposed individuals may be estimated by a/m_1 and c/m_2 respectively. The total risk is n_1/n and the excess risk among the exposed individuals is $(a/m_1)-(c/m_2)$. The relative risk of exposed to unexposed individuals is given by $(a/m_1)/(c/m_2)$. The odds ratio, or the relative odds of developing the disease in the exposed group as compared to the unexposed group, is $(ad)/(bc)$. Finally, the population attributable risk is $1-((c/-m_2)/(n_1/n)$. This represents the proportion of the total disease burden which may be attributed to exposure to the risk factor of interest.

In retrospective studies, the values of m_1 and m_2 in the population at large are not known, so that the relative risk is not estimable. With rare diseases, however, the odds ratio will provide a good approximation to the relative risk. In order to estimate the population attributable risk with cohort studies, the frequency of exposure in the population at large is required. Similarly, with case-control studies, supplementary information on the frequency of disease occurrence in the population at large must be obtained.

4.3.2 Health Risks in Canada

Many of the environmental risks which have received public attention lie in the area of public health (Richmond et al., 1981).

Since accurate measures of morbidity are largely unavailable, such health risks are usually expressed in terms of various measures of mortality. In Canada, for example, almost 90% of deaths are due to familiar medical disorders with the predominant causes of death being cardiovascular disease and cancer (Health and Welfare Canada and Statistics Canada, 1980). Taken as a group accidents, poisoning and violence represent the third leading cause of death in Canada and are becoming relatively more important as other causes of mortality decline at a faster rate (Nicholls & Davies, 1980). Occupational deaths account for a small proportion of all accidental deaths and have been decreasing in recent years (Pochin, 1981).

While our overall national risk profile may differ from that of other nations, the health risks faced by Canadians are remarkably similar to those in other industrialized countries with comparable lifestyles. Elsewhere, in less developed societies, the predominant health risks remain those associated with the lack of a safe and nutritious food and water supply, the prevalence of communicable disease and other natural hazards.

Of the many diseases for which epidemiological data has been acquired, cancer occupies a place of special concern. Sufficient epidemiological evidence has now accumulated to demonstrate that ionizing radiation as well as some 36 chemicals and industrial processes are known or strongly suspected to be associated with the

occurrence of one or more forms of human cancer (Bartsch et al., 1982). Of these agents, all but one or two have also been shown to be effective in one or more animal species. Many more chemical compounds are also known to be carcinogenic in animals (Sax, 1981), and may therefore be considered as presumptive human carcinogens.

Taking this and other information into account, Doll & Peto (1981) attempted to estimate the proportion of the total cancer burden which could be attributable to specific causes in the U.S. population under age 65 (Table 4.1). Similar estimates have also been provided by Miller (1984) and Cole (1984). Although these figures involve a considerable degree of judgment on the part of these investigators, there seems to be consensus on the fact that tobacco is responsible for nearly 30% of the total cancer burden. Estimates of the proportion of cancer mortality attributable to diet were less consistent, reflecting our relatively poor understanding of the relationship between diet and cancer. (Although their best estimate of cancer mortality related to diet was 30%, Doll & Peto further indicated this figure could be as low as 10% or as high as 70%.) With the possible exception of occupation; the remaining determinants of cancer were all considered to have attributable risks of less than 10%.

Table 4.1 Estimates of the Percentage of Cancer Deaths Attributable to Known Causal Factors

Factors	Canada	United States	
	Miller (1984)	Cole (1984)	Doll & Peto (1981)
Tobacco	26	30	30
Diet	32	8	35
Occupation	9	3	4
Radiation*	2	2	3
Alcohol	6	2	3
Reproductive patterns	4	1	7
Sexual activity	3	?	
Licit drugs	1	1	1
Viruses	?	1	5
General environment	?	1	2

* Including sunlight

4.3.3 Canadian Health System Statistics

Federal, provincial and municipal governments in Canada share the responsibility for matters relating to public health. At the federal level, the Department of National Health and Welfare is the principal agency in this regard and is responsible for the overall promotion, preservation and restoration of the health of Canadians as well as for their social security and social welfare. The Department is concerned with data on health products, health hazards and certain disease areas; they also operate a medical care data bank, and integrate health statistical data from various sources.

The main federal agency responsible for health statistics is Statistics Canada. Through its Health Division, it has established information systems relating to vital statistics, special diseases, health personnel and hospital and institutional care (Statistics Canada 1980). Although the collection of the original data in these areas is the responsibility of the provinces, the Statistics Canada Act of 1971 authorizes the national statistical agency to collate this information and publishes a number of national reports. In practice, this is done through federal-provincial agreements prepared to facilitate national reporting.

Statistics Canada maintains complete records on deaths which occur in Canada, including age, location and cause as identified by the widely recognized International Classification of Disease Code. From this information on mortality, the life expectancy of Canadians can be estimated. The current life expectancy is about 70 and 77 years for males and females, respectively (Statistics Canada 1979).

Hospital statistics can provide much information on specific illnesses and disabilities as well as patterns of treatment. This allows tabulation of the number of patient admissions and separations (by death or discharge) and of the length of stay in hospitals. Some provinces also keep detailed records of diagnoses of patients' conditions derived from physicians' medical care insurance claim forms. Although these hospital morbidity data are a very comprehensive source of information on illness and disability patterns, this source has limitations. There are no data on illnesses which are self-treated or

on those which improve before admission to hospital. There is also relatively little information on the chronically disabled or on the number of days Canadians remain at home because of illness.

Besides compiling general mortality and hospital morbidity data, Statistics Canada maintains special registries and undertakes special analyses relating to particular disease conditions and their treatment. Some of these information systems are developed in cooperation with voluntary agencies. In 1969, for example, Statistics Canada started a national cancer reporting system in cooperation with the National Cancer Institute of Canada and nine existing provincial tumour registries. Statistics Canada also operates a renal failure register in cooperation with the Kidney Foundation of Canada and Health and Welfare Canada. Its purpose is to register and follow all patients requiring artificial kidney treatment (chronic peritoneal or hemodialysis) or receiving kidney transplants since 1973.

Other data are derived from notification which physicians are required under provincial laws to make to public health authorities. These include venereal diseases, streptococcal sore throat and scarlet fever, measles, rubella, hepatitis, tuberculosis, whooping cough and several others. Thus, additional information is available on the incidence and treatment of these diseases, although it has been estimated that the number of cases of venereal disease may be subject to significant underreporting.

In addition to these ongoing programs, health statistics may also be obtained through special surveys conducted from time to time. The Nutrition Canada survey conducted during the period 1970-1972, for example, was designed to assess the nutritional status of the Canadian population by means of personal interviews with over 22,000 individuals selected from all parts of Canada. This survey provided data on food consumption patterns and corresponding nutrient intake (Health and Welfare Canada, 1975, 1976· Myers & Kroetsch 1978) as well as descriptive information and dental health (Health & Welfare Canada, 1977a) and height, weight and other body dimensions (Health & Welfare Canada, 1980). Similarly, the Canada Health Survey conducted during 1978-1979 provided information on the general health status of the Canadian population (Health & Welfare Canada and Statistics Canada, 1981).

4.4 SUMMARY AND CONCLUSIONS

While epidemiological studies apply directly to man, they can be subject to certain biases as well as the confounding effects of unidentified risk factors present in the environment. While such effects can be more stringently controlled in toxicological experiments conducted in a controlled laboratory setting, the use of toxicological data for purposes of human risk assessment presents the difficult problem of trans-species extrapolation. Both approaches can provide quantitative estimates of human health risk, although the use of

toxicological data for this purpose is generally subject to the further uncertainties involved in extrapolating from high experimental doses to lower human exposure levels.

The determination of risks associated with low levels of exposure is difficult with either toxicological or epidemiological data since this necessarily entails extrapolation from higher exposure levels where risks can be accurately measured. With toxic effects other than carcinogenesis or mutagenesis, acceptable levels of human exposure have traditionally been established through the application of a suitable safety factor to the dose level at which no adverse health effects were observed in toxicological tests. This is based on the assumption that there exists a threshold dose below which the risk is zero.

With carcinogens and mutagens, the existence of a threshold is not considered a prudent assumption. In this case, it is generally assumed that the excess risk will be directly proportional to the level of exposure in the low dose region. While absolute safety can thus be achieved only by reducing exposure to zero, a virtually safe dose corresponding to some suitably low level of risk may still be considered.

5. REGULATORY AND NONREGULATORY OPTIONS FOR RISK MANAGEMENT

5.1 INTRODUCTION

The effective control of hazards to human health may be achieved in a variety of ways. Historically, common law actions such as claims of negligence or product liability have been employed in an attempt to ensure product safety (Baram, 1982). The inadequacy of such measures has contributed to the subsequent proliferation of regulations, promulgated by government agencies in an attempt to provide better public protection from unnecessary health risks. More recently, the current trend towards regulatory reform and deregulation has prompted governments to give increasing consideration to nonregulatory alternatives for risk management.

Options for the control of risks may be broadly classified as regulatory, economic, advisory, or technological (Krewski, 1986). Unlike regulation, which prescribes safety criteria to be enforced by government sanctions, economic approaches to managing risk employ economic incentives or disincentives to limit the introduction of anthropogenic pollutants into the environment. Advisory options for risk management are based on the provision of advice to either producers or consumers of risk, so as to promote risk avoidance. The development of new technology for pollution abatement may also serve to reduce risk without invoking any of the preceeding options for risk

management. The selection of the most suitable control option for a particular risk management situation must take into account a variety of factors, including the nature of the risk, public acceptability of the option under consideration, and the mandate of the responsible authority to implement the option (Lave & Menkes, 1985).

In this chapter, we review the risk management options currently available to public health decision makers. The regulatory, economic, advisory and technological approaches for controlling risk are discussed in sections 5.2 - 5.5 respectively. Consideration is also given to factors which impact upon the selection of a spetific option (section 5.6).

5.2 REGULATORY OPTIONS

Regulatory approaches to risk management rely on governmental authority to enforce compliance with specific health and safety requirements (Marchant, 1984). The most stringent regulations utilize prosecution or direct orders to comply as means of enforcement. However, other regulatory options such as self regulation and the issuance of permits or approvals for certain undertakings may provide more flexible means of regulatory control, in that they allow for greater industrial participation in decisions regarding what level of control is feasible and how to achieve that level of control.

5.2.1 Direct Regulation

In the past, direct regulation has been the most common approach to risk management. Although a detailed discussion of regulatory statutes in Canada is deferred to Chapter 6, we note here that regulations generally allow comparable standards to be applied to risk producers, yet may permit some flexibility to accommodate specific situations (Marchant, 1984). In some cases, regulation is an effective and necessary way of addressing problems and circumstances that cannot be handled by any other means (Reschenthaler, 1979). Governments have the mandate to ensure the social and economic well-being of their citizens; consequently the use of regulation attempts both to protect society and to allow industry the freedom to operate efficiently and prosperously (Ashford et al., 1985).

A number of factors have contributed to the continuing use of regulation, including society's growing consciousness over health, safety, and environmental concerns coupled with increasing public demand for greater protection from environmental hazards (Covello & Mumpower, 1985). The rapid growth of our complex technological society and the rising cost of technological risks have served to promote the use of regulation as a means of coping with scientific and technological developments. Uncertainties as to the immediate and long term effects of chemical substances and other environmental hazards

have also raised concerns which in the past have often been the object of regulation.

Although the use of regulation is appropriate in many situations, it is subject to certain limitations. For example, regulation may not allow for the economically efficient distribution of clean up costs amongst effluent sources (Spence & Weitzman, 1978). Regulation can engender significant costs, institutional changes, job dislocations or loss, more stringent government control of industry, and growth of government bureaucracy (Baram, 1982). The use of regulation may also discourage the development of industrial initiatives to reduce emissions and to undertake research and development programs to identify new pollution abatement technologies.

Once enacted, regulation is generally not readily amenable to change. Further, the effectiveness of any regulation depends on how well it can be enforced. In short, the cost, clumsiness, and coercive nature of direct regulation has served to open the door to the use of less draconian alternatives (Anderson et al., 1978).

5.2.2 Self Regulation

Private industry frequently develops technical documents which provide guidance concerning use of their products. Self regulation allows industry to utilize such standards for risk management (Baram,

1982). While standards may be required to meet with the approval of government, the acceptance of private standards does not preclude the subsequent introduction of regulations if required.

The use of private standards as a regulatory device in effect increases government's technical and economic expertise, while reducing the drain on its own internal resources. Use of such standards provides flexibility, as they can be tailored to the needs of individual risk producers. This acts to facilitate compliance in that risk producers are encouraged rather than forced to be good corporate citizens (Baram, 1982).

As an example, many airlines and intercity bus lines in Canada have recently acted on their own initiative to prohibit cigarette smoking on some of their vehicles. Such policies provide effective protection from tobacco smoke for many travellers, at no cost to government.

Despite its advantages, self regulation has been subject to many criticisms. In some situations, government and industry may fail to negotiate mutually acceptable standards, thereby rendering self regulation infeasible (Marchant, 1984). Even if compatible standards are produced, arrangements between government and industry may be seen to lack public scrutiny. Further, allowing industry to assume a major role in the development of its own standards may result in

nondisclosure, although this may be avoided if appropriate safeguards are built into the regulatory system (Baram, 1982).

5.2.3 Permits/Approvals

This regulatory option requires risk producers to obtain permits or approvals from government prior to engaging in a particular activity. The specific requirements for permission or approval are usually negotiated between government and industry on a case-by-case basis within the limits of established guidelines (Marchant, 1984).

The permit/approval system is useful in that it addresses risk problems before they arise. Further, this option provides sufficient flexibility to allow industry to choose its own technology for risk management. Requirements can be tailored to specific circumstances and may provide an opportunity for consultative inputs by other interested parties. This approach can be problematic, however, in that an equitable treatment of risk producers may be difficult to achieve (Marchant, 1984). Further, technical solutions may also become obsolete over time and sanctions for noncompliance may weaken.

5.3 ECONOMIC OPTIONS

5.3.1 Insurance and Other Compensatory Schemes

Insurance and other compensatory schemes basically serve to ensure that those parties who are adversely affected by the risk producing behavior of others are duly compensated for their losses, regardless of the financial status of the risk producer (Baram, 1982). Such approaches also ensure that the offender does not suffer severe financial loss as a result of a single claim.

The most common economic options of this type include liability insurance, compensation funds, and automatic indemnity mechanisms such as bonding, escrow and restoration funds. While such options can serve to produce strong incentives for risk reduction, they only do so in cases where the cost associated with implementing the option outweighs the economic benefits associated with the risk producing activity.

Liability Insurance. Liability insurance consists of a premium-based public or private insurance aimed at compensating people or property subjected to injury (Marchant, 1984). Such a system is advantageous in that it increases the likelihood of compensation for injured parties, and forces the risk producer to pay for damages as well as risk alleviation.

The cost of such insurance may, however, be very large. Further, the system may be costly to implement or adjudicate through litigation,

and may suffer from the difficulties associated with legally demonstrating cause and effect. The existence of insurance against one risk may also generate undue pressure for other risks to be covered, ultimately leading to mandatory no fault underwriting of all risks.

Liability insurance has been used with some success in the United States, particularly with regard to the Black Lung Program and Price Anderson Insurance (Baram, 1982). The Black Lung Program provides cash benefits to miners suffering from pneumoconiosis, or black lung disease, as a result of chronic occupational exposure to coal dust. The financial responsibility for compensation is allocated between the risk source and the federal government. Such a program provides a means of dealing with the substantial risk arising from necessary but dangerous mining activities which are not readily amenable to technological solutions. Although the program is advantageous for its beneficiaries, the financial burden of compensation ultimately rests with the public.

Price Anderson Insurance attempts to ensure that the general public would be compensated if an accident occurred at a licensed nuclear energy facility, and sets a limit on the liability of private industry so as to encourage research and development. The program requires each nuclear facility to possess insurance coverage of $140 million (1982 dollars) for public liability claims. The federal government then provides indemnity protection in excess of private insurance to a fixed limit of $560 million. The program has a built-in sunset mechanism: each facility pays $5 million in the event of a

disaster, thus reducing government subsidization as the number of facilities increases (Baram, 1982).

Price Anderson Insurance creates extensive peer pressure to reduce risk, as premium rates are influenced by the operating experience of the entire industry. Although the scheme facilitates an equitable distribution of compensation, there are few instances where public policy will allow such an indemnity partnership between government and industry. Traditional liability insurance mechanisms also promote a cautious approach to the adoption of new hazardous technologies and may thus discourage rapid technological development which may entail increased risk.

Compensation Funds. Compensation funds are government administered mechanisms for imposing liability without fault. In Ontario, for example, the Workmen's Compensation Board provides cash benefits and medical costs to victims of work-related disabilities (Workmen's Compensation Act, 1980). Employers are required to contribute a fixed sum to the fund for each employee, with compensation paid from an industry-wide pool. Employer premiums vary with the amount of compensation paid across the industry.

Compensation funds promptly provide necessary financial benefits to injured workers, serve to avoid litigation, and provide an economic incentive for employers to reduce risks. As a result, they indirectly

contribute to alleviating the effects of injury to workers, employers, and society at large.

Despite their advantages, compensation funds are plagued with certain difficulties. Uniform definition of conditions required for compensation must be established to ensure industry-wide equity. Economic incentives may only stimulate an immediate rather than a continuous effort for risk reduction. Further, the program is only truly effective to the extent that costs are internalized or absorbed by risk producers and not transferred to risk consumers. In cases of chronic disability, a causal relationship may be difficult to establish.

Automatic Indemnity Mechanisms. Automatic indemnity mechanisms serve as security for compliance with prescribed requirements for risk minimization, in accordance with a specified deadline (Marchant, 1984). "Bonding" guarantees the performance of a contract. It is a written obligation binding the signatories to pay a predetermined sum upon occurrence of particular event. "Escrow" is a written instrument imposing certain legal obligations, and is deposited together with a specific amount of funds, and held by a nonparty to the contract until the occurrence of a specified event. A "restoration fund" is a trust fund created to ensure that money will be available to rectify problems caused by the occurrence of a specific event (Baram, 1982). With each of these three methods, refunds are progressively allotted to the risk

source as conditions of compliance are met. Similarly, funds are forfeited for noncompliance, and thus provide an economic incentive for risk management.

Automatic indemnity mechanisms can provide a simple, relatively inexpensive means of risk management. Their effectiveness however, requires strict monitoring of activities and enforcement of penalties, which may present problems in cases where noncompliance is difficult to measure (Baram, 1982). Automatic indemnity mechanisms may not be feasible in cases where the deposit of funds interferes with the financial base of the proposed undertaking, or where the economics of compliance/noncompliance change so as to render the deposit inadequate (Marchant, 1984).

5.3.2 Levies and Other Cost Structures

Some critics argue that the most effective means of dealing with health and environmental risks is to compel offending parties to bear the full costs of damages through the imposition of an economic penalty (Reschenthaler, 1979). Levies and other cost structures basically serve to increase the cost of risk producing activities, with the intent of providing an economic incentive for reduction or elimination of risk producing behavior (Anderson et al., 1978). Using this approach the government is responsible for monitoring hazards and imposing economic sanctions when required. Amongst the most common

cost structures are polluter-financed cleanup funds, effluent charges, waste disposal pricing, delay penalties, marketable permits, and raw material input pricing.

Polluter Financed Cleanup Funds. Polluter-financed cleanup funds are monies collected through the imposition of levies on polluters as a form of compensation for the amount of health or environmental damage which has resulted from their past activities. Such funds are used to finance in whole or in part the cleanup of existing pollution, through some government-administered mechanism.

Although the use of polluter-financed cleanup funds represents an attempt to make polluters share in the cost of treating or removing existing pollution, such funds suffer from several shortcomings in practice. For example, since liability is based on existing pollution levels, it may be difficult to establish the exact source of the problem, or to locate and charge polluters whose industries no longer exist. Apportioning costs on a fair basis may also prove difficult, particularly in cases where all pollution sites in a given area are not eligible for clean up. Finally, the government may end up supplementing the cost of clean up in cases where the fund is insufficient, even if such a move is not economically feasible nor prudent, given other government priorities.

Effluent Charges. The use of effluent charges involves imposing a fee per unit of discharge, or per unit of discharge above a specified emission level (Marchant, 1984). This option provides flexibility in that the charge can vary according to the type and location of discharge such that an equitable economic burden is placed on sources of risk having different abatement costs.

An example of the imposition of effluent charges may be found in the Canada Water Act (Somers & MacDonald, 1983). This Act requires payment of an effluent charge for the dumping of specified quantities of waste into specific water quality management areas.

Effluent charges are also employed in the case of water pollution in West Germany, Italy, France, and the Netherlands (OECD, 1986a). Similar charges are being levied in France, Japan, the Netherlands and Sweden in an attempt to reduce industrial emissions of sulfur dioxide into the atmosphere (OECD, 1986ab).

In order for a system of effluent charges to be effective, the charge should reflect all social costs that result from the activity so that it pays for all of the resources it consumes (Wrage, 1985). This would encourage risk producers to reduce risk until the marginal costs of further abatement exceed the amount of the charge itself (Economic Council of Canada, 1981). This notion is encompassed in the "Polluter-Pays-Principle" advocated by the OECD (1983a, 1984a).

While the idea of polluters paying for the true cost of their discharges is a very appealing one, there are practical problems associated with such a system. Determination of the charge required to achieve the desired level of abatement or control is difficult (OECD, 1983b). The traditional argument in favour of effluent charges is often based on an unrealistic assessment of the amount of information actually available to regulators regarding pollution sources, the extent of pollution, and the costs of clean up (Spence & Weitzman, 1978). Once established, it may be difficult to change fee schedules; further, the costs of charges may be passed on to the consumer in the form of increased prices, inflation or unemployment (Reschenthaler, 1979). Finally, the implementation of a charge system is costly in terms of the time and resources required for continued monitoring of effluent levels.

Waste Disposal Pricing. Waste disposal pricing increases the costs associated with waste disposal using such methods as landfill sites or waste processing facilities. This option has been used in several industries in Italy, France and the Netherlands so as to encourage innovations in recovery, recycling, reuse of waste, development of new products, or other means to reduce waste streams (OECD, 1986ac).

The advantages of waste disposal pricing are similar to those of effluent charges in that it internalizes pollution costs, stimulates

innovation, and employs the Polluter-Pays-Principle. This option also creates some incentive for private response as opposed to government intervention.

Waste disposal pricing is not suitable for controlling all forms of risk. Even in cases where it is applicable, the costs of waste disposal may not reflect the true social and economic costs of waste production. Further, such charges may give the appearance of providing a license to pollute, and may thus lack social acceptability (Marchant, 1984).

Delay Penalties. Delay penalties represent a variant of emission charges in which fees are imposed only if stipulated emission standards are not met within a predetermined schedule (Marchant, 1984). Each risk producer receives a schedule specifying maximum allowable emissions, the required rate of reduction, and ultimate risk objective (OECD, 1983a). This system has been used successfully in the control of both air and water pollution in Connecticut (Marchant, 1984), where the value of the penalty is proportional to the savings afforded the industry due to delayed capital investment. The rate of return on the industry's pollution-related, revenue-producing investments is also taken into consideration (Clark, 1978).

This option has the advantage of converting the issue of emission controls in offending firms into a matter of economic decision making, in that polluters must decide to what extent the benefits associated with their activities outweigh the cost of violating the required emission standards. In order to be effective, the penalty rate should be high enough to make delays in compliance unprofitable. However, the schedule and target emissions must be realistic, and economically practicable (Economic Council of Canada, 1981). Delay penalties also suffer from the same limitations as effluent charges.

Marketable Permits. Using a system of marketable permits, government officials define overall cumulative and dispersive pollution objectives for particular discharges (OECD, 1983a, 1984b). A permit system is then established so as to limit the total discharge within any specific geographical area in accordance with the established pollution objectives. Emission permits are distributed or auctioned off to offenders who must adjust their emissions accordingly.

The total cost of reducing emissions to a specified level depends on the allocation of permits, the nature of emission reduction schedules, and the incentives for risk producers to develop and utilize less expensive control methods (Economic Council of Canada, 1981). Initial auctioning of permits, as opposed to free distribution, generates revenue which may be used for government research and development, and may serve to encourage the development and

installation of new pollution control equipment or the adoption of new technologies. The extent to which cost are transferred to consumers depends on the market structure and the demand for permits.

Marketable permits have been used to a limited extent in the case of air pollution through the National Ambient Air Quality Standards administered by the Environmental Protection Agency in the United States (OECD, 1986d). They have also been used to some extent with point sources of water pollution in the State of Wisconsin, with permissible discharge levels varying according to ambient water conditions.

Two national policies have been utilized for permit trading in the United States. The "bubble policy" allows the Environmental Protection Agency or individual states to designate multiple sources of emission in one or more firms as a single source or bubble for regulatory purposes. The limitations imposed on each bubble can be reallocated amongst individual sources within the bubble. The "offset policy" permits the location of new and expanded sources of pollution in existing industrialized areas, provided compensatory reductions can be achieved at other locations within the same region (OECD, 1986d).

Marketable permits have not been widely used due to difficulties associated with their implementation. These include questions of how to determine the cost of the permits for any given geographical area or

type of pollution, and how to allocate discharge allowances from the point of view of economic efficiency. Generally, industry has opposed the use of marketable permits due to the belief that they are charged twice for the same emission. Environmentalists have also opposed the system as it appears to sanction pollution.

Raw Material Input Pricing. Raw material input pricing attempts to induce conservative use of raw materials so as to reduce the emissions associated with process outputs. Such a system has been used in Canada in cases where gas and oil are used either as energy sources or as petrochemical feedstocks (Marchant, 1984).

Manipulation of raw material prices affords the same economic benefits as do other penalty-type options. However, there are problems associated with the determination of appropriate prices and the extreme sensitivity of these prices to rapid rises in the costs of basic commodities.

5.3.3 Fiscal Support

Financial assistance or subsidies generally exist to facilitate the development or adoption of new technologies or procedures designed to reduce risk. Subsidies may take the form of grants to reduce the capital cost of pollution abatement, loans below market price, or special accelerated depreciation tax policies for investments (OECD,

1983c). Charge-type instruments may be preferable to subsidies if it is felt that risk producers should bear the full costs of abatement.

From the economic perspective, subsidies are acceptable provided that the marginal conditions for efficiency are met. In this regard, subsidies are preferable if they are transitory, do not significantly disturb trade and investments, and are designed to promote socio-economic objectives (OECD, 1984a). Typically, subsidies are financed through general revenues or through other charge-related programs. The choice of the financing scheme will necessarily determine to whom the costs of risk reduction are redistributed (Economic Council of Canada, 1981).

While fiscal support options may induce investment in risk reduction facilities, they can be inefficient because the incentives provided for process-related changes tend to result in amelioration rather than prevention of risk. For example, various forms of fiscal support for the tobacco industry such as tax-deductible tobacco advertising have served to inadvertently promote risk in terms of cigarette smoking. The linking of subsidies to specific risk reduction objectives could, however, help to optimize the use of public funds (Anderson et al., 1978).

Amongst the most common options for fiscal support are tax deductions, rebates and credits, equity or debt financing, tax exempt

bonds, government contributions and related funding, government loans, and loan guarantees and insurance (Marchant, 1984). Each of these particular options is discussed briefly below.

Tax Deductions, Rebates and Credits. Enhanced deductions or credits, such as the accelerated capital cost allowances associated with investments or with other expenses which are intended to reduce risk, are useful in that they can be tailored to suit corporate priorities through self-administration (Marchant, 1984). Self-administration reduces government overhead costs and avoids the need for governmental review or intervention.

An Accelerated Capital Cost Allowance Program (ACCA) for pollution control equipment, machinery, and structures, is administered in Canada by Environment Canada. This program provides a tax write-off for eligible capital expenses to polluters over a three year period, and may be applied to any property used primarily for the purpose of preventing, reducing, or eliminating pollution (Law Reform Commission of Canada, 1986).

One difficulty with such non flow-through tax structures is that they are of no benefit to firms without taxable income or investment outlays at particular points in time. In addition, tax deductions tend to be applicable only in the upswing of the business cycle and may result in large financial losses for government. Further, tax

advantages may provide only an indirect incentive for risk reduction, in that the private sector may not regard them as subsidies.

Equity or Debt Financing. Flow-through tax features such as equity or debt financing can be transferred by the taxable entity to sources of equity or debt financing through shares or other instruments. As such, this option provides tax-supported equity financing for specific purposes, is flexible enough to accommodate individual firms, and may benefit those without taxable incomes.

However, the cost of such a program can be high, and government may find it difficult to retain control over the system. Further, flow-through tax features complicate the tax system, and may interfere with the principle of progressive taxation by effectively providing tax shelters to industry.

Tax Exempt Bonds. Tax exempt bonds are not considered to be taxable income from the point of view of the investor, and thus effectively reduce the interest rate to the issuer. This option is widely used for municipal and industrial development, and for environmental purposes in the area of water pollution in the United States (OECD, 1986d).

Tax exempt bonds are similar to flow-through tax features for equity financing, but provide a much higher degree of repayment

security, and are generally suitable for public authorities with a secure tax base. Although government retains control over bond issuance, public support for bonds can be obtained via investment from the private sector.

Despite their advantage, tax exempt bonds can be costly. In addition, they appear to subsidize risk producers, undermine the progressivity of the tax system, and increase its complexity.

Government Contributions and Related Funding. Contribution programs involve provision of funds conditional upon compliance with specific criteria and conditions. Refundable taxes are similar to grants, except that taxable income is not required for eligibility. Forgiveable loans act as contributions if specified conditions are met; otherwise they are recoverable as loans.

These options have been employed to some extent in Canada, most notably in the case of Economic and Regional Development Agreements (ERDA's) administered through the Department of Regional Industrial Expansion. ERDA's are 10 year agreements under which federal and provincial governments jointly plan, coordinate and operationalize a variety of economic development policies, programs, and activities. There are currently over 100 such agreements in place, with a total federal-provincial commitment of over $4 billion (First Ministers, 1985).

Contribution-type options are useful in situations where governments can obtain visibility and credit for their actions. Such options are flexible in that support packages and the scale of financial assistance can be tailored to specific clients. However, contribution options violate the Polluter-Pays-Principle, and are often viewed as giveaways to nonperformers. In addition, such measures may prolong nonviable situations and depend on risk producers being motivated to reduce risks and apply for assistance.

Government Loan Programs. Government loan programs provide direct assistance to qualifying risk producers for specific purposes such as industrial development and agriculture. Such programs are beneficial to industries who otherwise may not be capable of attracting the necessary capital for risk reduction activities, and allow for government assistance without violating the principle that the polluter pays. Canada maintains a government loan program through the Federal Business Development Bank, a crown corporation. Such loans are provided to qualifying firms at the prime rate of interest as set by the Bank of Canada.

Government loans can be very costly when default occurs, especially in high risk lending situations, and may not be equally available to all firms. In cases where governments do not have established lending practices, it may be difficult to modify administrative procedures so as to accommodate this option.

Loan Guarantees and Insurance. A loan guarantee is a public guarantee covering some percentage of the principle of loans provided by private lenders for specific purposes (Wrage, 1985). Loan insurance involves negotiation with the borrower, and may include specific conditions. Loan guarantees have frequently been used for industrial development purposes at both federal and provincial levels in Canada (Marchant, 1984).

Loan guarantees and insurance are useful in that government need not compete with conventional lenders, but can take advantage of their administrative and approval capabilities. In addition, they permit government to provide support to the private sector at arm's length. As with government loan programs however, loan guarantees and insurance can lead to differential treatment of firms, force the government to bear the opportunity cost of its investment, and create significant liabilities for the public guarantor (Law Reform Commission of Canada, 1986).

5.4. ADVISORY OPTIONS

Advisory or informational approaches to risk management may serve one of two purposes. First, they may provide information and advice to risk producers in lieu of a stricter form of control in an attempt to persuade offenders to reduce risks. Second, they may provide information and advice to the risk consumers so as to increase

awareness, facilitate informed decision making, or encourage exertion of pressure on risk producers. Risk management options aimed directly at risk producers include consultative and administrative approaches. Those aimed at the public include disclosure and adverse publicity, worker information, and public awareness.

5.4.1 Consultative Approach

With the consultative approach, government undertakes research, identifies hazards, and develops guidelines which are offered to industry on a voluntary compliance basis. By doing so, government assumes an active advisory role, and uses moral suasion to induce offenders to reduce risks (Reschenthaler, 1979).

The consultative approach is appealing to industry as it reduces research expenditures and offers great flexibility for risk reduction. However, it is not certain that offenders will act to reduce risks to an acceptable level, if at all. Further, it is questionable whether government would or could reallocate funds so as to permit research, and assemble and publicize comprehensive information for the benefit of individual offenders.

5.4.2 Administrative Approach

Using the administrative approach, government assumes responsibility for designing and implementing formal safety standards, and for enforcing these standards through a professional inspectorate. While government acts to police risk producers, the ultimate resolution of the problem is left to the offender because of the belief that risk producers, having richer information sources, can create more practically reasonable and economically efficient solutions. Such an approach is particularly useful when the nature of the hazard is uncertain and when risk-related decisions are complex (Reschenthaler, 1979).

In order for this approach to be effective, sufficient resources must be available for government agencies to conduct the necessary research, formulate standards, maintain a competent inspectorate, and conduct inspections at required intervals. The agency must also have available to it an adequate system of sanctions to ensure compliance with standards.

5.4.3 Disclosure and Adverse Publicity

Disclosure refers to the release of nonaccusatory information regarding risk production. In response to disclosures, risk producers may choose to voluntarily modify operations so as to reduce risk, or

forego offending activities. Adverse publicity involves the release of risk-related information as it pertains to specific offenders. Such publicity may not be accompanied by prior notice nor an opportunity to be heard. Regardless of the situation, named offenders must comply with publicized requirements (Baram, 1982).

Disclosure and adverse publicity provide the public with essential information, using moral suasion or more stringent tactics to achieve compliance. As such, these options reduce government control of private transactions, and are less costly and time consuming than are regulations.

Both disclosure and adverse publicity have been employed in Canada by the Environmental Protection Service of the Department of the Environment. These techniques have been used as a means of public persuasion through the use of such instruments as education and advertising campaigns intended to alter or influence the behaviour of the private sector (Law Reform Commission of Canada, 1986).

The use of such options requires government to find a balance between the public's right to warning of and protection from risk and industry's right to due process. Proprietary information must be protected against release except in situations where particular activities pose a real threat to human health or the environment.

5.4.4 Worker Information

Risks in the workplace may be minimized by affording workers a direct role in risk reduction. Such an approach relies heavily on safety committees and may include a provision for a meaningful right of work refusal. This option is based on the assumption that employees who are given adequate information on occupational hazards can act in conjunction with management to reduce or eliminate the corresponding risks (Reschenthaler, 1979).

In order to be effective, safety committees must have direct links to appropriate medical and environmental health services which can provide information, technical expertise and monitoring. Committees must also be sufficiently independent such that they are not subservient to management. They must be allowed to participate in decision making and must have recourse to appeal. Using this approach, government takes on the reduced role of advisor or referee.

5.4.5 Public Awareness

Public awareness of potential risks to human health and the environment may be stimulated through the provision of information on such risks for the population at large. For example, public awareness programs sponsored by government agencies provide useful information on such topics as nutrition, smoking, alcohol abuse, and in general health

protection (WHO, 1984). Government agencies may employ various mass media to do so directly or may attempt to promote a social environment that advocates health promotion and environmental protection. Public awareness campaigns are useful in reaching large segments of the population. To be effective, sufficient information should be available to increase knowledge to the point where individual behavior problems concerning risk may be altered. In practice, however, public awareness initiatives may not be sufficiently powerful to persuade some individuals to minimize their exposure to those risks over which they may exert some control.

5.5. TECHNOLOGICAL OPTIONS

Technological approaches to risk management rely on technological solutions to reduce risk, with the availability of different technologies affording flexibility in compliance (Reschenthaler, 1979). Use of the technological approach allows each polluter to reduce its level of emissions by different amounts, using different technologies, depending on the individual's abatement costs (Wrage, 1985). This approach stems from the notion of moving directly from hazard identification to engineering control, and is favorable to both government and industry because it allows for negotiation in implementing technologically based risk reduction measures.

The development of new technological processes or equipment may serve to reduce risk generation in particular situations. Such actions can minimize government intervention in private industry while at the same time acting as a viable economic alternative for offenders. In the United States, for example, Union Carbide has developed a new technology to transform certain wastes and byproducts into marketable commodities, thereby increasing the firm's revenue and reducing its disposal problem (Reschenthaler, 1979).

In addition to encouraging new technological developments, technological solutions may involve transfer of information about existing technology in conjunction with the use of financial and other incentives to promote its adoption (Reschenthaler, 1979). Environment Canada, for example, performs extensive research into water pollution control technologies, and makes this information available to industry, which may lack the resources or the interest to develop such technologies on their own.

Technological approaches to risk reduction are being used in Canada, notably in the case of the National Ambient Air Quality Objectives established under the Clean Air Act (Somers & MacDonald, 1983). The establishment and enforcement of the specific emission standards promulgated under the Act take into account the limitations of the best available technology as well as the cost associated with compliance to the standards.

Another recent example of how new technological development can serve as a means of reducing risk is the use of activated-carbon filters and reverse osmosis units for water purification and sewage treatment (Tobin, 1984). Such innovations often occur even in the absence of regulatory pressures or economic incentives, and offer considerable promise for reducing health risks following their introduction.

For specific applications, it has been suggested that the "best practical technology" be selected on the preferred method of risk management. The primary problem associated with this notion lies in the ambiguity of the term "best practical technology". This term could mean: do what is easy; do what industry leaders already do; do everything short of industry bankruptcy; or use whatever standard technology is available regardless of cost. The apparent simplicity of this idea is thus at the same time its fundamental weakness (Lave and Menkes, 1985).

5.6. SUMMARY AND CONCLUSIONS

In this chapter, we have considered a variety of risk management options available for the control of risks to health, safety and the the environment. All of the options considered may be broadly characterized as regulatory, economic, advisory or technological. Although direct regulation has been widely employed in the past, it can

represent an expensive control mechanism which is subject to a certain degree of resistance, particularly within the current atmosphere of deregulation. As such, nonregulatory options for risk management are being given careful consideration in order to develop as viable alternatives to regulatory action.

In general, economic incentives may be used as a means of altering business decisions regarding pollution control investments or by making it more profitable for firms to abate rather than pollute. Insurance and other related schemes may be invoked to compensate risk consumers without placing excessive financial burdens on individual risk producers. Levies and other cost structures serve to increase the cost of risk producing activities, thereby providing an incentive for risk reduction. Fiscal support may also be offered by government to facilitate the development and installation of new technology designed to reduce risk.

Advisory approaches to risk management may provide information either to risk producers in an attempt to encourage risk reduction or to risk consumers so as to promote risk avoidance. This method of dealing with risk avoids the need to develop formal standards as with most regulatory and economic approaches, and leaves individuals free to make their own decisions concerning risk. The advisory approach is likely to be most useful with hazards of relatively minor consequence

and will work best when the public perception of risk corresponds well with the magnitude and nature of the risk at hand.

Technological solutions to risk management problems may also be effective in reducing risk, using different technical means of pollution abatement. Technological approaches to relieving risk may be applied as part of a regulatory initiative, but can also be used independently of other approaches to limit the emission of pollutants to the extent possible.

The selection of the most appropriate risk management option to apply in any particular cases requires consideration of the nature of the hazard in question. For example, health risks involving serious irreversible damage may not be suitable choices for control options other than regulation, whereas less serious risks may be more amenable to the use of nonregulatory alternatives.

The efficiency of each option in reducing or preventing risk should be given consideration, along with the resources required for its implementation. The capacity of the existing infrastructure to respond to the task of implementing the option is also important. This requires consideration of mandate, clarity of the governing doctrines, and administrative simplicity.

The extent to which the option selected and the results achieved are consistent with public values, including cultural and social risk acceptability, is important. The equitable distribution of burdens related to the implementation of the option also needs to be considered, as do the precedent-setting implications for similar problems which may arise in the future.

Application of these criteria to specific situations can constitute the core of public decision making in the selection of risk management options. The selection of a particular alternative will then depend on the specific circumstances surrounding the risk management problem and the ability of the responsible agency to ensure that the selected alternative will be effectively operationalized. The appeal of each alternative is dependent upon the social, political, and economic climate within which risk is to be controlled.

6. REGULATORY STATUTES IN CANADA

6.1 INTRODUCTION

In the previous chapter, risk management options were broadly categorized as regulatory, economic, advisory or technological. Of these, regulation is the most widely employed approach to controlling health risks. In this chapter, the legislative basis for regulatory action in Canada is reviewed in detail.

We begin with a discussion of the division of legislative power between the Federal Parliament and the Provincial Legislatures based on the Constitution Acts of 1867 and 1982 (section 6.2). The major regulatory statutes which have been enacted for the protection of human health and the environment are then discussed in section 6.3. Because the Constitution Acts lead to some overlap of regulatory responsibility between the federal and provincial governments, federal-provincial regulatory relationships are examined in section 6.4.

Recent initiatives in the direction of regulatory reform are discussed in section 6.5, with a summary of the current regulatory apparatus in Canada provided in section 6.6.

6.2 FEDERAL-PROVINCIAL DIVISION OF POWER

The Constitution Acts of 1982 and 1867 (formerly the British North America Act) are the principle documents of the Canadian Constitution

and divide legislative power between the Federal Parliament and Provincial Legislatures. The Acts delineate both general and specific areas of federal and provincial jurisdiction, primarily through sections 91 and 92. Although the Charter of Rights enacted by the Constitution Act (1982) also describes the constraints of federal and provincial power, the nature and full extent of these constitutionally guaranteed rights remain to be delineated by the courts.

Section 91 of the Constitution Act provides the federal government with the authority to regulate hazardous substances, hazardous products and environmental contaminants, primarily through its jurisdiction over interprovincial and international trade and commerce (section 91(2)) and criminal law (section 91(27)). It has also been suggested that the constitutional authority granted the federal government "to make laws for the peace, order and good government of Canada" provides the federal government with the responsibility to deal with all forms of pollution which cross provincial boundaries and international borders.

Over time, the courts have interpreted these general powers as emergency, residual, and national dimension doctrines (Wylie, 1984). Emergency power is exercised for a finite period of time in response to demonstrable crises. Residual powers refer to all matters not specifically assigned to provincial governments. The national dimension applies to issues which require national resolution. The extent of federal authority regarding these last two doctrines has

never been explored fully. Delineation of such authority would be particularly useful in the case of interprovincial or international transmission of aquatic or atmospheric pollutants, as provincial governments lack sufficient legislative breadth to deal effectively with such matters.

Section 92 assigns the provinces wide domains of jurisdiction. Provincial legislatures have authority within their boundaries over matters of manufacturing, municipal institutions, property and civil rights, the working environment (except for federal works, undertakings and businesses as discussed in section 92(10)), waste disposal, and the provision of health care services (Nemetz et al. 1982).

The Constitution Acts do not deal specifically with management of risks to health and the environment. However, the federal government is afforded some authority over environmental management on the basis of several specific areas of legislative jurisdiction including those pertaining to federal property, taxation, statistics, navigation and shipping, seacoast and inland fisheries, Indian lands, criminal law, federal works, international and interprovincial trade, and the implementation of Empire treaties (sections 91 and 92; Wylie, 1984).

The provinces have control over environmental management through ownership of natural resources within their boundaries (section 92(109)), and from their authority over property and civil rights

(section 92(13)). They also have control over direct taxation within their boundaries and the power to spend these revenues for provincial purposes. As such, provincial legislatures have regulatory power which is comparable to that of the Federal Parliament in the area of environmental risk management.

Federal authority over water quality is in some cases incidental to its authority over navigation and shipping (section 91(10)), seacoast and inland fisheries (section 91(12)), and the treaty powers (section 91(132)). Federal authority takes precedence regarding coastal and boundary waters. Provincial authority, largely through ownership of natural resources, applies only to intraboundary waters (Wylie, 1984).

Federal responsibility for air quality falls under and the peace, order and good government clause noted previously, and generally extends only to interboundary matters. Stationary sources of air pollution are the responsibility of the provinces.

The Constitution Acts give both the federal and provincial governments powers with respect to agriculture. Thus, both levels of government can use this power to develop legislation dealing with fertilizers, feed products, and pesticides. Federal authority over pesticide regulation stems from its powers in both trade and commerce and criminal law. Provincial authority is a function of the provinces' authority over the work environment. In cases of conflict, section 95

expressly states that any provincial act shall have effect only if it does not contradict federal law.

Parliament's responsibility for criminal law power affords it the authority to establish health and safety standards for foods. Until recently, this power was assumed to afford Parliament authority over food compositional standards as well. However, a 1979 Supreme Court Ruling (the Labatt Lite Beer Decision) revoked this latter area of authority as it applies to beer, ale, and related products. This case involved the sale of lite beer containing more than the allowed 2½% alcohol content. In effect, the Court ruled that Parliament acted in an ultra vives fashion to establish standards which are inconsistent with the criminal laws under which the Food and Drugs Act and Regulation are based. As a result, the provinces can successfully challenge Parliament's authority over compositional standards for any food and can assume such authority for themselves. Otherwise, provincial authority over food regulation stems only from their authority over health and safety. In 1987, however, the Food and Drug Act was amended so as to permit standards on food composition under the trade and commerce limitations applicable to inter-provincial trade. (Products sold in the same province as they were produced were unaffected by this change.)

6.3 FEDERAL STATUTES

The major federal statutes which have been enacted for the protection of the human health and the environment are summarized in Table 6.1. These are discussed individually in detail below.

Table 6.1 Federal Statutes for the Protection of Human Health and the Environment

Statute	Minister(s) Responsible	Year Enacted	Purpose of Legislation	Nature of Regulations
Fisheries Act	Fisheries and Oceans	1939	To protect and manage the Canadian Fisheries resource.	The Act provides authority to form regulations respecting designation of substances, quantities of substances and treatments as "deletrious substances".
Food and Drugs Act	Health & Welfare Consumer and Corporate Affairs (responsible for economic aspects)	1953	To protect the consumer from health hazard and fraud, use and sale of foods, drugs, cosmetics, medical devices.	Regulation-making power been used to establish standards for foods, food additives, food ingredients and drugs and to elaborate positive listings of maximum residue levels for agricultural chemicals in food products.
Canada Labour Code	Labour	1966-67	To recognize unions and the rights of employees working for firms and Crown Corporations carrying out or engaged in federal government business. (It does not cover public servants employed by federal departments.) Part IV of the Act deals with employee safety.	Power to establish regulations dealing, inter alia, with the handling, transport, storage, use and disposal of substances or devices dangerous to the safety or health of employees.

Table 6.1 (cont'd). Federal Statutes for the Protection of Human Health and the Environment

Statute	Minister(s) Responsible	Year Enacted	Purpose of Legislation	Nature of Regulations
Pest Control Products Act	Agriculture	1968	To regulate products used for control of pests and the organic functioning of plants and animals.	The Act permits promulgations of regulations which may, inter alia, deal with registration of both products and establishments, labelling and packaging of pest control products, standards for safety and efficacy of control products.
Hazardous Products Act	Consumer and Corporate Health and Welfare (Section 9 and 10)	1968	To protect the consumer in the use and sale of hazardous products which fall outside the jurisdiction of the Explosive Act, the Food and Drugs Act, the Pest Control Products Act and the Atomic Energy Act.	Regulations have been established with respect to labelling and packaging of products designated as hazardous substances.
Canada Water Act	Environment	1969	To provide for the management of water resources of Canada.	The Act provides authority to make regulations respecting the designation of substances, quantities of substances and treatments as "waste".
Clean Air Act	Environment	1971	To control ambient air quality and air pollution.	The Act provides authority to make regulations concerning the maximum concentration of any element or additive in fuels which would result in significant contribution to air pollution resulting from the combustion of the fuel under ordinary circumstances.

Table 6.1 (cont'd). Federal Statutes for the Protection of Human Health and the Environment

Statute	Minister(s) Responsible	Year Enacted	Purpose of Legislation	Nature of Regulations
Environmental Contaminants Act	Environment Health and Welfare (as an advisor as designated under Sections 3,4,5,6,7)	1975	To protect human health and the environment from substances that contaminate the environment.	The Act contains authority to prescribe regulations which schedule substances and classes of substances as environmental contaminants.
Ocean Dumping Control Act	Environment	1975	To control the dumping of waste and other substances in the ocean.	The Act provides authority, inter alia, for promulgation of regulations prescribing maximum quantities of prohibited and restricted substances.
Transportation of Dangerous Goods Act	Transport	1980	To promote public safety in the transportation of dangerous goods.	The act provides for the promulgation of regulations dealing with, inter alia, classification of dangerous substances or organisms and specifying dangerous goods that shall not be handled, offered for transport or transported in any circumstances.

6.3.1 Fisheries Act

The 1969 Amendment to the Fisheries Act prohibited the deposition of a "deleterious substance" of any type in water frequented by fish. In addition, the power to make regulations respecting substances, or classes of substances, quantities and concentrations of substances or classes of substances and treatments, processes and changes of water was also included.

6.3.2 Food and Drugs Act

This Act differs significantly from the more recent environmental control statutes in that the Food and Drugs Act is a mature piece of legislation. The Food and Drugs Act itself is quite a brief document covering only eleven pages. Section 25 of the Act, however, provides authority for the writing and passage of regulations for carrying out the purposes and provisions of the Act. Over the years, full advantage has been taken of the provision as some 175 pages of regulations have been drafted.

The earliest legislation in this area was passed in 1875 to control the widespread sale of grossly adulterated liquor. The current Act was passed in 1954 following revisions of the original statute in 1920 and 1949. As more than 100 years have elapsed since the passage of the original food control legislation, considerable experience has been gained with respect to promulgation of specific regulations and standards to protect the consumer from health hazards and fraud with respect to the sale and use of foods, drugs, cosmetics and medical devices.

Two specific areas of regulation under the Food and Drugs Act impact upon, or interact with, environmental control legislation. Division 15 of the Regulations prescribes maximum residue limits for agricultural chemicals on food products. With the exception of certain agricultural chemicals, the pest control substances themselves are registered in terms of use patterns, and other parameters under the Pest Control Products Act.

Section B.01.046 is also significant in that it lists two substances of environmental origin, namely ethylene thiourea and chlorinated-p-dioxins, the presence of which constitutes adulteration of the food.

It is important to note that control under the Food and Drugs Act or regulations promulgated pursuant to this statue focus upon the deleterious material in or upon the food product rather than control of the contaminant itself.

6.3.3 Canada Labour Code

This act applies to labour and workplace safety conditions for employees involved in federal work, undertaking or business that is within the authority of the Parliament of Canada. Section 84 of the Act provides the Governor in Council with authority to make regulations for the safety and health of persons employed in federal work including provisions respecting the handling, transportation, storage, use and

disposal of substances or devices dangerous to the safety or health of employees. The Act also provides authority for the Minister of Labour to authorize any employer to establish a safety and health committee for federal work, undertaking or business. Section 96 of the Act provides employees with rights of redress and complaint through the Canada Labour Relations Board respecting violations or alleged violations of the Act or regulations.

6.3.4 Pest Control Products Act

This Act is designed to control products used for the control of pests and the organic functions of plants and animals. It is somewhat unique in that Section 4(2) require control products exported out of Canada (or conveyed from one province to another) to be manufactured in an establishment which (a) complies with prescribed conditions and (b) is registered and operated as prescribed. While no regulations have been made pursuant to Section 4(2), it does provide a mechanism to prohibit the sale of de-registered or banned agricultural chemicals to other (often developing) countries.

Detailed regulations have been promulgated under this Act prescribing registration requirements. The Health Protection Branch of the Department of National Health and Welfare provides advice respecting the toxicology of pesticides and related substances and is responsible for regulating residue levels of agricultural chemicals on food products under the Food and Drugs Act.

6.3.5 Hazardous Products Act

This Act is a product-oriented statute which differs significantly from the Food and Drugs Act in that the basic Act does not provide broad powers of prohibition but rather relies upon the use of schedules to the Act and regulations to deal with specific hazardous substances. Schedule I to the Act prohibits the advertising, selling and importing of certain hazardous products such as jequirty beans or furniture and toys created with a lead containing material. Schedule II prohibit the advertising, selling and importing of products such as cleaners containing chlorine compounds and glues containing ketone solvents.

When a product is added to Part I or Part II of the Schedules, any manufacturer, distributor of that product or any person having that product or substance for sale may, within sixty days of the date of the order of the Governor in Council, request that the Minister refer the order to a Hazardous Products Board of Review.

6.3.6 Canada Water Act

This statute authorizes the establishment of water quality management areas by the federal government in collaboration with provincial governments. Some of the steps to ensure effective management include monitoring, research and the establishment of a water quality plan. It is interesting to note that the Act requires prepublication of the plan in the Canada Gazette as well as a concise summary in a newspaper of general circulation in the area affected by the plan at least once a week for a period of four weeks.

The second part of the Act deals with contaminants resulting from the manufacture and use of laundry detergents and other cleaning agents which may cause eutrophication. Regulations may be promulgated prescribing the nature of permissible contaminants and maximum permissible concentration of such contaminants in any cleaning agent or water conditioner.

Section 8 of the Act is important in that it contains a general prohibition against depositing waste in any waters comprising a water quality management area except in quantities and under conditions specifically prescribed. Additionally, this Section provides for the payment of effluent discharge fees as a control measure against persons depositing waste in a designated water quality management area.

6.3.7 Clean Air Act

This Act, like the Canada Water Act, is cast in general terms and implementation depends upon promulgation of specific emission standards. The Minister of the Environment is responsible for formulation of air quality objectives reflecting tolerable ranges of concentration of contaminants, acceptable ranges of concentration of contaminants and desirable ranges of concentration of contaminants.

When the emission into the ambient air of an air pollutant in quantities and concentrations which constitute a danger to human health or violate the term of an international agreement, national emission

standards may be prescribed. It is an offence to violate a national emission standard as defined in Section 7 of the Act.

This Act also provides for the federal government to enter into agreements with provincial governments for the purpose of facilitating, coordinating and implementing policies and programs designed for the control and abatement of air pollution and adopting and applying, throughout Canada, national ambient air quality objectives.

6.3.8 Environmental Contaminants Act

This is a relatively new statute (1975) which is intended to cover problems that cannot be effectively handled under other environmental legislation, particularly new or recently identified pollutants. It is jointly administered by the Ministers of the Environment and National Health and Welfare.

This Act provides the Minister of National Health and Welfare with broad powers to collect data and conduct investigations with respect to substances entering or likely to enter the environment in quantities or concentrations or under conditions which may endanger human health or the environment. The Minister of National Health and Welfare is also given joint authority with the Minister of the Environment to appoint advisory committees to review data collected, receive representation from interested parties and obtain advice concerning control of environmental contaminants.

As is the case with most modern statutes, all regulations made pursuant to this Act must be pre-published in Part I of the Canada Gazette. If an objection to a proposed regulation is filed, the Minister of National Health and Welfare shall establish an Environmental Contaminants Board of Review composed of not less than three people to study and review the objection.

Important regulations have been promulgated under this Act to prohibit specified uses of persistent environmental contaminants such as polychlorinated biphenyl compounds (PCB's).

6.3.9 Ocean Dumping Control Act

This is a relatively recent statute designed to control dumping of waste and other substances in the ocean. Three schedules have been included within the Act which define prohibited substances which cannot be dumped (Schedule I); restricted substances which may only be dumped upon receipt of a permit (Schedule II) and (Schedule III) which factors are to be taken into account in granting permits.

The Act also provides for the Minister to convene a Board of Review when a dumping permit has been refused; has been granted but is unacceptable to the applicant or has been suspended or revoked. The Act also provides a range of control actions including seizure of ships and aircraft involved in unauthorized dumping.

6.3.10 Transportation of Dangerous Goods Act

This act has been designed to promote public safety in the transportation of dangerous goods. It is a new piece of legislation and may be considered as one of the control measures directed against hazard to life resulting from spillage or contamination from hazardous substances during transport. The Act contains broad prohibition against handling, offering for transport or transport of dangerous goods unless all applicable safety standards are complied with and all containers, packaging and means of transport comply with all applicable prescribed safety standards.

The Act provides extensive power to make regulations necessary to carry out the purpose and provisions of the Act. It currently includes nine classes of substances considered to be dangerous goods: explosives, gases, flammable liquids, flammable solvents, oxiding substances, poisonous and infectious substances, radioactive materials, corrosives and miscellaneous products.

6.4 FEDERAL-PROVINCIAL RELATIONS

Given the scope of the regulatory statutes discussed in section 6.3, it is clear that the federal government has broad powers in the regulation of health and environmental hazards in Canada. At the same time, the Constitution Acts have conferred related responsibilities on the provinces. While a detailed review of the numerous pieces of legislation enacted at the provincial level for the protection of

human health and the environment will not be attempted here, it will be instructive to briefly examine existing federal provincial relationships in the areas of environmental quality, pesticide usage, and food safety.

6.4.1 Environmental Quality

At the federal level, the Department of the Environment (DOE) has primary authority over environmental management. DOE administers the most pertinent federal Acts for management of air and water quality, namely the Clean Air Act, Environmental Contaminants Act, Canada Water Act, Fisheries Act, and Ocean Dumping Control Act. In addition, certain Acts administered by other federal departments impact upon water quality management, including the Arctic Waters Pollution Prevention Act and Northern Inland Waters Act (Department of Indian Affairs and Northern Development); the International Boundary Waters Treaty Act (External Affairs); the Navigable Waters Protection Act (Department of Transport); and the Pest Control Products Act (Agriculture Canada)(Wylie, 1984). Each province, largely through its Ministry of Resources and Environment, also administers specific legislation directed towards environmental management (see Table 6.2).

The Canadian Council of Resource and Environment Ministries (CCREM) represents the primary federal-provincial forum for the discussion of environmental issues. This committee establishes

Table 6.2. Selected Provincial Statutes for the Protection of Human Health and the Environment in the Province of Ontario

Area	Statute
Environmental Management	Environment Protection Act (1971)
	Air Pollution Control Regulations (1970)
	Environmental Protection (General) Regulations (1970
	Guidelines and Criteria for Water Quality
	Ontario Waste Management Corporation Act (1981)
	Ontario Water Resources Act (1970)
	Pollution Abatement Incentives Act (1970)
	Public Health Act (1970)
Pesticide Usage	Pesticides Act (1973) and General Regulations (1974)
Food Safety	The Fish Inspection Act (1975)
	The Milk Act and Regulations (1978)
	The Liquor Licence Act and Regulations (1983)
	The Liquor Control Act (1975)
	The Pesticides Act (1981) and Regulations (1982)
	The Farm Products Marketing Act (1979)
	The Meat Inspection Act (1980) and Regulations (1982)
	The Oleomargerine Act (1970) and Amendments (1971)
	The Edible Oil Products Act (1970)

mutual objectives and guidelines for air quality and water quality. In addition, there exist several more specific federal/provincial agreements for air and water quality management, such as the Agreements for Great Lakes Water Quality (with Ontario) and for the Long Range Transport of Air Pollutants (with Manitoba, Ontario, Quebec and New Brunswick). Such federal/provincial agreements usually involve cost recovery services provided by one level of government to the other, cost-shared programs in areas of mutual interest, and memoranda of understanding to avoid duplication in area of concurrent jurisdictions (Wylie 1984).

The Clean Air Act and Regulations enable the Minister of the Environment to enter into cooperative arrangements with the provinces and other bodies, establish contaminant objectives and natural emission standards, and form advisory boards and committees. As provinces have authority over stationary sources of pollution, the federal government can only prescribe non-mandatory natural ambient air quality objectives for common pollutants. In cases where emissions present a danger to human health or violate an internal agreement, however, the federal government may prescribe mandatory national emissions standards. In recent years, the federal government has chosen to issue guidelines only, in order to repatriate areas of concurrent jurisdiction to provincial authorities.

A recent Amendment to the Clean Air Act enables the federal government to take some action on interboundary transport of air pollutants, as in the case of acid rain. As well, the Amendment provides for establishment of a cash fund to be used for a cost sharing program between the federal and provincial governments aimed at reducing acid deposition. Ontario and Quebec have agreed to share the federal/provincial cost of reducing SO_2 emissions by 50% by 1993.

6.4.2 Pesticide Usage

Agriculture Canada is primarily responsible for pesticide regulation at the federal level. Under the Pest Control Products Act and Regulations (1972), products must be registered with the Department before they can be sold in Canada, thereby providing the Minister of Agriculture with broad authority to prevent the manufacture, storage, display, distribution or use of any pesticide under unsafe conditions. The Provinces have no role in the initial registration process and as a result are not privy to information supplied by registrants, unless registrants agree to release such information. In cases where new information is obtained on established product, the Provinces are however included in the registration review process by virtue of section 19 of the Regulations.

Several provinces have their own legislation for control of pesticide use (see Table 6.2). Provincial Ministers of Agriculture

have authority over the Acts, with the primary regulatory tool being licensing of sales outlets and professional applicators.

Pest control product management is facilitated to a large extent through several committees. At the federal level, all Departments with an interest in pesticides have a representative on the Federal Interdepartmental Committee on Pesticides (FICP), an advisory body reporting to the host Departments. There are also several federal/provincial committees such as The Canada Committee on Pesticide Use in Agriculture and the Canada Associations of Pest Control Officials (Agriculture Canada, et al., 1978).

All provinces also have established groups which meet regularly to elaborate provincial recommendations on pesticides use. Most provinces also have interdepartmental committees similar to FICP, which consider problems of a regional nature and review significant provincial pest control programs. The recommendations of these committees are reflected in the regulatory status of pesticides under the Pest Control Product Act and other relevant legislation at both levels of government.

6.4.3 Food Safety

At the federal level, the Department of National Health and Welfare has sole authority over food regulation through the Food and

Drugs Act (1954) and Regulations. The Food and Drugs Act is a consumer-oriented statute intended to protect consumers from health hazards and fraud in the sale and use of foods (as well as drugs, cosmetics, and medical devices). Certain other federal Acts indirectly affect food regulation, most notably those Acts dealing with commodities and the Pest Control Products Act.

Although the provinces do have some authority for food regulation within their boundaries, Newfoundland is the only province with its own Food and Drugs Act. Newfoundland developed its own Act, and, due to its late entry into Confederation, has chosen to maintain it. All provinces also have some indirect control over food safety, through various commodities Acts (see Table 6.2).

While there exist no federal-provincial committees or working groups dealing with food regulations, both levels of government cooperate to ensure that the food supplied to Canadians has high standards of safety and quality. Although the federal government alone conducts food research and formulates standards, the provinces are empowered to enforce federal food regulations as well as to apply stricter food standards of their own.

6.5 REGULATORY REFORM

Recent years have witnessed trends not only towards deregulation and the use of nonregulatory options for risk management, but an

increased emphasis on regulatory reform. These initiatives have been motivated largely by a sense that the regulatory apparatus in Canada may have become too cumbersome and unwieldy. As such, public sentiment seemed to be in favour of streamlining regulation with a view to increasing both efficiency and effectiveness.

In August, 1980 a special Parliamentary Task Force on Regulatory Reform under the chairmanship of Mr. James Peterson, the Member of Parliament from Willowdale, was established to review the regulatory process and Parliament's role in the process. Executives from regulated industries, trade associations and senior officials from government departments gave testimony before the Committee. In December 1980, this Task Force presented its reports which contained some 29 recommendations (Report, 1980) to Parliament. In terms of the work of the public health, the following recommendations were particularly significant.

- Each Department and Agency should publish twice a year a regulatory agenda giving notice of upcoming regulatory initiatives.
- Departments and Agencies should prepublish regulations whenever appropriate and the onus should be on them to justify why prepublication is not desirable or possible.
- All proposed regulations should be subject to appropriate impact assessment to be performed by the sponsoring Department.
- Each Department and Agency should review immediately its regulatory statutes and regulations.

- Departments and Agencies (should) ensure that all their regulatory activities are evaluated periodically under the program evaluation system.

- Annual reports of Departments and Agencies should include a summary of progress made on regulatory reform, a summary of major regulatory initiatives undertaken in the past year, identification of those contemplated, identification of program evaluations completed in the past year and a schedule of future program evaluations.

- Public interest group participation in the Federal regulatory process should be encouraged and supported by higher levels of financial assistance.

- Departments should consider the use of the consensus process wherever they have determined that a regulatory standard is an appropriate policy.

- Where overlap duplication or conflict of regulatory requirements exists within the Federal government or between Federal and Provincial jurisdictions, the Government should take action to reduce or eliminate the burden in the private sector. In particular, immediate action should be taken in the areas of occupational health and safety, food labelling and advertising and food production and processing.

A particularly noticeable theme throughout this report was the need for the Government to improve communication concerning regulatory proposals with the regulated industries, public advocacy groups and other interested parties. This is in keeping with current trends towards improved risk communication practices and greater public partipation in decision making (see chapter 9).

The parliamentary Task Force also suggested that all regulatory proposals should be subjected to the following questions.

- What is the problem?
- What alternative solutions have been considered?

- How will this proposal help?
- What are its drawbacks?
- What are its advantages?
- Who will gain and who will lose?
- Why is it the best means of dealing with the problem?

In response to these recommendations, the responsible regulatory review committees within the Health Protection Branch incorporated these questions into a standardized protocol for application in the establishment and review of regulatory proposals.

In summary, this Task Force concluded that more discipline, better communication and more openness is needed in the regulatory process. Furthermore, regulatory proposals should always give consideration to alternative solutions, including non-regulatory options as discussed in Chapter 5.

More recently, the Ministerial Task Force on Program Review re-examined the patterns of regulatory control of health and environmental hazards at the federal level in 1985. This Task Force consisted of the Deputy Prime Minister, the Ministers of Finance and Justice, and the President of the Treasury Board, and utilized 19 public/private sector study teams to comprehensively review all aspects of federal regulation as well as regulatory relationships with the provinces, territories and independent regulatory agencies. The

purpose of the undertaking was to produce a revised inventory of federal programs so as to improve service to the public, increase efficiency, and reduce red tape.

Generally, the study teams found Canada to be both over-regulated and badly regulated. These problems stemmed largely from the cumulative effect of regulation at three levels of government, and from the existence of outdated regulations. Overlap and duplication of regulation between levels of government was found to be significant. The Task Force note that the current regulatory situation was acting as an enormous impediment to economic growth, initiative, enterprise and personal freedom, and that intergovernmental cooperation was required to reduce the overall regulatory burden.

As a result of the Task Force findings, the federal government has decided to implement a comprehensive Regulation Reform Strategy. While bearing in mind the benefits of regulation, the reforms will act to limit the rate of growth and proliferation of new regulations, alter existing regulations as required, increase the public's role in the regulatory process, streamline the federal regulatory system and increase regulatory cooperation with the provinces, especially as it pertains to overlap and duplication (Office of the Deputy Prime Minister, 1986).

6.6 SUMMARY AND CONCLUSIONS

Regulation represents one of the most widely used and most forceful option available for risk management. In this chapter, we have reviewed the major pieces of federal legislation for the protection of human health and the environment in Canada, including the following.

- Fisheries Act (1939)
- Food and Drugs Act (1953)
- Canada Labour Code (1966-67)
- Pest Control Products Act (1968)
- Hazardous Products Act (1968)
- Canada Water Act (1969)
- Clean Air Act (1971)
- Environmental Contaminants Act (1975)
- Ocean Dumping Control Act (1975)
- Transportation of Dangerous Goods Act (1980)

Many of these Acts are complementary in nature and may be jointly administered by one or more federal government departments. Taken as a whole, these statutes provide a firm basis for the establishment of regulatory controls over potential hazards to human health and the environment.

In addition to federal legislation, many provinces have also enacted related statutes by virtue of the authority nested in them under the Constitution Acts of 1982 and 1867. Since the Constitution Acts generally fail to explicitly assign a clear line of authority to manage risks to either the federal or provincial governments, they frequently fall within the legislative jurisdiction of both levels of government. Although federal legislation generally takes precedence in cases of conflict, the problem of overlapping jurisdictions has frequently been resolved through the close cooperation of federal and provincial officials. Frequently, federal activity has been directed toward establishing national standards for adoption and enforcement by the provinces. The federal government provides scientific information to form the basis of cooperative actions and encourages standards across the country.

As a result of the First Ministers Conference in 1981, the federal government has begun to withdraw its exercise of authority from areas of concurrent jurisdiction, both as a shown of good faith and as a cost saving measure. This effort has been welcomed by the richer provinces such as Ontario, Alberta and British Columbia, who have greater resources with which to develop risk management programs. Other provinces have not appreciated such efforts, as they lack the resources to develop their own regulatory agenda.

At present there exists a strong movement towards regulatory reform and deregulation in Canada, particularly at the federal level. This was most apparent in the recommendation of the Ministerial Task Force on Program Review, which submitted its report to Parliament in 1985. The Task Force put forward a large number of specific proposals for streamlining the federal regulatory apparatus, many of which are still under active consideration.

7. DECISION ANALYSIS

7.1 INTRODUCTION

The rapid pace of technological development in modern industrialized nations has resulted in the synthesis and use of many new chemical compounds in recent decades. This has been accompanied by a heightened awareness among the public of the potential health risks posed by chemical substances, including food additives, drugs and pesticides, as well as contaminants present in the air, water and the environment in general.

Regulatory agencies charged with the responsibility to control chemical exposures may employ different risk management strategies. These agencies must both choose among the available control options in a reasonable and cost-effective manner, and determine the appropriate level of vigour and expense with which to pursue the approach taken (Munro & Krewski, 1981). Depending on the nature of the problem at hand, the control mechanisms can be advisory, technological, economic or legislative (chapter 5).

Programs to improve human health by reducing the risks posed by environmental hazards can be evaluated within the same basic framework used to assess other health care programs. That framework has evolved and developed over the years (see, for example, Torrance et al., 1972; Weinstein & Stason 1977; Drummond 1980; Weinstein, 1981; Warner & Luce 1982) to the point where it has now been applied to a wide range of health care programs. This chapter describes how that framework can be adapted and applied to evaluate programs designed to reduce the risks of environmental hazards in general (Torrance & Krewski, 1986).

The basic framework within which toxic chemical control programs may be evaluated is described in section 7.2 including identification of the major costs and consequences of a risk reduction program. Measurement of the main categories of cost is discussed in section 7.3 along with the measurement of health and other benefits. The use of this information in evaluating alternative programs by formal decision analysis procedures is described in section 7.4 with guidelines for their implementation provided in section 7.5. Section 7.6 provides a brief summary along with the main conclusions of this chapter.

7.2 A GENERAL FRAMEWORK FOR DECISION ANALYSIS

Economic evaluation is the comparative analysis of alternative courses of action considering both their costs and their consequences. There must always be at least two alternatives, otherwise there is no

need for an analysis. In the simplest case, one of the alternatives is represented by the status quo, while the other alternative is the new program under consideration. The evaluation is then based on the incremental costs and consequences of the new program as compared to those for the existing situation.

Frequently there are more than two alternatives under consideration so that a number of comparisons are required. Each alternative can also be considered at several different levels of intensity. For example, three alternatives for a particular problem may be to do nothing, to launch a public information program, or to impose formal regulations. Moreover, a public information program may be invoked at different levels of vigour and expense, while regulations can be proposed at different levels of stringency. Multiple alternatives and multiple levels of scale within alternatives add to the complexity of the analysis, but do not change the fundamental approach. For clarity of exposition, this paper will deal only with the simple case of one program being compared to the status quo.

Because society's resources for health improving programs are scarce, both costs and consequences must be considered in evaluating a risk reduction program. It is also essential to specify the viewpoint of the analysis. Possible viewpoints include that of the producer, consumer, government, or society as a whole. Although costs and consequences can differ from one viewpoint to another, the societal

viewpoint would seem most appropriate for purposes of public policy decision making.

A schematic representation of the major costs and consequences of a toxic chemical control program is shown in Figure 7.1. Three categories of cost are identified: program costs, producer costs and consumer costs. A good program will have desirable consequences in terms of reduced human exposure and subsequent improvements in human health and environmental status.

The program costs incurred by the responsible agency are those that would be required for a particular toxic chemical control program. These costs include those associated with program design, implementation and operation, including the enforcement of any regulations that may be established. Producer costs are additional costs to industry of program compliance, including both direct costs associated with modifications to the production process and indirect costs due to reduced productivity. Consumers may incur higher costs as a result of increased production costs, a reduction in the quality or availability of goods, or a preference for more expensive but less hazardous alternatives.

The most immediate consequence of a good toxic chemical control program will be a reduction in human exposure. This will be followed

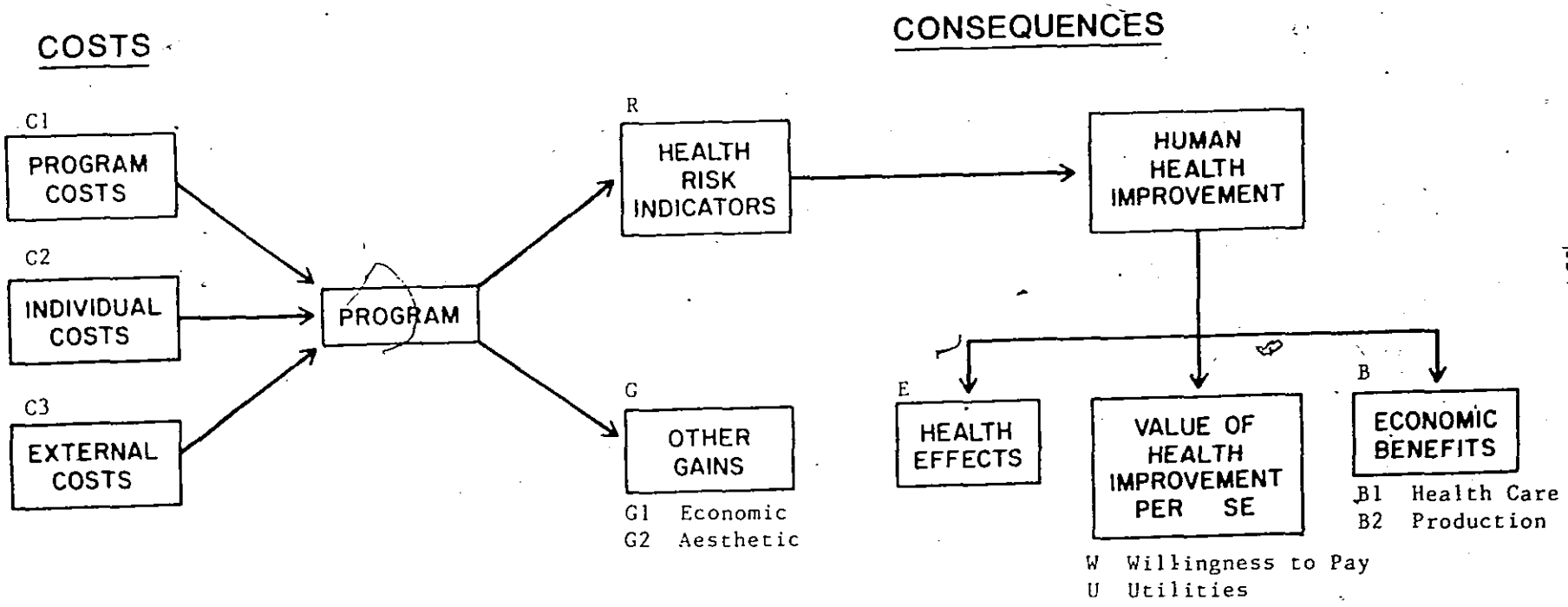


Figure 7.1 Major Components in the Economic Evaluation of Risk Reduction Programs

by a corresponding improvement in human health status expressed in terms of direct health effects, the intrinsic value of improved human health to the individuals affected, and the economic benefits of improved health in the population at large. The improvement in human health due to the control program is defined in terms of conventional health indicators such as disability days prevented, reduction in morbidity and mortality, or life-years gained. The value of health improvements per se is the value people attach to being healthy rather than sick. Direct economic benefits may occur due to reductions in health care costs while indirect benefits may arise as a result of increased productivity in a healthier population.

Other benefits unrelated to human health are also possible. For example, there are economic benefits associated with healthier crops and livestock arising from reduced production costs as well as greater yields. There are also intangible benefits such as the aesthetic value of a cleaner environment.

7.3 MEASURING COSTS AND CONSEQUENCES

The process of determining costs can be broken into three distinct steps: identification, measurement and valuation. The first step is simply to identify all items of cost without concern for how they might be measured. It is important to be comprehensive at this step, and to

be uninhibited by potential measurement difficulties. In this way a complete itemization of costs is developed.

Cost measurement consists of determining the quantity of each cost item in its natural units. For example, the development of a particular regulation might require the use of scientific and technical personnel, laboratory equipment and related supplies and overhead.

Valuation is the step of attaching dollar values to the items of cost. Generally, market values are used for the valuation of costs. Nonmarket items, like consumer volunteer time, must be valued using imputed prices -- often what you would have to pay to obtain equivalent services. Joint costs, like overhead, are difficult to value because there is no satisfactory method of allocating these costs to the relevant program components (Kaplan, 1982). For each joint cost an allocation basis must be selected which is judged to be approximately proportional to usage of the joint resource. If the allocated joint costs are relatively large (generally they are not), sensitivity analysis can determine the robustness of the final conclusions to the allocation judgments.

Present costs and future costs cannot be simply added together. Future costs must be discounted to their equivalent present value. Alternatively, initial startup and equipment costs can be annuitized over future years (Richardson & Gafni, 1983). Either process requires

a discount rate, to remove the effect of inflation. Although the choice of an appropriate rate is a controversial, theoretical issue (Sumner 1980ab; Jenkins 1980, 1981; Burgess, 1981), recent practice in health care studies has been to use a central discount rate of 5% per annum and to carry out a sensitivity analysis of this assumption (Boyle et al., 1983).

7.3.1 Measuring Exposure

Levels of exposure to toxic chemicals present in the environment can be measured in a variety of ways. These chemicals may enter the biosphere either as naturally occuring substances or as the result of man's activities. Surveys of environmental quality can be used to evaluate the presence of chemicals in the general environment, including those present in air (Munn, 1981) and water (American Public Health Association, 1980). These compounds may result in subsequent human exposure via inhalation or the consumption of drinking water. Ingestion of food additives and contaminants can be evaluated directly using data on food consumption patterns (Nutrition Canada, 1974) coupled with information on the levels of these chemicals in food (Food Safety Council, 1978).

Occupational exposures can be assessed using a variety of monitoring instruments, including personal monitors carried by individual workers. Biological monitoring can also be used to confirm chemical exposures via the detection of certain metabolites in urine or other tissue samples (Tannenbann & Skipper, 1984), although the specificity of this approach remains to be established.

Programs designed to reduce chemical exposures can take a variety of forms, including restrictions on usage, avoidance of exposure, and reductions in the level of exposure using technological controls. The purpose of this paper is not to describe in detail the range of possible alternatives, but rather to provide possible approaches to choosing among these alternatives.

7.3.2 Measuring Human Health Improvement

Health improvement can be measured in terms of health effects in health units, the economic benefits of the health improvement in dollars, and a measure of the value of the health improvement per se using either dollars or utilities. Health effects are measures of health improvement expressed in traditional units such as disability-days prevented, lives saved, or life-years gained. Risk reduction programs might often create a stream of such health benefits each year into the future. For example, a program might be expected to

save fifty lives per year following its implementation. It is important in this case to discount future health effects just as future costs are discounted. Failure to do so can lead to bizarre policy recommendations (Keeler & Cretin, 1983). Theoretical considerations suggest that the same discount rate should be used for both program costs and consequences (Weinstein & Stason, 1977).

Measurements of the health effects associated with exposure to toxic substances present in the environment can in some cases be subject to considerable uncertainty. For example, on the basis of the epidemiological data available to date, it has been estimated that 4300 deaths may be attributable to air pollution each year in Canada (Hamilton, 1985), although the plausible values range from 0 to 13,000. This is due in part to difficulties in accurately monitoring human exposures and in part to the uncertainty surrounding dose response relationships, particularly at low levels of exposure. In general, it is important that the uncertainty associated with measurements of health effects be determined and taken into consideration in the overall analysis.

The direct economic benefits of improved health are the savings in costs of health care and social services realized because the

population is healthier (Fraser et al., 1976; Cooper & Rice, 1976). These savings are estimated in the same way that costs are determined: all items are identified, measured and valued. Frequently, these savings come from reduced utilization of physician services, drugs and hospitals. The first two are relatively easy to value. The savings due to reduced physician utilization can be determined from the relevant fee schedule; savings in drug usage is simply the retail cost of the drugs.

Reductions in hospital utilization can be more complicated. A gross approximation to the financial savings can be obtained by multiplying the hospital days saved by the average per diem hospital rate which is readily available for all hospitals (Statistics Canada, 1983). However, if more accurate estimates of hospital savings are desired, special studies are required to determine the actual costs for the types of patients involved. In many cases the financial savings associated with reduced hospitalization are never actually realized in the short run, because the hospital beds are simply filled with other patients.

Indirect economic benefits are the increases in productive output because the population is healthier. This is normally measured by the increased earnings which result from the program under evaluation. There has been a great deal of criticism of this approach because it discriminates against programs that improve the health of the non-work-

ing: children, the elderly, and the unemployed (Klarman, 1982; Evans, 1984). This criticism is appropriate when indirect economic benefits are used as a proxy for the value of improved health per se, as it has been in many studies. However, it does provide a measure of productive resources returned to society as a result of allocating other resources (C1 and C2) to the program.

The value of health improvement for its own sake can be measured in dollars using willingness-to-pay, or in utilities using preference measurement instruments. The willingness-to-pay approach has not been widely attempted due to certain measurement difficulties (Klarman, 1982). Faced with the certainty of adverse health effects, for example, an individual's willingness-to-pay will depend strongly on income. By foregoing relatively inexpensive preventive measures but accepting greater costs for curative procedures, the value of health seems to be viewed differently depending on one's health status (Evans, 1984).

Utilities for health states are cardinal values that are assigned to each health state on a scale that is established by assigning a value of 1.0 to being healthy and 0.0 to being dead. The utility value reflects the quality of the health state, and allows morbidity and mortality improvements to be combined into a single weighted measure called Quality-Adjusted Life-Years (QALY's) gained (Weinstein & Stason, 1977; Wilkins & Adams, 1978; Dillard, 1983). For example, if a

program improved the health of individual A from a 0.50 utility to a 0.75 utility for one year and extended the life of individual B for one year in a 0.50 utility state, the total QALY's gained for that year would be 0.25 for individual A plus 0.50 for individual B for a sum of 0.75.

Analysts have three choices for determining the utility values for use in a study. One approach is to use values that have been measured and published in other studies (Kaplan et al. 1979). A second approach which has been used in a number of studies is to determine the utility values by judgment and to perform extensive sensitivity analyses to assess the robustness of the conclusions to these judgmental values (Weinstein, 1981). A third approach is to measure the necessary utilities using preference measurement instruments on an appropriate sample of respondents (~~Sackett &~~ Torrance, 1978; Kaplan & Bush, 1982; Churchill et al., 1984). As with other health effects, future QALY increases must be discounted to their present value. Consideration should also be given to the uncertainty associated with any estimates of such measures of health improvement.

7.3.3 Measuring Nonhealth Effects

In addition to the benefits of human health improvement, a toxic chemical control program may create other benefits which should be identified and, to the extent possible, quantified. The value of

healthier crops, commercial forests, farm animals and other nonhealth benefits can be converted into dollars by determining their economic value. These economic gains can be viewed as a partial offset to the social costs of the program (C1 + C2).

Healthier plants, animals, forests and a cleaner environment have aesthetic value in addition to commerical value. Although this value can in theory be measured by using either willingness-to-pay or utilities, it would likely prove very difficult in practice to make these assessments. Given the current state of the art, it is probably better simply to identify these benefits and not attempt to value them. Nevertheless, they should form part of the discussion and the interpretation of the results leading up to a policy recommendation (Torrance & Oxman, 1986).

7.4 DECISION ANALYSIS

Programs directed at the improvement of human health are normally evaluated using cost-effectiveness analysis (CEA), cost-utility analysis (CUA) or cost-benefit analysis (CBA) (Thompson, 1982). Frequently, several techniques and several viewpoints are investigated to broaden the base of information on which to form a policy recommendation. The different techniques and viewpoints are simply different methods of combining and displaying the data from Figure 7.1 to assist in decision making. Recall that for simplicity, we are

concerned only with the generic case of one alternative (one toxic chemical control program) being compared to the status quo (the do nothing alternative). In this case the data in Figure 7.1 are all incremental quantities indicating the extra costs and consequences of the program as compared to the status quo.

7.4.1 Cost-Effectiveness Analysis

Cost-effectiveness analysis (CEA) may be based on the costs required either to reduce exposure R or to effect improvements in health status E. If little is known about linkage of a particular exposure reduction with subsequent health improvement, it may be wise to restrict the measure of health benefits to a measure of exposure reduction only. In this way alternative programs can be compared using CEA to determine the alternative with the lowest cost per unit reduction in exposure level. Cost-effectiveness analysis can also determine a priority ranking for additional programs and levels of intensity for the case where a greater exposure reduction is desired than that provided by the most cost-effective program.

This approach was used by Logan et al. (1981) to compare the cost-effectiveness of alternative programs for reducing blood pressure in hypertensives. Thus, rather than attempting to estimate the possible ultimate health impact in terms of cardiovascular disease, Logan restricted his measures to the reduction of blood pressure,

The advantage of this approach is that it is easier to obtain the necessary data. The disadvantage is that it has a more restricted usefulness. While it can be used to determine the best approach for a particular toxicant, it cannot be used to compare different compounds and it cannot compare toxic chemical control programs with other health improving strategies.

In cost-effectiveness analysis, the dollar cost of a program is compared to the nondollar consequences of the program by means of a cost/effectiveness ratio, CE. If there are several alternatives directed at the same type of effect, this approach can identify which of those is the most cost-effective. If there is only one alternative, an external standard or a judgment by management is required to determine whether or not the program is worthwhile in terms of its cost-effectiveness ratio.

A number of cost-effectiveness formulations are available depending upon the viewpoint taken as well as the extent and quality of available data. The primary viewpoint for making public policy recommendations should be that of society. Three CEA formulations from society's viewpoint are shown below.

$CE_1 = (C1+C2+C3-G1)/R$ (CEA-1)

$CE_2 = (C1+C2+C3-G1-B1)/E$ (CEA-2)

$CE_3 = (C1+C2+C3-G1-B1-B2)/E$ (CEA-3)

] Societal Viewpoint

CEA-1 would be appropriate if quantitative data on the human health impact is not available. Here, R would represent a numerical measure of the impact of the program in terms of exposure reduction. Since health outcomes are not available, economic benefits B1 and B2 cannot be incorporated in this formulation. The remaining economic items must be handled with care to avoid double counting. For example, higher prices as a component of C3 should not be counted if they are simply due to higher production costs already incorporated in C2. Similarly, lower production costs identified as an economic benefit in G1 should only be counted once, either as a cost offset in C2 or as a gain in G1, but not both. The increased value of harvest identified as a potential economic gain in G1 must be treated carefully. If livestock and crops are healthier and heavier as a result of the program, the increased value of the harvest is an appropriate economic gain. However, if the product is not improved but the price simply increases to compensate producers for higher costs of production, there is no economic gain. For example, suppose a particular program increases both the cost of producing wheat and the average quality of the wheat, and as a result

the price per bushel increases. The higher production costs would be included on the cost side in C2. The price increase to offset these costs would be listed under C3, but would not be counted twice in the numerator of the CEA-1 formulation. However, the price increase due to the quality improvement is really not a price increase because the product is different and better, and it should count as a economic gain under G1.

If quantitative estimates on the human health impact of the program are available, formulation CEA-2 or CEA-3 can be used. Here E would be the health improvement attributable to the program measured in physical units such as disability-days prevented or life-years gained depending on the nature of the health impact. The only difference between CEA-2 and CEA-3 is that the latter includes indirect economic benefits B2. The argument for incorporating this term is that B2, represents resources returned to society as a result of the program and these should be shown as an offset to the resources which society must invest in the program. Thus, the numerator of CEA-3 is the net resource consumption of the program from society's viewpoint. Although inclusion of B2 seems appropriate from society's point of view, this is subject to some controversy (Klarman, 1982), so that CEA-2 is included as an alternative formulation.

Cost-effectiveness analysis can also be used to investigate other viewpoints. For example, from the federal government's viewpoint the

formulation would be CEA-4 or CEA-5, where the numerator represents only those costs borne by the government. If a joint federal-provincial viewpoint is desired, CEA-6 could be used. This incorporates the economic benefits of reduced costs for health care and social services, a provincial jurisdiction.

$CE_4 = C1/R$ (CEA-4)

$CE_5 = C1/E$ (CEA-5)

} Federal Government Viewpoint

$CE_6 = (C1-B1)/E$ (CEA-6) Federal-Provincial Viewpoint

7.4.2 Cost-Utility Analysis

Cost-utility analysis (CUA) is simply a special form of cost-effectiveness analysis in which the measure of effect is quality-adjusted life-years (QALY's) gained. The CUA formulae are identical to CEA-2,3,5,6 with E replaced by U. The advantage of CUA over CEA is that it uses a common unit of measurement for all programs and thus allows comparisons across all programs. Indeed, it would allow comparisons of toxic chemical risk reduction programs to other health enhancing programs, such as neonatal intensive care or coronary artery bypass surgery. The main disadvantage of this approach is the difficulty in obtaining accurate data on QALY's as discussed in section 7.3.2.

7.4.3 Cost-Benefit Analysis

In cost-benefit analysis (CBA) all costs and consequences are converted to dollars and summarized in the net benefit (NB) figure. A program is then considered to be cost-beneficial if the net benefit is positive. If there are multiple alternatives of comparable scale, the one with the largest NB is the best.

Cost-benefit analysis, like CEA and CUA, can be undertaken from a variety of viewpoints, although the societal viewpoint is the most common and the most appropriate for public policy decision making.

$NB_1 = B1 + B2 + G1 - C1 - C2 - C3$ (CBA-1)

$NB_2 = B1 + W + G1 - C1 - C2 - C3$ (CBA-2) Societal viewpoint

$NB_3 = B1 + B2 + W + G1 - G1 - C2 - C3$ (CBA-3)

Formulation CBA-1 represents the conventional approach to cost-benefit analysis as applied to health care programs over the last fifteen years. However, this approach has been severely criticized as being biased in favour of programs for those who work and earn income, as opposed to programs that improve the health of children, housewives and the elderly. The criticism centers on the validity or lack of validity of item B2. However, the criticism is inappropriate if formulation CBA-1 is used as a measure of the net resource drain of a particular program on society.

Since net resource drain as measured by CBA-1 provides only one side of the picture (costs but not consequences), it is insufficient for decision making. The value of the improved health itself must be entered into the analysis, theoretically using willingness-to-pay. Formulations CBA-2 and CBA-3 both incorporate the willingness to pay factor W, although this has proven difficult to implement in practice. The difference between these two formulations is that CBA-2 omits B2. This approach is favoured by some analysts because of the controversial nature of item B2. However, because B2 is a valid measure of resource gains due to the program, formulation CBA-3 seems the most appropriate decision oriented cost-benefit analysis formulation.

Cost-benefit analyses can also be calculated from other viewpoints, in which case they become an economic impact analysis for a particular organization or segment of society.

$NB_4 = B1 - C1$	(CBA-4)	Federal-provincial viewpoint
$NB_5 = G1 - C2$	(CBA-5)	Producers viewpoint

For example, the net economic impact for the federal and provincial governments is given by CBA-4. Actually CBA-4 is a somewhat gross approximation in that a full analysis would incorporate the extra taxes collected on the extra earnings B2, the increase (decrease) in taxes due to increased (decreased) profits by producers, and other relevant changes in transfer payments. However, CBA-4 is intended for

governmental budgetary planning and control, not for program decision making.

Similarly, formulation CBA-5 investigates the overall economic impact on the producers. Basically, this impact is the economic gains due to healthier crops (G1) less the added costs of production (C2), being careful not to double count. A more sophisticated analysis would consider the impact on producers of other factors such as government incentives and subsidies. Again, however, the point of this analysis would be to better assess the producers' position rather than to make a decision on the overall net benefit of the program.

7.4.4 Risk-Benefit Analysis

As with CEA, risk-benefit analysis (RBA) recognizes the difficulties of measuring health effects. In contrast with CUA and CBA, this is done in an ad hoc manner without making any attempt to quantify health effects. As defined by the Treasury Board of Canada (1979, p. 10), RBA is

> ". . a special case (of CBA) where explicit attention is devoted to categories of human costs (risk or loss of life or limb) for which established techniques of evaluation do not yet exist. In risk-benefit analysis, benefits should be interpreted in a net sense (i.e., the balance of the ordinary benefits over the ordinary costs), but the risk of loss of life and of human suffering represent extraordinary costs, to be evaluated separately".

The model of RBA is consistent with that proposed by the United States Office of Technology Assessment (OTA, 1981), in which health risks are considered on a qualitative basis, only when comparing the net dollar benefit of all other factors.

Within the context of our evaluation framework, the net dollar value of the nonhealth benefits would be summarized in terms of a risk net benefit value (RNB) to be evaluated in light of the concommitant but nonmonetary information on health effects. The RNB figure is similar to that in CBA, excluding the valuations of human health improvement (B1, B2, and W) used in CBA. From the societal viewpoint, for example, the RNB value would be as follows.

$RNB_1 = G1-(C1+C2+C3)$ (RBA-1) Societal Viewpoint

It is instructive to note that RNB_I, is simply the negative of the numerator in CE_1. Thus, risk-benefit analysis is related to cost-effectiveness analysis in that, although not forming a ratio, it effectively makes use of formula CEA-1. The difference is that in risk-benefit analysis, explicit consideration is given to health risks and benefits, albeit in a qualitative fashion.

7.4.5 Socio-Economic Impact Analysis

In Canada, a socio-economic impact analysis (SEIA) is now required for any new government regulation pertaining to health, safety or fairness which is anticipated to result in major costs. Even if specified cost criteria are not met, a SEIA may be required if the proposed action would "have other important implications of potential concern to interested groups or the public at large" (Treasury Board of Canada, 1979, pp. 23-24).

Formally, a SEIA involves an evaluation of the assessment of both the allocative and nonallocative effects of the proposed regulation. Allocative effects on market efficiency can be measured in terms of production and consumption and are the traditional domain of CBA. Nonallocative effects are also of concern to government, including those involving the distribution of income, market structure, international trade, and inflation. These latter effects are however less well suited to formal methods of economic evaluation.

7.4.6 Analyses Based on Risks

Techniques for arriving at decisions involving toxic programs to control environmental hazards which consider only information on possible health risks have also been discussed in the literature (Office of Technology Assessment, 1981; Hendrick et al., 1984). Such

strategies will be referred to here under the general heading of risk analysis. Risk minimization aims at reducing as far as possible exposures resulting in adverse health effects. A special case of this approach is the zero-risk principle, which calls for the complete elimination of all exposures resulting in some level of risk, no matter how small. Risk/risk analysis seeks to put the risk in question in perspective by comparison with other known risks. A variation on this theme is to compare the risk of using a particular chemical with the risk of not using it, as with the use of drugs with the potential for serious side effects to treat life-threatening diseases. While these procedures can be useful, they consider only consequences and not costs, and are thus outside the scope of this chapter.

7.5 GUIDELINES FOR IMPLEMENTATION

As an aid in understanding how the material in this chapter might be applied in the evaluation of a particular toxic chemical control program, the hypothetical example of a program to regulate the emission of environmental contaminants is considered briefly here. The purpose of this discussion is to demonstrate how the methods proposed in this paper might be applied in general terms, not to develop the ultimate approach.

The program could operate at a number of levels of stringency with P_0 represent the status quo (no regulation). The first task is to

assemble as much of the data as possible for each program alternatives $P_i(i=1...,k)$. C1 would include the initial cost of designing and implementing the regulation, annuitized on an annual basis, plus the annual cost of enforcing and monitoring the regulation. C2 would include the annuitized value of equipment modifications plus the annual increase in operating costs. C3 would include price increases, if any, multiplied by annual sales. R would represent the annual reduction in population exposure. E would be the annual reduction in ill-health or premature death as a result of reduction in exposure. B1 would be the present value of the stream of savings in health care costs and social services costs attributable to the health improvement E in the year under study. B2 would be the present value of future production as measured by earnings attributable to E, the health improvements which occurred in the year under study. W would be the sum that people would be willing to pay for health improvement E. Q would be the quality-adjusted life-years (QALY's) gained in reduced disability days and reduced mortality. G1 would represent the annual economic benefits from healthier crops, forests and animals due to the improved environment.

Obviously, much of this data would be quite crude and should be accompanied by a measure of its uncertainty for subsequent use in

sensitivity analysis. If the human health improvement data is considered so uncertain as to be unreliable, the evaluation would be limited to CEA with cost-effectiveness ratios expressed in terms of the cost per unit reduction in exposure R. Otherwise, the data can be analyzed using the various models described in section 4.

Further illustration of the use of the techniques described in this chapter has been given by Krewski et al. (1988), who examined decisions taken in Canada, the United States and the United Kingdom with respect to different options available for reducing the levels of lead in gasoline. This analysis indicated that the detailed evaluations carried out in these three countries all fall within the general framework provided by the model given in this chapter. The use of a common framework to systematically analyze the three decisions also served to highlight different assumptions and judgments used, and to clarify the reasons for the different decisions reached in the three countries.

7.6 SUMMARY AND CONCLUSIONS

In recent years, methods for measuring health benefits and evaluating health care programs have undergone rapid development. Organized consideration of all of the relevant costs and consequences is important for purposes of identifying the relevant alternatives, establishing the perspective to be taken in the analysis, and evaluating the uncertainty surrounding the potential costs and benefits of the program under consideration.

The two fundamental forms for this work are cost-effectiveness analysis (CEA) and cost-utility analysis (CUA). Cost-benefit analysis may also be considered if sufficient information is available to place a value on the human health improvement. Toxic chemical control programs designed to improve human health can be evaluated using the same approaches. Methods have been illustrated in this paper to show how these approaches can be applied in a relatively straightforward manner.

Although not immediately clear from their definitions, there is a systematic relationship among the different approaches to decision analysis discussed in this paper. CEA and CUA consider only nonallocative effects and seek to determine the program providing the most cost effective approach to risk reduction, with risk expressed in terms of QALY's or surrogate measures of health risk such as reduction in hypertension. Risk-benefit analysis (RBA) and CBA include consideration of health benefits, although RBA does so in only a qualitative fashion. Socio-economic impact analysis (SEIA) considers both allocative and nonallocative effects, and would be of most interest to broader programs with national or international economic implications.

The practical application of these procedures will no doubt raise certain methodological issues, particulary in the area of linking program proposals with specific health improvements. In some cases, this latter relationship may be subject to considerable uncertainty. As experience with these procedures accumulates, however, these difficulties may be overcome and sounder strategies for the control of environmental hazards may be achieved.

8. RISK PERCEPTION AND RISK ACCEPTABILITY

When the public worries, it is our responsibility to worry.

Dan Beardsly,
Environmental Protection Agency

8.1 INTRODUCTION

As discussed in chapters 3 and 4, risk analysis attempts to provide rational scientific estimates of health risks, and to identify sources of uncertainty inherent in scientific data. Such measures may subsequently be used in determining the appropriate degree of control that may be required to manage risk.

Despite the objective nature of risk analysis, quantitative estimates of health risk do not take into account how the public views that risk. In contrast to risk analysis, risk perception is a process in which individuals subjectively or intuitively comprehend, estimate, and evaluate the probabilities and consequences of risks. Since risk analysis fails to consider the subjective elements of risk perception, it is important for decision makers to remain cognizant of public concern for health risks in order that risk management decisions may receive greater public acceptance.

In this chapter, we review several recent studies designed to gauge public perception of health and environmental risks in Canada (section 8.2). Psychological and sociological factors known to influence risk perception are considered in section 8.3. Levels of risk which the public appears to consider acceptable or which have been allowed for regulatory purposes are discussed in section 8.4. A discussion of the role of risk perception in the risk assessment and risk management process is given in section 8.5 with a brief summary of the main findings of this chapter provided in section 8.6.

8.2 PUBLIC PERCEPTION OF HEALTH AND ENVIRONMENTAL RISKS IN CANADA

A number of studies have been conducted to gauge the public's perception of health and environmental risks in Canada, primarily in the form of public opinion surveys focusing on specific issues of concern. Although a systematic review of this research will not be attempted here, an examination of the findings of several such investigations will provide some insight into the Canadian public's perception of health risks. Specifically, we consider three separate studies focussing on the perception of general health hazards, food additives, and water quality.

8.2.1 General Health Hazards

In order to examine the public's perception of health risks in Canada, the Policy, Planning and Information Branch of the Department

of National Health and Welfare conducted a public opinion survey involving detailed personal interviews with individuals from across Canada (Health & Welfare Canada, 1984b). A multi-stage sampling technique was used to gather the data, with interviews obtained from 200 of the 600 respondents contacted. The specific objectives of this study were to ascertain the criteria people use to evaluate risks, to collect data the Branch could use to improve its communication with the public, and to provide data for internal policy development.

Each respondent was asked to rate 12 activities and substances on a scale of one to seven, ranging from no health risk at the lower limit to an extremely high health risk at the upper limit (Figure 8.1). Of the 12 potential risk factors considered, illegal drugs, smoking and alcohol were given the highest risk ratings. The majority of these risk factors were perceived to be somewhat hazardous, in the sense that their average rating was above the mid-point (4.0) of the scale.

In order to further delineate those characteristics which affect risk perception, the respondents were asked to rate the extent to which thirteen such characteristics were relevant to each of the 12 risk factors considered. By averaging the ratings of these characteristics over respondents, a risk profile for each of the 12 risk factors considered was constructed.

The risk profile for smoking, for example, is shown in Table 8.1. These results suggest that smoking is perceived as a health risk

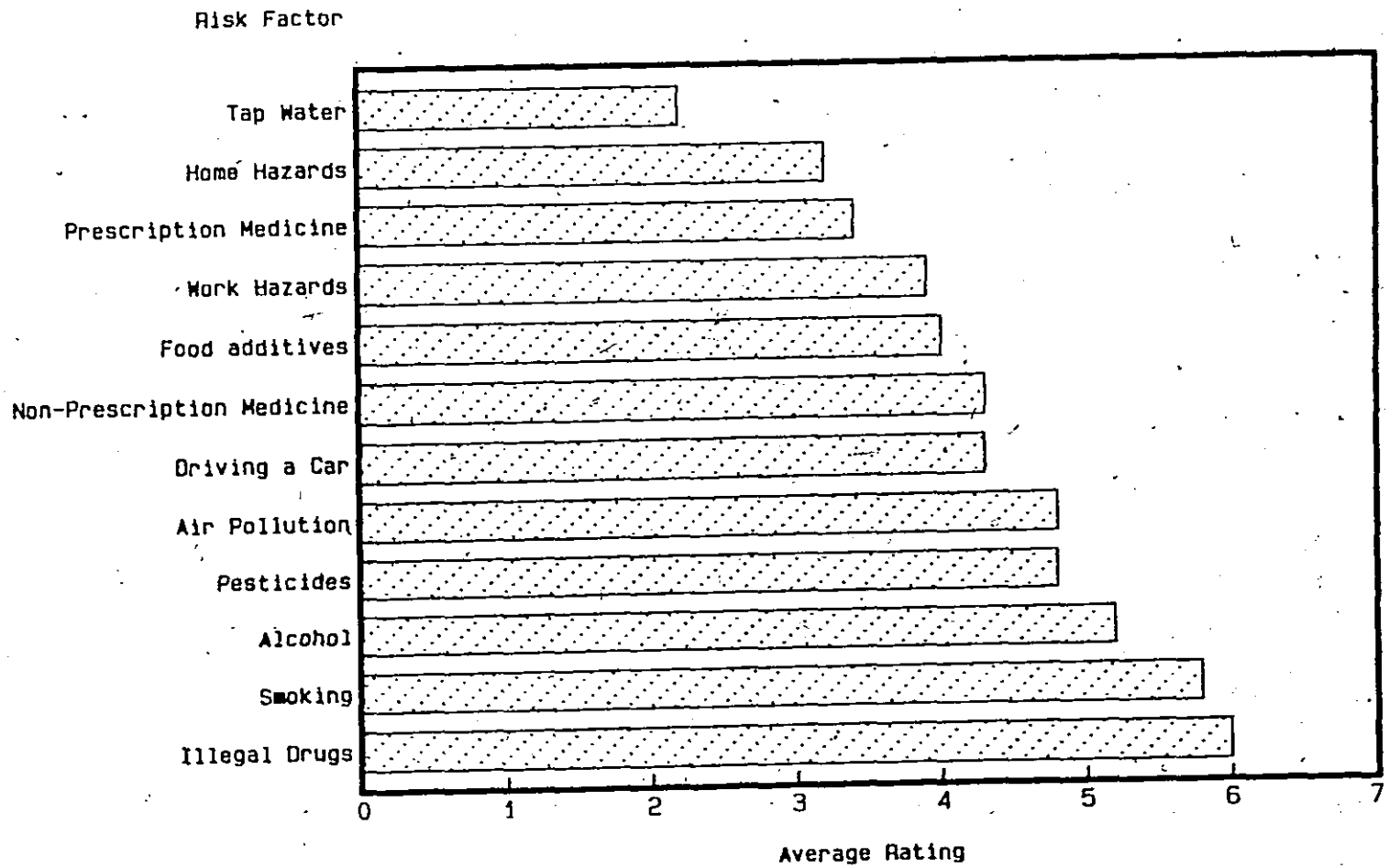


Figure 8.1 Average Ratings Assigned to Twelve Health Risk Factors

Table 8.1 A Risk Profile for Smoking

Characteristics	Average Rating (1 to 7)
Risk is known to science	6.2
Risk has little compensating benefit	5.6
Risk affects many people	5.4
Public is aware of and understands the risk	5.4
Risk is likely to adversely affect health	5.3
Magnitude of the risk is increasing	4.8
Consequences of the risk are likely to affect future generations	4.8
The government (or other body with responsibility) controls risk minimally	4.7
Harmful effects of the risk are readily visible	4.2
Risk is likely to cause a disaster (i.e. many people affected concurrently)	3.7
Risk affects the respondent personally	3.6
The individual respondent has control over the risk	3.0
Adverse effects of risk occur immediately	2.6

largely because the risk is known to science, there is little compensatory benefit, it affects many people, the public is aware of the risk and it is considered likely to harm health. On the other hand, the concern over smoking is reduced by the belief that the adverse health effects are delayed rather than immediate, and that the risk is entirely voluntary.

An examination of the risk profiles for the remaining risk factors revealed that the scientific community was considered to be aware of the risks involved in all cases. This observation suggests that the provision of scientific evidence of risk can play an important role in enhancing perceived risk.

Further examination of the risk profiles for the 12 risk factors considered revealed that the risk factors tended to cluster within four broad groupings involving bad habits, environmental risks, medicine and day-to-day risks.

Bad Habits. The risk factors which had the highest perceived risk (illegal drugs, smoking and alcohol) all fell within the category of bad habits. These risks are of concern largely because they are considered to be known to science, confer little benefit and harm health. These factors are, however, offset by the beliefs that the individual can exert control over these risks, their effects are delayed, and they do not personally affect the respondents.

Environmental Risks. The environmental hazards (air pollution, pesticides, tap water and food additives) were, with the exception of tap water, perceived as posing risks of intermediate concern. These risks were considered to affect many people, present a risk to future generations, and be known to science. On the other hand, it was believed that these environmental hazards induced only limited adverse health effects which are both delayed and not readily discernible.

Medicine. Within the category of medical hazards, non-prescription drugs were considered to present much lower risks than prescription drugs. Medical risks were felt to be known to science, affect many people, and affect future generations. The belief that prescription and non-prescription drugs have little effect on respondents personally and are largely under individual control tended to reduce their perceived risk.

Day-to-Day Hazards. With the exception of driving, day-to-day hazards were generally perceived as low risks. These hazards were considered to present scientifically known risks, affect many people, and cause immediate effects. They were felt to be under individual control and have little effect on future generations.

In order to examine the sources which people use to obtain information on risk, respondents were asked to identify their primary sources of information from a list of six potential sources (Table 8.2). These results identify the news media is the main source,

Table 8.2 Primary Sources of Information on Health Risks

Source	Percentage of Respondents
News Media	64
Health & Welfare Canada	22
Consumer Groups	18
Doctor	15
University Scientist/Professor	4
Local Medical Officer	3

consulted three times as often as any other single source. Health & Welfare Canada was the second most popular source of information, providing information on a wide range of health risks, most often on smoking, alcohol and illegal drugs.

Respondents were also asked to rank the credibility of each source on a scale of 1-6, least to most trustworthy (Table 8.3). These results indicate that the six sources of health risk information differ in credibility, but not dramatically. Although the most popular source of information, the news media ranked lowest in credibility.

In order to evaluate public exposure to risk information, respondents were asked to identify which risk factors they had heard about most often from each of their primary sources of information. These results indicate that the three risk factors assigned the highest perceived risk score (illegal drugs, smoking and alcohol) were also the three risks the public had heard most about. Also, the two risks assigned the lowest perceived risk ratings (home hazards and tap water) were the ones the public had heard the least about. These observations support the hypothesis that public exposure to risk information is an important determinant of risk perception.

8.2.2 Food Additives

Health & Welfare Canada (1979) conducted a cross-Canada survey of consumers to measure their knowledge of food additives, their concern

Table 8.3 Credibility of Sources of Health Risk Information

Source	Average Rank (1 to 6)
Doctor	4.0
Health & Welfare Canada	3.7
Local Medical Officer	3.5
Consumer Groups	3.4
University Scientist/Professor	3.4
News Media	3.1

over food additives and their perspective of additives with regard to safety of the food supply. The survey involved a group of 24,940 self-selected adult Canadian shoppers, representing about 0.1% of the Canadian population. Information on public views towards food additives was obtained by means of a brief self-administered questionnaire.

This survey rendered that most people felt that there is inadequate control of food additives, and that the addition of colours to food is not justifiable. Almost 90% of those surveyed were concerned over possible adverse health effects from food additives. Most people indicated they made an effort to eat food with less additives and were prepared to pay more for food with no additives. Over 90% of respondents wanted more information on food additives.

The respondents were also asked to rank food additives in relation to other potential health hazards in food (Figure 8.2). Hazards associated with pesticides and bacteria were rated about the same as those from food additives, with industrial pollutants and other hazards being of much less concern. (Note that the sum of the response rates for each hazard exceeds 100% since some respondents selected more than one item as being of most concern to them.)

8.2.3 Water Quality

Environment Canada (1981) sponsored a study of public opinion and attitudes concerning water quality in the Canadian Lower Great Lakes

"My greatest concern about the safety of the food supply is the presence of:"

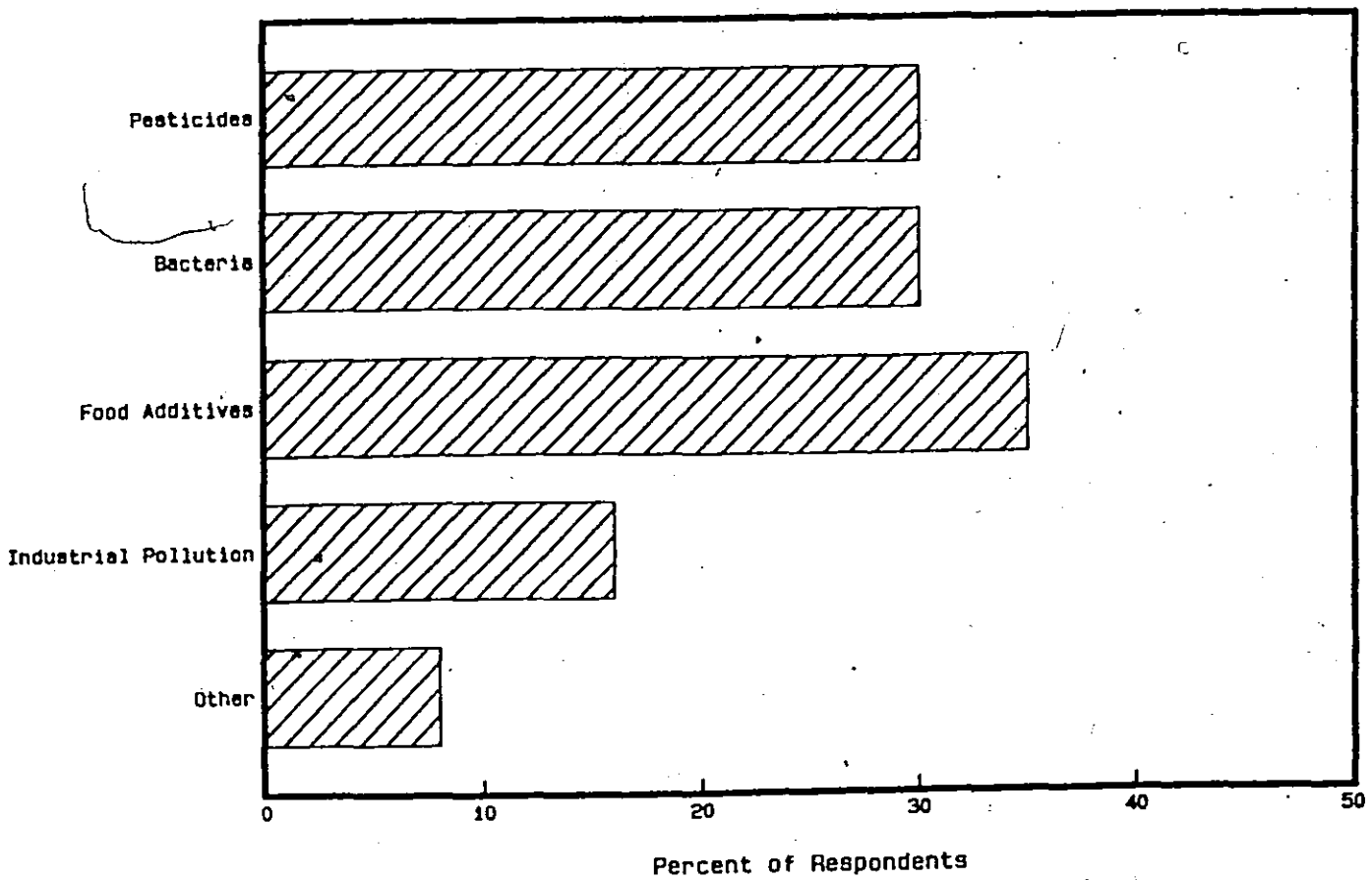


Figure 8.2 Public Perception of Health Risks Associated with the Food Supply

Basin in 1978. Information was obtained by means of telephone interviews with 3,066 people selected at random in Southern Ontario. The objectives of the study included an assessment of current public perception of Great Lakes water quality and public attitudes towards improving water quality.

Over half of the respondents indicated a general dissatisfaction with water quality (Figure 8.3). While the public's perception of water quality was influenced somewhat by personal experience, about 65% of respondents cited the mass media as their major source of water quality information (Table 8.4). The public identified the control of industrial and general pollution as the most important strategies for improving water quality (Table 8.5).

Over half of the respondents felt that government was not doing enough to improve water quality, and indicated a willingness to pay for better water. However, a later study conducted by Environment Canada (1983) revealed that while the majority of Canadians were willing to support extra taxes of $10 per year to reduce water and air pollution, only 13% had actually donated funds to an environmental organization.

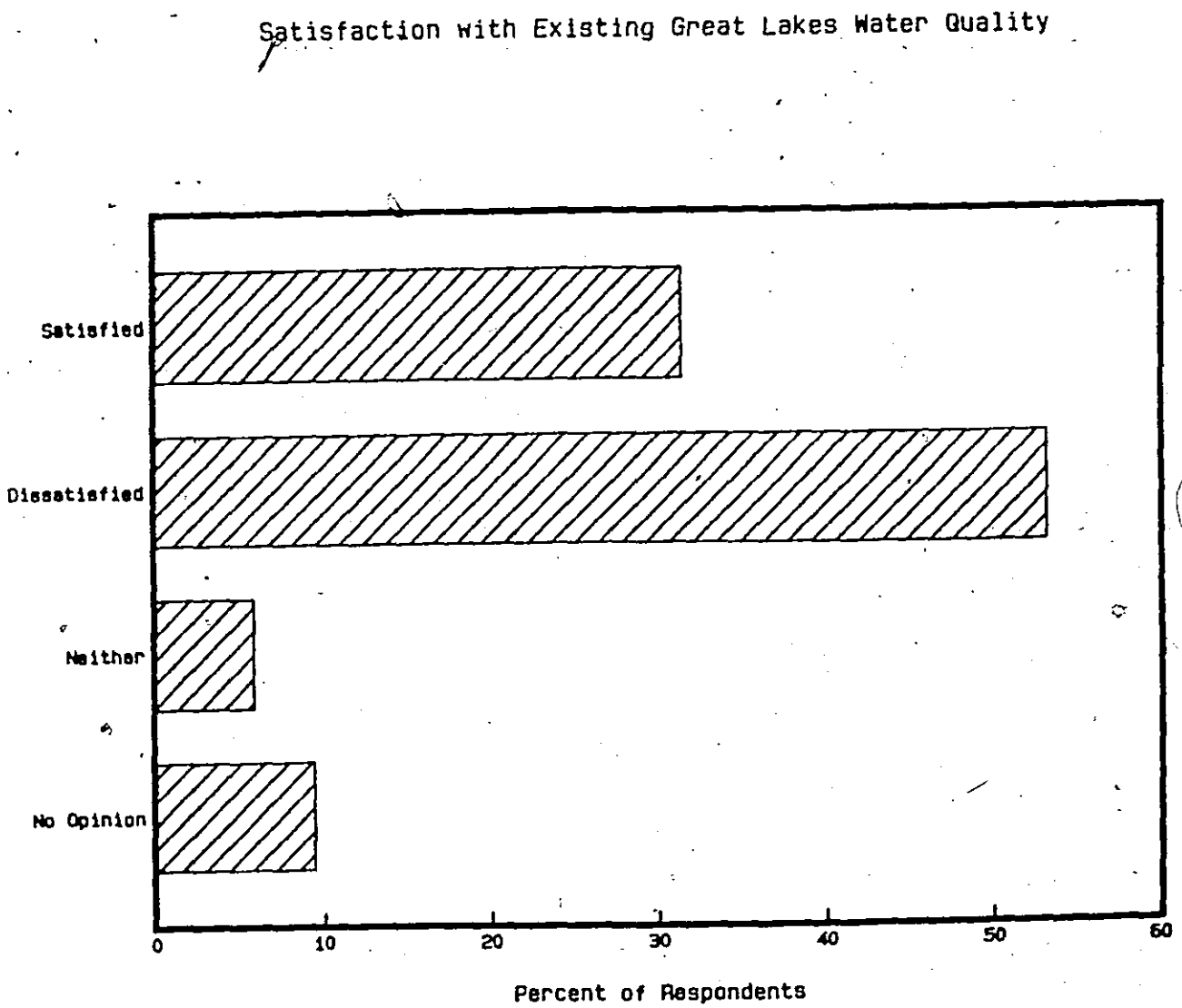


Figure 8.3 Public Perception of Great Lakes Water Quality

Table 8-4 Primary Sources of Information on Water Quality

Source	Percentage of Respondents
Newspapers/Magazines/Books	47.6
Personal Experience	20.2
TV/Radio	17.6
Other People	4.8
Government	0.9
Water Tests	0.7
Educational Institution	0.6
Do Not Know	7.6

Table 8.5 Risk Management Strategies Perceived as Improving Water Quality

Strategy	Percentage of Respondents
Reduce Industrial Pollution	29.9
Control Polluting	17.7
Stricter Enforcement of Regulations	8.6
Improved Waste Treatment	7.9
Public Education	7.3
Stricter Regulations	3.1
Control Pollution from Birds	3.3
Ensure U.S. Co-operation	2.7
Do Not Know	19.5

8.3 FACTORS INFLUENCING RISK PERCEPTION

Difficulties in understanding probabilistic processes, biased media coverage, misleading personal experiences, and the anxieties generated by life's gambles, frequently lead individuals to deny uncertainty, misjudge risks, and maintain unwarranted confidence in judgements of fact (Slovic et al., 1982). Information processing in humans is hindered by biases and limitations which affect their subjective evaluations of probabilities. People do not perceive all lives to be of equal value, nor do they perceive all forms of death as equal. Frequently, nonfatal health impairment such as permanent brain damage is seen as more serious than death itself. In the public's view, the significance of the probability of an event tends to decrease as conceivable consequences increase, until what is possible becomes more important than what is probable (Whyte, 1984).

8.3.1 Psychological Determinants of Perceived Risk

Several laboratory experiments conducted by Lichtenstein et al. (1978) have provided insight into the public's perception of the frequency of lethal events. These studies have revealed two highly consistent but systematic biases in judgements of frequency. First, individuals tended to overestimate the frequency of occurrence of low probability risks such as floods and underestimate the frequency of high probability risks such as cancer and heart disease. Second,

individuals tended to exaggerate the frequency of some risks and to underestimate the frequency of others at different levels of objectively determined frequency. The test subjects in these studies were unable to correct for these sources of bias when specifically instructed to do so.

Errors in the subjective estimates of risks frequently result from heuristic biases, which occur due to the unconscious need to make sense of new information (Slovic et al., 1982). Heuristics are inferential rules used to generalize using limited information, and to reduce difficult mental tasks into simpler ones. Such a process is often valid, but can lead to large and persistent biases in judgement, with serious implications for risk assessment.

The availability heuristic, for example, comes into play when an event is perceived as probable to the extent that instances of it are easily recalled or imagined. This heuristic is greatly affected by selectivity of information presentation, sensationalism, drama, and recency of experience. In the case of low probability hazards, the very discussion of the event may increase the perceived probability of occurrence (Van Horn & Wilson, 1977).

Scientific experiments and casual observations indicate that individuals have difficulty thinking about and resolving risk-benefit

conflicts, even in simple gambling situations (Slovic et al., 1979). In order to deal with the anxieties associated with uncertainty, individuals frequently perceive patterns in event occurrence. Such perceptions increase feelings of control and beliefs that it is possible to predict the occurrence of future events. This principle is embodied in the gambler's fallacy that a long losing streak necessarily heralds a change of luck.

Another means of coping with uncertainty is over-confidence. Individuals frequently believe themselves to be less subject to risks than others. Consequently, there is a tendency to accept higher risks for themselves than they would accept for society as a whole. The problem with over-confidence is that individuals tend to be insensitive of how little information they have in making their risk decisions (Slovic et al., 1979).

Decisions made under conditions of uncertainty are frequently affected by the way in which the problem is framed. A decision frame reflects an individual's conception of the acts, outcomes, and contingencies associated with a particular choice (Tversky & Kahneman, 1981). The decision frame adopted by an individual for any decision is controlled partly by the formulation of the problem, and partly by the norms, habits, and personal characteristics of the decision maker. Rational choice requires that preferences between options not be reversed when frames are changed. However, due to the imperfections of

human perception and decision making, changes in perspective often reverse the relative desirability of options, usually unbeknownst to the individual.

The framing of a problem is particularly important for new and complex risk issues, for which people have no well defined preferences. In such situations, individuals may be unfamiliar with the terms in which the issue is represented, or may hold contradictory values due to their occupancy of different roles in life. They may also experience shifting views with time and be uncertain as to which should form the basis of their decisions. Even when their own inconsistencies in judgement are recognized, they may not want to resolve the corresponding conflicts, resulting in an impasse (Fischhoff et al., 1979).

8.3.2 Sociological Determinants of Perceived Risk

Knowledge and attitude are related to socioeconomic and demographic variables (Whyte & Burton, 1982). Knowledge about the technical, scientific, and medical aspects of hazards tends to be low, but is generally higher for males, younger adults, and better educated individuals. Despite their general lack of knowledge, most individuals feel capable of making risk decisions. Attitude, especially concern about risk, is less clearly defined than is knowledge, although older individuals and women, particularly those with young children, generally express the most concern.

The passage of time generally has the effect of increasing population awareness, and sometimes knowledge, about hazards. Since knowledge is cumulative, the introduction of new information over time has the effect of redefining risk problems and priorities. For example, prior to July 1979, the problem of acid rain was virtually unknown to the Canadian public. Following media publicization of the acid rain problem, awareness increased to the point that, in 1982, acid rain was believed to be the principle risk to the environment (Whyte & Burton, 1982).

According to a recent study, Canadians perceive the severity of risks in relation to the level at which they feel responsibility for protection lies (federal, provincial, local, or individual). Thus, differences in the perceived importance of risk problems across Canada may reflect variations in economic and political conditions. For example, public awareness of acid rain in 1982 was highest in Ontario, and lowest in Quebec, possibly due to differences in scientific, government, and media attention to the issue at provincial levels. Strong regional and local differences have been found for environmental and energy related risks in general, since they are perceived to be within the jurisdiction of the provincial or local governments (Whyte & Burton, 1982).

Given the increasing scientific and technical complexity of many current risks, individuals must accept much of the information and

interpretations of facts by experts on faith. The perceived credibility of information sources influences how information is received. Credibility appears to be high among doctors and government agencies, with the news media having the lowest credibility (see Table 8.3). The level of trust that the public places in government is important, as the public tends to believe warnings of danger, but not reassurances of safety (Whyte & Burton, 1982).

8.3.3 Characteristics of Perceived Risk

Since the public clearly does not perceive all risks in the same lights, it is important to understand the specific characteristics of risks which influence how they are perceived. Many risk characteristics are highly correlated with one another. Analyses of their interrelationships indicates that in general, three main factors largely influence risk perception: the degree to which a risk is understood, the degree to which a risk invokes feelings of dread, and the number of people exposed to the hazard (Slovic et al., 1982).

A detailed review of factors known to influence risk perception was conducted by Whyte & Burton (1982). Their findings are consistent with those of Slovic et al. (1982) and are summarized in Table 8.6. These results indicate that a risk is perceived to be more serious if its consequences are immediate and it directly impacts on the individual, as opposed to causing only an indirect effect through a

complex chain of events. Dread hazards which create great fear or anxiety are of greater concern as are those involving a large number of fatalities simultaneously particularly if the fatalities are grouped in space or time as is generally the case in a major accident.

Concern is enhanced if the process or mechanism leading to the risk in question is not understood or if the individual has little or no control over the risks. Similarly, involuntary risks are less readily accepted than those which are voluntary, and unfamiliar risks are of greater concern than familiar ones. Situations in which children are at risk are viewed with greater concern than otherwise as are situations in which the victims are identifiable as opposed to statistical.

Concern is also heightened when there is little knowledge about the risk. This issue is complex: initial awareness may cause alarm, which decreases once understanding is gained. When more knowledge is obtained, however, the uncertainty associated with scientific knowledge becomes more significant than the gain in reassurance.

Risks for which the information source is not perceived as credible tend to be viewed with greater concern than those for which the source of information is reliable. Concern may also be heightened when there is much media attention, although the net effect depends on the kind and content of coverage.

Table 8.6 Factors Influencing Risk Perception

Factors tending to increase risks as perceived	Factors tending to reduce risks as perceived	Examples
Immediate	Latent or delayed	Tornado-drought; fire asbestos
Direct	Indirect	Flood; drought; bridge failure; mercury in lakes
Dread hazards	Common hazards	Cancer; influenza
Large number of fatalities per event	Small number of fatalities per event	Air crash; auto crash; avalanche; snowstorm
Fatalities grouped in space and time	Fatalities scattered or random in space/time	
Mechanisms or process not understood	Mechanism or process understood	
Uncontrollable	Controllable	
Involuntary	Voluntary	Food additives; smoking; radioactive fallout; mountain climbing
Children at risk	Children not at risk	DDT in human mother's milk
Identifiable victims	Statistical victims	In-plant workers; cancer from environmental radiation
Lack of knowledge	Knowledge	
Lack of belief in authority of source of information	Belief in authority of source of information	Private industry; university scientists
Much media attention	Little media attention	PCB's; LNG transport
Unfamiliar	Familiar	Nuclear accident; house fire
Major accident	No major accident	TMI reactor; Pickering; Flixborough; Dow plant at Sarnia; Mississauga chlorine derailment; LNG transport

8.4 RISK ACCEPTABILITY

While it is important to take into account public perception of risk in the risk assessment process, it is also necessary to consider what level of risk may be considered acceptable for risk management purposes. Although zero risk is clearly the ideal, this is impossible to achieve with certain naturally occurring contaminants such as dioxin which are ubiquitous in the environment. In other cases, zero risk may only be achieved at great expense, thereby raising the question as to whether or not some small but nonzero level of risk may represent a satisfactory goal in practice. One may also question the advisability of controlling manmade hazards with such vigour that the resulting level of risk is insignificant in relation to risks from common natural hazards or voluntary human activities. For these reasons, it is important to consider what level of risk should be considered as socially acceptable.

8.4.1 Revealed and Expressed Preferences

Traditionally, two proxy measures have been utilized in an attempt to determine what level of risk the public finds acceptable (Whyte & Burton, 1982). The revealed preference approach, first propagated by Starr (1969) is based on the assumption that society, through a process of trial and error, has arrived at a nearly optimal and thus acceptable balance of the risks and benefits associated with any activity.

Consequently, it should be possible to use economic risk-benefit data from recent years to reveal patterns of acceptable risk-benefit trade offs.

Starr's findings suggested that the acceptability of any risk is roughly proportional to the third power of its benefits; that the public seems willing to accept risks from voluntary activities roughly 1000 times greater than those tolerated from involuntary activities that produce the same level of benefit; that the level of risk tolerated for voluntarily accepted hazards is similar to that from disease; and that the acceptable level of risk for any hazard is inversely related to the number of persons exposed.

Although the revealed preference approach utilizes objective data, it has a number of drawbacks (Van Horn & Wilson, 1977; Fischhoff et al., 1979). In general, it infers public values indirectly, using procedures that may be theoretically and politically untenable. It also assumes that past behaviour is a valid predictor of present preferences, which may not be true in light of rapidly changing technology and changing societal goals. It assumes that income distribution and social structures of the past were satisfactory to all parties. The revealed preference approach assumes that reason or choice would not have led to different preferences if risks and benefits had been analyzed in advance and it assumes that people have full information and can use it optimally. It may also undervalue

risks to which the market responds slowly, such as those involving long time lags between exposure and consequences, and it makes strong, not always supported assumptions about the rationality of public decision making in the marketplace.

The expressed preference approach uses such tools as opinion surveys, detailed questioning of selected groups, and interviews with public interest advocates to directly elicit current perceptions about risks. The expressed preference method is useful in that it allows for widespread public participation, and allows consideration of all types of risks and benefits, not merely those which are measured in dollars or in annual fatalities (Fischhoff et al., 1979). The expressed preference method suffers from sampling difficulties and is sensitive both to the formulation and wording of questions as well as the failure of individuals to present their true preferences (Van Horn & Wilson, 1977).

Findings in expressed preferences studies have nonetheless generally concurred with those in revealed preference studies, in that society will tolerate greater risks for greater benefits and that double standards exist for voluntary and involuntary activities. However, such studies did find that perceived risk decreases slightly with overall benefit so that high levels of risks which might be acceptable in Starr's theory would not be acceptable using the expressed preference theory (Whyte & Burton, 1982).

8.4.2 De Minimis Risk

Recognizing that the goal of zero risk is often unattainable or even undesirable, risk management policy makers have debated the concept of a de minimis or trivial level of risk with which society need not concern itself (Spangler, 1987). This is an analogy with the legal dictum de minimis non curat lex which states that "the law is not concerned with trifles".

As illustrated in Figure 8.4, there exists some small level of risk which most people would consider acceptable in the sense of being insignificant or not worth worrying about. What the actual de minimis level of risk should be is not clear, although it may be comparable to or less than the roughly one in a million chance of being killed by lightning each year (Wilson, 1984). With respect to this point, the Royal Society Study Group on Risk Assessment (1983, p. 181) suggested that

> "...the figure of 10^{-6} is probably... appropriate, except perhaps if clear causal links are established in the risks from certain consumer products. In such circumstances we would consider 10^{-7} to be an annual level below which further control was certainly not justified, but even there the problem of the very salient situation (i.e. that certain risks are feared at any level) may well remain."

At the other end of the spectrum, there are clearly risks sufficiently high so as to be deemed unacceptable by the majority of the population. This includes annual risks of death in the order of one in a thousand or one in a hundred, which are greater than the

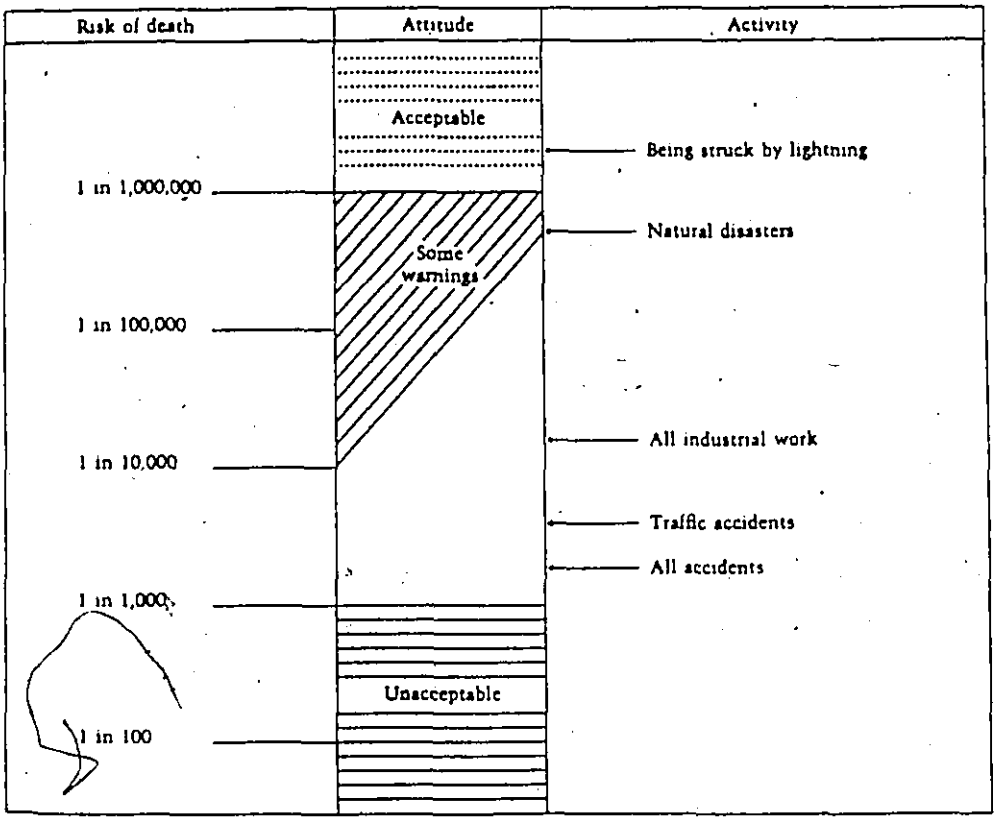


Figure 8.4 Acceptable and Unacceptable Risks

annual risk of a fatal accident. The greatest challenge for decision makers lies not with very small or very large risks, but for those which lie in between these two extremes. In this regard, the Royal Society Study Group further stated that

> "... it is for annual risks of between 10^{-3} and 10^{-6} that risk management should consist of comparing risks, detriments, costs, and benefits, rather than in seeking prohibitions at the upper level, or concluding that no special action is needed at the lower figure".

The establishment of an acceptable level of risk for exposure to substances in the environment capable of causing cancer has assumed particular prominence in discussion of de minimis risk. This debate has its origins in the 1957 Delaney Amendment to the U.S. Food and Drug Act, which prohibits the use of food additives which have been shown to induce cancer in either animals or humans. While such a zero risk philosophy seemed eminently reasonable from the public health point of view, this legislation was later seen by some as being unduly restrictive.

Mantel & Bryan (1961) subsequently took the bold step of proposing that a lifetime cancer risk of one in 100 million might be viewed as insignificant. While this proposal stimulated further discussion on the topic of de minimis risk, no regulating initiatives were taken in this direction until 1977 when the U.S. Food & Drug Administration (FDA, 1977) proposed using the Mantel-Bryan procedure to establish

regulations for carcinogenic residues of feed animals found in the edible tissues of food producing animals, with the lifetime risk limited to one in a million. While the Delaney Amdendment would still apply to direct food additives intentionally incorporated into processed food, non-zero residues of indirect food additives in commodities such as beef and poultry could now be allowed at de minimis levels.

Subsequently, the U.S. Environmental Protection Agency suggested that trace levels of carcinogenic contaminants in drinking water might be tolerated provided that the lifetime cancer risk did not exceed one in 100 thousand (EPA, 1979). The following year, the Agency issued revised guidelines for establishing water quality criteria which cautioned that while exposures corresponding to lifeline risks ranging from 10^{-7} to 10^{-5} would be estimated, these figures did not represent an Agency judgement on what constitutes an acceptable level of risk (EPA, 1980).

This view reflects that currently held by other regulatory agencies in both the United States and Canada. Thus, although no nonzero level of increased cancer risk is likely to be condoned as acceptable, lifetime risks in the range 10^{-7} to 10^{-5} are often implicit guidelines established for exposure to environmental contaminants in water, air and soil.

One concern often expressed about such analysis is the potential for accumulation of risk as a result of exposure to several components. For example, exposure to ten chemicals each having an estimated risk of 10^{-6} would lead to an accumulated risk of 10^{-5} in the absence of any synergistic effects. While this argument is superficially correct, it does presume that the individual risk estimates are accurate, and makes no allowance for the fact that the individual estimates are actually upper bounds rather than best estimates. In general, risk assessment techniques for joint exposures are not well established and require further development before this and other issues relating to mixtures can be adequately addressed (NAS, 1988).

8.5 IMPLICATIONS FOR RISK MANAGEMENT

A fundamental objective of government is to provide regulations which ensure an adequate degree of public protection and which enjoy a broad base of public support. The goal of regulation acceptability implies that regulations must, in some manner, be responsive to the preferences and beliefs of the public (Otway & Thomas, 1982).

Measurement of public perception of risk can provide decision makers with an important tool to evaluate the significance of the state of the environment and of their proposals for development and conservation of the environment (Whyte, 1984). Studies of public attitudes can highlight the concerns of people at risk, and be used to forecast their reactions to hazards and their management. Lack of such

information could increase the likelihood that well intentioned policies will fail to meet such goals (Slovic et al., 1982).

Covello et al. (1982) suggest that an understanding of risk perception is crucial because it contributes, through both societal and political processes, to societal decisions about risk acceptability. Risk management policies governing various types of hazards are in fact determined to a large extent by people who are not scientists or experts in risk assessment (Hohenemser et al., 1983).

Although risk perception does have a place in risk management decision making, it should not replace objective risk analysis (Royal Society, Study Group 1983). Governments frequently have information that is generally not available to the public, and generally enjoy a broader perspective on risk issues. Further, governments have a responsibility to lead, and consequently have permission to act at times in a manner contrary to the wishes of segments of the population. Policy directed primarily at alleviating public anxieties can result in large expenditures having low cost effectiveness. Conversely, serious risks may also be neglected due to public indifference. In general, risk managers should thus strive to keep risk perception broadly in line with the actual magnitude of hazards so as to avoid distortion of public health priorities.

If risk perceptions is to be considered in risk assessment and risk management, decision makers must determine how best to measure these perceptions, how to reconcile conflicting perceptions, and how to balance public perception, expert opinion and other inputs in the decision making process (Whyte, 1984). These are not easy questions to answer, but they must be addressed if decisions are to be accepted by the public.

8.6 SUMMARY AND CONCLUSIONS

In contrast to the scientific endeavour of risk analysis, risk perception is a subjective process by which individuals intuitively assess risk. Because of human limitations in information processing, the public often misjudges risk and arrives at perception of risk which can be at odds with objective estimates. Nonetheless, decision makers need to be aware of the public's perception of the risk associated with different hazards, and give their views proper consideration in selecting an appropriate risk management strategy. This is important in order that decisions on the control of risk enjoy public acceptance.

Information on public perception of health and environmental risks in Canada may be obtained from public opinion surveys. In a recent study of twelve activities and substances, illegal drugs, smoking and alcohol were rated as being of greatest concern, whereas home hazards

and tap water were ranked lowest. While the majority of respondents in this survey identified the news media as being their primary source of information on health risks, the news media ranked lowest in terms of credibility.

A number of studies designed to elucidate the factors influencing risk perception have been conducted over the years. Psychological studies have demonstrated that people tend to overestimate the frequency of rare events and underestimate the frequency of common events. Perceptions of risk are also known to change over time as new information becomes available.

A number of particular risk characteristics are known to influence risk perception. For example, individuals tend to voluntarily accept higher risk levels for themselves than for society as a whole, frequently because they feel that the benefits are worthwhile and that they have autonomy over their lives. In general, three main factors have been shown to influence perceived risk: the degree to which the hazard is understood, the degree to which it involves feelings of dread, and the size of the population at risk.

In an attempt to develop risk policies which are satisfactory to the public, decision makers have frequently adopted the notion of acceptable risk. Acceptable risk is based on the assumption that there exists a nonzero level of probability of occurrence for any event,

below which the public as a whole is willing to accept the risk. This concept has been widely debated as a means of establishing acceptable levels of exposure to trace levels of carcinogenic agents present in the human environment. Although regulatory authorities are reluctant to establish a precise level of risk which they would support as tolerable, lifetime risks in the order of one in a million seem to have been accepted in previous regulatory actions.

Although an important component of risk assessment, risk perception should not replace risk analysis. Those charged with making critical risk decisions generally enjoy a broader perspective and access to more information than the public. Since risk perceptiondoes contribute to societal decisions, the challenge for decision makers is to remain attuned to the public's perception of risk, and to balance that perception not only against the scientific inputs into the risk assessment process, but also against the social, economic and political factors inherent in most decisions.

9. RISK COMMUNICATION

9.1 INTRODUCTION

Communication of risk related information is clearly a critical part of the risk assessment process. In particular, it is important that risk data be expressed in understandable terms, that uncertainties surrounding estimates of risk be understood, and that effective channels of communication be established among individual and corporate actors involved in the assessment of risk.

The term risk communication has come into use in recent years to describe this process, and has been formally defined by Covello et al. (1987) as "any purposeful exchange of information about health or environmental risks between interested parties". More specifically, they state that

> "...risk communication is the act of conveying or transmitting information between interested parties about (a) levels or health or environmental risks; (b) the significance or meaning of health or environmental risks; or (c) decisions, action, or policies aimed at managing or controlling health or environmental risks. Interested parties include government agencies, corporations and industry groups, unions, the media, scientists, professional organizations, public interest groups, and individual citizens."

With this broad definition, risk communication encompasses most forms of communication within the process of risk assessment and risk management, including information flows between academic experts, regulatory authorities, special interest groups and the general public. However, it also focuses on the communicative processes through which individuals and institutional actors negotiate their interests and concerns about the assessment and management of risks in contemporary society. Thus, risk communication is what all participants in the risk assessment process engage in by virtue of their public involvement. This includes their attempts to inform, persuade, or warn others, to analyze problems and circulate findings on new knowledge, to change attitudes or behaviour, to set charges or rates for costs, or to carry out legally-mandated responsibilities.

It is well known that there are fundamental disagreements within contemporary society over the proper assessment and management of health and environmental risks. While expert knowledge of many different kinds is called upon to address the complexity of such risks, non-experts are also called upon to approve or disapprove, ultimately through political processes, decisions based on accumulated technical fact and reasoning.

In such situations, the risk communication process itself often becomes an explicit focus of controversy. Charges of media bias or sensationalism, of distorted or selective use of information by

advocates, of hidden agendas or irrational standpoints, and of the inability or unwillingness of regulatory authorities to communicate vital information in a language that the public can understand, are common (Slovic, 1986; Keeney & von Winterfeldt, 1986; Mazur, 1984; Sharlin, 1986). Such charges are traded frequently at public hearings, judicial proceedings, and conferences, expressing the general and pervasive sense of mistrust felt by many participants towards others. Of course there are also genuine differences of principle, outlook and values among the citizenry; disagreements will persist even with the most complete and dispassionate knowledge of others' views.

As a discipline, risk communication tries to achieve an adequate understanding of the communication processes in the risk area, an understanding that responds to its inherent complexity and array of participants. In terms of its practical orientation, risk communication seeks to improve the workings of these processes, and so reduce the level of mistrust among participants. The ultimate objective is to assist in the formation of a reasonable consensus in contemporary society on how to assess and manage risk.

In this chapter, we attempt to elucidate the role of risk communication in the process of risk assessment and risk management. We begin with a discussion of three theoretical models which have been proposed to describe the process of risk communication (section 9.2). This is followed by a review of current risk communication

practices used by individuals and institutions (section 9.3). A summary of the main points of the chapter is provided in section 9.4 along with a discussion of the importance of good risk communication practices in risk assessment.

9.2 RISK COMMUNICATION MODELS

A number of models have been developed to describe the nature of the risk communication process. These include the information flow or institutional model, the message transmission model, and the communications processes model (Leiss & Krewski, 1987).

The information flow model is oriented towards the flow of information on technological risk among institutional actors (Baram, 1984, 1986ab). Its focus is on the ways in which such information is transmitted from those who initially possess it to those who may ultimately acquire it.

The message transmission model draws upon a well-known engineering interpretation of communications theory (Shannon & Weaver, 1949). This theory was adapted to the special case of risk communication by Covello et al. (1986, 1987). The message transmission theory is based on the categories of source, channel, and receiver, together with the message that is carried in the system.

A third model seeks to combine the essential features of the previous two, namely institutional structures and message transmission processes, to provide a more complete and dynamic representation of risk communication. The basic idea underlying this concept is that risk communication is a process that essentially involves the interplay of two social domains, called the expert sphere and the public sphere. This interaction is constrained both by the nature of risk and by the conventional institutional channels that direct the flow of information and opinion in contemporary society. These three models are discussed briefly below.

9.2.1 The Information Flow Model

In the information flow model, the primary communication pathways are from the institutions which generate the basic knowledge about risk - industry, researchers or experts, and regulatory agencies - to individuals and the general public (paths A, B and C in Figure 9.1). There is also a two-way information flow between industry and agencies and between agencies and researchers (paths B' and C'). The media acquire information about risk from industry, researchers and regulatory agencies (path D') and transmit that information to interested parties and the general public along path D.

This model is rooted within a legal framework in which duties, responsibilities, culpability, and liability can be assigned to the

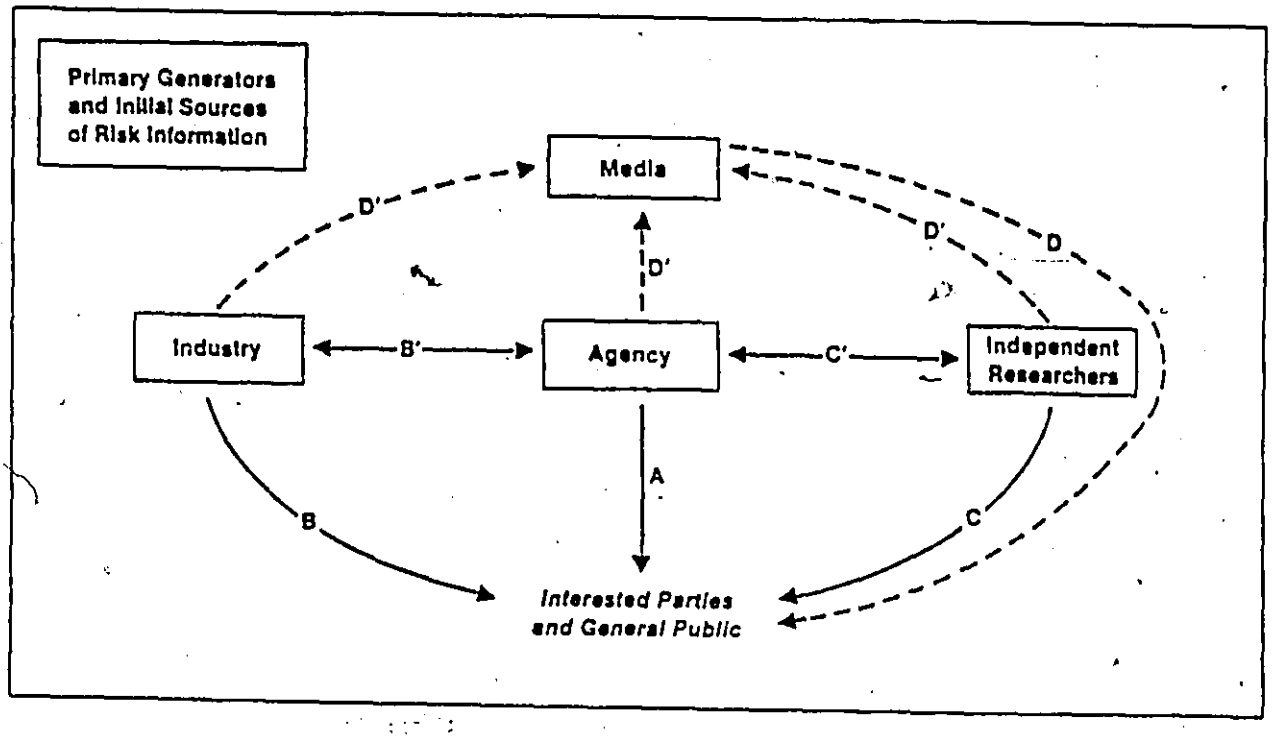


Figure 9.1 The Information Flow Model of Risk Communication

various institutional or individual actors, on the basis of the risk information which they transmit (or fail to transmit) to others. This framework reflects the fact that society has attempted to face the dilemma of risk generation by creating institutional and legal structures of increasing complexity by which to monitor and manage risks, and to compensate those harmed by the accompanying hazards.

In the United States, the responsibilities of regulatory agencies tend to be mandated in legislation (National Research Council, 1983). This includes generic requirements for information generation under the Administrative Procedures Act and the Freedom of Information Act. The Administrative Procedures Act gives rise to elaborate rule-making procedures with the corresponding documentation published regularly in the Federal Register. Other more specific statutes such as the Toxic Substances Control Act establish authority for setting environmental standards and impose requirements for information generation, notice of hazards, and disclosure of information. These responsibilities are supplemented by obligations born by industry under the common law of torts in the U.S., especially the "duty to warn" of product-related or occupational hazards, and the corresponding entitlements to compensation enforceable through individual or class-action lawsuits by those who suffer injury as a result of them.

In Canada, the corresponding instruments are the Canada Gazette and the Access to Information Act. However, a very different

constitutional structure means that the purely legal exercise of these types of duties exists in a much more restricted form. In contrast to the separation-of-powers doctrine in the U.S., the unified authority of the parliamentary system means that generally neither the judiciary nor legislative committees will challenge the administrative acts and procedures of government departments, or force them to disclose the information base or rationale for their decisions. For the same reason, individuals and special interest groups by and large have been unable to force disclosure of additional information from either government agencies or private industry through legal action. To date there has been a relatively limited flow of risk information in Canada along all of the communications pathways identified in the information flow model. However, both government agencies and industry recently have been moving in the direction of enhanced risk information flow in an attempt to increase public confidence in industry practices and in regulatory decision making.

The information flow model does have several limitations. First, this is an effective risk communication model only when the capability exists to stimulate an adequate level of information flow through frequent resort to legal action, or the threat of such action. In this regard, it describes the U.S. situation well, but not that of Canada or of Western European nations (O'Hare, 1980). Second, it represents the media as a simple transmission mechanism for risk information, rather than as an active player in the dynamic tension of risk

communication processes. And third, it represents the category of "interested parties and the general public" as the purely passive recipient of the sum total of all the risk information travelling along the A-B-C-D pathways. This overlooks the crucial impact of public understanding and perception of risk on societal decision making.

9.2.2 The Message Transmission Model

The message transmission model is based on a well-known engineering theory of communications. In the pure version of this model, the message is simply an electronic signal, and the system objective is to reproduce the source signal without distortion at the receiving end (Figure 9.2).

In human communications, the basic idea for this theory is illustrated by telephone and telegraph communications. A message ("I will arrive at the airport at 9 o'clock") is carried from sender to receiver via a specific technological system. Problems may arise in any one of four dimensions: (1) source (sender): speaks poorly and is misunderstood; (2) channel (transmission technology): static on the line; (3) receiver: is inattentive and misses the words or their intended meaning; (4) message: is ambiguous or incomplete (morning or evening?). In the telegraph version, possible problems at the sending or receiving points involve encoding and decoding.

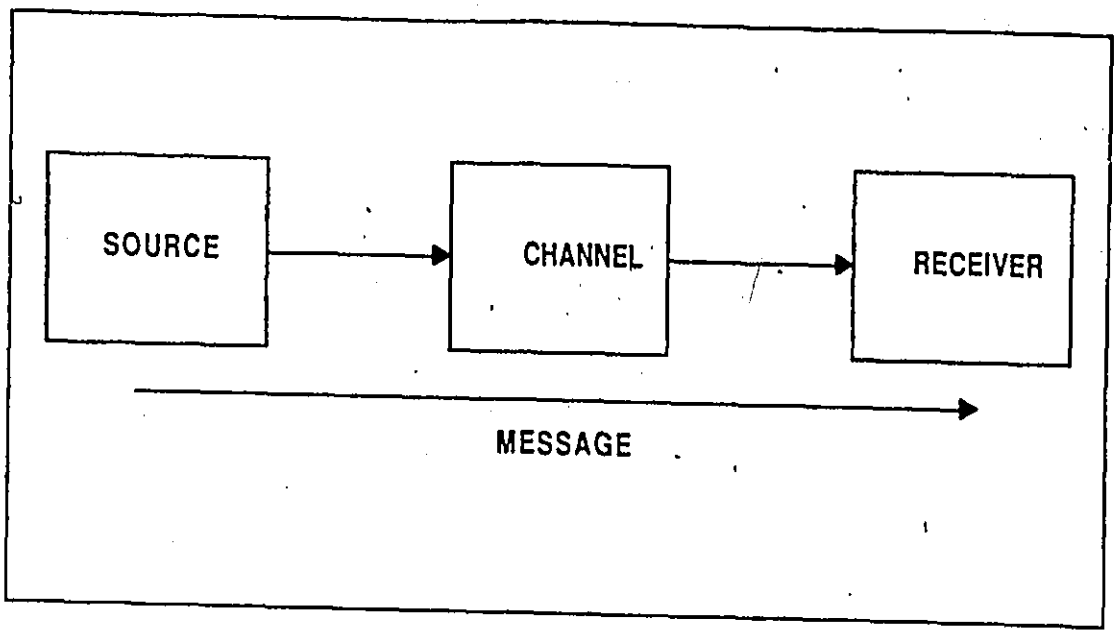


Figure 9.2 The Message Transmission Model of Risk Communication

Covello et al. (1986, 1987) adopted this model for their comprehensive review of the risk communication. In particular, they have organized their discussion of outstanding problems in this field in terms of the four dimensions given above.

Message Problems. Message problems include both deficiencies in the knowledge base and analytical techniques used by experts to assess risk, as well as difficulties in expressing inherently complex risk assessments in a way that the general public can understand. There are deficiencies in the current state of scientific understanding of risk and in the methods used for assessing risk. The difficulties experienced by the general public attempting to understand highly technical analyses, especially quantitative analyses, represent a barrier to risk communication.

Source Problems. Source problems include such regular occurrences as disagreements among experts which fuel public uncertainty and concern. In many cases, resource limitations mean that these uncertainties cannot be reduced. There is also a lack of data addressing the specific fears and concerns of individuals and communities and a limited understanding on the part of many experts of the interests, concerns, fears, values, priorities, and preferences of individual citizens and public interest groups. Finally, the use of bureaucratic, legalistic, and technical language can lead to a lack of trust and credibility in experts on the part of members of the general public.

Channel Problems. In this model, channel problems are primarily a function of the role and practices of the mass media in risk communication. The main problems are thought to include selective or biased media reporting, premature disclosure of information and oversimplifications, distortions, or inaccuracies in interpreting technical risk information (Mazur, 1984; Sharlin, 1986; Slovic, 1986; Keeney & von Winterfeldt, 1986). However, these phenomena are not well understood and further research on the role of the media in risk communication is needed.

Receiver Problems. The most serious problems associated with the receiver include inaccurate perceptions of risk and indeed lack of sufficient interest in risk on the part of many citizens. Research on risk perception has shown a significant level of overconfidence in one's ability to avoid harm, as well as strong beliefs based on this overconfidence which are resistant to change; both of these factors make the tasks of risk communicators very difficult to carry out. There are also unrealistic expectations about the effectiveness of regulatory action; demands for levels of scientific certainty that are impossible to achieve; and a reluctance to accept trade-offs.

In the light of these problems, Covello et al. (1987) suggest that the most promising approaches to better risk communication are to effect improvements in the following areas: information and education; behaviour change and protective action (to encourage risk-avoidance);

disaster warnings and emergency information; and joint problem-solving and conflict resolution in which the public is actively involved in risk management decision making.

The model has many strengths, especially in comparison with the information flow model. First, it identifies a single basic structure that is at the core of all risk communication processes - namely, the tension between experts or expert knowledge and the public or various publics, which operate with what we might call common-sense understanding. Second, the preceding review of problems shows that difficulties arise at every point along the message transmission line. This allows us not just to assign legal or ethical responsibility to actors in these processes, but also to pinpoint specific types of deficiencies and thus to explore remedies for them. Third, and most important, this model represents the message or the communications process as the dynamic core of risk communication. This is because in all four domains (source, channel, receiver, and the message proper), both opportunities and problems actually arise when the system is activated for the purpose of message transmission.

There are some notable weaknesses, such as the fact that the theory on which the model is based portrays communications processes as a collection of one-way transmissions. Thus, although the model's proponents are certainly well aware that most problems arise in the interactions among participants, the message transmission theory does not and indeed cannot represent them well.

Another principal difficulty with this model is its inadequate representation of the role of the media, which is placed in the domain of channel problems. In the message transmission theory, the channel is seen as simply a technological solution to the system requirement for undistorted signal reproduction. However, the mass media in society are not, and do not pretend to be, a neutral transmitter of information. The media are, in the fullest sense, independent actors in social communications processes, with the various forms of print and electronic media each having its own organization agenda and modus operandi. An adequate model for risk communications must represent them as such.

9.2.3 The Communication Processes Model

The communication processes model proposed by Leiss & Krewski (1987) seeks to incorporate the best feature of the previous two, and to improve upon them. As illustrated in Figure 9.3, this model essentially involves the interplay of two domains called the "expert sphere" and the "public sphere" (Covello et al. 1986, 1987; Fischoff et al. 1983). The reasons for holding such a view will become apparent if we examine each side of this interplay in turn.

On the expert side, we must recognize that hazard identification and risk estimation are matters requiring considerable technical knowledge, including measurements and analyses employing highly

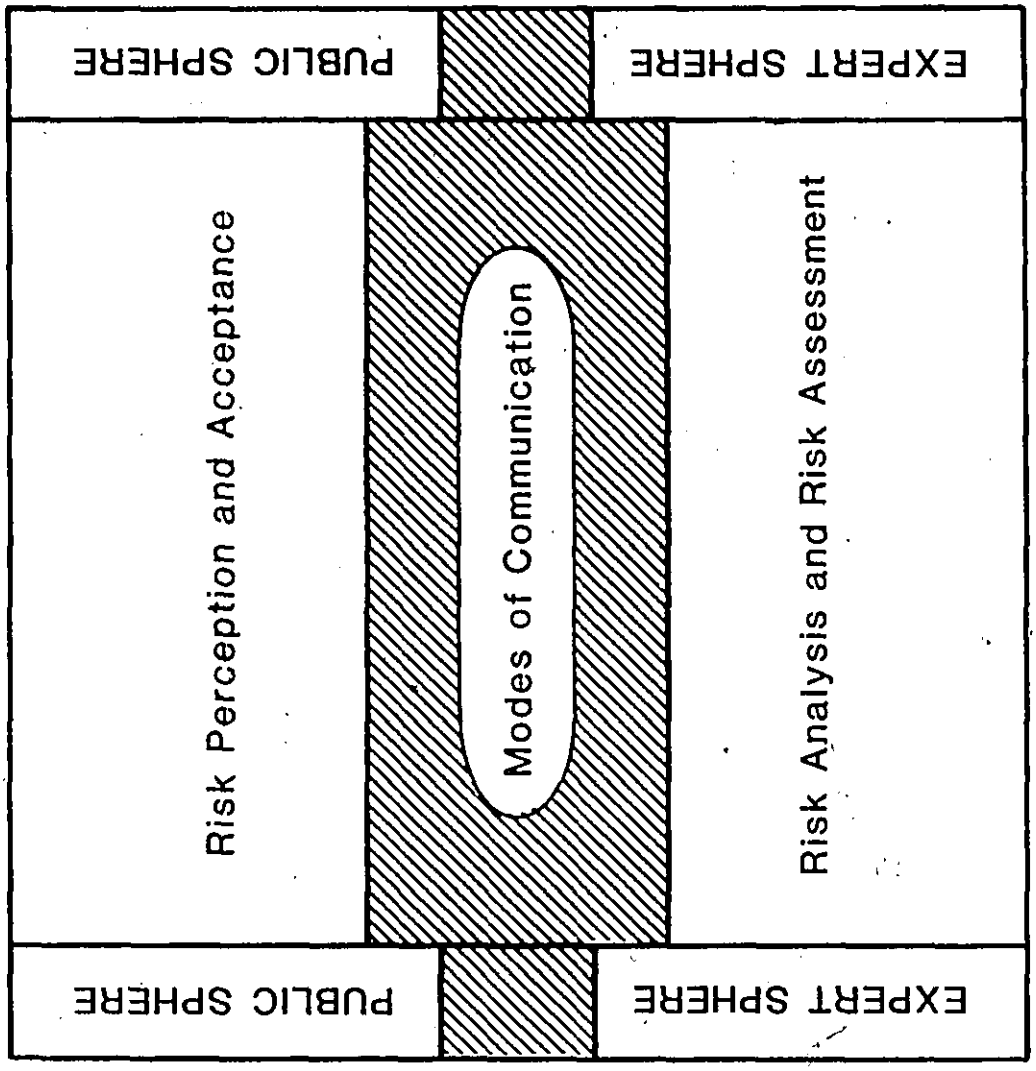


Figure 9.3 The Communications Processes Model of Risk Communication

sophisticated methodologies. The probabilistic dimension of risk necessarily entails the use of technical language for its description. In addition, insurance coverage and legal liability mean that non-scientific but nonetheless highly professional expertise also will be called upon in the assessment of risks and their consequences.

On the public side, the determination of risk acceptability is, in the final analysis, made by the citizenry as a whole through political decision making processes, including the electoral support of politicians and parties with certain stances on the management of risk. Of course, most decisions will still be made by public- and private-sector officials, acting with the advice of experts, in a great variety of forums. However, in a democratic society the parameters within which the discretionary authority of these officials may be exercised at any time will be set by the general character of risk acceptance and perception in society as a whole, as well as by relevant economic, social, and political factors. The state of societal risk perception and acceptance must be factored into risk management decision making; if risk managers do not do so, they themselves run the risk of igniting bitter public controversy and possibly the reversal of their decisions.

The model highlights the tension between expert and public spheres which, it is submitted, is a distinctive feature of risk communication. (Other forms of social communication, such as political discourse or advertising, are not structured in this way.) This is an implicit

feature of the message transmission model as well; however, that model also embodies, in a form that is not fully clarified, an unacceptable presupposition - namely, that the process originates with a straightforward "signal" (message content) that is subject to distortion as it travels onwards to the recipients. This suggests a degree of apathy on the part of the senders that is unrealistic. The communications processes model, on the other hand, assumes that all participants have an identifiable set of interests that bear upon their involvement, and that these interests play a part in the outcome of risk communications processes.

The message transmission model is deficient because it locates the primary basis for distortion or bias of the message in the channel, which usually is assumed to be the mass media. Selectivity in the choice of emphasis or in the presentation of evidence is, by no means the monopoly of a single group of actors; among other things, this view ignores the fact that a good deal of valuable risk information - including some that otherwise would never have seen the light of day - has been forced into circulation as a result of media efforts.

The main point is that an adequate model of communications processes must assume that all participants are generally interested in risk communication and that all play the game pretty much in the same way. A complete set of case studies on risk communication would show, without a doubt, that all actors - industry, government, and the public

- have behaved with complete candour at some times, and at others have been demonstrably biased or selective in their communications on risk.

9.3 RISK COMMUNICATION IN PRACTICE

As indicated in section 9.2, institutions and individuals have been engaged in communicating information about risk in various ways for a long time. What follows is a sketch of some risk communication strategies and practices being used in Canada today (see also Grima et al., 1986; Krewski & Birkwood, 1987; Salter, 1985b). This overview does not pretend to be a complete survey of risk communication practices, but rather is intended to illustrate some of the information flow patterns identified in the various models discussed earlier.

Within the context of the communication processes model, information flows between the expert and public spheres is illustrated by print-media information distribution mechanisms and consultation and feedback strategies. Within the expert sphere, the activities of expert committees may be used to illustrate risk communication practices among technical specialists. Within the public sphere, the interactions between the media and interest groups represent an obvious form of risk communication. Specific illustrations drawn from risk communication practices currently employed by departments of the federal government of Canada are given below.

9.3.1 Print-Media Information Distribution Mechanisms

A wide variety of information distribution formats have been developed for communicating with an array of publics, including industry, provincial and federal officials, and environmental groups. To begin, let us consider some formats used by the Pesticides Directorate of Agriculture Canada for matters relating to the assessment and registration of pesticides.

The Department issues formal memoranda providing up-to-date information on matters of special concern (such as the memorandum of September 11, 1986 on 2,4-D) for distribution to public interest and pesticides user groups as well as members of the Canadian Association of Pesticide Control Officers (CAPCO). Trade memoranda and memoranda to registrants are directed at the manufacturers of registered pesticides, users, and government agencies, and contain information on all matters affecting specific regulatory practices for registered products under the Pest Control Products Act, such as changes in data requirements for product registration. Discussion documents used to summarize the background and rationale for a particular regulatory decision are available to the general public. New product notes are similar in format to the discussion documents, but also include directions for use, storage and disposal of pesticides, and discuss limitations on uses and first aid treatments for emergencies. The Department also issues a regular bulletin directed at the members of

the Canadian Association of Pesticide Control Officers called CAPCO Notes. A regular publication called Pesticide Information contains current information for applicators on all aspects of pesticides use, as do more specialized reports on current research.

Health & Welfare Canada's Information Letters are directed primarily to scientific researchers in industry, doctors, and pharmacists. Such reports are authored by various units in the Health Protection Branch and contain up-to-date information on all aspects of health concerns. Health & Welfare Canada's programs for public information on medication and prescription drugs, on the other hand, are directed at the general public. The program for supplementary information on medication deals with such matters as side effects and interactions between drugs. For example, a patient package insert is provided for oral contraceptives; and there is a special advisory on acetylsalicylic acid and Reye's syndrome.

The Health Promotion Branch of the Department is charged with motivating Canadians to adopt healthy lifestyles and has a great array of communications programs in all forms of media (leaflets and bulletins, magazines, booklets, displays and audio-visual materials) directed at the general public. There are major programs on smoking, drinking, the workplace, diet, and physical exercise. It is generally accepted today that visually-oriented communications strategies have a greater impact than do print-media formats, especially for messages

directed at younger persons (Leiss et al., 1986), although this varies somewhat according to the type of message. Unfortunately, government information campaigns still are often designed without explicit provisions to measure the likelihood of their effectiveness as, for example, by testing alternative communications strategies. In general, major investments in public-information campaigns should always be accompanied by evaluation programs that seek to test their comparative effectiveness.

9.3.2 Consultative and Feedback Strategies

Environment Canada has formulated a "Public Consultation and Information Availability Policy" which forms an integral part of the formulation of Departmental regulations. This program has a standing provision for consultation between officials and members of the public, including regular discussions on matters of mutual interest at Departmental headquarters and in the regions. It provides an opportunity for members of the public to comment on all significant regulations proposed by the Department. It allows access by members of the public to most Departmental data and published materials. The program also provides financial assistance for public interest groups and professional associations to attend designated Departmental meetings.

There are two recent examples of the implementation of this policy. In September 1986, the Department issued a report entitled

From Cradle to Grave: A Management Approach to Chemicals, which was prepared by a task force with representatives from industry, government, labour, consumers, and environmental groups. This report recommends the adoption of a "life cycle" approach to toxic chemicals management, which seeks to track the uses and dispositions of chemicals through all of the typical stages that have been identified: research and development, introduction into the marketplace, manufacture, transportation, distribution, use, and disposal.

In October 1986, the Ministers of Environment Canada and Health and Welfare Canada issued the Final Report of the Environmental Contaminants Act Consultative Committee. This Committee, consisting of representatives of the federal and provincial governments, industry, labour, and environmental interest groups, was charged with advising the two Departments on proposed changes to the Environmental Contaminants Act in areas where a substantial amount of consensus agreement exists. For example, a recommendation has been made for establishing a notification system whereby new chemicals being put into use in Canada must be accompanied by a specific data package that will permit assessment of potential environmental hazards.

To take another example, the mandate of Agriculture Canada's Pest Management Advisory Board is "to improve the pest management process in Canada by assessing pest management issues with its constituents in a fair, open, balanced and complete manner, and by making comprehensive

recommendations concerning the resolution of these issues". The Board seeks to identify matters of significant general concern within its mandate (such as proprietary information), and to utilize a variety of consultative mechanisms - workshops and regional public meetings, commissioned studies, and the facilitation of exchanges among members of different constituencies - to carry out that mandate (Pest Management Advisory Board, 1986).

9.3.3 Expert Committees

Departments such as Health and Welfare Canada have established many expert advisory committees, usually composed of outside experts with a Departmental official serving as secretary. These committees undertake detailed assessments of particular areas of concern such as analgesics, coughs and colds, heroin availability, dermatological products, herbal medication, human reproduction, and biotechnology.

Many technical publications arise from the work of such expert committees, which aim at giving a state-of-the-art assessment of scientific knowledge in an area that is, or may become, a matter of regulatory concern. The Federal-Provincial Working Group on the Development of Guidelines to Control Risks for Women in Industry (a Subcommittee of Health and Welfare Canada's Federal-Provincial Advisory Committee on Environmental and Occupational Health) recently published a special report entitled The Pregnant Worker: A Resource Document

for Health Professionals. This report evaluates potential chemical, physical, microbiological, ergonomic, and physcosocial hazards to pregnant women in the workplace. Health and Welfare Canada, together with Environment Canada, has established an Environmental Contaminants Advisory Committee on Mutagenesis, which issued a report entitled Guidelines on the Use of Mutagenicity Tests in the Toxicological Evaluation of Chemicals in 1986. This report proposes a system for classifying and testing chemicals according to four "levels of concern", arranged according to types of human exposure; testing and data reporting requirements for chemicals would be dependent upon the place assigned to them in the levels-of-concern scheme.

Interdepartmental committees have been formed in such areas as biotechnology and pesticides where several departments have responsibilities for different aspects of an area. Federal-Provincial Advisory Committees, such as Health & Welfare Canada's Committee on Environmental and Occupational Health, regularly exchange information and advice in all areas of health concerns. Finally, most departments have officials who serve on intergovernmental technical or expert committees at the international level, such as the Codex Alimentarius, the Joint Expert Committee on Food Additives, and International Joint Program on Chemical Safety.

9.4 SUMMARY AND CONCLUSIONS

Risk communication practices of various kinds have long been undertaken by governments, industry, researchers, interest groups, and the media. The phrase "risk communication" itself, however, has come into popular use only within the last five years or so. During that time, risk communication has become more clearly separated from other areas that are related closely to it but which have quite a different focus, such as risk perception. The acceptance of the phrase "risk communication" is a positive step, for it helps to call attention to a vital area of concern shared by all those who are involved in risk management endeavours, providing a common language with which they can discuss issues and perspectives. And the rapidly growing body of literature on risk communication is helping to clarify those issues and result in recommendations for improvements in practices.

In this chapter, three theoretical models of risk communication were discussed in an attempt to provide some insight into the risk communication processes. The first model is oriented towards the flow of information on technological risk among institutional actors. Its focus is on the ways in which such information is transmitted from those who generate it to those who may ultimately acquire it.

The second model draws upon a well-known engineering interpretation of communications theory. This message transmission theory was adapted to the discussion of risk communication and is based on the categories of source, channel, and receiver, together with the message that is carried in the system.

A third kind of model seeks to combine the essential features of the other two - namely, message transmission processes and institutional structures - to produce thereby a more complete and dynamic representation of risk communication. The basic idea is that risk communication is a process that is defined by the interplay of two social domains, called the expert sphere and the public sphere. This process is constrained both by the nature of the risk problematic and by the conventional institutional channels that direct the flow of information and opinion in contemporary society.

Current risk communication practices in Canadian federal government departments confirm that the pathways between experts and public spheres in the third model are in practical use. In particular, various print-media information distribution formats serve as a means of communication from experts to the various publics, as well as for communication among experts in different sectors. The consultation and feedback forums operate largely in the public sphere, where non-expert languages predominate. On the other hand, expert committees, by their very nature, are concerned largely with discussions at a highly technical level.

The importance of effective risk communication in risk assessment should not be understated. The current limitations of risk assessment result from the inherent difficulties in communicating research findings from experts to a lay audience which are based on complex

theoretical constructs that necessarily incorporate assumption, probabilities, ranges of uncertainty, and subjective judgements. Current limitations of public understanding occur because people's perceptions of risk are often simply inaccurate, because risk information by its very nature often frightens the public, because strongly-held beliefs are hard to modify, and because views are easily influenced by the ways in which information is presented.

At this point, several steps may be taken to improve risk communication practices in the future (Slovic, 1986). With respect to information presentation, there is a strong need for delivering quantitative risk estimates in intuitively meaningful terms that do not oversimplify problems of uncertainty. We need to know more about how to present the consequences of risk, as well as the probabilities of occurrence, in order to make the appropriate impact at the individual level at which people evaluate how they should respond personally to risk information. We need to know more about the bases of informed consent with respect to establishing an acceptable risk for the public, evaluating both the public's information needs on risk and also the adequacy of information presentations. More research should also be done on the ways in which messages about risk are received by the public, including tests for measuring the "comprehensibility" of different types of messages.

10. CASE STUDY NO. 1: ACID RAIN

10.1 INTRODUCTION

The phenomenon of acid deposition, commonly know as acid rain, is one of the most pressing environmental issues facing Eastern North America, Western Europe and Scandinavia (Office of Technology Assessment, 1984). Most of the acid rain in Eastern North American may be attributed to pollution from sulphuric and nitric acid. The problem begins with anthropogenic emissions of sulphur and nitrogen oxides relating primarily to industrial activities and the use of modern transportation vehicles (Ontario Ministry of the Environment, 1980).

Acid precipitation itself evolves through a cycle of four consecutive stages beginning with the emissions of sulphur and nitrogen compounds, emitted primarily in the form of sulphur dioxide (SO_2) and oxides of nitrogen (NO_x), and transported by winds and air currents at both high and low altitudes. Meteorological conditions can carry these pollutants hundreds and even thousands of miles form their point of origin, allowing time for chemical transformation to acids and other secondary pollutants. They return to earth in the form of acidified rain or snow (wet deposition) or as particulate matter or gases (dry deposition).

Since SO_2 and NO_x are the dominant precursors of sulphates, fine particles and ozone, the basic oxidation mechanisms involving SO_2 and NO_x are of interest. Energy from sunlight facilitates a number of chemical reactions that transform sulphur and nitrogen oxides into sulfuric acid and nitric acid respectively. Homogenous gas phase and heterogeneous reactions are the two basic processes in the oxidation of SO_2 to sulphuric acid or neutralized sulphates (Ozkanyak, 1986). NO_x (mostly NO) is directly emitted from combustion sources and automobiles, and converted to NO_2 following a series of reaction steps.

Nitrate formation in the atmosphere is less well understood than sulfates. Like sulfates, both homogenous and heterogeneous reactions contribute to nitrate aerosol formation. Various nitrogen oxides, volatile acids, and gaseous nitrates are involved as precursors in nitric acid and nitrate aerosol formation reactions. The ultimate production of nitrate aerosols is assumed to involve condensable species such as NH_4NO_3 in the gas phase or complex set of reactions including particulates. Due to limitations of available data base and inherent measurement difficulties, it is difficult to evaluate the nitrate formation mechanisms in the troposphere (Ozkaynak, 1986).

Acid precipitation is known to have serious effects on the natural environment, particularly lakes, rivers and fisheries, as well as

man-made structures and buildings. There is also concern over long-term effects on forests and other vegetation. The effects of acidic precipitation on vegetation and forests include toxic effects on plant cells, disturbances of normal growth, interference with reproductive processes and increased susceptibility to other environmental stress factors (Mchaughlin, 1985). The effects of acidification on aquatic systems range from the reduction of food sources by accelerated fungi growth to the decline of fish stocks in lakes and streams (Ghelardi and Murphy, 1981). Hydrogen and sulfate ions in rain can stimulate corrosion of building materials such as steel, copper and wood. Furthermore, limestone and concrete are vulnerable to accelerated deterioration from acid rain (Ghelardi and Murphy, 1981).

At present, neither the direct health impacts of dry deposition nor the indirect effects of wet deposition are as clearly delineated as those in the environment. In this chapter, the existing body of knowledge concerning the potential health effects of acid deposition, both of a direct and indirect nature, is reviewed (section 10.2). Epidemiological and toxicological studies currently underway within Health and Welfare Canada are also considered, along with the special problems involved in the design of studies intended to detect subtle abnormalities. Data on human exposure to acid deposition is considered

in section 10.3. This information is used to evaluate possible risk management strategies for the control of acid deposition from the point of view of human health (section 10.4). An analysis and evaluation of the acid rain problem within the context of the framework for risk assessment and risk management discussed in the previous chapters is presented in section 10.5. A brief summary of the key issues involved the assessment of human health effects attributable to acid rain is given in section 10.6.

10.2 HEALTH EFFECTS OF AIR POLLUTION

Epidemiological studies have provided much of the evidence in establishing an effect of air pollution on health. The strongest evidence of the public health hazard posed by ambient air pollution was provided by a series of short-term pollution episodes. Between 1930 and 1960 it became evident that exposure to very high levels of air pollution over a period of several days was potentially fatal in certain sub-populations, namely the elderly. In London, 1952, a fog episode was associated with 4000 excess premature deaths. Other studies throughout the world, including Donora, the Meuse Valley, New York and Tokyo have consistently demonstrated a pattern of increased respiratory mortality and/or morbidity on days or during periods of increased pollution burden. Although healthy persons are at risk of adverse

health responses to air pollution exposures, the highest levels of mortality and morbidity occurred in those with pre-existing cardiorespiratory disease.

Epidemiological research into the potential health effects of air pollutants has focused on the study of general or selected populations exposed to various pollutants in ambient air. The type of pollutants studied are generally dictated by industrial, geographical and topographical factors within the area of interest. In these studies, the dominant pollutants are generally perceived as being causally related to a quantifiable health effect, usually respiratory health.

10.2.1 Mortality Attributable to Air Pollution

Despite many years of multidisciplinary study, health effects from air pollutions are not well understood. Problems in measuring exposure, investigating human effects at low exposures, and in translating animal data to humans combine to yield uncertainties in dose-response relationships and in mechanisms of damage.

Several studies (Firket, 1931; Schrenk et al., 1949; Ciocco and Thompson 1961; Waller and Commins, 1966; Gore and Shaddick, 1958; Scott, 1963; Martin and Bradley, 1960; Appling et al. 1975; ; Glasser and Greenburg, 1971; Schimmel and Greenburg, 1972) have demonstrated a relationship between air pollution (particularly sulphur dioxides and

total suspended particulate matter) and mortality in as diverse situations as the Meuse Valley in Belgium in 1930, Donora, Pennsylvania in 1948, London in 1952 and 1962, and New York City in 1953 and 1963. These are isolated instances of air pollution episodes influencing human health. However, there exists evidence that air pollution in general is associated with mortality (Lave and Seskin, 1977). However, the influence of transported pollution is not as clearly understood.

To date, the only report attempting to quantify the total health risk of airborne acidic deposition in Canada has been Hamilton's (1985) analysis of excess mortality attributable to transported air pollution. Transported pollution is composed of several areometric factors (such as fine particles, sulphates, nitrates, ozone, sulphuric acid, temperature, barometric pressure and relative humidity) which may not act independently of each other.

The health effects of each of these factors have not been separated from the mix. Sulphates are used as an indicator of the pollution mix but it is the mix itself that is most likely related to any harmful health effects.

The estimated deposition of sulphates in Canada is determined in the following manner. Sulphates are formed by chemical reactions of SO_2 emissions with other atmospheric pollutants. SO_2 emissions from power plants are used as an estimate of total SO_2 emissions. A steady

state diffusion equation is used in conjunction with average wind speed and direction to predict the deposition of sulphates in an area from local, regional and distant sources.

In order to translate these exposure estimates into estimates of mortality attributable to air pollution, an estimate of the level of mortality resulting from a given unit of exposure was required. Because of the lack of scientific consensus, Hamilton (1985) used a subjective probability distribution as described by Morgan et al., (1978), to arrive at a measure of sulphate health damage. This health damage function is essentially a compilation of expert opinion of five scientists concerning the relationship between sulphate pollution and premature mortality. On average, these results suggest an excess of 3.7 deaths per 10^5 persons exposed to 1 $\mu g/m^3$ of sulphates. There is a considerable probability of a smaller effect and a 14% chance of no effect. The range of uncertainty based on the central 90% of this distribution is 0-11 excess deaths per 10^5 persons $\mu g/m^3$. The range reflects a divergence of opinion over the validity of epidemiological studies indicating a relationship between mortality and air pollution levels. It includes estimates from scientists who believe there is a negligible effect at prevailing sulphate concentrations as well as those believing these levels can result in excess mortality.

This health damage function was primarily derived from studies of mortality on large, heavily polluted, American cities. In these studies, sulphates correlate with SO_2 and TSP. The health impact of

this local pollutant mix may not be the same as the transported mix. Thus, in the absence of direct information on the influence of the long range transport of air pollutants (LRTAP) alone on human mortality, estimates of health damage can only be made using the sulphate-health damage function of Morgan et al. (1978).

Use of linear non-threshold dose-response functions for pollutants other than carcinogens can be criticized as unrealistic. Many pollutants, particularly irritants, are likely to have physiological thresholds - exposure levels below which the body's natural defense mechanisms are able to counteract their harmful effects. Thus the dose-response function of Morgan et al. (1978) may not be representative of the LRTAP situation. Furthermore, since the central 90% of the health damage distribution includes zero, there does not exist strong evidence of an association between LRTAP and mortality.

This health damage function may nonetheless be used in conjunction with exposure data to estimate the level of mortality attributable to air pollution in Canada. The estimated sulphate pollution concentration in each of the ten Canadian provinces based on 1978 SO_2 emissions for Canada and the U.S.A. are given in Table 10.1. In addition to total exposure, the likely contributions of local, Canadian and U.S. sources are also provided. Note that for each province, Canada receives more sulphate deposition from the U.S. than is generated within its own borders. The corresponding estimates of

excess mortality based on 1980 population figures are given in Table 10.2. Although it is estimated that there may be 4300 excess Canadian deaths from air pollution, the range of uncertainty surrounding this figure is 0-13,000. Eleven percent of the excess deaths are due to local pollution, 10% are due to pollution transported from other parts of Canada and about 79% are due to pollution originating in the U.S. The Hamilton model predicts that 79% of sulphate deposition in Canada originate from the U.S. However other estimates are much lower at 50% (Ontario Ministry of the Environment, 1986a). This may be due to the fact that 65% of U.S. SO_2 emissions is due to power plants while only 15% of SO_2 emissions is due to this source in Canada (Franklin et al. 1985). Since Hamilton (1985) uses only emissions of SO_2 from power plants he may be overestimating the U.S. contribution to SO_2 emissions and thus sulphate deposition.

Due to the wide range of uncertainty surrounding these figures, the estimate of 4300 excess Canadian deaths must be viewed with caution. In fact, there is no direct evidence of any Canadian mortality due to LRTAP. Clearly more research is needed which focuses directly on the LRTAP mixture and Canadian mortality data.

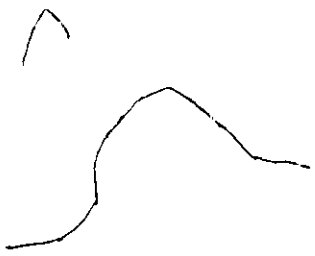
Table 10.1 Estimated Sulfate Pollution Concentrations in Canada by Province and Source (1978 SO_2 Emissions)

Province	Concentration by Source ($\mu g/m^3$)			
	Local	Canada	USA	Total
British Columbia	0.15	0.09	0.60	0.84
Alberta and Saskatchewan	0.45	0.15	0.95	1.5
Manitoba	0.37	0.32	1.1	1.8
Ontario	0.95	0.36	6.0	7.3
Quebec	0.48	0.97	4.5	5.9
New Brunswick	0.12	1.1	5.0	6.2
Newfoundland and Labrador	0.40	0.63	1.9	2.6
Nova Scotia and P.E.I.	0.14	0.92	4.0	5.1

Table 10.2 Estimated Excess Mortality from Transported Air Pollution in Canada
(1978 SO_2 Emission, 1980 Population)

Province	Excess Deaths Per Year*			
	Local	Canada	USA	Total
British Columbia	14	8	57	79
Alberta and Saskatchewan	50	16	100	170
Manitoba	14	12	44	70
Ontario	300	110	1900	2300
Quebec	110	230	1000	1300
New Brunswick	3	29	130	160
Newfoundland and Labrador	1	13	41	55
Nova Scotia and P.E.I.	5	33	150	190
Total	500	450	3400	4300

* The 90% uncertainty interval is 0-3 times the expected value shown in the table.



10.2.2 Morbidity Attributable to Air Pollution

Recent work in the Netherlands, France, the U.S. and Canada has suggested associations between components of acidic deposition and adverse health responses. Van der Lende et al. (1981) has monitored two areas in the Netherlands of differing levels of air pollution for 15 years. At intervals of three years, 2,000 subjects aged 15-54 completed respiratory questionnaires and performed lung tests. After nine years, decreases in lung function were greater in the polluted area. Similarly, the prevalence of respiratory symptoms was also higher in subjects living in the polluted region.

Two large U.S. studies have reported associations between pollutants and health responses. Detels et al (1984) have demonstrated a positive association between oxidants and a decline in pulmonary function in residents of Los Angeles. Ware et al. (1986) have studied over 8,000 children in six cities in the U.S. with varying degrees of air pollution. They have shown that total suspended particulate matter and sulphur dioxide are positively related to an increase in the frequency of chronic respiratory illness and symptoms but no such association was found with pulmonary function.

Bates and Sizto (1983) reported positive associations between ambient sulfate levels and hospital admissions in southwestern Ontario. The analysis focused on two time intervals namely July/August and January/February for the years 1974, and 1976 to 1984. The strength of the association was not dependent on asthma since the correlation was still significant without considering asthma-related admissions. These findings are relevant to the Canadian transported pollutant problem since the sulfates present in this region are predominantly due to long range transport.

Sharratt and Cerny (1979) studied the respiratory health and function of 100 grade one children living in two cities also in southwestern Ontario. Using local monitoring stations in Windsor (high pollution) and London (low pollution) children were compared on the basis of a series of health parameters. The authors reported higher prevalences of respiratory-related symptoms and lower pulmonary function indices in children residing in Windsor.

Neri et al. (1976) studied 6,000 adolescents and adults living in Sudbury and Ottawa, Ontario. The main health metrics were spirometry values and the responses to a questionnaire on chronic respiratory symptoms. The two cities varied in the level of SO_2 measured at standard monitoring stations, with Sudbury having higher concentrations. The authors concluded that a history of residing in the more polluted area was associated with a higher risk of adverse respiratory health.

Kerigan et al. (1983) initiated a three year prospective study of 3200 school children between seven and ten years of age in Hamilton, a steel producing town in southern Ontario with high levels of air pollution, primarily SO_2 and particulate matter. Children in different socio-economic groups were selected from each of four quadrants of the city corresponding to different air pollutant levels. The respiratory status of each child was assessed by means of a detailed questionnaire and pulmonary lung function testing. Total suspended particulates ranged from 35-76 $\mu g/m^3$ on an average annual basis during this period with peaks of 110-120 $\mu g/m^3$. In this study, factors such as outdoor air pollution and the use of gas stoves for cooking were overwhelmed by the effects of maternal smoking in the home, and previous history of respiratory illness.

A 10 day investigation focusing specifically on transported pollutants was performed by Health and Welfare Canada in the summer of 1983. Using serial measures of spirometry and self-reported respiratory symptomatology, 52 campers ages 8 to 16 attending a residential summer camp were studied. Twenty-three of the subjects were diagnosed asthmatics. Using monitoring equipment located at the camp, pollution levels were monitored continuously. The LRTAP mix was associated with decrements in pulmonary function.

A pilot study to assess the chronic respiratory health effects in children from LRTAP was undertaken by Health and Welfare Canada in 1983-84. The respiratory status of over 1400 children aged 7-12 years was examined in Tillsonburg, Ontario (high LRTAP exposure) and Portage

la Prairie, Manitoba (low LRTAP exposure). Respiratory status was determined by three means: pulmonary function testing, questionnaires assessing chronic respiratory symptoms, and bi-weekly telephone interviews assessing acute effects. Inhalable particles (with elemental analysis of sulphates and nitrates), SO_2 and NO_x were monitored in both towns from October 1983 to March 1984. While NO_x and inhalable particles differed little between the towns, Tillsonburg had somewhat elevated levels of SO_2, sulphates and nitrates. The children in Tillsonburg had increased incidence of early hospitalization due to respiratory illness, inhalant allergies, chest colds and a higher incidence of stuffy nose and cough with phlegm than their counterparts in Portage la Prairie. Furthermore, children in Tillsonburg demonstrated a decrement in pulmonary lung function when compared to children in Portage la Praire (Burnett et al., 1988). Due to the confounding effects of region and the clustering effects of community the results of this study do not conclusively demonstrate an association between LRTAP and respiratory health.

Based on the results from the Portage/Tillsonburg pilot study Health and Welfare Canada recently initiated a study to assess the potential health effects of transported pollution on the respiratory health in 5000, seven to eleven year old children in ten communities with populations between 3,000-15,000 people in Canada. Each of these towns generates little of its own pollution. The five communities in south-east Saskatchewan receive little transported pollution and the five towns in southwest Ontario are exposed to the highest levels of transported pollution anywhere in Canada.

10.2.3 Indirect Health Effects of Acid Deposition

Indirect effects of acid precipitation result from the corrosiveness of low pH precipitation and runoff, which may dissolve toxic metals such as aluminum, cadmium, copper, lead and mercury from soils and rocks in lakes and from drinking water distribution systems. In areas characterized by acid precipitation, the drinking water criteria for toxic heavy metals are being exceeded in Canada (Méranger et al., 1983), the United States (Morse et al., 1979; Fuhs & Reddy, 1980) and in southern Scandinavia (Hultberg and Wenblad, 1980).

Lakes in Canada's Precambrian Shield with limited buffering capacity are sensitive to acidification from LRTAP. A study of drinking water pumped from such lakes in Ontario showed concentrations of copper and lead to exceed the levels recommended in the Guidelines for Canadian Drinking Water Quality (Méranger et al., 1983).

Acid leaching of plumbing was implicated as a potential source of elevated copper, lead, and aluminum concentrations in drinking water sampled from wells and shallow springs in the Old Forge-Eagle Bay areas (Fuhs & Olsen, 1979). Copper exceeded the U.S. water quality criteria by a factor of 7 and lead exceeded the lead criterion by a factor of 100.

Elevated ground water and tapwater concentrations of aluminum and copper as well as elevated tapwater concentrations of cadmium and zinc are reported in southwestern Sweden (Hultberg and Wenblad, 1980). Hultberg and Wenblad also reported water quality results from surveys of predominantly shallow wells in three counties in southwestern Sweden. The main source of acid precipitation in the study areas was long range transport of sulphur from other countries. Of the 494 sampled wells, 315 had pH values below 6.0. In one county 49% of the wells had a pH below 5.5 and 13% of the owners reported severe corrosion of copper water pipes.

Studies which attempt to establish a direct relationship between acid deposition and degraded drinking water quality are rare and inconclusive. Even though no deleterious health effects have been associated with these metal concentrations, the potential for metal toxemia exists. Health and Welfare Canada is currently undertaking a program to examine the potential health effects resulting from heavy metals leached into drinking water due to acidic deposition.

10.3 HUMAN EXPOSURE

Currently, there exists several air pollution monitoring networks in Canada operating on an ongoing basis. The National Air Pollution Surveillance (NAPS) monitoring network (Environment Canada, 1984a)

maintains air monitoring stations in most Canadian cities with a population base in excess of 50,000 people. The pollutants measured include sulphur dioxide, nitrogen dioxide and total suspended particulates, all of which are relevant to the problem of acid deposition.

Every two years emission data on these pollutants is compiled by Environment Canada (Environment Canada, 1983). In 1978, the highest SO_2 emissions in Canada occurred in the major population areas of Southwestern Ontario and in the city of Montreal. The mining regions of Quebec, Ontario, Manitoba, Saskatchewan and Alberta also have high emissions. High levels of nitrogen dioxide are mostly centered in large population areas, while high levels of particulate matter are associated with both large population and mining regions. In recent years, the levels of sulphur dioxide, nitrogen dioxide and suspended particulates have fallen. As of 1981, all these pollutants were at or below the maximum desirable objectives (Environment Canada, 1984a).

The Air and Precipitation Monitoring Network (APN) maintained by Environment Canada monitors air quality in areas containing no local source pollution. As such, pollutants monitored at these sites will have been transported from other regions. Data from this network indicate that southwestern Ontario is subject to the highest levels of LRTAP exposure, the Maritimes has moderate exposure and Western Canada receives little transported pollution (Barrie et al., 1982).

The Canadian Network for Sampling Precipitation (CANSAP), Canada's largest monitoring network (54 stations), is a long-term, countrywide system and provides data on the chemical composition of precipitation. The CANSAP program began in 1977 and is designed to sample wet deposition only. In general, pH values have decreased from 1955 to 1977 and pH's of 5.6 or less (the pH of natural rain water) have spread from the Ohio Valley to the entire eastern U.S. and central Canada.

10.4 RISK MANAGEMENT STRATEGIES

10.4.1 Canadian Risk Management Strategies

There are a variety of potential strategies available for management of the acid rain situation. As discussed in chapter 5, these strategies may be broadly classified as regulatory, economic, advisory, and technological. The current Canadian effort relies on a combination of all four strategies in the context of a Federal Emissions Reduction Program.

In March 1984, the Canadian government announced its intent to develop and implement controls to reduce SO_2 emissions. Computer simulation models revealed that a 50% reduction in SO_2 emissions in eastern North America would meet the annual wet sulphate deposition target of 20 kilograms per hectare, the loading rate which would protect all but the most sensitive aquatic ecosystems. Federal and

Provincial Environment Ministers agreed to implement such a decrease by 1994 in two phases (25% by 1990 and 25% by 1994) for those provinces east of Saskatchewan, and agreed in February 1985 to an initial interprovincial allocation of reductions. Federal and provincial governments also agreed to financially support abatement efforts (Environment Canada, 1984b, 1985ab; Lewis & Davis, 1986).

Regulation. There exists legislation at both the federal and provincial levels which influences the nature of acid rain control measures. The Clean Air Act (1970) allows the federal government to set ambient air quality objectives as guidelines for the provinces. Provinces, through their own clean air legislation, control emissions within their borders; the federal government is responsible for international matters. Although provinces maintain responsibility for stationary sources of emissions, that for vehicle emissions is a shared one. Both levels of government also play a role in interprovincial matters (Environment Canada, 1984b, 1985a).

The Clean Air Act was amended in 1980 in order to afford the federal government authority over transboundary pollution originating in Canada. However, the provinces are given the first opportunity to control such pollution. In addition, the Amendment allows the federal Minister of the Environment to recommend site specific standards for sources that might reasonably be anticipated to constitute a significant danger to the health, safety, and welfare of persons in

another country (Clean Air Act, 1980). Although the Clean Air Act has successfully reduced local air quality problems, it has not fully resolved problems of long-range transport and long-term loading of air pollutants, in part because only source emissions, not transformation products, are controlled (Ontario Ministry of the Environment, 1986b).

In response to the acid rain situation, the federal government has issued NO_x emissions standards for all new (i.e. 1988) motor vehicles. Three way catalytic converters will be placed on all such vehicles sold in Canada, reducing NO_x emissions by 20%. Ontario and Quebec, the two largest emitters of acidic gases, have regulated SO_2 emission reductions at INCO (Sudbury), Ontario Hydro, and Noranda (Rouyn and Murdochville). In addition, the Ontario government is currently preparing legislation to control new and modified boilers. The regulation will place a constraint of 1% sulphur content on fuel or require removal of an equivalent amount of SO_2 from flue gas (Environment Canada, 1985a; Lewis & Davis, 1986).

Economic Measures. Federal and provincial governments are using fiscal support to finance the development and implementation of new technologies through cost sharing contributions. As part of the Emissions Reduction Program, the federal government has allocated $25 million to cost share, with industry, the development and demonstration of new processes and pollution control technologies for nonferrous smelters; $150 million to cost share implementation of these new

techniques with the provinces and industry; $470 million for development and testing of new coal burning processes, blending of different varieties of coal, and cleaning and removal of sulphur from coal: and extended a $30 million federal-provincial scientific research program, aimed in part at monitoring the efficacy of the Canadian abatement program (Environment Canada, 1985a; Lewis & Davis, 1986). According to Turner (1988) the Emission Reduction Program goes beyond the traditional concerns of improving local air quality, and specifically deals with the cause of the acid rain problem: total pollution loading.

Advisory Efforts. The federal LRTAP program transfers research information to industry and promotes public awareness of the acid rain problem. In both cases, the program serves in an advisory capacity.

The LRTAP program endeavours to reduce annual wet sulphate deposition to under 20 kilograms per hectare so as to protect moderately sensitive areas, secure necessary emission reductions in both Canada and the United States, monitor acid deposition and its effects, provide the data required to refine abatement programs, and support public information efforts (especially in the U.S.). The program also has the mandate to assess the risks to health posed by airborne pollutants which are subjected to long-range transport and to monitor the impact of abatement programs on human health (Franklin et al., 1985).

Technological Controls. Federal and provincial governments are utilizing a technological approach by allowing polluters to select their own emission control technologies and processes. As a result, the smelter industry is considering such alternatives as process modernization, increasing sulphuric acid production, more selective mining, and enhanced mineral separation during mining. The utilities industry is examining the use of load management, operational changes in generating stations (greater use of low sulphur coal or washed coal), and increased use of hydraulic and nuclear capacity. Both types of industry are also considering expansion of the techniques used to improve local air quality (Environment Canada, 1985ab).

10.4.2 Canada - United States Relations

Joint Canadian - American efforts to address the problems associated with long-range transboundary transport of airborne pollutants have occurred within the context of a longstanding tradition of environmental cooperation. The Boundary Waters Treaty Act of 1909 set a precedent for this relationship, and was followed by the Great Lakes Water Quality Agreements (1971, 1978). The desire of both countries to reduce transboundary air pollution was further revealed by their subscription to the philosophies of the Stockholm Declaration Act (Principle 21, 1972) and the Convention on Long Range Transport of Air Pollution (1979). The Stockholm Declaration Act noted the responsibility of countries to ensure that activities within their

jurisdiction did not damage the environment of others. At the 1979 Convention, signatory states from the Economic Commission for Europe expressed their intent to control transboundary air pollution as far as possible (Environment Canada, 1984b: Lewis & Davis, 1986).

Acid rain was first publicly recognized as an important bilateral environmental problem in the mid-1970's. By 1976, the Canadian Network for Sampling Precipitation had collected sufficient data to indicate the transboundary nature of the acid rain problem, at least in eastern Canada. In that same year recommendations made by the Departments of Fisheries and Environment, along with the potential implications of a proposed large scale oil to coal conversion in the U.S., prompted the establishment of an integrated program on long-range transport of air pollutants in these Canadian Departments (Turner, 1988).

Late in 1978, the U.S. Congress passed a resolution requiring the State Department to negotiate a cooperative air quality agreement with Canada. In response, a Joint Canada - United States Statement on Transboundary Air Quality was issued in July 1979, the emphasis being on long-range transport of air pollution and acid rain (Lewis & Davis, 1986). The statement was followed in August 1980 by a Memorandum of Intent, which formally committed both countries to active enforcement of existing air pollution legislation and to working toward a bilateral agreement. Following the Memorandum, the Canadian Parliament amended

the federal Clean Air Act so as to permit reciprocity with similar U.S. legislation (Lewis & Davis, 1986).

An indication of future Canadian - American cooperation in the matter of acid rain was provided during the first Shamrock Summit talks in March 1985. Both President Reagan and Prime Minister Mulroney noted the severity of the problem as it pertained to bilateral relations. Consequently, they appointed Special Envoys Drew Lewis and William Davis to assess the international environmental problems associated with transboundary air pollution, and to recommend actions to help solve these problems.

In January 1986, the envoys reported that acid rain is a serious transboundary, environmental problem in both countries, and that a limited number of potential avenues exist for achieving major reductions in acidic air emissions, each bringing with it high socioeconomic costs. The envoys issued recommendations in the areas of innovative control technologies, cooperative actions, and research (Lewis & Davis, 1986).

Although both countries accepted the document, the U.S. has not yet committed itself to the recommended five year government-private sector plan for technology research. The second Shamrock Summit in March 1986 produced only an acknowledgement from that acid rain is a problem from President Reagan. The U.S. government did not indicate

when or if it would create an acid rain control program having specific target dates for SO_2 emission reductions.

In late May, the U.S. House of Representatives Energy and Commerce Subcommittee on Health and Environment approved new acid rain legislation. The bill proposed a 50% reduction in SO_2 emissions from coal fired power plants, smelters and boilers, and a 25% reduction in NO_x emissions from motor vehicles. Costs would be absorbed largely through a fifty cent surcharge on all electricity bills. This is the first time that a major acid rain bill had been passed in the House. However, the bill still faces strong opposition from the House, Senate, and the Reagan administration before becoming law (Canadian Environmental Control Newsletter, 1986).

10.4.3 Future Perspectives

Based on current scientific evidence, there is no question as to the severity of the acid deposition problem in Eastern North America; the key policy question is not so much whether to act, but when and how to act (Office of Technology Assessment, 1984). Such a decision must be based on a number of factors, many of which are plagued with uncertainty. These factors include the extent and location of existing acid rain-induced damage; the risk of further damage to human health and the environment; the likelihood that significant advances in

science and control technologies will occur within a reasonable timeframe; the effectiveness of alternative control strategies; and the magnitude and distribution of the socioeconomic impacts of any action on affected parties. These uncertainties, combined with disagreements over the costs and benefits of control measures in relation to other societal priorities, make management of the acid rain situation difficult (Office of Technology Assessment, 1984). The existence of uncertainties does not necessarily imply that decision making should be postponed, however. Rather, it implies that policymakers should be aware of such uncertainties and their implications in development of any strategies for the control of acid deposition.

The current Canadian strategy for SO_2 emission reduction is expected to result in capital expenditures of $1.1-1.5 billion in the nonferrous smelter industry, and $1 billion or more in the utilities sector. The overall cost is expected to be $600 million to $1 billion per year (Turner, 1986). Proposed control measures for reducing NO_x emissions from motor vehicles is expected to increase the costs of vehicles to consumers by $160-$200 million per year for the first ten years. During that time, capital and operating costs to industry may be as high as $494 and $42 million per year respectively (Turner, 1986).

10.5 ANALYSIS AND EVALUATION

The phenomenon of acid rain has evolved in recent years from a relatively unknown problem as recently as ten years ago to a crisis of major proportions throughout Eastern North America and much of Western Europe. The problem originates with anthopogenic emission of sulphur and nitrogen oxides which are subsequently transported over long distances and return to earth as acidified wet or dry deposition.

The environmental effects of acid rain were identified long before suggestions of potential adverse impacts on human health became of concern. These environmental impacts included widespread damage to forests and other vegetation, marked declines in fish stocks in many lakes and streams, and corrosion of buildings. In the presence of these wide ranging environmental effects, questions arose as to the potential for adverse effects on human health.

While many epidemiological studies had previously demonstrated increased morbidity and mortality from air pollution in general, few studies had focused specifically on transported air pollution. Although preliminary studies conducted by Health & Welfare Canada suggest that small decrements in preliminary lung function may occur in children exposed to LRTAP, further research is required both in Canada and abroad to corroborate these initial results. Because of the

complex nature of the acidic deposition, toxicological studies have been of limited value in explaining the health effects of exposure to LRTAP. Given the vagueness of the current information on human health risks, it is not surprising that quantitative estimates of LRTAP mortality are highly uncertain and thus of only limited use for purposes of risk assessment. However, despite the uncertainty in relating health damage to exposure, it is clear that the greatest contribution to overall exposure in Canada is provided by emission sources in the United States.

It is of interest to note that the current Canadian strategy for controlling acid rain employs a combination of the risk management options discussed in chapter 5. Specifically, the regulatory option has been employed through ambient air quality objectives established under the Clean Air Act (see chapter 6). New technologies for the control of source emissions are being developed with some fiscal support from the federal government. Finally, the government has also been active in promoting public awareness of the acid rain problem and in advising industry on the need for new emission controls.

Another interesting dimension of the acid rain problem is its transboundary nature. Because emissions of sulphur and nitrogen oxides are transported from Canada to the United States and vice versa, it would be impossible for Canadian actions to resolve the problem without concomitant action in the United States. Although this has led to

bilateral negotiation on acid rain, it is only in recent months that any positive steps have been taken in the United States to reduce emission levels. If approved, the proposed 50% reduction in SO_2 emissions in the United States would match a previous unilateral commitment of this type taken by Canada in 1983.

The acid rain case also underscores the need for more information for effective risk management. Confidence in any decision pertaining to the management of environmental problems is enhanced when the outcomes of various alternative courses of action can be predicted (Turner, 1988). In the case of acid rain, scientific uncertainty regarding the severity and geographical extent of damage as well as the time frame for future damage have been subject to much debate (Office of Technology Assessment, 1984).

Scientific uncertainties impact significantly on several policy concerns. These include making the control action as fair as possible, minimizing cumulative and irreversible damage and its impact on future generations, weighing the risks of damage against gains which might be made by waiting for the development of better information and technologies, and assuring that the social benefits of the program justify its costs (Office of Technology Assessment, 1984).

Further investigation of key scientific issues could facilitate development of a more effective control strategy for the acid rain

problem. Amongst these key issues are the effects of ions other than SO_2 (especially NO_x and ammonium ions) on acidity the contribution of dry acid deposition to acidity: the nature of the specific sensitivities of natural resources: the relationship between deposition rate and damage to sensitive receptors; and the linking of specific pollution sources and receptor sites (Office of Technology Assessment, 1984; Turner, 1988).

10.6 SUMMARY AND CONCLUSIONS

A brief description of the phenomenon of acid rain and its environmental and health impact is given in this chapter for the Canadian context. The emission distribution of several pollutants in Canada is illustrated and areas of highest long range transported pollution are identified.

In general, there is much uncertainty regarding the health effects of specific pollutants, especially those which are created by chemical and physical reactions in the atmosphere and transported long distances (Hamilton, 1985). While several epidemiological, clinical, and animal studies have demonstrated the respiratory effects of sulphur and nitrogen oxides on humans, few studies have examined the effects of their atmospheric transformation products (Franklin et al., 1985).

Although elevated levels of air pollution have been linked to increased respiratory morbidity and mortality in both North America and Europe, no single pollutant or specific atmospheric reaction has been positively shown to cause these health outcomes (Franklin et al., 1985). Problems are associated with measuring exposures, investigating human effects at low exposures, and translating animal data to the human situation. Such difficulties combine to produce uncertainty in the dose response relationship and in proposed mechanisms of damage. Further complicating the matter are uncertainties associated with the location of the pollution source, the nature of its transport patterns, transformation products, and deposition sites, and with identifying the demographic variables which affect human exposure (Hamilton, 1985).

Several studies have revealed significant correlations between atmospheric air pollution concentrations and mortality. The strongest evidence is provided by several short term episodes of pollution which occurred between 1945-65. High levels of air pollution over a period of several days were found to be fatal, especially in the elderly and in individuals with preexisting cardiorespiratory disease. A recent analysis suggests that the number of excess deaths in Canada from LRTAP is estimated to be 4300 (with a 90% uncertainty interval of 0-13,000), with 79% of these deaths caused by pollution originating in the U.S.A.

Recent studies have suggested an association between the components of acid deposition and adverse respiratory health effects.

In general, these studies found that individuals living in areas having high air pollution levels, had lower levels of lung function and/or higher instances of respiratory illness, relative to those living in less polluted areas. However, relatively few studies have addressed the question of separating the effects of local and transported air pollution.

The current Canadian strategy for management of the acid rain situation using a combination of regulatory, economic, advisory and technological options appears to be a reasonable one. The use of regulation provides a legal imperative for emission reduction. Provision of funds to industry for development and implementation of new technologies increases the likelihood that action will be taken within the specified timeframe without imposing a significant financial burden on polluters. Provision of government financed scientific information to industry through the LRTAP program serves to broaden industry's information base without utilizing its own research funds. Finally, flexibility regarding the choice of technological options allows individual companies to select that which is best suited to their particular situation, and thus increases the likelihood of compliance.

The recommendations of the Special Envoys on acid rain largely advocate advisory options, advocating greater sharing of scientific and technological information between the government and industry in Canada

and the United States. Thus, the recommendations are a well suited supplement to the current Canadian strategy.

The decision to implement a control strategy at present has been made in light of the risks associated with delaying action. However, it is not clear whether independent Canadian action will have any significant impact on the acid rain problem without corresponding controls being implemented in the United States.

The potential impact of the uncertainties associated with the acid rain situation on both human health and the environment highlight the need for further research. Adaptation of current control strategies to accommodate new findings can facilitate the development of better management strategies and better use of the limited resources available for improving environmental health.

11. CASE STUDY NO. 2: SACCHARIN

Anybody who thinks saccharin is injurious to health is an idiot.

Theodore Roosevelt
President of the United States

11.1 INTRODUCTION

Saccharin was discovered accidentally in 1879 by Constantin Fahlberg while working in Ira Remsen's laboratory at Johns Hopkins University in Baltimore (Remsen & Fahlberg, 1879-1880). Fahlberg was investigating the oxidation of toluenesulphonamide when a compound he was working with spilt on his fingers. Fahlberg, who apparently was not the most fastidious of individuals, failed to wash his hands adequately. As a consequence, while eating his supper that evening he noted the sweet taste of his bread and traced the sweetness to the chemical commonly known as saccharin (Munro et al., 1975).

Being 200-700 times sweeter than sugar, its potential as a non-nutritive sweetening agent was quickly recognized. One of saccharin's first commercial uses in North America was in fact as a sugar substitute in canned vegetables and beverages that were subject to heat damage due to lack of refrigeration (ACSH, 1979).

In the intervening years, saccharin has been alternately castigated and approved for human use by various regulatory authorities. The controversy regarding the safety of saccharin for human consumption was well underway by 1890 (Hunter, 1971) and has not appreciably abated in the intervening years. This melodrama has

involved members of the scientific community, trade associations, legislative and regulatory authorities, the press, the public and even a president of the United States.

11.2 EARLY HISTORY

In one of the first tests in which saccharin was administered to man, diabetics were reported to have tolerated five grams of saccharin per day for a period of five months. Another report claimed that the only adverse effect observed in an individual who consumed 52 grams of saccharin over a period of nine days was diarrhea (Neumann, 1927). However, there was apparently sufficient concern about saccharin to hold an international meeting in Brussels in 1908 for the purpose of recommending ways of controlling saccharin in food and beverages (de Boer & Borgsma, 1943).

Some early testing techniques required a degree of self sacrifice rarely seen today. It is recounted that in 1902 the renowned Harvey Wiley, then Chief Chemist with the United States Department of Agriculture, formed a "poison squad" of volunteers to test food ingredients (Marine & van Allen, 1972). After one such test, Wiley recommended to President Theodore Roosevelt that saccharin should be banned because it was "highly injurious to health". Roosevelt declared that "anybody who says saccharin is injurious to health is an idiot" on

grounds that his personal physician, Dr. Riley, prescribed it for him every day (Coon, 1975).

Although President Roosevelt was apparently convinced that saccharin was safe for human use, he nonetheless formed a Referee Board in 1907 to evaluate the influence of saccharin on nutrition and health. The chairman was the co-discoverer of saccharin, Ira Remsen. The Board's findings were announced in 1911 during the presidency of W.H. Taft, and saccharin was banned from use in soft drinks and other foods as an adulterant under the Pure Food and Drugs Act (Munro et al., 1974).

Since sugar was not readily available during World War I, restrictions on the use of saccharin were lifted both in Europe and North America. After the war, its use as a food additive continued and the United States government brought suit against the Monsanto Company for violating the Pure Food and Drugs Act in 1919; the case was eventually dismissed in 1925 (Munro et al., 1974). Subsequently, numerous animal tests of relatively short duration were conducted to ascertain potential adverse health effects in man. In 1951, Fitzhugh et al. reported an excess incidence of lymphomas in certain groups of rats fed diets containing saccharin for prolonged periods.

In 1955, the U.S. National Academy of Sciences/National Research Council (NAS/NRC) Committee on Food Protection reviewed the scientific evidence on the safety of saccharin. They concluded that the maximum

expected consumption was not likely to be hazardous and suggested that an acceptable intake of saccharin was at least one gram per day.

The consumption of saccharin had been increasing steadily in North America after the case against Monsanto was dismissed in 1925, and its use further escalated after 1959 when the U.S. Food and Drug Administration (FDA) allowed artificial sweeteners to be used in a variety of prepared foods initially designed for use in special diets. Later, in the diet soft-drink revolution of the sixties, such foods were marketed not only for consumption by people who had to restrict their sugar or caloric intake, but as non-nutritive or non-caloric products for use by the general public (Munro et al., 1974).

Due to the increased use of saccharin, either alone or in combination with cyclamate, the United States Food & Drug Administration requested a re-evaluation of all of the toxicological data on saccharin by an ad hoc Subcommittee of the NAS/NRC Committee on Food Protection in 1968. This committee concluded that an intake of less than one gram of saccharin per day by an adult should present no hazard, but recommended further studies on the basis that the existing animal experiments concerning chronic toxicity and potential carcinogenicity of saccharin were outmoded by current standards (National Research Council, 1968).

The bulk of the information on which saccharin's current regulatory status is based became available subsequent to this review by the United States National Academy of Sciences in 1968. As discussed in the next section, the major toxicological finding was the induction of tumours of the urinary bladder in second generation rats whose parents were also fed saccharin. These toxicological results prompted an extensive series of epidemiological investigations of human populations in attempt to confirm the results of the animal studies.

11.3 HEALTH EFFECTS

11.3.1 Toxicological Studies

Saccharin has been the subject of extensive toxicological investigation over the years (see Arnold et al., 1983, for a detailed review). Of the many studies conducted, the most important toxicological findings have been the induction of tumours of the urinary bladder in rats exposed to high doses of saccharin in the laboratory. These results were first observed in two-generation studies in which offspring of the parent generation were subjected to dietary exposure to saccharin for the duration of their lifespan. Because the parent generation is also exposed in this test system, the second generation animals may also be exposed in utero prior to birth as well as via their mother's milk following parturition.

The first two-generation bioassay was conducted by the Wisconsin Alumni Research Foundation. In this study, groups of Sprague-Dawley rats were fed diets containing 0.0, 0.05, 0.5 or 5.0% sodium saccharin produced by the Remsen-Fahlberg procedure for 14 weeks prior to mating, plus during mating and lactation. Following parturition, the second generation was weaned onto their parent's diet, which they consumed until the study was terminated 100 weeks later. Thus, the second generation animals were exposed to saccharin and its impurities during gestation, throughout lactation via the dam's milk, and for the remainder of their lifetime via the diet. In this study, the incidence of tumours was apparently increased by the consumption of the 5% saccharin diet. In particular, squamous cell carcinomas of the uterus and transitional cell carcinomas of the urinary bladder were seen exclusively in the treated groups and occurred with greater frequency at the higher dosage levels.

Since this experiment was the first chronic study undertaken using the two-generation model, it seemed natural that the results would be critically evaluated and the subject of confirmatory studies. Thus, the United States Food & Drug Administration carried out a similar study with Sprague-Dawley rats being fed diets containing 0.0, 0.01, 0.1, 1.0 and 5.0 and 7.5% sodium saccharin. The pathological findings included 9 urinary bladder neoplasms (7 males and 2 females) in the high dose groups fed 7% saccharin, as well as one such lesion in each of the 0 and 5% dose groups.

The high dosages of saccharin required to elicit bladder tumours in the two-generation studies, as well as the apparent predilection for the second generation males, resulted in more questions than answers as to the carcinogenic potential of saccharin. These findings suggested that an impurity in saccharin might be responsible for the observed tumourigenic effects. Studies conducted in the Health Protection Branch (HPB) laboratories indicated that o-toluenesulphonamide (commonly called o-TS) was the major impurity in all of the saccharin samples used in the various long-term feeding studies. These and other observations led to the hypothesis that o-TS, a known inhibitor of carbonic anhydrase, the enzyme involved in the acidification of urine, would increase the excretion of bicarbonate. This action could produce an alkaline urine, predisposing the animals to urolithiasis in the kidneys and bladder. Irritation from these stones over a long period of time could produce hyperplasia and ultimately tumours.

HPB scientists proposed that an additional two-generation chronic feeding study be undertaken to ascertain whether o-TS did in fact have tumourigenic potential. The initial protocol for this study called for groups of rats to be fed either a standard control diet or one containing graded levels of o-TS. In addition, ammonium chloride was to be added to the drinking water of one of the highest o-TS dosed groups to prevent the formation of alkaline urine. Comments on this experimental protocol were solicited from other regulatory

agencies, a number of internationally recognized toxicologists, and from several industrial organizations.

Just prior to the start of this study, a quantity of saccharin produced by the Maumee procedure became available. This method of saccharin production results in no detectable levels of o-TS and the total amount of impurities is less than with the Remsen-Fahlberg procedures. This saccharin was to be fed to an additional group of rats at a level of 5% in the diet, a level comparable to the highest dose used in the previous two-generation studies.

The results of this experiment included a significant increase in bladder tumours in both first and second generation male rats fed the diet containing 5% sodium saccharin as compared to their respective control groups (Arnold et al., 1980). The incidence of bladder tumours in all of the other treatment groups was not significantly different from that of the respective control groups.

Since saccharin had never been fed to rats at dietary levels between 1 and 5% in a chronic feeding study prior to 1977, there was some interest in whether it might produce bladder tumours at dietary levels below 5%. The only chronic feeding study conducted since that time was financed by an industrial organization, the Calorie Control Council (CCC), and conducted under contract by the International Research and Development Corporation (IRDC) in Michigan. This study

used an unbalanced design in which increasing numbers of animals were assigned to the lower dose groups, thereby making it more sensitive for risk assessment purposes at the lower dose levels (Shubik, 1985). In addition, it contained one group of animals exposed to saccharin from birth onwards only, thereby allowing for an assessment of in utero exposure regarding bladder tumour induction.

In this study, there was a statistically significant increase in the incidence of bladder tumours at the 3% dose level. In addition there was a numerical increase in the number of bladder tumours at the 1% dose level compared to controls which, while not statistically significant, may be of some biological significance. It was also conclusively demonstrated that in utero exposure to saccharin was not necessary to elicit bladder tumours in the rat. Previously, the only convincing data demonstrating that saccharin was responsible for bladder tumour induction were studies in which the two-generation or in utero exposure model had been employed. Unfortunately, the results of the study did not provide any appreciable insight into the mechanism by which saccharin produced the bladder tumours.

Studies which have attempted to elucidate the mechanism by which saccharin produces bladder tumours generally revolve around animal models wherein the bladder is sensitized by an initiating dose of a known bladder carcinogen followed by exposure to saccharin in the drinking water (Hicks and Chowaniec, 1977) or feed (Cohen et al., 1979;

Nakanishi et al., 1980). Conceptually, this model can be considered to occur in two steps (Hicks, 1984). The first step is to administer a chemical which is a known bladder carcinogen via the diet or drinking water for a few days or weeks. The dose chosen is one at which, if the treatment was discontinued, the incidence of bladder tumors will be extremely low when the animals reach two years of age. This first step is termed initiation. If another chemical is now added to the diet after the initiator is no longer administered, and an appreciable number of bladder tumours subsequently occur, this latter chemical would be termed a promoter in that it was responsible for the development of a bladder tumour in initiated cells.

Recent studies by Cohen and colleagues (Cohen et al., 1982; Murasaki and Cohen, 1983ab) have demonstrated that sodium saccharin will increase the incidence of bladder tumours when administered to rats following an initiating dose of the chemical FANFT ((N-[4-(5-nitro-2-furyl)-2-thiazolyl]) formamide) or following a traumatic physical event such as freeze ulceration. Both initiating events result in regenerated hyperplasia of the bladder ureothelium. Unlike models wherein chemicals are used to sensitize the bladder, Hasegawa et al., (1984) have also shown that the carcinogenic process resulting from urinary bladder freeze ulceration followed by the feeding of saccharin appears to proceed without any mutagenic agent being detected in the urine. In addition, Hasegawa et al., (1985) have recently reported promotion of the freeze ulcerated bladder tissue by saccharin

even when the induced regenerated hyperplasia appeared to be morphologically normal.

Clayson (1984) has reviewed the data concerning the toxicological effects of saccharin in rodent studies and has suggested that the induction of bladder cancer in male rats as a result of adding saccharin to their diet is related to the integrity of the urothelial barrier. If this remains intact, then saccharin would not attain a sufficient concentration in the urothelium to exert its carcinogenic potential. He suggests that the carcinogenic effects may be mediated by saccharin's ability to inhibit enzymic processes.

Both the commercial grade and highly purified saccharin have been tested in a number of in vivo and in vitro test systems to ascertain its mutagenic potential, with equivocal results. It has been suggested that these variable observations may be attributed either to impurities or weak mutagenic activity (Arnold et al., 1983). The interpretation of these results, and the corresponding implications for human health is thus not readily apparent at this time.

11.3.2 Epidemiological Studies

Since the finding of carcinogenicity in animal experiments, saccharin has been subjected of extensive epidemiological investigation (see Armstrong, 1985, for a detailed review). Such studies have

involved time trend analysis of bladder cancer mortality rates in relation to per capita consumption of saccharin; comparison of cancer mortality rates in diabetics with those in the general population; and case-control studies in which past consumption of artificial sweeteners by bladder cancer patients is compared with that in disease free control subjects.

Although studies of average trends in bladder cancer mortality have been negative, such studies are of limited value due to the small proportion of the population at large exposed to large quantities of artificial sweeteners, and the uncertain influence of other known risk factors for bladder carcinogenesis such as smoking and occupation. Changes in diagnostic criteria and methods of treatment may also affect the interpretation of data collected over a period of time.

Because of their greater consumption of artificial sweeteners, diabetic populations have been selected for study in several investigations. Although clearly negative, these studies did not monitor artificial sweetener consumption on an individual basis, and could not control for the possible effects of smoking or other known factors for bladder cancer.

In addition to these investigations, the review and evaluation of the epidemiological data conducted by Armstrong (1985) considered thirteen case-control studies conducted between 1974 and 1982 relating

individual dosages of artificial sweeteners to the risk of bladder cancer. Since that time, one further study has been conducted (Mommisen et al., 1983). An additional case-control study involving cancers of all types was reported by Morrison (1979), including 13 cases of bladder cancer.

Most of these studies were unable to distinguish between use of saccharin and cyclamate. Since information on exposure to artificial sweeteners was obtained either via a mail questionnaire or personal interview in all of these studies, the data on exposure may be subject to recall or interviewer bias. Another potential source of error is nonresponse, which was as high as 50% in some studies. Selection bias may also occur in those studies using controls comprised of hospital patients if their disease states were in any way related to the use of artificial sweeteners (Silverman et al., 1983).

Taken as a whole, this extensive series of case-control studies provides little evidence for the carcinogenic effects of artificial sweeteners on the human urinary bladder (Cordle & Miller, 1984). Nonetheless, Howe et al. (1977) reported an excess risk in males using artificial sweeteners, although other suggestions of a positive association have been limited to population subgroups. Hoover & Strasser (1980) noted elevated risks in nonsmoking white females otherwise at low risk for bladder cancer and male heavy smokers with a high baseline risk. While the same findings for the former

subpopulation were also reported by Mommsen et al. (1983), other studies have implicated different subgroups. For example, Cartwright et al. (1981) reported an increased risk in nonsmoking males, but tended to discount this finding on the basis of the inconsistency between males and females and between smokers and nonsmokers.

11.3.3 Human Exposure

The first significant commercial use of saccharin in North America involved its addition to canned vegetable products including infant foods, as a sweetening and bacteriostatic agent (Ross, 1915). Because saccharin does not result in calorie production, it has been prescribed for diabetics (Lepowski, 1977) and used for the treatment of obesity in Great Britain (Ross, 1915). However, its use as a food additive was somewhat limited due to the bitter aftertaste that follows the ingestion of larger quantities.

In 1944, cyclamate was discovered and subsequently introduced into the marketplace as a second artificial sweetener. Soon it was found that when cyclamate was added to saccharin, the unpleasant aftertaste of saccharin was eliminated. This cyclamate-saccharin mixture was known commercially as Sucaryl (Munro et al., 1974).

Artificial sweeteners are currently used in a variety of food products (Table 11.1) and as a table top sweetener (Arnold et al.

1983). Additionally, saccharin has been tested as an additive to livestock rations for the purpose of improving body weight gain.

Table 11.1 Uses of Artificial Sweeteners

1. Beverages: soft drinks, powdered juices and drinks, wine.
2. Sweets/Desserts: ice cream, popsicles, canned fruit, dessert toppings, cookies, candies, pudding.
3. Condiments: sauces, dressings.
4. Miscellaneous: chewing gums, pickled vegetables, jams, jellies, flavour enhancer.
5. Table-top Sweeteners: tablets, solutions
6. Foods: canned vegetables, ketchup, mint sauce, sweet ham, bacon.
7. Pharmaceutical Products: oral liquid pediatric preparations, cough syrups, chewable tablets.
8. Cosmetics: toothpaste, dentifrice powders, lipstick, mouthwash.
9. Tobacco Products: chewing tobacco, snuff, wrapper paper, mouth-pieces.

The consumption of saccharin has increased dramatically in many countries since 1950, but probably more so in North America than in Europe. For example, the consumption of saccharin in the United States in 1953 was 21,000 lb. whereas in 1962, 2.5 million lb. were consumed. Following the ban on cyclamate in 1969, consumption rose to 4.0 million lb. in 1970. Of the saccharin consumed by North Americans, approximately 70% is ingested in diet soft drinks, 13% in various dietetic foods, 12% as a table top sweetener, and the remaining 5% used

in miscellaneous cosmetic, tobacco and medicinal preparations. These consumption patterns do not appear to have changed appreciably in the ensuing years prior to March, 1977.

The best data on recent consumption patterns come from a 1972 survey of beverage consumption conducted in the U.S. and Canada (National Research Council, 1978). This report indicated that 49.7% of the population drank carbonated beverages, of which only 12% used low-calorie beverages. It may thus be presumed that approximately 6% of the population ingested approximately 70% of all the saccharin consumed as a food additive. Barkin et al. (1977) further reported that 3 of their patients had consumed 8-12 bottles of diet soda pop, 1-2 cans of diet fruit, and upwards of 20 tablets containing cyclamate on a daily basis for up to 6 years. When evaluating any potential risk associated with the ingestion of saccharin, it is necessary to examine the consumption patterns for particular segments of the population rather than gauging exposure simply in terms of per capita consumption.

The best available data on artificial sweetener consumption by Western Europeans indicate that on a per capita basis, they consume less artificial sweeteners than do North Americans (Armstrong & Doll, 1974). However, saccharin was extensively used by Europeans during the First and Second World Wars when sugar was unobtainable and the laws

regarding the sale and use of saccharin were generally more liberal than in North America.

11.4 REGULATORY STATUS

11.4.1 The Decision to Take Regulatory Action

When the results of the Canadian two-generation study became available in the mid-seventies, officials of the U.S. Food & Drug Administration met with their Canadian counterparts and concurred that sodium saccharin itself rather than any impurity was most likely to be responsible for the induction of bladder tumour in rats (U.S. Food & Drug Administration, 1977).

Direct epidemiological evidence was looked for and the results from a then unpublished study in progress at the Canadian National Cancer Institute were drawn on. Although the study was comparatively small-scale, it was consistent with saccharin being a bladder carcinogen in man with the relative risk of exposed to unexposed males estimated to be about 1.6 (Howe et al., 1977).

It was well recognized that saccharin was used commercially not only as a substitute for sugar, but also to replace sugar in certain foods, such as carbonated beverages consumed by diabetics and other people who wanted a sweet low-calorie drink. People bought saccharin

or saccharin-cyclamate tablets and used them in the preparation of food, notably to sweeten hot beverages. In these circumstances a more overt decision to purchase and consume saccharin was made. Further uses of saccharin in drugs and cosmetics varied from seemingly trivial benefits to masking drug tastes that could interfere with maintenance of therapy for geriatric patients or the consumption of vitamins and mineral supplements by infants.

The question as to whether or not saccharin was of unique or even significant benefit in the treatment of obesity, the maintenance of ideal weight, or the maintenance of the health or quality of lifestyle for the diabetic, was considered. The scientific literature available on this subject prior to March 9, 1977, however, did not support the existence of these purported benefits to any appreciable degree (Farkas & Forbes, 1965; see also Office of Technology Assessment, 1977; National Academy of Sciences, 1978; Rosenman, 1978).

Discussion with representatives of the Canadian Medical Association, the Canadian Dental Association, the Canadian Diabetic Association and the Registrars of Pharmacy were held to discuss the above information, mutual concerns and the options available. The value of saccharin to diabetics versus its potential health risks was given particular consideration.

In addition to an evaluation encompassing all of the available toxicological and epidemiological data, many other factors were given due consideration. These included the regulatory status of saccharin under the Food and Drugs Act and Regulations, the health status of individuals consuming various saccharin-sweetened products, the potential health benefits derived from consumption of these products, the tendency of consumers to continue consumption of products they habitually consume (such as tobacco) despite the potential health hazards, the economic impact of changes in the Food and Drug Regulations, the use of warning labels designed to promote informed choice by consumers, and the potential availability of less hazardous substitutes or alternatives (Munro & Krewski, 1981). It was recognized that in the long run, saccharin might be replaced by other nutritive sweeteners currently being developed and undergoing toxicological testing (Health and Welfare Canada, 1981a). It was also recogized that any changes in the regulatory status of a product should allow for disposal of existing stocks provided that the potential hazard was not judged to be unduly great.

While there was little doubt that saccharin per se was the agent responsible for the bladder tumours observed in all three of the two-generation animal feeding studies conducted in the seventies, and that some form of regulatory action was therefore necessary, it was also recognized that further research was required in several areas. One of the primary needs was further epidemiological research to

supplement the preliminary findings by Howe et al. (1977) that bladder tumours in man could be associated with the consumption of saccharin. Other areas for study concerned the efficacy and potential health benefits of saccharin use (Office of Technology Assessment, 1977) as well as studies to ascertain the mechanism by which it elicits its tumourigenic response in rats (Arnold et al., 1980).

The Minister of National Health and Welfare announced his decision regarding saccharin on March 9, 1977, wherein he scheduled a phasing-out of saccharin as a food additive and restricted sale as a table-top sweetener to pharmacies. The sale of saccharin in soft drinks, beverage mixes and beverage bases would be phased out by July 1, 1977 and in other foods by November 1, 1977. The sale of saccharin alone or in combination with cyclamate would be restricted to pharmacies as of September 1, 1977 (Health and Welfare Canada, 1977b). He later agreed to a request from Canadian food manufacturers for a short-term extension of these deadlines to October 1, 1977, December 1, 1977 and February 1, 1978, respectively (Health and Welfare Canada 1977c). Even with the extensions allowed, not all products would be sold within the time limit, and small specialized companies suffered significant losses.

The decision that the sale of saccharin for use as a table-top sweetener be restricted to pharmacies was in part attributable to the benefits of saccharin perceived by certain segments of the population,

primarily diabetics. Such an outlet restricted general availability while leaving an avenue open for those who perceived a benefit from saccharin and would result in such consumers making their own benefit-risk evaluations when purchasing such products.

Concurrently, on March 9, 1977, the U.S. Food and Drug Administration also announced its intention "to ban saccharin because the artificial sweetener may cause cancer in humans" (Consumer Reports 1977). The ensuing public and congressional debate (Consumer Reports 1977; Food Chemical News, 1977a) ultimately resulted in a congressionally mandated 18 months moratorium on FDA's proposed ban of the artificial sweetener (Food Chemical News, 1977b; U.S. Food & Drug Administration, 1977). This moratorium has been periodically extended through to the present day.

11.4.2 The Saccharin Controversy

The initial public response in Canada was largely favourable towards the saccharin decision, although in the United States, the response was more emotional (Consumer Reports, 1977). In an attempt to minimize the anticipated fears of the saccharin-consuming public, especially pregnant women, regulatory officials attempted to place the risk of saccharin consumption in a context which may have down-played such concerns. This course of action resulted in charges from industry that the regulatory decision was made hastily. On the other hand,

various consumer groups in the United States charged that the government had waited too long and the results of the HPB study only confirmed results found almost five years previously (Rhein & Marion, 1977).

In addition, a substantial number of arguments regarding risk-benefit considerations were put to the Ministers in Parliament and became a matter of public record (Hansard, 1977). Press comment at the time very largely took the form of straightforward reporting of Parliamentary exchanges. Later lobbying and publicity in the United States designed to stay the FDA's proposed controls on saccharin was headed by the diet food industry supported by the U.S. Calorie Control Council (Consumer Reports, 1977; Rhein & Marion, 1977). It provided Canadian readers with media exposes that sometimes ridiculed the Canadian studies as irrelevant or unscientific, and indicted control measures as bureaucracy run wild (Food Chemical News, 1977a). One U.S. Senator even criticized the HPB studies because of the use of "Canadian" rats (Rhein & Marion, 1977) whereas in point of fact, the animals used were actually of United States origin (Arnold et al. 1980)!

11.4.3 Recent Developments

In November of 1977, the U.S. Congress passed the Saccharin Study and Labeling Act. This Act requested the U.S. National Academy of Sciences (NAS) to examine the risks and benefits accruing from the use of saccharin, and to consider other issues surrounding federal food safety policy in general.

The committee convened by the NAS accepted the validity of the animal tests regarding saccharin. Saccharin not only acts by itself toproduce bladder tumours, but saccharin promotes the cancer-causing effects of some other carcinogenic compounds in rats. Whether as an initiator or promoter, saccharin was viewed by the committee as a potential cause of cancer in humans.

The committee also evaluated various procedures currently available for extrapolating results from animal test to human populations and concluded that the art of extrapolation does not permit confident estimates of saccharin's potential to cause cancer in humans. Although saccharin would be expected to be of low potency in humans based on the animal studies, the committee pointed out that even low risks applied to a large number of exposed persons may lead to public health concerns. The committee also concluded that saccharin itself, and not its impurities, is most likely responsible for saccharin's carcinogenic properties.

The committee was unable to find any studies that permitted an objective assessment of the alleged health benefits of saccharin use by the general public. The committee did recognize that there is a perceived need or psychological reliance on nonnutritive sweeteners by certain segments of the population. However, at the time of their deliberations, the committee was unable to draw conclusions about the implications of the perceived need. The committee also accepted the

premise that the use of nonnutritive sweeteners in drugs and dentifices presents insignificant risks and involves possible benefits.

The committee concluded that the epidemiology studies available did not provide clear evidence to support or refute an association between past saccharin use and bladder cancer in males. All but two of the studies either suffered from methological difficulties or were too insensitive for reliable measurement of potentially low-level effects from saccharin usage. The committee noted that when health effects in the human population are weak, such effects are difficult to distinguish from either random variation or from the influence of more potent carcinogens, particularly in the case of bladder cancer. (Bladder cancer is known to be associated with cigarette smoking and occupational exposure to various chemicals, each of which appear to be more potent carcinogens than saccharin).

The International Agency for Research on Cancer (IARC) also conducted a detailed review of the carcinogenic risks posed by saccharin. IARC was established in 1965 by the World Health Assembly as an independently financed organization within the framework of the World Health Organization. The Agency conducts a program of research concentrating particularly on the study of potential carcinogens in the human environment and prepares monographs on the evaluation of carcinogenic risks to humans posed by industrial chemicals (IARC, 1982a). In a 1980 publication, IARC concluded that:

> "there is sufficient evidence that saccharin alone, given at high doses, produces tumours of the urinary tract in male rats and can promote the action of known carcinogens in the bladder of rats of both sexes; and there is limited evidence that ortho-toluenesulphonamide is carcinogenic when given orally to rats; but the available data suggest that impurities at the levels normally found in commercial saccharin do not contribute to the carcinogenicity of saccharin.
>
> Although a small increase in the risk of urinary bladder cancer in the general population or a larger increase in some individuals consuming very high doses of saccharin and cyclamate cannot be excluded, the epidemiological data provide no clear evidence that saccharin alone, or in combination with cyclamates, causes urinary bladder cancer. There are no epidemiological studies on a possible association between use of saccharin and cyclamates and cancer at other sites in humans."

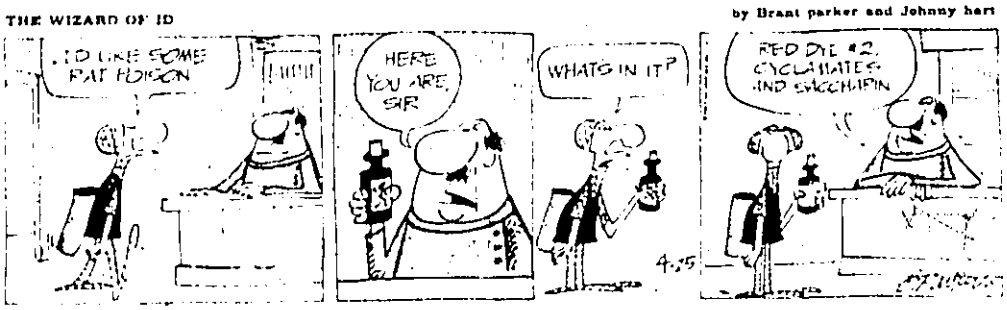
Reconciliation of the toxicological and epidemiological evidence on the carcinogenic potential of saccharin was subsequently attempted at a symposium sponsored by the International Agency for Research on Cancer (Wald & Doll, 1985). The consensus of opinion among the participants was that the epidemiological data was on balance of greater relevance to the human situation. However, although it was concluded that "saccharin, used either alone or in combination with cyclamates, is not a demonstrable cause of bladder cancer in the human population at large under past conditions of use", it was noted that the possibility of a weak effect could not be completely excluded as in the previous IARC review in 1982 (Krewski, 1985).

Although considerable data on the potential risks posed by saccharin has become available since 1977, this has served primarily to support the regulatory decision taken at that time. In particular, the

Calorie Control Council sponsored study has served to support the evidence regarding the induction of urinary bladder tumours in the rat, while ruling out the necessity of *in utero* exposure in order for this to occur. Similarly, although largely negative, the epidemiological studies have been unable to rule out the possibility of carcinogenic effects in man. As a result, the regulatory status of saccharin in Canada remains unchanged.

11.5 ANALYSIS AND EVALUATION

Since its synthesis in 1879, saccharin has been the subject of numerous toxicological investigations and reviews by public health authorities. The findings of the experimental studies conducted with saccharin in the seventies have led to recommendations from several national health authorities for curtailment of the use and availability of saccharin. These recommendations and their application through the force of law have been the subject of considerable controversy. This grew rather than diminished as successive studies appeared in the literature, diabetes and obesity control, consumer protection, law, public administration and politics responded in print and the electronic media. Their response often arose from positions based on science, philosophy, personal judgement, public interest, sectional interest and self-interest. Saccharin made its way from the small print on the labels of food products to leading articles and even to that touchstone of society, the funny pages.



Within the context of the ICTC model for risk assessment and risk management as described in chapter 2 (Figure 2.2), the potential health hazards posed by saccharin were first identified using toxicological investigation. Of particular concern, and the basis of the current status of saccharin in Canada, was the observation of tumours of the urinary bladder in second generation male rats whose parents had also been exposed to saccharin. Because most known human carcinogens have been shown to be active in one or more animal species, saccharin was thus considered to pose a potential hazard to human health. Although subsequent epidemiological studies were unable to provide direct evidence that saccharin is a significant factor in the production of bladder cancer in man, these studies are also unable to exclude the possibility of some small increase in risk in the human population and are therefore not necessarily inconsistent with the animal data.

Risk estimation based on the extrapolation of animal data to man is always difficult, even with the traditional one-generation chronic toxicity/carcinogenicity bioassay. For the two-generation bioassay, the problem of inter-species conversion takes on new dimensions due to differences in the physiological characteristics of the yolk sac and the fact that the fetus may or may not be exposed to metabolites which the human fetus can either further metabolize or eliminate.

The options available for the control of food additives such as saccharin with demonstrated carcinogenic potential are limited. In the United States, the Delaney Amendment proscribes by law that no substance known to cause cancer in animal or man may be permitted for use as a direct food additive. Although the regulatory environment in Canada is less rigid, there has been a clear tendency to adhere to this policy in practice. As such, Canada took action to ban the use of saccharin as a direct food additive under the Food and Drugs Act.

Perceived benefits that have been associated with the use of saccharin include the prevention of diabetes, obesity, dental caries, hyperlipidemia and hypoglycemia, and its role in drug formation. These perceived benefits were evaluated vis a vis the potential health risks of saccharin consumption, and resulted in the availability of saccharin in pharmacies for those who felt a need for its use.

The saccharin decision was based on the latest scientific information available at the time. The initial public reaction in Canada was largely in favour of this decision, although greater public opposition orchestrated by certain special interest groups was encountered in the United States.

While the curtailment of the uses of saccharin is still controversial, it is not considered to be an inappropriate response to the problem at hand. In retrospect, it seems that by today's standards the decision was not accompanied by a sufficiently comprehensive attempt to communicate its scientific basis and rationale to the public. The use of detailed Information Letters such as those issued in the case of the food additive aspartame and the packaging contaminant acrylonitrile, may provide a vehicle for greater public awareness into the safety evaluation process and regulatory decision-making.

This case study clearly shows that the regulation of food additives can be a highly complex issue, involving some of the most fundamental and challenging problems in human risk assessment and social decision-making. Briefly, these include the appropriate determination of toxic effects in animals, the extrapolation from these effects to what might be seen in man (and the lower intakes), the evaluation of scanty and sometimes inconsistent epidemiological evidence, the assessment of health and other benefits, consideration of

the concerns of special groups such as diabetics and the impact on industry and trade, conformity with legal constraints, and the timing of the decision and its implementation. The saccharin example also illustrates the feasibility of dealing with such problems through comprehensive regulatory action which takes cognizance of the balance between risks and benefits as well as the degree of informed choice that consumers are able to exercise.

11.6 SUMMARY AND CONCLUSIONS

The present review of the history of saccharin has shown that it has been alternately endorsed or banned by various regulatory agencies during the last one hundred years and that after a century of use, scientific studies are still clarifying the nature of the toxicological effects of saccharin. At this time, saccharin is available in pharmacies in Canada but it is not permitted in foods. In other countries saccharin is usually subject to varying degrees of control, often severely limiting the use or requiring warning labels. Saccharin is an example of a chemical whose use has been managed so that in these countries, any risk to the consumer is through individual choice rather than inadvertent exposure.

Regulatory agencies are often confronted with problems for which there is either a lack of data or less than the desired amount of information, as is clearly the case regarding any risk-benefit

assessment pertaining to saccharin. The present unknowns apply to toxicological and epidemiological studies, both of which have large gaps in the scientific database against which the safety of saccharin must be assessed. These unknowns have been a large part of saccharin's past and will likely remain so during the foreseable future.

While it is not yet possible to quantify the potential risk to human health from ingesting saccharin with any degree of certainty, the data available today permits a somewhat better estimate than in 1977. Thus, the decision to ban saccharin in 1977 has not been contradicted by the data which has become available in the intervening years. Nevertheless, the availability of saccharin in pharmacies continues to allow individuals the freedom of choice to evaluate the various alternatives on a personal basis, which is in keeping with the attitudes prevalent in many segments of our current day society. Conversely, the benefits attributed to saccharin remain largely unsubstantiated. Under these circumstances, and the fact that a perceived majority of consumers desire an "absolutely safe" food supply (Williamson, 1981), regulatory agencies in North America are often viewed as being somewhat cautious in their regulatory actions. However, until more definite data on the potential human health risks of saccharin are developed, this prudent course of action appears warranted when viewed from the perspective of public health protection.

12. CASE STUDY NO. 3: FORMALDEHYDE

12.1 INTRODUCTION

Formaldehyde is ubiquitous in the human environment, occurring in both indoor and outdoor air as a result of release from industrial processes, and formaldehyde-containing materials such as urea-formaldehyde foam insulation (UFFI). It is also emitted as a by-product during combustion, is formed naturally in the atmosphere and is present at low levels as a normal body constitutent.

The fact that formaldehyde causes sensory irritation in humans has long been recognized. More recently, formaldehyde has been found to be carcinogenic in certain animal species, thereby prompting concern as to its potential carcinogenicity in humans. Although several epidemiological studies have been conducted to investigate this possibility, the available results are not definitive. The resulting uncertainty regarding the possibility of carcinogenic effects in humans has had considerable influence on the manner in which formaldehyde and related products have been regulated in Canada.

In this chapter, the available data on the health risks associated with exposure to formaldehyde are examined. This involves consideration of general characteristics (section 12.2); data on human exposure (section 12.3); toxicological and epidemiological studies of

adverse health effects (section 12.4); and regulatory status (section 12.5). In view of the uncertainty surrounding the potential human health risks posed by exposure to formaldehyde, we also consider areas in which further information may serve to facilitate our understanding thereby providing a stronger base upon which regulations may be based in section 12.6. A brief summary of this case is presented in section 12.7.

12.2 PROPERTIES, SOURCES AND USES

Formaldehyde, the simplest and most common aliphatic aldehyde in the environment, is a colorless gas with a pungent suffocating odor. It is formed at low levels in ambient air as an intermediate in the oxidation of methane with higher concentrations occurring in polluted atmospheres due to the presence of increased levels of methane and oxygen precursors (Squire & Cameron, 1984). Formaldehyde is also emitted during the incomplete combustion of wood, coal, hydrocarbon fuels, and tobacco.

Formaldehyde is generated industrially by superheating air and methanol mixtures, using either silver crystal or mixed metal oxide catalysts. It is released into the atmosphere during the manufacture and use of both the gas itself and formaldehyde-containing products. Formaldehyde was first produced commercially in 1901 in the United States for use primarily as an embalming agent and disinfectant

(Morris et al., 1975). Most of the formaldehyde generated in recent years has been used in the production of urea-, phenol-, actyl-, and melamine-resins.

Urea-formaldehyde is primarily used for the manufacture of particle board, medium density fibre board, and hardwood plywood (IARC, 1982b). It is also used in the production of foam insulation, moulding compounds and foundry resins as well as in the treating and coating of paper, textiles and surfaces. Phenolic resins are used as adhesives in water resistant plywood and as binders in fibrous glass insulation. Polyacetal resins are used as unreinforced thermoplastic resins, while thermosetting melamine resins are used in surface coatings, moulding compounds, laminates, dinnerware, and the treating and coating of paper and textiles (Meek et al., 1985).

Formaldehyde is also used in the production of chemicals, most notably 1,4- butanediol, and to a lesser extent in the production of elastomers, explosives, plastics, fibers, fertilizers, textiles, cosmetics, and dyes (Wong, 1983).

12.3 EXPOSURE

Formaldehyde levels in remote outdoor areas are generally in the 2 ppb range, the value predicted for unpolluted air (Meek et al., 1985). Concentrations in populated areas consistently fall between 2-12 ppb

(Cleveland et al., 1977; Guicherit & Schulting, 1985; Tuazon et al., 1978; Desuiel, 1985; Bortoli et al., 1985).

Formaldehyde concentrations in indoor air are influenced by the type, quality and age of building materials and furniture present, the level of occupancy and human activity, and environmental factors such as temperature, humidity, and ventilation rate. The use of gas stoves, fireplaces, wood stoves, and kerosene heaters, as well as indoor tobacco smoking, also affect formaldehyde levels (Meek et al., 1985).

Formaldehyde concentrations in indoor air are generally less than 0.05 ppm in conventional homes, although higher concentrations have been found in some homes containing particleboard or urea-formaldehyde foam insulation (UFFI)(Health & Welfare Canada, 1986). However, the higher levels sometimes found in newer homes resulting from release mainly from particleboard tend to decrease with time. Formaldehyde levels in mobile homes tend to be somewhat higher than in conventional homes, with levels ranging up to 1 ppm. This is believed to be a result of the higher proportion of particleboard and plywood used in mobile home construction and due to lower ventilation rates.

12.4 HEALTH EFFECTS

12.4.1 Toxicological Studies

Acute Toxicity. Acute exposure of laboratory animals to airborne concentrations of formaldehyde greater than 100 ppm produces salivation, acute dypsnoea, vomiting, cramps, and death (IARC, 1982b). At concentrations of 10-50 ppm, decreases in respiratory rates, adverse effects on pulmonary function (Chang et al., 1981; Barrow & Steinhagen, 1981; Amdur, 1960; Kane & Alarie, 1977; Davis et al., 1967) and eye irritation (Salem & Collumbine, 1960) have been observed. Acute exposure to concentrations of less than 1 ppm also result in decreased respiratory rates in both rats and mice (Barrow & Steinhagen, 1981) and adverse effects on pulmonary function in guinea pigs (Amdur, 1960).

The results of short term studies reveal species differences in the adaptive response of rodents to formaldehyde irritancy, with mice being more responsive than rats (Chang et al., 1981). The dose response relationship for sensory irritation in mice appears to be quite similar to that in humans (Gamble, 1983).

Subchronic and Chronic Toxicity. The effects of subchronic and chronic exposure to formaldehyde in animal species are confined mainly to lesions in the nasal cavity and decreases in body weight gain (Health & Welfare Canada, 1986). These effects have been observed in some species following exposures of 1-2 ppm for six months or more.

There are clear species differences in the development of nasal lesions, with rats and monkeys being most susceptible and hamsters being least susceptible. Mice are of intermediate susceptibility. These species differences correlate well with differences in adaptive responses (i.e., decreases in respiratory rates) observed in short term studies and with differences in formaldehyde deposition in the nasal cavity (Chang et al., 1983).

Formaldehyde has been carcinogenic in two strains of rats, producing a high incidence of nasal tumours (38-50%) in both sexes following long-term exposure to approximately 15 ppm (Kerns et al., 1983; Sellakumar et al., 1985; Tobe et al., 1986). In mice, nasal tumours have also been observed at 15 ppm (Kerns et al., 1983) although the increase was not statistically significant and was restricted to one sex. More limited studies have provided no indication of carcinogenic effects in hamsters (Dalbey, 1982). There is currently no evidence to indicate that formaldehyde increases the occurrence of tumours at sites other than the nasal cavity.

In rats, the dose response relationship for nasal tumours over the range 0-15 ppm is highly curvilinear, with a disproportionate increase in tumour incidence occurring at higher concentrations. There is some evidence that protective mechanisms such as mucociliary clearance and DNA repair are saturated at the concentration at which tumours develop. Inhibition of nasal mucociliary function (Morgan et al., 1983), extensive tissue damage (Swenberg et al., 1982), and an increase in cell proliferation have been observed in the nasal cavities of rats

exposed to this level for periods as short as six hours. Similar tissue injury has not been observed in the hamster at this level of exposure.

Genotoxicity. Formaldehyde has been shown to cause damage to genetic material in a number of assays including those in cultured mammalian cells, Drosophila, fungi, and bacteria (Auerbach et al., 1977; Consensus Workshop on Formaldehyde, 1984). It should be noted, however, that the genotoxic potential of formaldehyde has not been consistently demonstrated in related assays. For example, although formaldehyde leads to sister chromatid exchange in Chinese hamster ovary cells, mutation of these cells has not been observed (Boreiko & Ragan, 1985). Based on a recent review of the in vitro studies, it was concluded that formaldehyde may be considered a weak mutagen, inducing less than a 10-fold increase over background in those assays where it tested positive (Consensus Workshop on Formaldehyde, 1984).

The results of the in vivo studies conducted to date are mixed. While formaldehyde is mutagenic in Drosophila under certain conditions (Boreiko & Ragan, 1985), other tests have been either negative or marginally positive (Consensus Workshop on Formaldehyde, 1984). No increase in the frequency of either chromosome aberrations or sister chromatid exchanges were observed in bone marrow cells or spermatocytes in six pathology workers as compared to five unexposed controls (Thomson et al., 1984).

Metabolism and Pharmacokinetics. Formaldehyde is an important metabolic intermediate and a required precursor in the endogenous synthesis of formic acid; consequently, it is normally present in low concentrations in body fluids (Meek et al., 1985). It is rapidly metabolized to formic acid by enzymes in the liver, and to a lesser extent in the brain, kidney, muscles, and erythrocytes. Formic acid is then further oxidized to carbon dioxide and water. Carbon dioxide may be eliminated in urine as a sodium salt, or enter the single carbon metabolic pool, a biosynthetic pathway which leads to the formation of amino acids such as serine, choline, and glycine (Squire & Cameron, 1984).

In addition to being oxidized to formic acid, formaldehyde can form adducts with proteins, histones, and nucleic acids. Reaction with DNA has been found to result in irreversible denaturation of the DNA helix (Newell, 1983). If a formaldehyde molecule reacts with two paired strands of a DNA helix, the adduct forms a cross-link which could interfere with DNA replication, and lead to mutations.

12.4.2 Epidemiological Studies

Irritancy and Pulmonary Sensitization. Symptoms of irritation, particularly of the eyes, nose and throat, have been reported in a number of cross sectional studies of workers exposed to levels of formaldehyde ranging from less than 1 ppm to greater than 5 ppm (Meek

[illegible]

Cases of pulmonary sensitization involving severe and prolonged asthma attacks have been reported in several studies among workers exposed to very low concentrations of formaldehyde (Nagy et al., 1965). However, it is not possible on the basis of available data, to conclude that the association is causal.

Symptoms of irritation in individuals exposed to formaldehyde in public or residential buildings have been investigated in several studies (Goodbody & Zenz, 1981; Dally et al., 1981; Garry et al., 1980; Godish et al., 1979; Grayson, 1977). However, only limited conclusions can be drawn as many of these studies lacked suitable controls and in fact were simply surveys of residents who attributed their symptoms to formaldehyde exposure in their homes.

In other studies (Main & Hogan, 1981; Olsen & Dossing, 1982; Thun et al., 1981; Burdach & Kerkerbord, 1980) with proper controls, adverse effects were not observed in populations exposed to

concentrations less than approximately 0.5 ppm. However, the potential for subject bias (overreporting of symptoms based on knowledge of exposure) and lack of specificity of the acute effects of formaldehyde, complicate the interpretation of the results (Meek et al., 1985).

Mild symptoms of eye irritation at levels as low as 0.3 ppm were noted in early clinical studies (Rader, 1974; Schuck et al., 1966). In more recent and better controlled studies, significant differences between exposed and control groups have been detected in the frequency of symptoms of irritation only at levels of exposure approaching 1 ppm for periods up to 30 minutes in length (Bender et al., 1983; Weber-Tschopp et al., 1977).

Carcinogenicity. A series of historical cohort studies have been performed to examine the potential carcinogenic effects of formaldehyde in humans (Blair et al., 1987; Acheson et al., 1984; Harrington & Oakes, 1984; Levine et al., 1984; Wong, 1983; Harrington & Shannon, 1975). None of these studies revealed any excess of nasal or nasopharnygeal cancer associated with exposure to formaldehyde. However, given the rarity of such cancers, the power of these studies to detect small increases in risk was limited. Exposure levels were poorly determined and the effects of confounding factors could not easily be taken into consideration in the analyses. These studies also generally involved exposure periods shorter than desired for a disease such as cancer which can have a relatively long latent period.

In some of the cohort studies, carcinogenic effects were reported at sites distant from the nasal cavity. Harrington & Shannon (1975) noted excess lymphatic and hematopoietic lesions in British pathologists and Acheson et al. (1984) reported increased lung, rectal, bone, and thyroid tumours in British chemical workers. However, it is not clear that these tumours were attributable to formaldehyde exposure. For example, the incidence of lung tumours in chemical workers in the study by Acheson et al., (1984) was increased compared to the British population as a whole, but not when compared to local residents. Moreover, there was little evidence of an exposure response relationship and the number of deaths from cancer of the bone and thyroid was small.

Increases in nasal or nasopharnygeal cancers attributable to formaldehyde exposure have also not been found in the case-control studies performed to date (Hayes et al., 1984; Olsen et al., 1984; Brinton et al., 1983; Fayerweather et al., 1983; Hernberg et al., 1983; Anderson et al., 1982). While it was possible to adjust for exposure to confounders such as smoking and wood dust in some of these studies, confounding remained a serious problem in others. For example, in the case-control study of nasal cancer in Denmark, Finland and Sweden conducted by Hernberg et al., (1983), the association between formaldehyde exposure and nasal cancer was clearly confounded by wood dust exposure. Whereas 15 out of 18 of the formaldehyde-exposed cases

were also exposed to wood dust, none of the 6 formaldehyde-exposed controls had been exposed to wood dust.

An association between formaldehyde exposure and cancer incidence has been observed in several of the proportionate mortality studies conducted to date. Walrath & Fraumeni (1983) detected excess skin, kidney, and brain lesions in embalmers; Liebling et al., (1984) found excess colo-rectal as well as buccal and pharyngeal tumours in factory workers; Stayner et al., (1985) found excess cancers of the buccal cavity, biliary passage and liver as well as the lymphatic and hematopoietic system in embalmers. These results add little to the weight of the evidence concerning the carcinogenicity of formaldehyde however, because of the potential for bias in selection of the exposed population and the tendency to overestimate the mortality experience of occupational groups, such studies should be considered only as preliminary screening tools. In this case, these findings were not corroborated by the inherently more sensitive cohort or case-control studies discussed above.

Taken as a whole, epidemiological studies conducted to date provide little convincing evidence of the carcinogenicity of formaldehyde in human populations. Positive results have been reported in certain proportionate mortality studies; however, the number of excess deaths has been small, the pattern has varied from study to study, and the findings have not been reproduced in analytical studies.

Still, the possibility that formaldehyde is a human carcinogen cannot be excluded on the basis of the available data due to the limited sensitivity, lack of quantitative exposure data, insufficient exposure periods, and confounding in studies conducted to date.

12.4.3 Risk Estimation

Evidence for carcinogenicity in humans is most persuasive if derived from analytical epidemiological studies (Squire & Cameron, 1984). For formaldehyde, such evidence is lacking; any assessment of potential cancer risk must therefore be based on the analysis of animal bioassay data. Although there are data available for administered doses of formaldehyde ranging from 0.3 to 15 ppm there is still considerable uncertainty regarding the shape of the dose response curve at lower doses, largely because of the lack of understanding regarding the mechanisms of formaldehyde-induced cancer. Often because of this lack of understanding, some form of linear extrapolation is used to estimate risks at low doses (Krewski et al., 1986). Such an approach provides an upper bound for low dose risk estimates such that the true risk to humans exposed to formaldehyde by inhalation is not likely to be higher than this limit (Consensus Workshop on Formaldehyde, 1984).

It is also generally assumed at least implicitly that the administered dose (the external measure of exposure that is directly controlled in laboratory studies of toxicity) is proportional to the

delivered dose to the target tissue (in this case, the amount of formaldehyde covalently bound to respiratory mucosal DNA). The delivered dose is presumed to be the direct causative variable in mechanistic descriptions of the carcinogenesis process at the cellular and molecular levels (Starr & Buck, 1984).

Extrapolation of the risks associated with formaldehyde exposure from rodents to humans requires consideration of interspecies differences in formaldehyde metabolism, nasal anatomy and physiology, and adaptive response (Squire & Cameron, 1984; Starr et al., 1985). The nasal exposure of rodents to airborne substances such as formaldehyde is higher than that of humans, as the former are obligatory nose breathers. Rodents have internal nasal structures which act as baffles to deflect large volumes of air; humans do not. Rodents also have a much larger surface area for filtering air relative to their respiratory rate, which implies that if both species were exposed to similar concentrations of airborne material, rodents would receive a higher dose per unit area. It is possible however, that in humans, formaldehyde might induce cancers of the respiratory tract other than nasal and nasopharyngeal cancers, most notably those of the buccal cavity, larynx, trachea, esophagus, and bronchi (Acheson, et al., 1984), due to the fact that humans breathe partially through the mouth.

Information on the mechanisms of formaldehyde toxicity is also relevant to interpreting the risk estimates for formaldehyde. Such data include the effects of the sensory irritation response and the mucociliary clearance mechanism on delivery of formaldehyde to target tissues, the disposition of delivered formaldehyde in target cells via metabolism and macromolecular binding, and the cellular proliferative response to cytotoxicity (Starr & Buck, 1984). In an attempt to incorporate some of the mechanistic data, Starr and Buck have quantitatively estimated the cancer risks at low levels of formaldehyde exposure incorporating delivered dose (amount of formaldehyde covalently bound to respiratory mucosal DNA) as the measure of exposure. This approach takes into consideration the non-uniform relationship between administered and delivered dose observed in a recent study by Casanova-Schmitz et al. (1984).

The risk of cancer associated with exposure to low airborne levels of formaldehyde has been quantitatively estimated on the basis of the incidence of nasal squamous cell carcinomas in rats observed in the CIIT study. Using the multistage model, the estimated lower 95% confidence limits for virtually safe concentrations associated with a lifetime risk of 10^{-5} is 0.0064 ppm. Taking delivered dose as the measure of exposure (Starr and Buck, 1984), this value is increased to 0.016 ppm (Health and Welfare Canada, 1986).

12.5 REGULATORY STATUS

12.5.1 Occupational Exposure Limits

Over the years there has been a steady decrease in the levels for formaldehyde permitted in the workplace. In 1949, a Maximum Allowable Concentration of 10 - 20 ppm for an 8 hour exposure period was jointly recommended by the Department of National Health and Welfare and the Ontario Ministry of Health. In 1978, the American Conference of Governmental and Industrial Hygienists (ACGIH) established a Threshold Limit Value (TLV) of 2 ppm for an 8 hour exposure. This is a ceiling value and does not permit short term excursions at a higher level. This value has been adopted for federal workers under the Canada Labour Code and by most of the provinces. At the present time, however, the ACGIH Threshold Limit Value for formaldehyde is being reviewed (Corpus Information Services, 1982).

12.5.2 Non-occupational Exposure Limits

Guidelines for formaldehyde levels in indoor air have not been established in any province, with the exception of British Columbia which has introduced a value of 0.1 ppm. Such guidelines have, however, recently been established by the Federal-Provincial Advisory Committee on Environmental and Occupational Health. This Committee has recommended an action level of 0.10 ppm for formaldehyde in indoor air.

It was also recommended that every effort be made to reduce concentrations below a target value of 0.05 ppm. It is likely that these guidelines will prompt the introduction of ventilation requirements and product specifications to ensure that formaldehyde levels in indoor air are below the recommended values.

12.5.3 Urea Formaldehyde Foam Insulation (UFFI)

Increasing concern over possible health problems associated with residential uses of UFFI prompted formation of a Working Group on the Toxicology of Thermal Insulation, in 1978. In 1980, this Group recommended specific installation procedures for UFFI. They also recommended a guideline of 0.1 ppm for free formaldehyde in UFFI-containing homes based on the application of a 20-fold safety factor to the ACGIH TLV for occupational exposure. This limit was comparable to that established in Norway and the United States (Corpus Information Services, 1982).

In 1980, an Expert Advisory Committee was formed by Health and Welfare Canada, to assess potential risks to health to persons occupying buildings insulated with UFFI (Corpus Information Services, 1982). The committee recommended that a moratorium on the use of UFFI be imposed until it had completed its detailed investigation. In

response, the federal government announced a temporary ban on UFFI for residential homes, effective December 18, 1980.

In April of 1981, the Committee concluded that UFFI can be an unstable material of variable composition, being susceptible to breakdown and the release of formaldehyde gas. It was recommended that the ban remain in place until industry satisfactorily demonstrated that a safe, stable and defined product had been developed. Government and industry were also to develop standards to ensure that the recommended level of formaldehyde (0.1 ppm) would not be exceeded by installation of UFFI (Corpus Information Services, 1982).

On June 11 of that year, the Department of Consumer and Corporate Affairs (CCA), initiated a UFFI program which included a national indoor formaldehyde testing survey, a Hazardous Products Board of Review to hear representations from interested parties, and a toll free Across Canada Telephone System for computerized recording of inquiries and complaints (Corpus Information Services, 1982).

On December 14, 1981, the government released "The Report on the National Testing Survey to the Board of Review", which revealed that a small fraction of homes not containing UFFI had average indoor formaldehyde levels greater than 0.1 ppm, as did a large fraction of UFFI-containing homes in which health problems were reported (Corpus Information Services, 1982).

The following week, CCA announced details of a $110 million assistance plan for owners of UFFI homes. The plan included free screening of formaldehyde levels in homes, with a more comprehensive examination if levels were greater than 0.1 ppm, advice to homeowners on remedial work, along with up to $5,000 in grants to cover costs, and financial assistance and technical advice to persons in homes having levels less than 0.1 ppm if their health was affected by the formaldehyde gas (Corpus Information Services, 1982).

During its final meeting in January of 1982, the Board of Review concluded that the only effective solution to the problems associated with UFFI used, was to maintain the ban (Corpus Information Services, 1982). The ban remains in effect to date.

12.6 ANALYSIS AND EVALUATION

The fact that formaldehyde acts as a sensory irritant in humans has long been well documented in workers exposed to levels above 1 ppm. This epidemiological data on sensory health hazards is supported by toxicological studies in animals. More recently, long-term exposure to formaldehyde in rats has been shown to induce malignant tumours of the nasal cavity. These latter effects have not yet been observed in humans, although the epidemiological studies conducted to date are insufficient to preclude this possibility.

Based on the noncarcinogenic effects of formaldehyde, a TLV of 2 ppm over 8 hours has been established for occupational exposure. Based on estimates of carcinogenic risks obtained from the toxicological studies, a lower level of 0.1 ppm has been established for indoor air. These latter effects have also been responsible for the banning of UFFI, an action which is currently being challenged in the courts.

This action indicates that several strategies have been used jointly in order to control the health risks posed by formaldehyde. Recognizing that formaldehyde cannot be completely eliminated from ambient air, this has included the establishment of guidelines for occupational and nonoccupational exposure in order to ensure minimal risk. In contrast, a zero-risk philosophy was apparent with the banning of UFFI on the basis of the potential for carcinogenic effects of formaldehyde gas released from this product. This position was supported by the availability of other less hazardous insulating materials.

The establishment of regulations for formaldehyde use in Canada has been a difficult and controversial process largely due to the limitations of the pertinent scientific data. Greater understanding of the mechanisms of formaldehyde toxicity and of adverse health effects in humans could lead to the refinement of current regulations. In particular, more detailed scientific information concerning exposure,

pharmacokinetics and metabolism, irritancy and sensitization, carcinogenicity and genotoxicity, and risk estimation, is desirable.

More valid data are required on the extent and magnitude of occupational exposure to formaldehyde, particularly in industries such as textile manufacture where there are large numbers of people at risk. More data are also needed formaldehyde levels in workplaces such as mortuaries and foundaries in which high concentrations have been reported and in workplaces in which epidemiological investigations are being or have been conducted. More accurate data are also required on the variability of formaldehyde concentrations in indoor air, so as to better define both short term peak exposures and long term (seasonal) variations (Consensus Workshop on Formaldehyde, 1984). Development of more reliable portable methods for determination of formaldehyde levels in the air of public and residential buildings would also be desirable.

It has been difficult to precisely establish the levels of formaldehyde which induce symptoms of irritancy in human populations, due to the limitations of available clinical and epidemiological studies. More carefully designed clinical studies of sensitive individuals and epidemiological investigations of occupationally and residentially exposed populations would provide valuable additional information. Such studies should involve examination of populations with longer exposure periods, estimation of the degree of exposure to

formaldehyde and to other chemicals, and assessment of the effects of important confounders (especially smoking).

Further carcinogenesis bioassays in rats at doses above, below, and between 2-15 ppm would allow scientists to better define the dose response curve for the carcinogenicity of formaldehyde (Consensus Workshop on Formaldehyde, 1984). However, since rats are obligatory nose breathers, comparative toxicity/carcinogenicity studies should also be conducted in primates which have oral and nasal breathing patterns similar to those of humans.

The relationship between formaldehyde levels in ambient air and formaldehyde concentrations at target cells needs to be more clearly established in both animals and humans (Starr et al., 1985). In particular, additional data on the absorption, transport, tissue distribution, and metabolism of formaldehyde would be desirable (Starr, 1986). Comparative studies on related aldehydes may facilitate understanding of the metabolism, reaction with macromolecules, and toxic effects of formaldehyde. Since formaldehyde is known to form DNA adducts, the structure of such adducts, the response of DNA repair mechanisms, and the relationship between chromosome breaks and formaldehyde dose, should also be investigated (Consensus Workshop, on Formaldehyde 1984).

A better understanding of the nature of formaldehyde - induced DNA lesions is desirable (Starr, 1986). Experiments should be conducted to determine whether such adducts preferentially form on single stranded DNA within cells, whether these lesions are associated with the cytotoxic, mutagenic, and transforming activity of formaldehyde, and what repair mechanisms remove such lesions (Consensus Workshop on Formaldehyde, 1984).

Estimation of the risk of formaldehyde-induced carcinogenesis in humans could be improved through the expansion and refinement of existing epidemiological databases, and by consideration of additional data from animal studies involving short term or intermittent exposure in species with breathing patterns similar to those of humans (Consensus Workshop on Formaldehyde, 1984). Refinement of mathematical dose response models through incorporation of critical mechanistic data is also needed (Health and Welfare Canada, 1986; Starr et al., 1985).

12.7 SUMMARY AND CONCLUSIONS

Formaldehyde is formed naturally in the environment and is released from anthropogenic sources including the manufacture, use and decomposition of formaldehyde-containing products and combustion. Due largely to the extensive use of formaldehyde-containing resins in building products, levels of formaldehyde in indoor air are greater than those in ambient air.

Formaldehyde is a vitally important intermediate in the metabolism of cells. Exposure to exogenous sources, however, results in sensory irritation. In clinical studies, significant increases in symptoms of irritation of the eyes, nose and throat have been observed at levels greater than 1 ppm. Data derived from observational studies of populations exposed to formaldehyde in the occupational environment or in public or residential buildings are less reliable owing to limitations of the investigations conducted to date; however, in the well-conducted studies, symptoms of irritation have not been associated with exposure to levels less than 0.5 ppm. It has also been suggested that formaldehyde may cause asthmatic attacks in sensitive individuals, although the available data are inconclusive in this regard.

The database on the possible carcinogenicity of formaldehyde is, by comparison, much less complete than that concerning the irritancy of the compound. Formaldehyde has been shown to be carcinogenic in two strains of rats producing a high incidence of nasal squamous cell carcinomas following administration of approximately 15 ppm. Epidemiological studies conducted to date have not been able to unequivocally demonstrate that formaldehyde is carcinogenic in human populations; however, this possibility cannot be excluded, particularly in light of the recent suggestive results of Blair et al. (1987). Estimation of the potential risk of cancer associated with exposure to formaldehyde can still be done using data from the animal bioassays. These estimates are however also uncertain due to a lack of understanding of the mechanisms of formaldehyde-induced carcinogenesis

and interspecies differences in nasal structure and breathing patterns.

As data on the irritancy and possible carcinogenicity of formaldehyde have accumulated, however, increasingly more stringent regulations to limit occupational exposure and the use of certain formaldehyde-containing products in Canada have been introduced. A threshold limit value of 2 ppm for occupational exposure has been adopted in many of the provinces; this value is currently under review and may well be reduced. Indeed, in Ontario, a value of 1 ppm has been recommended to provincial occupational health authorities (Farber et al., 1985).

Following deliberation of an expert advisory committee, urea formaldehyde foam insulation was banned in Canada in 1980 and guidelines for formaldehyde levels in indoor air have recently been established by a Federal Provincial Advisory Committee. It is likely that these guidelines will prompt the introduction of ventilation requirements and product specifications to ensure that formaldehyde levels in indoor air are below the recommended value. Indeed, formaldehyde emissions from particleboard have, over the past few years, been reduced dramatically due to changes in production methods introduced by the Canadian particleboard industry.

It is apparent, therefore, that the uncertainty about the risks associated with exposure to levels of formaldehyde currently present in the occupational and general environment has prompted the adoption of a prudent course of action in Canada. Wherever possible, formaldehyde emissions and permitted levels of exposure have been reduced. Moreover, although additional epidemiological and mechanistic data on the carcinogenicity of formaldehyde are desirable, it seems unlikely that this information will be available in the near future and it is probable that current trends to reduce exposure to this possible carcinogen will continue.

13. CONCLUDING COMMENTARY

Safety assessment is a risky business.

John Kirschman
General Foods Corporation

13.1 RISK ASSESSMENT AND RISK MANAGEMENT

Recent years have witnessed a surge in public and professional interest in the assessment and management of health and environmental risks, as reflected in the marked increase in the appearance of newspaper articles and scientific publication on risk. Following the pioneering work of Lowrance (1976 and Rowe (1977), this trend has culminated in the birth of a new discipline called risk science focussing on the systematic study of risk. Several universities have established special programs in risk assessment of risk management. At the same time, professional associations such as the Society for Risk Analysis have been formed to sponsor conferences on risk and publish professional journals devoted exclusively to risk.

A natural component of this evolutionary process has been the development of conceptual models of risk assessment and risk management to describe the risk problematic. The first such model was proposed by Whyte & Burton (1980) who envisaged a simple four stage model comprised of hazard identification, risk estimation, risk evaluation, and risk management. The first two stages combined constitute the risk analysis phase; the first three comprise the risk assessment phase. Since that

time, this model has been refined by a number of national and international organizations with strong interests in health and environmental risks. While these later models portray the process in much greater detail, thereby clarifying our understanding of risk assessment and risk management, they all involve the original four broad steps of identification, estimation, assessment and management.

The first step in the process is hazard identification. Epidemiological studies of human populations and toxicological studies conducted in the laboratory currently provide the bulk of the information used to identify human health hazards. Since both of these approaches to hazard identification have distinct advantages, neither approach should be preferred to the exclusion of the other.

Epidemiological studies have the unique advantage of providing evidence of adverse health effects directly in humans. However, observations on human populations can be of limited sensitivity when exposures are low. Given that human beings are constantly exposed to a myriad of environmental health hazards, the analysis of such data must allow for the possibility that any adverse health effects observed in epidemiological studies may be attributable to some unsuspected confounding risk factor. Since human exposure must take place prior to the conduct of an epidemiological investigation, moreover, epidemiology cannot be used to evaluate new materials prior to their introduction into the human environment.

Toxicological tests conducted in the laboratory can be designed so as to be largely free from bias, and can achieve great sensitivity through the use of exaggerated doses. These results are however achieved at the expense of obtaining only indirect evidence of the potential for substances tested to harm humans. Nonetheless, since most agents known to induce adverse health effects in humans also do so in one or more toxicological tests, it has become standard practice to assume that materials which are shown to be active in toxicological tests should be regarded as presumptive human health hazards.

Once a potential human health hazard has been identified, it is often useful to determine the magnitude of the risk presented. This may be accomplished by a more quantitative analysis of the same toxicological and epidemiological data base used to identify health hazards. Since this generally requires analysis of dose response relationships, the data requirements for risk estimation may be more extensive than for hazard identification. In particular, while potential hazards may be identified using only one or two relatively high dose groups in toxicological tests, a series of increasing doses may be required in order to delineate the slope of the dose response curve for purpose of risk estimation.

One of the most difficult problems in risk estimation is to infer the level of risk associated with exposure to low levels of environmental toxicants. Traditionally, acceptable levels of human

exposure have been established by dividing the dose at which no adverse health effects were observed in toxicological test by a suitable safety factor designed to allow for both inter- and intra-species differences in sensitivity. This approach is based on the assumption that there exists a threshold dose below which there is no risk of an adverse health effect occurring.

The threshold assumption may not be valid for certain delayed irreversible health effects such as carcinogenesis and mutagenesis. For these effects, it is generally considered that the risk is directly proportional to the level of exposure in the low dose region, so that any nonzero level of exposure will be associated with some nonzero risk. While absolute safety can then only be guaranteed if exposure is completely eliminated, a virtually safe level of exposure corresponding to some suitably low or de minimis level of risk may still be considered.

Because of the uncertainties involved in estimating low dose cancer risks, there has been a general tendency towards conservatism in risk estimation. Specifically, linear extrapolation may lead to upper bounds rather than best estimates of risk in some cases. (Although this may be viewed as a conservative practice, the present state of the art of cancer risk estimation does not permit the calculation defensible best estimates.) Similarly, conservative assumptions are often made in extrapolation results obtained in animals to humans.

This can present problems in balancing risks against benefits since conservative practices are generally not employed in assessing benefits.

What an acceptable level of risk should be in this context is a difficult question. It seems reasonable to suggest that there exist trivial risks which are not worth worrying about, and, at the other extreme, that there are risks sufficiently high so as to be considered unacceptable by the majority of the population. While regulatory agencies are reluctant to endorse a specific level of risk as being acceptable, previous regulatory actions taken on certain carcinogens present in the environment have allowed for lifetime risks on the order of one in a million.

Once risk analysis is complete, risk managers must select a suitable risk management strategy. The options available to decision making are many and varied, and may be broadly classified as regulatory, economic, advisory or technological. In Canada, as in most developed nations, regulation represents perhaps the most commonly used and certainly the most forceful option for risk management. At the federal level, there exists ten major statutes for the protection of human health and the environment. Many provinces have also enacted related legislation under the authority vested in them by the Constitution Acts of 1982 and 1867. Although there is some overlap between jurisdictional boundaries, the federal government has begun to withdraw its authority in areas of concurrent jurisdiction.

At present, there is a strong movement towards regulatory reform and deregulation, resulting in an increased consideration of the nonregulatory options for risk management. In this regard, economic incentives and disincentives offer powerful tools for influencing industrial policies on pollution control. Similarly, insurance schemes may be used to provide compensation without unduly burdening individual risk producers. Advisory approaches to risk management rely on the provision of information either to risk producers in an attempt to encourage risk reduction or to risk consumers so as to promote risk avoidance. Technological approaches to pollution abatement represent another risk management option which is becoming increasingly effective. These approaches are not mutually exclusive and may be applied jointly, as with fiscal support programs sponsored by government to facilitate the development of new technologies for pollution control.

The selection of a particular option or combination of options for risk management generally requires consideration of factors other than risk. Since hazardous materials also carry with them significant economic benefit, it can be reasonably argued that decisions based on risk alone (other than situations in which zero risk is mandated as the only permissable objective) are incomplete. To achieve a balance between risks and benefits, program evaluation techniques such as cost-benefit analysis may be invoked. In the United States, this is required by law under several statutes. This legal motivation is less

conspicuous in Canada, although a detailed socio-economic impact analysis is required for regulations anticipated to have major economic consequences.

A major difficulty in the application of risk-benefit and cost-benefit analysis in the health area is the monetarization of human health effects. This is a controversial area for which no consensus has yet been attained or for which no generally accepted methodology presently exists. Thus, less ambitious program evaluation techniques such as cost-effectiveness analysis may be considered. By seeking the least cost solution to exposure reduction, cost-effectiveness stops short of quantifying health effects. However, this simplification isobtained at the expense of eliminating comparisons between risk reduction programs involving different exposures at different health effects.

In practice, risk management decisions must also consider factors other than risks and benefits. In particular, decision makers need to be aware of public perception of risk, and give their views consideration in selecting a risk management strategy. While there are clear differences between perceived and actual risks, it should by no means be assumed that scientific estimates of risk are to be treated as an immutable baseline against which to gauge public perception. Uncertainties in risk estimation abound, including those involved in extrapolating from the high doses used in laboratory tests to the lower doses corresponding to human exposure levels, and in converting animal data to the human situation.

Less well recognized is the fact that even basic scientific observations can be subject to considerable uncertainty. For example, the diagnosis of a neoplastic lesion in an animal following completion of a laboratory carcinogenicity test may be influenced by the degree of autolysis experienced during the cause of the study, the attention paid to tissue acquisition and preservation at necropsy, and the use of special stains or more detailed evaluations using electron microscopy or morphometric analyses. Such uncertainties should be clearly borne in mind when evaluating the results of objective scientific studies of risk.

Throughout the process of risk assessment and risk management, it is essential that information on risk be communicated in clear and understandable terms and that effective channels of communication be established among individuals and institutions involved in the assessment of risk. Despite its importance to successful risk assessment, it is only within recent years that risk communication has been the object of systematic study. Models of risk communication have been developed to examine the process of risk communication in detail. These models reflect the message transmission processes and institutional structures involved in risk communication, and highlight the interplay between experts and the public in risk assessment.

While risk communication has recently been viewed as being distinct from related areas such as risk perception, specific risk communication techniques have been in practical use for some time. These include print-media information distribution systems to convey data on risk primarily from experts to the public, consultative and feedback mechanisms for public participation in risk management, and highly technical discussions within expert committees. In the future, we need to know more about how to effectively present quantitative data on risk without oversimplifying uncertainty and on how this information is received an understood by the general public.

13.2 PRACTICAL IMPLICATIONS

In the preceding section, the main features of the model used here to describe the process of risk assessment and risk management were reviewed. The model itself is based on a systemic analysis of this process, and offers considerable insight into the key elements of risk management decisions involving environmental health hazards. To further evaluate the utility of our model, it was applied to three environmental hazards: acid deposition, saccharin and formaldehyde. These case studies support the validity of the model, and provide a basis for some general observations concerning its practical implications.

In two of the three cases (saccharin and formaldehyde), health hazards were first identified on the basis of toxicological experimentation. In particular, both compounds were shown to cause cancer in rodents following long term exposure. In both cases, these toxicological findings spawned a series of epidemiological investigations in suitably chosen human populations to the point where saccharin and formaldehyde are among the most extensively studied chemicals epidemiologically. This tendency to rely primarily on toxicological data for hazard identification may be reduced in future as epidemiological techniques for human health monitoring become more refined.

Despite the large epidemiological data bases assembled on these two substances, the human evidence remains somewhat ambiguous. Since this may be due to the relatively low levels of exposure within the human population, regulatory action has been taken on both compounds. This is not to say that the apparent discrepancy between the animal and human data is not a point of controversy. On the contrary, the question as to whether either saccharin or formaldehyde is a human carcinogen has been the subject of much scientific debate. For example, critics of toxicological testing point out that the dose of saccharin required to induce cancer in animals is roughly equivalent to that received by consuming eight hundred bottles of diet soda each day. This line of argument of course misses the point that high doses are used in toxicological tests to achieve maximal sensitivity and that

carcinogenic effects observed at high doses may well be indication of similar effects at lower doses, albeit with reduced frequency. Thus, public health officials prudently treat saccharin and formaldehyde as presumptive human carcinogens even at the low levels of exposure to which people are generally subjected.

Identification of the hazards associated with acid deposition is different in many respects. First, the most relevant health effects are respiratory disorders, not cancer. Second, since the atmospheric pollutants which characterize acid rain are not amenable to toxicological testing, it is necessary to rely almost exclusively on epidemiological data for hazard identification. In addition to limitations in sensitivity due to low levels of exposure, these studies are difficult to conduct because of the problems in separating out the effects of common air pollutants not associated with acid deposition. Nonetheless, the studies conducted to data are suggestive of a small but detectable reduction in lung function performance in certain segments of the population. Even if such effects can be confirmed, the biological significance of such changes remain to be established.

Having identified these hazards, we next considered the extent to which quantitative estimates of risk played a role in subsequent risk management decisions. Once saccharin was labelled as a presumptive human carcinogen, it could no longer be used as a direct food additive. Thus, in accordance with the zero risk principle, its use as a food additive was banned.

Since exposure to formaldehyde could not be so easily eliminated, quantitative estimates of cancer risk were prepared and used in establishing the threshold limit value for occupational exposures as well as guidelines for levels in indoor air. Using a pharmacokinetic adjustment to take into account the dose reaching the target tissues in the nasal cavity, the TLV was set to limit the lifetime risk to one in one hundred thousand (10^{-5}).

In the case of acid rain, estimates of the level of mortality attributable to acid deposition have been prepared and have received much publicity. Although it has been estimated that some 4,000 deaths may be caused by air pollution in Canada each year, this figure is highly speculative and could be as low as zero or as high as 13,000. Because of this uncertainty, and because of the problems in associating these effects with those air pollutants which characterize acid rain, these estimates have not played a major role in the management of acidic deposition.

The three case studies considered here showed that a variety of risk management options have been deployed to control the hazards involved. Because of its carcinogenic effects, saccharin was banned as a direct food additive under the Food and Drug Act in 1977. Because of its low potency and perceived benefit as a means of calorie control, however, it could continue to be sold in pharmacies without a prescription for personal use.

Recognizing that formaldehyde cannot be completely eliminated from indoor air, nonzero guidelines for both occupational and nonoccupational exposure have established in attempt to ensure minimal risk. At the same time, urea formaldehyde foam insulation was banned in 1980 under the Hazardous Products Act because of the availability of other equally effective yet less hazardous insulating materials.

The control of acidic deposition in Canada is based on a combination of regulatory, economic, advisory, and technological risk management strategies. In particular, air quality objectives for major pollutants associated with acidic deposition have been established under the Clean Air Act. Both federal and provincial governments are using fiscal support to finance the development of new pollution control technologies. Both levels of government have also implemented major programs to promote public awareness of the acid rain problem. Finally, governments have allowed the technologies used to control industrial emissions contributing to the acid rain problem to be selected by individual polluters.

The role of public perception of risk is apparent to some degree in each of these three case studies. In the case of saccharin, there was a widespread perception that this artificial sweetener was harmless, coupled with a perceived benefit in terms of calorie control. While the perceived benefit could not be documented scientifically, the public's perception of saccharin as a safe sweetener resulted in a Congressional moratorium on the 1977 ban in the United States, a moratorium which remains in effect to this day.

Unlike saccharin, the public perceived the risk associated with exposure to formaldehyde to be serious, as reflected in the deluge of inquiries received by regulatory authorities. This perception was reinforced by the marked drop in property values in homes insulated with UFFI.

The case of acidic deposition clearly illustrate how risk perception can change over time. Prior to 1979, acid rain was virtually unknown to the public. By the early 1980's, however, this issue was rated near the top of the public' list of concerns. Today, acid rain is known more for its demonstrated adverse effects on the environment rather than on human health.

Risk communication has also been an issue of interest in these three cases. There were clearly problems in communicating the nature of the risks posed by saccharin to the public, as reflected in the degree of public skepticism over the hazards of this artificial sweetener. In the United States, a massive advertising campaign was mounted by the Calorie Control Council to promote this perception. This phenomena provides support for the communication processes model of risk communication which revolves around the interplay between the expert and public spheres.

The formaldehyde case also highlights the communication between experts and the public. Once a potential hazard was identified,

regulatory authorities were besieged with requirements for information on the risks involved, and what could be done to eliminate UFFI from homes which have been insulated with this material.

The public awareness programs implemented by governments have served to raise the level of public consciousness regarding acid rain. This information has been provided in different print and electronic media formats, and has been highly successful in making Canadians aware of this important environmental issue. The success of these programs may be measured in part by the enthusiasm with which this issue has been embraced by environmental groups.

13.3 THE FUTURE OF RISK ASSESSMENT

By most measures of human mortality and morbidity, the current Canadian population is safer than that of previous generations. This is equally true for other developed nations. Much of this improvementin the quality of life can be attributed to impressive advances made in the biomedical and life sciences in recent decades, particularly in sanitation and hygiene.

While few would question the assertion that technological advances have provided us with a great number of health and other benefits, it is becoming increasingly evident that many such innovations are not without an attendant degree of hazard. Much

recent attention has been focussed on the risks and benefits of the many synthetic chemicals introduced into the human environment since the beginning of the post-war era; the hazards associated with many other technological advances are only now becoming better identified, including those relating to solid waste disposal, new medical device technology, and the transportation of hazardous products.

A number of recent major events have served to sharpen public awareness of certain risks. The unheralded incident at Three Mile Island in 1979, for example, served to heighten public concern as to the safety of nuclear reactors. The derailment of a chlorine tanker in metropolitan Mississauga raised the spectre of accidental spillage of hazardous materials during transport. The more recent tragedy of Bhopal showed how dangerous the uncontrolled release of toxic chemicals can be. While particulate and gaseous emissions into the atmosphere have long been recognized by environmental scientists, the dramatic effects of acid rain on our Canadian lakes and forests served to underscore the risks due to air pollution.

If the current debate over environmental hazards has accomplished one thing, it is widespread recognition of the ineluctable fact that life is not risk-free. The most trivial activities in life carry with them some attendant degree of risk. In the real world in which absolute safety is an unachievable goal, it is necessary to weigh health and other benefits in light of the risks.

In recent years, formal decision-making procedures which may be applied in environmental risk management have come under study by a number of groups, including the Royal Society in the United Kingdom and the U.S. National Academy of Sciences. Although the development of straightforward decison-making criteria which would satisfy all corners of the risk triangle comprised of government, industry and the public appears to be an unattainable goal for the immediate future, these investigations have at least clarified some of the complexities involved in risk management decisions.

Many problems in risk management, for example, are characterized by overlapping jurisdictional boundaries at both the intra and international level and by the need for interdisciplinary approaches to risk assessment involving experts in diverse fields ranging from toxicology and epidemiology to economics and sociology. Ill-defined problems are often subject to almost continual redefinition with the passage of time and the unfolding of new events and accumulation of knowledge.

Fundamental to the complexity of many risk management decisions is uncertainty: uncertainty as to the effects of long-term low-level exposure to environmental contaminants, uncertainty as to the relevance for human populations of the effects induced in toxicological test systems, and uncertainty as to environmental exposure pathways and individual susceptibility. The assessment of benefits and the

distribution or risks, benefits and costs among different segments of society as a whole can be even more vague. Nonetheless, critical decisions must be made now even in the face of incomplete knowledge.

While it is evident that not all of the environmental problems with which we are currently faced can be solved through the passage of an ever-increasing number of specific regulations, a solid armamentarium of formal statutes may be called upon should regulation be selected as the risk management strategy most appropriate to the problem at hand. Other mechanisms for conveying information which may come into greater play in the future include labelling of consumer products, media advertising and public service announcements as well as educational programs designed to foster informed public choice.

The recent groundswell of public concern over environmental hazards has been accompanied by demands for more information from governments of potential health risks and for increased public participation in the risk assessment process itself. Although only introduced within recent years, both the Access to Information Act and Charter of Rights of 1982 represent powerful new forces guaranteeing freedom of information and whose full impact remains to be established.

As we look to the future, it is clear that both the spectrum of risks and the processes of risk assessment will change over time. With

increasingly complex environmental issues becoming commonplace, it is possible that governments may be called upon to play an even larger role in the management of risk. This will require careful ranking of our concerns and the development of explicit and consistent strategies for the control of environmental hazards. Scientific research may be expected to reduce, although not eliminate, the considerable uncertainty involved in the identification and estimation of risk. Refinements in the methodologies for assessing health and other benefits may also serve to sharpen comparisons between risks and benefits. Greater understanding of the risk assessment process among all segments of society, coupled with new information on both risks and benefits, may foster more acceptable if not better decisions in the years to come.

14. GLOSSARY OF KEY TERMS

ACCEPTABLE RISK: A risk whose probability of occurrence is so small, whose consequences are so slight, or whose benefits (perceived or real) are so great, that persons or groups in society are willing to take or be subjected to that risk.

EXPOSURE: Contact between a receptor and an agent.

The receptor may be a molecule, cell, tissue, organ, individual, or population.

EXPOSURE ESTIMATION: Estimation of the amount and duration of contact between a receptor and an agent.

May consider such factors as concentration, route, receptor, population, and timescale.

EXPOSURE IDENTIFICATION: Identification of the conditions of contact between a receptor and an agent.

May involve identification of concentration, route, receptor population, and timescale.

HAZARD: The likely adverse impact on health that can result from exposure to an agent, or from an event, or from undertaking or not undertaking some activity.

HAZARD IDENTIFICATION: Identification of the likely adverse impact on health that can result from exposure to an agent, or from an event, or from undertaking or not undertaking some activity.

May utilize case reports, toxicological studies, epidemiological investigations, or structure/activity analysis. May involve qualitative extrapolation from nonhuman test systems or from one route of exposure to another.

OPTION EVALUATION: The process of developing and analyzing options for risk management.

Development of options may involve consideration of program objectives and current institutional policies, and the regulatory environment. Option analysis may involve consideration of risks and benefits; uncertainties in risk estimations; public awareness and perception; technical feasibility; and economic, social, political, and cultural impacts.

PROBABILITY: The likelihood or frequency of occurrence of an event.

RISK: A measure of both the probability of and hazard to health resulting from an event or activity.

May involve quantitative extrapolation from animals to humans and/or from high dose/short time to low dose/long time. May consider potency (eg. physical/chemical properties; biological reactivity); susceptibility (eg. metabolic activation; repair mechanisms; age; sex; hormonal factors; immunological status); level of exposure (eg. sources; concentration; initiating events; routes; pathways); and adverse health effects (eg nature; severity; onset; reversibility) of the event or activity.

RISK ASSESSMENT: Assessment of the overall risk resulting from an event or activity, by hazard identification, risk estimation, and option evaluation.

The process involves consideration of such factors as risk acceptability, public perception, socioeconomic impacts, benefits, and technical feasibility, and leads to a decision based on both quantitative and qualitative factors.

RISK COMMUNICATION: A purposeful exchange of information on health or environmental risks between interested parties.

This may involve the transmission of information on the estimation, assessment or management of risk between government agencies,

industry, public interest groups, professional organizations, and individual scientistis or members of the public.

RISK ESTIMATION: Determination of the hazard and probability of occurrence of an event or activity.

Involves statistical analysis of toxicological and epidemiological data and of the level of human exposure. Examines the severity, extent, and distribution of the effects of an event or activity. Leads to a specific numerical point estimate or to a range of values

RISK MANAGEMENT: The selection and implementation of a strategy for control of a risk, followed by monitoring and evaluation of the effectiveness of that strategy.

The decision to select a particular strategy may involve consideration of the information obtained during risk evaluation. Implementation may involve a commitment of resources and communication with affected parties. Monitoring and evaluation may utilize such techniques as environmental sampling; post-market surveillance; prospective epidemiology; and analysis of new health risk information; as well as efforts to ensure compliance with the decision.

15. REFERENCES

Acheson, E.D., Barnes, H.R., Gardner, M J., Osmond, C., Pannett, B., and Taylor, C.P. (1984). Formaldehyde in the British Chemical Industry. Lancet, March 17, 611-616.

ACSH: American Council on Science and Health (1979). Saccharin. ACSH, New York.

Agriculture Canada, Fisheries and Environment Canada, and Health and Welfare Canada (1978) Pesticide Use and Control in Canada. Minister of Supply and Services, Ottawa.

Amdur, M.O. (1960). The Response of Guinea Pigs to Inhalation of Formaldehyde and Formic Acid Alone and with a Sodium Chloride Aerosol. Int. J. Air Poll., 3(4), 201-220.

American Public Health Association (1980) Standard Methods for the Examination of Water and Wastewater. American Public Health Association, Washington, D.C.

Ames, B.N., Durston, W.E. Yamasaki, E. and Lee, F.D. (1973a) Carcinogens and mutagens: A simple test system combining liver homogenates for activation and bacteria detection. Proceedings of the National Academy of Sciences 70, 2281-2285.

Ames, B.N., Lee, F.D. and Durston, W.E. (1973b). An improved bacterial test system for the detection and classification of mutagens and carcinogens. Proceedings of the National Academy of Sciences 70, 782-786.

Andersen, G.E. (1983). Human Exposure to Atmospheric Concentrations of Selected Chemicals, Vol. II. Systems Applications Inc. for U.S. Environmental Protection Agency, Strategies and Air Standards Division, Office of Air Quality Planning and Standards Research Triangle Park, N.C. 27711.

Andersen, S.K., Jensen, O.M. and Oliva, D. (1982). Formaldehyde-eksponering og lungecancer blandt danske laeger. Ugeskrift for Laeger 144, 1571-1573.

Anderson, F., Kneese, A. Taylor, S., Reed, P , and Stevenson, R. (1978). Environmental Improvement Through Economic Incentives exerpted in Resources (1979) 59: 1-4.

Anderson, M.E., Clewell, H.J., Gargas, M.L. Smith, F.A. and Reitz, R.H. (1987). Physiologically based pharmacokinetics and the risk assessment process for methylene chloride. Toxicology and Applied Pharmacology 87, 185-205.

Appling, A.J., Keddie, A.W.C. and Weatherby M.L.P.M. (1975). The High Pollution Episode in London. Report LR 263(AP), Warren Spring Laboratory, Stevanage.

Armstrong, B. (1985). Saccharin/cyclamates: epidemiological evidence. In: Interpretation of Negative Epidemiological Evidence for Carcinogenicity (N.J. Wald and R. Doll, eds.). International Agency for Research in Cancer, Lyon, pp. 129-143.

Armstrong, B. and Doll, R. (1974). Bladder cancer mortality in England and Wales in relation to cigarette smoking and saccharin consumption. British Journal of Preventive and Social Medicine 28, 233-240.

Arnold, D.L., Krewski, D., and Munro, I.C. (1983). Saccharin: a toxicological and historical perspective. Toxicology 27, 179-256.

Arnold, D.L., Moodie, C.A., Grice, H.C., Charbonneau, S.M , Stavric, B., Collins, B.T., McGuire, P.F., Zawidzka, Z.Z. and Munro, I.C. (1980). Long-term toxicity of ortho-toluenesulfonamide and sodium saccharin in the rat. Toxicology and Applied Pharmacology 52, 113-152.

Ashford, N.A. Ayers, C., and Stone, R.F. (1985). Using Regulation to Change the Market for Innovation. Harvard Environmental Law Review 9, 419-466.

Auerbach, C., Moutschen-Dahmen, M. and Moutschen, J. (1977). Genetic and cytogenetical effects of formaldehyde and related compounds. Mutation Research 39, 317-362.

Baram, M.S. (1982). Alternatives to Regulation. Lexington Books, Massachusetts.

Baram, M.S. (1984). The right to know and the duty to disclose hazard information. American Journal of Public Health, 74, 385-390.

Baram, M.S. (1986a). Chemical industry accidents, liability, and community right to know. American Journal of Public Health 76, 568-572.

Baram, M.S., and Kenyon, P. (1986b). Risk communication and the law for chronic health and environmental hazards. Environmental Professional 8, 165-178.

Barkin, M., Comisarow, R.H., Taranger, L.A. and Canada, A. (1977). Three cases of human bladder cancer following high dose cyclamate ingestion. Journal of Urology 118, 258-259.

Barrie, L.A. (1982). Environment Canada's Long Range Transport of Atmospheric Pollutants Program: atmospheric studies. In: Acidic Precipitation: Effects on Ecological Systems (F.M. d'Itri, ed.). Ann Arbor Science, Ann Arbor, pp. 141-161.

Barrow C.S. and Steinhagen, W.S. (1981). Sensory Irritation by Inhaled Formaldehyde in B6C3F1 Mice and F-344 Rats Following Single or Repeated Exposure. Toxicologist, 1, 5-6.

Barrow, C.S., Steinhagen, W.H. and Chang, J.C.F. (1983). Formaldehyde Sensory Irritation In: Formaldehyde Toxicity, (J.E. Gibson, ed.) Hemisphere Publ. Corp., Washington, D.C., p. 16-25.

Bartsh, H., Tomatis, L. and Malaveille, C. (1982). Mutagenicity and carcinogenicity of environmental chemicals. Regulatory Toxicology and Pharmacology 2, 94-105.

Bates, D.V. and Sizto, R. (1983). Relationship between air pollutant levels and hospital submissions in southern Ontario. Canadian Journal of Public Health 74, 117-122.

Bell, B. (1876). Paraffin epithelioma of the scrotum. Edinburgh Medical Journal 22, 135.

Bender, J.R., Mullin, L.S., Graepel, G.J., and Wilson, W.E. (1983). Eye Irritation Response of Humans to Formaldehyde. American Industrial Hygiene Association Journal 44 463-465.

Bickis, M. and Krewski, D. (1985). Statistical design and analysis of the long-term carcinogenicity bioassay. In: Toxicological Risk Assessment, Vol. I, Biological and Statistical Criteria (D.D. Clayson, D. Krewski & I.C Munro, eds.). CRC Press, Boca Raton, Florida, pp. 125-147.

Blair, A. and Spirtas, R. (1981). Use of occupational cohort studies in risk assessment. In: Health Risk Analysis (C.R. Richmond, P.J. Walsh & E.D. Copenhaver, eds). Franklin Institute Press, Philadelphia, pp. 97-108.

Blair, A., Stewart, P., O'Berg, M , Gaffey, W., Walrath, J., Ward, J., Bales, R., Kaplan, S., and Cubit, D. Mortality Among Industrial Workers Exposed to Formaldehyde. Journal of the National Cancer Institute 76, 1071-1084.

Bonner, P.S. (1978). Legislative Basis for the Activities of the Environmental Health Directorate. Contract report, Health and Welfare Canada, Ottawa.

Booth, S.C., Bosenberg, H., Garner, R.C., Hertzog, P.J. and Norpoth, K. (1981). The activation of aflatoxin B_1 in liver slices and in bacterial mutogenicity assays using livers from different species including man. Carcinogenesis 2, 1063-1068.

Boreiko, C.J. and Ragan, D.L. (1985). Genetic and transforming activity of formaldehyde. In: Formaldehyde Toxicity (J.E. Gibson, ed.). McGraw-Hill, New York.

Bortoli, M.D., Knoppel, M. et al (1985). Measurement of Indoor Air Quality and Comparison with Ambient Air. Published by the Commission of the European Communities.

Boyle, M.H., Torrance, G.W., Sinclair, J.C., and Horwood, S.P. (1983). Economic evaluation of neonatal intensive care of very-low-birth-weight infants. New England Journal of Medicine 308, 1330-1337.

Breslow, N.E. & Day, N.E. (1980). Statistical Methods in Cancer Research, Vol. I: The Analysis of Case Control Studies. International Agency for Research on Cancer, Lyon.

Breysse, P. (1977). Formaldehyde in Mobile and Conventional Homes. Environ. Health Safety News 26, 1-6.

Brickman, R., Jasanoff, S. and Ilgen, T. (1985). Controlling Chemicals: The Politics of Regulation in Europe and the United States. Cornell University Press, Ithaca, New York.

Brinton, L., Becker, J., Blot, W , et al. (1983). A Case Control Study of Cancer of the Nasal Cavity and Sinuses. American Journal of Epidemiology 118, 436-437.

Brown, C.C. (1976). Mathematical aspects of dose-response studies in carcinogenesis - the concept of thresholds. Oncology 33, 62-65.

Brown, C.C. and Koziol, J.A. (1983). Statistical aspects of the estimation of human risk from suspected environmental carcinogens. SIAM Review 25, 157-181.

Bryan, W.R. and Shimkin, M.B. (1943). Quantitative analysis of dose-response data obtained with three carcinogenic hydrocarbons in strain C3H male mice. Journal of the National Cancer Institute 3, 503-531.

Burdach, S., and Wechselberg, K. (1980). Gesundheitsschäden in der Schule. Fortschrihe Medizine 98, 379-384.

Burgess, D.F (1981). The social discount rate for Canada: theory and evidence. Canadian Public Policy 7, 383-394.

Burnett, R.T., Krewski, D., Birkwood, P.L. and Franklin, C.A. (1988). Health risks from acid rain. In: Information Needs for Risk Management (L. Grima & R.T. Munn, eds). Environmental Monograph No. 8 Institute for Environmental Studies, Toronto. In Press.

Cairns, J. (1975). The cancer problem. Scientific American 233: 64-78.

Campbell, T.C. (1980). Chemical carcinogens and human risk assessment. Federation Proceedings 39, 2467-2484.

Canadian Environmental Control Newsletter (1986). U.S. Acid Rain Bill Approved by House. CECN 3138, 2631-2632.

Cantor, K.P. (1981). Human case-control studies in risk assessment In: Health Risk Analysis (C.R. Richmond, P.J. Walsh & E.D., Copenhaver, eds.). Franklin Institute Press, Philadelphia, pp. 109-120.

Carroll, K.K. (1975). Experimental evidence of dietary factors and hormone-dependent cancers. Cancer Research 35, 3374-3383.

Cartwright, R.A., Abid, R., Glashan, R. and Gray, B.K. (1981). The epidemiology of bladder cancer in West Yorkshire. A preliminary report on non-occuaptional aetiologies. Carcinogenesis 2, 343-347.

Casanova-Schmitz, M., Starr, T.B., and Heck, H.d'A. (1984). Differentiation Between Metabolic Incorporation and Covalent Binding in the Labeling of Macromolecules in the Rat Nasal Mucosa and Bone Marrow by Inhaled ^{14}C -and ^{3}H Formaldehyde. Toxicology and Applied Pharmacology 76, 26-44.

Chang, J.C.F., Gross, E.A., Swenberg, J.A., and Barrow, C.S. (1983). Nasal Cavity Deposition, Histopathology, and Cell Proliferation after Single or Repeated Formaldehyde Exposures in B6C3F1 Mice and F-344 Rats. Toxicology and Appled Pharmacology 68, 161-176.

Chang, J.C.F., Steinhagen, W.H., and Barrow, C.S. (1981). Effect of Single or Repeated Formaldehyde Exposure on Minute Volume of B6C3F1 Mice and F-344 Rats. Toxicology and Applied Pharmacology 61, 451-459.

Chappel, C., Grice, H., Miller, A. and Willes, R. (1985). An Appraisal of the Role of Epidemiology in the Regulatory Risk Assessment Process in Canada. Science Council of Canada, Ottawa.

Churchill, D.N., Morgan, J., and Torrance, G.W. (1984). Quality of life in end-stage renal-disease. Peritoneal Dialysis Bulletin 4, 20-23.

Ciocco, A. and Thompson, D.J. (1961). A follow-up of Donora ten years after: methodology and findings. American Journal of Public Health 51, 155-164.

Clark, E.H. (1978). Comment on "Regulatory strategies for pollution control". In: Approaches to Controlling Air Pollution (A.F. Friedlaender, ed.). MIT Press, Cambridge, pp. 220-230.

Clayson, D.B (1984). The mode of carcinogenic action of saccharin. Cancer Letters 22, 119-123.

Clayson, D.B., Krewski, D. and Munro, I.C. (1983). The power and interpretation of the carcinogenicity bioassay. Regulatory Toxicology and Pharmacology 3, 329-348.

Cleveland, W.S., Graedel, T.E., and Kleiner, B. (1977). Urban Formaldehyde: Observed Correlation with Source Emissions and Photochemistry. Atmospheric Environment 11, 357-360.

Cohen, S.M., Arai, M., Jacobs, J.B. and Friedell, G.H. (1979). Promoting effect of saccharin and DL-tryptophan in urinary bladder carcinogenesis. Cancer Research 39, 1207-1217.

Cohen, S.M. Murasaki, G., Fukushima, S. and Greenfield, R.E. (1982). Effect of regenerative hyperplasia on the urinary bladder: carcinogenicity of sodium saccharin and N- 4-(5-nitro-2-furyl)-2-thiazolyl formamide, Cancer Research 42, 65-71.

Cole, P. (1984). Factors and risks in cancer incidence. Paper presented at National Conference on Cancer Prevention Strategies, Ottawa, October 16-17, 1984.

Committee on Food Protection (1980). Risk assessment/safety evaluation of food chemicals. National Academy Press, Washington, D.C.

Conference of First Minister (1985). Economic and Regional Development Agreements: Partnership for Growth. Environment Canada, Ottawa.

Consensus Workshop on Formaldehyde (1984). Report on the Consensus Workshop on Formaldehyde. Environmental Health Perspectives 58, 323-381.

Consumer Reports (1977). The great saccharin snafu. 42, 410-414.

Coon, J.M. (1975). Sweeteners: issues and uncertainties. Academy Forum, National Academy of Sciences, Washington D.C., pp. 133-142.

Cooper, B.S. and Rice, D.P. (1976). The economic cost of illness revisited. Social Security Bulletin 39, 21-36.

Corpus Information Services (1982). The Formaldehyde Issue. M. Lauzon (ed.), Corpus Information Services, Toronto, 2-11 - 3.5.

Cordle, F. and Miller, S.A. (1984). Using epidemiology to regulate food additives: saccharin case-control studies. Public Health Reports 99, 365-369.

Cornfield, J., Carlborg, F.W. and Van Ryzin, J. (1978). Setting tolerances on the basis of mathematical treatment of dose-response data extrapolated to low doses. In: Proceedings of the First International Congress on Toxicology. (G.L. Plaa & W.A.M Duncan, eds.). Academic Press, New York, pp. 143-164.

Cornfield, J., Rai, K. and Van Ryzin, J. (1980). Procedures for assessing risks at low levels of exposure. In: Quantitative Aspects of Risk Assessment in Chemical Carcinogenesis. Archives of Toxicology, Supplement 3, pp. 295-303.

Coulston, F. (ed.). (1979). Regulatory Aspects of Carcinogenesis and Food Additives: The Delaney Clause. Academic Press, New York.

Covello, V. and Merkhofer, M. (eds) (1984). Risk Assessment and Risk Assessment Methods. The-State-of-the-Art. National Science Foundation, Washington, D.C.

Covello, V.T. and Mumpower, J. (1985). Risk Analysis and Risk Management: An Historical Perspective. Risk Analysis 5 103-120.

Covello, V.T., Menkes, J. and Nechnevasja, J. (1982). Risk analysis, philosophy, and the social and behavioural sciences: reflections on the scope of risk analysis research. Risk Analysis 2, 53-58.

Covello, V., von Winterfeldt, D. and Slovic, P. (1986). Risk Communication: An Assessment of the Literature on Communicating Information About Health, Safety and Environmental Risk. Preliminary Report to the Environmental Protection Agency. Los Angeles: Institute of Safety and Systems Management, 31 pp., January 11, 1986.

Covello, V. von Winterfeldt, P. and Slovic, P. (1987). Risk communication: a review of the literature. Risk Abstracts, 171-182.

Crouch, E. and Wilson, R. (1981). Regulation of Carcinogens. Risk Analysis 1, 47-57.

Crouch, E.A.C. and Wilson, R. (1981). Risk/Benefit Analysis. Ballanger Publishing, Cambridge, Massachusetts.

Crump, K.S., Hoel, D.G., Langley, C.H. and Peto, R. (1976). Fundamental carcinogenic processes and their implications for low dose risk assessment. Cancer Research 36, 2973-2479.

Dalbey, W E. (1982). Formaldehyde and Tumors in Hamster Respiratory Tract. Toxicology 24, 9-14.

Dally, K.A., Hanrahan, L.P., Woodbury, M.A , and Kanarek, M.S. (1981). Formaldehyde Exposure in Nonoccupational Environments. Archives of Environmental Health 36, 277-284.

Davis, J.C. (1983). Science and policy in risk control. In: Risk Management of Existing Chemicals. Chemical Manufacturer's Association, Government Institutes, pp. 13-21.

Davis, T.R.A., Battista, S.P., and Kensler, C.J. (1967). Mechanism of Respiratory Effects During Exposure of Guinea Pigs to Irritants. Archives of Environmental Health 15, 412-419.

de Boer, H.W. and Borgsma, O. (1943). Onderzoekingen betreffende de scadelijkheid van saccharine. Chemisch. Weekblad 40, 26-32.

Desuiel, M. (1985). Courtesy of Seifert, B. - Private Communication.

Detels, R., Sayre, J.W., Tashkin, D.P., Massey, F.J. Coulson, A.H. and Rokaw, S.N. (1984). Relationship of physiologic factors to rate of change in forced expiratory volume in one 2nd and forced vital capacity. American Review of Respiratory Disease 129, 533-537.

Dillard, S. (1983). Durée ou Qualité de la Vie? Conseil des affaires sociales et de la famille, Québec.

Doll, R. (1985). Purpose of symposium. In: Interpretation of Negative Evidence for Carcinogenicity (N.J. Wald & R. Doll, eds.). IARC Scientific Publication No. 65, International Agency for Research on Cancer. Lyon, pp. 3-10.

Doll, R., and Peto, R. (1976). Mortality in relation to smoking: 20 years' observations on male British doctors. British Medical Journal 2, 1525-1536.

Doll, R. and Peto, R. (1981). The causes of cancer: Quantitative estimates of avoidable risks of cancer in the United States today. Journal of the National Cancer Institute 66, 1191-1308.

Dourson, M.L. and Stara, J.F. (1983). Regulatory history and experimental support of uncertainty (safety) factors. Regulatory, Toxicology and Pharmacology 3, 224-238.

Drummond, M.F. (1980). Principles of Economic Appraisal in Health Care. Oxford University Press, Oxford, England.

Economic Council of Canada (1981). Reforming Regulation. Canadian Government Publishing Centre, Ottawa.

Environment Canada (1981). Public Perception of Water Quality in the Great Lakes. Environment Canada, Ottawa.

Environment Canada (1983a). Study of Trends in Canadian Environmental and Water Issues Concerning Ontario and the Great Lakes Region. Environment Canada, Ottawa.

Environment Canada (1983b). A nationwide inventory of emissions of air contaminants (1978). Economic and Technical Review Report, EPS 3-EP-83-10 Environment Canada, Ottawa.

Environment Canada (1984a). National urban air quality trends, 1974-1981. Environmental Protection Service Report EPS 7/AP/14. Environment Canada, Ottawa.

Environment Canada (1984b). Acid rain: the Canadian perspective. Environment Canada, Ottawa.

Environment Canada (1985a). Acid rain: the statistics. Environment Canada, Ottawa.

Environment Canada (1985b). Acid rain: the Canadian control program. Environment Canada, Ottawa.

EPA: Environmental Protection Agency (1986). Guidelines for carcinogen risk assessment. Federal Register 51, 33992-34003.

Evans, R.G. (1984). Strained Mercy: The Economics of Canadian Health Care. Butterworths, Toronto.

Farber, E., Golberg, L. and Munro, I.C. (1985). Report of a Panel: Review of the available information on the health effects of occupational exposure to formaldehyde. Ontario Ministry of Labour, Toronto.

Farkas, C.S. and Forbes, C.E. (1965). Do non-caloric sweetners aid patients with diabetes to adhere to their diets? Journal of the American Dietetic Association 46, 482-484.

Fayerweather, W.E., Pell, S. and Bender, J.R. (1983). Case Control Study of Cancer Deaths in Du Pont Workers with Potential Exposure to Formaldehyde, In: Formaldehyde, Toxicology, Epidemiology, Mechanisms, J.J. Clary, J.E. Gibson, and R.S. Waritz, eds.). Marcel Dekker Inc., New York, (1983), pp. 47-125.

Ferguson, T.S. (1967). Mathematical Statistics: A Decision Theoretic Approach. Academic Press, New York.

Firket, J. (1931). Sur les causes des accidents survenus dans la vallee la Meuse, lors des brouillards de decembre, 1930. Bulletin de l'Academie Royale de Belgique 11, 683-739.

Fischhoff, B Slovic, P. and Lichtenstein, S. (1983). The public vs. the experts: perceived vs. actual disagreements about riskss. In: The Analysis of Actual versus Perceived Risks (V. Covello et al., (Plenum Prss, New York), pp. 235-249.

Fischhoff, B Slovic, P. & Lichtenstein, S. (1979). Weighing the risks, Environment 21, 17-20, 32-38.

Fiserova-Bergenova, V. (1983). Physiological models for pulmonary administration of inert vapors and gases. In: Modeling of Inhalation Exposure to Vapors: Uptake, Distribution and Elimination, Vol. I. (V. Fiserova-Bergerova, ed.). CRC Press, Boca Raton, Florida.

Fitzhugh, O.G., Nelson, A.A. and Frawley, J.P. (1951). A comparison of the chronic toxicities of synthetic sweetening agents. Journal of the American Pharmaceutical Association 40, 583-586.

Fleiss, J.L. (1981). Statistical Methods for Rates and Proportions. Wiley, New York.

Food Chemical News (1977a). Saccharin ban proposal expected within three weeks. 19, pp. 21-29.

Food Chemical News (1977b). Saccharin guidelines provide for stick-on labels, hang-tags. 19, pp. 47-50.

Food Safety Council (1978). Proposed System for Food Safety Assessment. Chapter 4. Human Exposure Assessment. Food and Cosmetics Toxicology 16, (Supplement 2), 25-28

Food Safety Council (1982). A Proposed Food Safety Evaluation Process. Food Safety Council, Washington, D.C.

Franklin, C.A., Burnett, R.T., Paolini, R.J.P. and Raizenne, M.E. (1985). Health risks fro acid rain A Canadian perspective. Environmental Health Perspectives 63, 166-168.

Fraser, R.D., Spasoff, R.A. and Prime, M.G. (1976). Toward the establishment of health research priorities: an Estimate of the Economic Burden of Ill-Health. Ontario Council of Health, Toronto.

Freundt, K.J. (1982). Mixed exposures to chemical hazards. Occupational Health and Safety, 10-13; 39-42.

Fries, G.F (1985). The PBB episode in Michigan: an overall appraisal. CRC Critical Reviews in Toxicology 16, 105-156.

Fuhs, G.W and Olsen, R.A. (1979). Technical memorandum: Acid precipitation effects on drinking water in the Adirondack Mountains of New York State. Department of Health, New York State, Albany, N.Y., 1979.

Fuhs, G.W and Reddy, M.M. (1980). Distribution of mercury and 14 other elements in remote watersheds in the Adirondack mountains, New York. Abstracts of Papers of the American Chemical Society 100, 130.

Gamble, J. (1983). Effects of Formaldehyde on the Respiratory System. In: Formaldehyde Toxicity (J.E. Gibson, ed.), Hemisphere, Washington, D.C., pp. 175-197.

Garner, R.C., Martin, C.N. and Clayson, D.B. (1984). Carcinogenic aromatic amines and related components. In: Chemical Carcinogens (2nd edition) Vol. I (C.E. Searle, eds.). American Chemical Society, Washington, D.C., pp. 175-276.

Gart, J.J., Krewski, D., Lee, L.N., Tarone, R.E. and Wahrendorf, J. (1986). Statistical Methods in Cancer Research, Vol. III, The Design and Analysis of Long-Term Animal Experiments. International Agency for Research on Cancer, Lyon.

Garry, V.F. Oatman, L., Pleus, R. and Gray, D. (1980). Formaldehyde in the home - some environmental disease perspectives. Minnisota Medicine 63, 107-111.

Ghelardi, R.E. and Murphy, B.L. (1981). Acid rain. Dangerous Properties of Industrial Materials Report, 14-27.

Glasser, M. and Greenburg, L. (1971). Air pollution, mortality, and weather New York City, 1960-1964. Archives of Environmental Health 22, 334-343.

Gold, L.S., Sawyer, C.B., Magaw, R., Backman, G.M., de Veciana, M., Levinson, R. Hooper, N.K., Havendar, W.R., Bernstein, L., Peto, R., Pike, M.C. and Ames, B.N. (1984). A carcinogenic potency database of standardized results of animal bioassays. Environmental Health Perspectives 58, 9-319.

Gore, A.T. and Shaddick, C.W. (1958). Atmospheric pollution and mortality in the County of London. British Journal of Preventive and Social Medicine 12, 104-113.

Grima, A.P., Timmerman, P., Fowle, C.D., and Byer, P. (1986). Risk management and EIA: research needs and opportunities. Canadian Environmental Assessment Research Council, Ottawa.

Guicherit, R., and Schulting, F.L. (1985). The Occurrence of Organic Chemicals in the Atmosphere of the Netherlands. The Science of the Total Environment 43, 193-219.

Hamilton, L. (1985). Health effects of long-range air pollution transport in Canada. Report prepared under contract with Health & Welfare Canada, Health & Welfare Canada, Ottawa.

Hansard (1972). House of Commons debates official reports, Queen's Printer for Canada, Ottawa, March 10.

Harrington, J.M., and Shannon, H.S. (1975). Mortality Study of Pathologists and Medical Laboratory Technicians. British Medical Journal 4, 329-332.

Harrington, J.M., and Oakes, D. (1984). Mortality Study of British Pathologists 1974. British Journal of Industrial Medicine 41, 188-191.

Hartley, H.O., Tolley, H.D. and Sielken, R.L., Jr. (1981). The product form of the hazard rate model in carcinogenic testing. In: Statistics and Related Topics (M. Csorgo, D.A. Dawson, J.N.K. Rao & A.K.Md.E Saleh, eds). North-Holland, Amsterdam, pp. 185-200.

Hasegawa, R., Greenfield, R.E., Murasaki, G., Suzuki, T. and Cohen, S.M. (1985). Initiation of urinary bladder carcinogenesis in rats by freeze ulceration with sodium saccharin promotion. Cancer Research 45, 1469-1473.

Hasegawa, R., St. John, M.K., Caro, M Issenberg, P., Klein, D.A. Walker, B.A., Jones, J.W., Schnell, R.C., Merrick, B.A., Davies, M.H., McMillan, D.T. and Cohen, S.M. (1984). Bladder freeze ulceration and sodium saccharin feeding in the rat: examination for urinary nitrosamines, mutagens and bacteria, and effects on hepatic microsomal enzymes. Food and Chemical Toxicology 22, 935-942.

Haseman, J.K. and Hoel, D.G. (1979). Statistical design of toxicity assays: role of genetic structure of test animal population. Journal of Toxicology and Environmental Health 5, 89-101.

Hayes, A.W. (ed.)(1984). Principles and Methods of Toxicology. Raven Press, New York.

Hayes, R.B., Raatgever, J.W., and de Bruyn, A.E. (1984). Tumours of the nose and nasal sinuses. Technical Report, Department of Public Health and Social Medicine, Medical Faculty, Erasmus University, Rotterdam The Netherlands.

Health and Welfare Canada (1975). Nutrition Canada Provincial Reports. Health & Welfare Canada, Ottawa.

Health and Welfare Canada (1976). Nutrition Canada Food Consumption Patterns Report. Health & Welfare Canada, Ottawa.

Health and Welfare Canada (1977a). Nutrition Canada Dental Report. Health and Welfare Canada, Ottawa.

Health and Welfare Canada (1977b). Canadian position on saccharin. News Release 1977-40, March 9, Health and Welfare Canada, Ottawa.

Health and Welfare Canada (1977c). Short-term extension of permitted use of saccharin. News Release 77-66, April 19. Health and Welfare Canada. Ottawa.

Health and Welfare Canada (1979). Food Additives: What do you Think? Health & Welfare Canada, Ottawa.

Health and Welfare Canada (1980). Nutrition Canada Anthropometry Report. Health & Welfare Canada, Ottawa..

Health and Welfare Canada (1984a). Mortality Atlas of Canada. Vol. 3: Urban Mortality. Supply and Services Canada, Ottawa.

Health and Welfare Canada (1984b). Health Risk Study, Phase II: National Survey. Health & Welfare Canada. Ottawa.

Health and Welfare Canada (1986). Report on Formaldehyde, prepared for the Federal-Provincial Working Group on Indoor Air Quality, Bureau of Chemical Hazards, Environmental Health Directorate, Department of Health and Welfare Canada.

Health and Welfare Canada and Statistics Canada (1980). Mortality Atlas of Canada (Vols. I & II). Supply & Services Canada, Ottawa.

Health and Welfare Canada and Statistics Canada (1981). The Health of Canadians: Report of the Canada Health Survey. Supply & Services Canada, Ottawa.

Hendrick, D. Dunnett, E., Rivers, R.Z., Krewski, D. and Muller, E.F. (1984). Risk-Benefit Analysis in the Management of Toxic Chemicals. Agriculture Canada, Ottawa

Herbst, A.L. and Ulfelder, H. and Poskanzer, D.C. (1971). Adenocarcinoma of the vagina. Association of maternal stilbestrol therapy with tumor appearance in young women. New England Journal of Medicine 284, 878-881.

Hernberg, S., Westerholm, P., Schultz-Larsen, K., Degerth, R., Kuosma, E., Englund, A., Engzell, U., Hansen, H.S., and Mutanen, P. (1983). Nasal and sinonasal cancer. Connection with occupational exposures in Denmark, Finland and Sweden. Scandinavian Journal of Work, Environment and Health 9, 315-326.

Hicks, R.M (1984). Promotion: is saccharin a promoter in the urinary bladder? Food and Chemcial Toxicology 22, 755-760.

Hicks, R.M. and Chowaniec, J. (1977). The importance of synergy between weak carcinogens in the induction of bladder cancer in experimental animals and humans. Cancer Research 37, 2943-2949.

Hirayama, T. (1981). Non-smoking wives of heavy smokers have a higher risk of lung cancer: A study from Japan. British Medical Journal, 282 183-185.

Hoel, D.G. (1985). The incorporation of pharmacokinetics in low dose risk assessment. In: Toxicological Risk Assessment, Vol. I, Biological and Statistical Criteria (D.B. Clayson, D. Krewski & I.C. Munro, eds.). CRC Press, Boca Raton, Florida, pp. 205-214.

Hoel, D.G., Kaplan, N.L. and Anderson, M.W. (1983). Implication of nonlinear kinetics on risk estimation in carcinogenesis. Science 219, 1032-1037.

Hohenemser, C., Kates, R.W. and Slovic, P. (1983). The nature of technological hazard. Science 220, 378-384.

Hoover, R.N. and Strasser, P.H. (1980). Artificial sweeteners and human bladder cancer. Preliminary Results. Lancet, pp. 837-840.

Horton, A.W., Tye, R. and Stemmer, K.L. (1963). Experimental Carcinogenesis of the Lung. Inhalation of Gaseous Formaldehyde or an Aerosol of Coal Tar by C3H Mice. Journal of the National Cancer Institute 30, 31-43.

Howe, G.R., Burch, J.D., Miller, A.B., Morrison, B., Gordon, P., Weldon, L., Chambers, L.W., Fodor, G. and Winsor, G.M. (1977). Artificial sweeteners and human bladder cancer. Lancet, pp. 578-581.

Howe, G.R., Lindsay, J., Coppock, E., and Miller, A.B. (1979). Isoniazid exposure in relation to cancer incidence and mortality in a cohort of tuberculosis patients. International Journal of Epidemiology 8, 305-312.

Hultberg, H. and Wenblad, A. (1980). Acid ground water in southwestern Sweden. In: Ecological Impact of Acid Precipitation, Proceedings of an International Conference, 11-14 March, 1980, Sande/gord, Norway, 1980.

Hunter, B.T. (1971). Consumers Beware. Your Food and What's Being Done to It. Bantam Press, New York.

IARC: International Agency for Research on Cancer (1982). Chemicals, Industrial Processes and Industries Associated with Cancer in Humans IARC Monographs, Volumes 1 to 29. IARC Monographs Supplement 4, IARC, Lyon.

IARC: International Agency for Research on Cancer (1982b). Formaldehyde. In: IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans, Some Industrial Chemicals and Dyestuffs. Vol. 29, IARC, Lyon, France, 1982, pp. 345-389.

Ibau, J. (1939). The relative potency of carcinogenic compounds. American Journal of Cancer 35, 188-190.

ICPEMC: International Commission for Protection against Environmental Mutagens and Carcinogens (1982). Mutagenesis testing as an approach to carcinogenesis. Mutation Research 99, 73-91.

ILSI: International Life Sciences Institute (1984a). The use of short-term tests for mutagenicity and carcinogenicity in chemical hazard evaluation. In: Current Issues in Toxicology (H.G. Grice, ed.). Springer-Verlag, New York, pp. 156-197.

Interdepartmental Working Group on Risk-Benefit Analysis (1984). Risk-Benefit Analysis in the Management of Toxic Chemicals. Agriculture Canada, Ottawa.

Irwin, J.O. and Goodman, N. (1946). The statistical treatment of measurements of the carcinogenic properties of tars (Part I) and mineral oils (Part II). Journal of Hygiene 44, 362-420.

Jenkins, G.P. (1980). Discount rates for economic appraisal of public sector expenditures. Canadian Public Policy 6, 549-555.

Jenkins, G.P. (1981). The public-sector discount rate for Canada: some further observations. Canadian Public Policy 7, 399-407.

Jones, C.A., Marlino, P.T., Lijinsky, W. and Huberman, E. (1981). The relationship between the carcinogenicity and mutagenicity of nitrosamines in a hepatocyte mediated mutagenicity assay. Carcinogenesis 2, 1075-1077.

Jurs, P.C., Hasan, M.N., Henry, D.R., Stouch, T.R. and Whalen-Pederson, E.K. (1983). Computer assisted studies of molecular structure and carcinogenic activity. Fundamental and Applied Toxicology 3, 343-349.

Kalbfleisch, J.D. & Prentice, R.L. (1980). The Statistical Analysis of Failure Time Data. Wiley, New York.

Kane, L.E. and Alarie, Y. (1977). Sensory Irritation to Formaldehyde and Acrolein during Single and Repeated Exposures in Mice. American Industrial Hygiene Association Journal 38, 509-522.

Kane, T.G. (1980). Consumers and the Regulators: Intervention in the Federal Regulatory Process. Institute for Research on Public Policy, Montreal.

Kanarek, M.S. (1981). Indirect epidemiology studies in environmental hazard risk assessment. In Health Risk Analysis (C.R. Richmond, P.J. Walsh, and E.D. Copenhaver, eds.). Franklin Institute Press, Philadelphia, pp. 87-96.

Kaplan, S. and Garrick, B.J. (1981). On the quantitative definition of risk. Risk Analysis 1, 11-27.

Kaplan, R.M., Bush, J.W. and Berry, C.C. (1979). Health status index category rating versus magnitude estimation for measuring levels of well-being. Medical Care 17, 501-525.

Kaplan, R.S. (1982). Advanced Management Accounting. Prentice-Hall, Englewood Cliffs, New Jersey.

Kaplan, R.M. and Bush, J.W. (1982). Health related quality of life measurement for evaluation research and policy analysis. Health Psychology 1, 61-80.

Kates, R.W. (1978). Risk Assessment of Environmental Hazard. SCOPE Report No. 8. Wiley, Chichester.

Keeler, E.B., and Cretin, S. (1983). Discounting of life-saving and other nonmonetary effects. Management Science 29, 300-306.

Keeney, R.L. and von Winterfeldt, D. (1986). Improving risk communication. Risk Analysis 6, 417-424.

Kerigan, A.T., Pengelly, L.D., Goldsmith, C.H. and Garside, B.K. (1983). The Hamilton study: The effect of environmental factors of the respiratory health of school children. Final Report, June 30, 1983. McMaster University, Urban Air Environment Group, Hamilton.

Kerns, W.D., Donofrio, D.J., and Pavkov, K.L. (1983). The Chronic Effects of Formaldehyde Inhalation in Rats and Mice: A Preliminary Report. In: Formaldehyde Toxicity. (J.E. Gibson, ed.) Hemisphere, Washington, D.C., pp. 111-131.

Klarman, H.E. (1982). The road to cost-effectiveness analysis. Milbank Memorial Fund Quarterly/Health and Society 60, 585-603.

Krewski, D. (1985). Conclusion. In: Interpretation of Negative Epidemiological Evidence for Carcinogenicity (N.J. Wald and R. Doll, eds.). International Agency for Research on Cancer, Lyon, pp. 145-148.

Krewski, D.R. and Van Ryzin, J. (1981). Dose response models for quantal response toxicity data. In: Statistics and Related Topics (M. Csorgo, D. Dawson, J.N.K. Rao & E. Saleh, eds.), North Holland, Amsterdam, pp. 201-231.

Krewski, D. Clayson, D., and McCullough, R.S. (1982). Identification and measurement of Risk. In: Living With Risk: Environmental Risk Management in Canada. (I. Burton, C.D. Fowle & R.S. McCullough, eds). Institute for Environmental Studies, Toronto, pp. 7-23.

Krewski, D., Murdoch, D., and Dewanji, A. (1986). Statistical Modelling and Extrapolation of Carcinogenesis Data. In: Modern Statistical Methods in Chronic Disease Epidemiology. (S.H. Moolgavkar and R.L. Prentice, (eds.) Wiley, New York, 259-282.

Krewski, D., Murdoch, D.J. and Withey, J.R. (1987). The application of pharmacokinetic data in carcinogenic risk assessment. In: Pharmacokinetics in Risk Assessment (Drinking Water and Health, Vol. 8), National Academy Press, Washington, D.C., pp. 441-468.

Krewski, D., Oxman, A. and Torrance, G. (1988). A decision oriented framework for the evaluation of environmental risk management strategies: a case study of lead in gasoline. In: Environmental Risk Assessment: A Textbook of Case Studies (D.J. Paustenbach, ed.). Wiley, New York. In press.

Krewski, D. and Somers, E. (1984). Risk Assessment and the control of toxic chemicals in the environment presented at CMEA/IRPTC/IPCS Seminar on Methodology of Optimal Use of Internationally-Prepared Health Risk Evaluations, Moscow, USSR, November 19-30.

Krewski, D., Brown, C., and Murdoch, D. (1984). Determining "safe" levels of exposure: Safety factors or mathematical models? Fundamental and Applied Toxicology 4, S383-S394.

Krewski, D. (1987). Risk and risk management: issues and approaches. In: Environmental Health Risks: Assessment and Management (S. McColl, ed.). Institute for Risk Research, University of Waterloo Press Waterloo, pp. 29-51.

Krewski, D. and Birkwood, P.L. (1987). Risk assessment and risk management. Risk Abstracts 4, 53-61.

Krewski, D. and Birkwood, P.L. (1988). Regulatory and nonregulatory options for risk management. In: Risk Management: Estimation, Evaluation and Assessment, University of Waterloo Press, Waterloo. In press.

Lave, L.B. (1981). The Strategy of Social Regulation: Decision Frameworks for Policy. Brookings Institution, Washington. D.C.

Lave, L.B. (ed.) (1982). Quantitative Risk Assessment in Regulation. Brookings Institution, Washington, D.C.

Lave, L.B. and Menkes, J. (1985). Managing risk: a joint U.S. - German perspective. Risk Analysis 5, 17-23.

Lave, L.B and Seskin, E.P. (1977). Air-Pollution and Human Mortality. Carnegie-Mellen University, Pittsburgh.

Law Reform Commission of Canada (1986). Policy Implementation, Compliance and Administrative Law. Law Reform Commission of Canada, Ottawa.

Lehman, A J. and Fitzhugh, O.F. (1954). 100-fold margin of safety. Quarterly Bulletin of the Association of Food and Drug Officials of the United States 18, 33-35.

Leiss, W. (1985). The risk management process. Working Paper. Agriculture Canada Ottawa.

Leiss, W. Kline, S., and Jhally, S. (1986). Social Communication in Advertising. Methuen, Toronto.

Leiss, W.L. and Krewski, D. (1988). Risk communication: theory and practice. In: Risk Communication in Canada (W.L. Leiss, ed.). University of Waterloo Press, Waterloo. To appear.

Lepkowski, W.C. (1977). Saccharin ban goes beyond issue of cancer. Chemical and Engineering News 55, 17-21.

Levine, R.J., Dal Croso, R.D., Blunden, P.B., and Battigelli, M.C. (1984). The Effects of Occupational Exposure on the Respiratory Health of West Virginia Morticians. Journal of Occupational Medicine 26, 91-98.

Leviton, A. (1984). Epidemiology: use and utility in the process Fundamental and Applied Toxicology 4, S357-S366.

Lewis, D. and Davis, W. (1986). Joint report of the special envoys on acid rain.

Lichtenstein, S., Slovic, P., Fischhoff, B., Layman, M. and Combs, B. (1978). Judged frequency of lethal events. Journal of Experimental Psychology: Human Learning and Memory 4, 551-578.

Liebling, T., Rosenman, K.D., Pastides, H., Griffith, R.G., and Lemeshow, S. (1984). Cancer Mortality Among Workers Exposed to Formaldehyde. American Journal of Industrial Medicine 5, 423-428.

Logan, A.G., Milne, B.J., Achber, C., Campbell, W.P. and Haynes, R.B. (1981). Cost-effectiveness of a worksite hypertension treatment program. Hypertension 3, 211-218.

Lowrance, W.W. (1976). Of Acceptable Risk: Science and the Determination of Safety. William Kaufman Inc., Los Altos, California.

MacMahon, B. and Pugh, T.F. (1970. Epidemiology: Principles and Methods. Little, Brown & Company, Boston.

Main, D.M., and Hogan, T.J. (1983). Health Effects of Low-Level Exposure to Formaldehyde. Journal of Occupational Medicine 25, 896-900

Maltoni, C., Lefemine, G., Ciliberti, A., Cotti, G., and Carretti, D. (1981). Carcinogenicity bioassays of vinyl chloride monomer: A model of risk assessment on an experimental basis. Environmental Health Perspectives 41, 3-29.

Marchant, C.K. (1984). Environment, Economy, Employment Policy Tool Kit, prepared for discussion purposes Niagara Institute/ Environmental Canada Consultation Workshop.

Maron, D.M. and Ames, B.N. (1983). Revised methods for the salmonella mutagenicity test. Mutation Research 113, 173-215.

Marine, G. and van Allen, J. (1972). Food Pollution: The Violation of our Inner Ecology. Holt, Rhinehart and Winston, New York.

Marsh, G.M. (1982). Proportional Mortality Patterns Among Chemical Plant Workers Exposed to Formaldehyde. British Journal of Industrial Medicine 39, 313-322

Martin, A.E. and Bradley, W.H. (1960). Mortality, fog and atmospheric pollution - am investigation during the winter of 1958-59. Monthly Bulletin of the Ministry of Health, Public Health Service, Laboratory Service 19, 56-72.

Mazur, A. (1984). The journalists and technology: reporting about Love Canada and Three Mile Island. Minerva 22, 45-66.

McLaughlin, S.B., Morris, S.C., Meier, A.K. and Shenk, D.L. (1978). A probabilistic methodology for estimating air pollution health effects from coal-fired power plants. Energy Systems and Policy 2, 287-310.

Meek, M.E., Atkinson, A., and Sitwell, J. (1985). Background Paper on Formaldehyde: Prepared for the WHO Working Group on Indoor Air Quality: Radon and Formaldehyde, Dubrovnik August 26-30.

Meranger, J.C., Khan, T.R., Vairo, C., Jackson, R. and Li, W.C. (1983). International Journal of Environmental Analytical Chemistry 15, 185 212.

Meselson, M. and Russel, K. (1977). Comparisons of carcinogenic and mutagenic potency. In: Origins of Human Cancer, Book C (H.H. Hiatt, J.D. Watson and J.A. Winston, eds.). Cold Spring Harbor Laboratory Cold Spring Harbor, pp. 1473-1481.

Miller, A.B. (1984). The information explosion - the role of the epidemiologist. Cancer Forum 8, 67-75.

Mommsen, S., Aagaard, J. and Sell, A. (1983). A case-control study of female bladder cancer. European Journal of Cancer and Clinical Oncology 19, 725-729.

Morgan, K.T., Patterson, D.L., and Gross, E.A. (1983). Formaldehyde and the Nasal Mucociliary Apparatus. In: Formaldehyde, Toxicology, Epidemiology and Mechanism. (J.J. Clary, J.E. Gibson, and R.S. Waritz, eds.), Marcel Dekker Inc., New York, pp. 193-209.

Morgan, M.C., Morris, S.C., Meier, A.K. and Shenk, D.L. (1978). A probabilistic methodology for estimating air pollution health effects from coal-fired power plants. Energy Systems and Policy 2, 287-310.

Morris, R.B., Higgings, F.B., Jr., Lee, J.A., Newirth, R., and Pervier, J.W. (1975). Engineering and Cost Study of Air Pollution Control for the Petrochemical Industry. Vol. 4: Formaldehyde Manufacture with the Silver Catalyst Process. U.S. Environmental Protection Agency Report No. EPA 450/3-73-006-d. Office of Air Quality Planning and Standards Industrial Studies Branch, Research Triangle Park.

Morrison, A.S. (1979). Use of artificial sweeteners by cancer patients. Journal of the National Cancer Institute 62, 1397-1399.

Morse, D.L., Watson, W.N., Houseworth, J., Witherell, L.E. and Landrigan, P.J. (1979). Exposure of children to lead in drinking water. American Journal of Public Health 69, 711-712.

Müller, P., Raabe, G., and Schumann, D. (1978). Leukoplakia Induced by Repeated Deposition of Formalin in Rabbit Oral Mucosa. Experimentelle Pathologie 16, 36-42.

Munn, R.E. (1981). The Design of Air Quality Monitoring Networks. MacMillan Publishers, London.

Munro, I.C., and Krewski, D.R. (1981), Risk assessment and regulatory decision making. Food and Cosmetics Tox

Munro, I.C. and Krewski, D. (1982). Regulatory considerations in risk management. In: Carcinogens and Mutagens in the Environment, Vol. II: Naturally Occurring Compounds (H. Stich, ed.). CRC Press, Boca Raton, Florida, pp. 443-460.

Munro, I.C., Moodie, C.A., Krewski, D. and Grice, H.C. (1975). A carcinogenicity study of commercial saccharin in the rat. Toxicology and Applied Pharmacology 32, 513-526.

Munro, I.C., Stavric, B. and Lacombe, R. (1974). The current status of saccharin. Toxicity Annual (C.L. Winek, ed.). Marcel Dekker, New York pp. 71-89.

Murasaki, G. and Cohen, S.M. (1983a). Co-carcinogenicity of sodium saccharin and N- 4-(5-nitro-2-furyl)-2-thiazolyl formamide for the urinary bladder. Carcinogenesis 4, 97-99.

Murasaki, G. and Cohen, S.M. (1983b). Effect of sodium saccharin on urinary bladder epithelial regenerative hyperplasia following freeze ulceration. Cancer Research 43 182-187.

Myers, A.W. and Kroetsch, D. (1978). The influence of family incomes on food consumption patterns and nutrient intakes in Canada. Canadian Journal of Public Health 69, 208-221.

Nakanishi, K., Hagiwara, A., Shibata, M., Imada, K., Takematsu, M. and Ito, N. (1980). Dose response of saccharin in induction of urinary bladder hyperplasias in Fisher 344 rats pretreated with N-butyl-N-(4-hydroxybutyl)nitrosamine. Journal of the National Cancer Institute 65, 1005-1010.

NAS: National Academy of Sciences (1978). Saccharin: technical assessment of risks and benefits. National Academy Press, Washington, D.C.

NAS: National Academy of Sciences, Committee on Nitrites, and Alternative Curing Agents in Food (1981). The Health Effects of Nitrate, Nitrite, and N-Nitroso Compounds. National Academy Press. Washington, D.C.

NAS: National Academy of Sciences (1983). Risk Assessment in the Federal Government: Managing the Process. National Academy Press, Washington, D.C.

NAS: National Academy of Sciences, Committee on Identification of Toxic and Potentially Toxic Chemicals for Consideration by the National Toxicology Program (1984). Toxicity Testing: Strategies to Determine Needs and Priorities. National Academy Press, Washington, D.C.

NAS National Academy of Sciences, Complex Mixtures Committee (1988). Methods for the In Vivo Toxicity Testing of Complex Mixtures. National Academy Press, Washington, D.C.

NRC: National Research Council (1968). Non-nutritive sweeteners. Interim report to FDA, Dept. of Health Education and Welfare, Washington, D.C. (unpublished).

NRC: National Research Council. (U.S.) Committee on the Institutional Means for Assessment of Risks to Public Health (1983) isk Assessment in the Federal Government: Managing the Process. National Academy Press, Washington, D.C.

Nemetz, P.N., Vertinsky, I.B. and Vertinsky, P. (1982). Health-risk management in Canada: current practices. In: Living with Risk: Environmental Risk Management in Canada (I. Burton, C.D. Fowle & R.S. McCullough, eds.). Environmental Monograph No. 3, Institute for Environmental Studies, University of Toronto.

Neri, L.C., Mandel, J.S., Hewitt, D. and Jurowski, D. (1976). Chronic obstructive pulmonary disease in two cities of contrasting air quality. Report published for Health and Welfare Canada, 605-7-5920.

Neumann, R.O. (1927). Wird die ausnutzung des nahrungseiureibes durch saccharin beeinfluft? Archiv für Hygiene 96, 265-276.

Newcombe, H.B. (1967). Record linking: The design of efficient systems for linking records into individual and family histories. American Journal of Human Genetics 19, 335-359.

Newell, G.W. (1983). Overview of Formaldehyde. In: Formaldehyde Toxicity, (J.E. Gibson, ed.), Hemisphere, Washington, pp. 3-12.

Nicholls, E.S. and Davies, J.W. (1980). Accidents, Poisoning and Violence in Canada. Health and Welfare Canada, Ottawa.

Nordman, H., Keskinen, H., and Tuppuraninen M. (1984). Asthma Caused by Formaldehyde. 3rd Int. Conf. Indoor Air Quality and Climate. Stockholm. August 20-24, 1984, 3, 127.

NRC: National Research Council, Committee on Chemical Environmental Mutagens (U.S.)(1983b). Identifying and Estimating the Genetic Impace of Chemical Mutagens. National Academy Press, Washington, D.C.

Nutrition Canada (1974). Food Consumption Patterns Report. Health & Welfare Canada, Ottawa.

OECD: Organization for Economic Cooperation and Development, Environmental Committee Group of Economic Experts (1983a) ncentives for Environmental Management: The Recent U.S. Experience. ENV/ECO/CB 83.8, Background Paper - Session 9, International Conference on Environment and Economics, Paris, June 7.

OECD: Environmental Committee Group of Economic Experts (1983b). Use of Economic Instruments in the Environmental Policy in Sweden. ENV/ECO/CB/83.16 Background Paper - Session 9, International Conference on Environment and Economics, Paris, October 17.

OECD: Environmental Committee Group of Economic Experts (1983c). Economic Instruments - Review and Outlook. ENV/ C /CB/83.20 Background Paper - Session 9, International Conference of Environment and Economics, Paris, December 30.

OECD: Environmental Committee Group of Economic xperts (1984a). Economic Instruments - Review and Outlook. ENV/ECO/CB/83 9 Issue Paper - Session 9, International Conference on Environment and Economics, Paris, March 14.

OECD: Environmental Committee Group of Economic Experts (1984b). The Envorcement of Environmental Policies in the United States. ENV/ECO/84.6 Discussion Paper, October 16.

OECD: Organization for Economic Cooperation and Development Environment Committee Group of Economic Experts (1986a). Use of Economic Instruments for Environmental Protection. ENV/ECO/86.16, OECD. Discussion Paper. Paris.

OECD: Organization for Economic Cooperation and Development, Environment Committee Group of Economic Experts (1986b). Economic Instruments for Environmental Protection in Sweden. ENV/ECO/86.13, OECD, Paris.

OECD: Organization for Economic Cooperation and Development, Environment Committee Group of Economic Experts (1986c). Economic Instruments for Environmental Protection in Italy. ENV/ECO/86.11, OECD, Paris.

OECD: Organization for Economic Cooperation and Development, Environment Committee Group of Economic Experts (1986d). Economic Instruments for Environmental Protection in the United States. ENV/ECO/86.14, OECD, Paris.

Office of the Deputy Prime Minister (1986). Background to the Regulatory Reform Strategy. Ottawa.

Office of Science and Technology Policy (1985). Chemical carcinogens: a review of the science and its associated principles. Federal Register 50, 10372-10442.

Office of Technology Assessment (1977). Cancer Testing Technology and Saccharin. Congress of the United States, Washington, D.C., Supt. of Docs.

Office of Technology Assessment (1981). Assessment of Technologies for Determining Cancer Risks from the Environment. U.S. Government Printing Office, Washington, D.C.

Office of Technology Assessment (1984). Acid Rain and Transported Air Pollutants: Implications for Public Policy. U.S. Government Printing Office, Washington, D.C.

O'Hare, M. (1980). Improving the use of information in environmental design making. Environmental Impact Assessment Review 1, 229-250.

Okrent, D. (1980). Comment on societal risk. Science 208, 372-375.

Olsen, J.H. and Dossing, M. (1982). Formaldehyde Induced Symptoms in Day Care Centers. American Industrial Hygiene Association Journal 43, 366-370.

Olsen, J.H., Plough Jensen, S., Hink, M., Faurbo, K., Breum, N.O. and Holler Jensen, O. (1984). Occupational Formaldehyde Exposure and Increased Nasal Cancer Risk in Man. International Journal of Cancer 34, 639-644.

Ontario Ministry of the Environment (1980). The case against acid rain. Ministry of Government Services, Toronto.

O'Riordan (1979). The scope of environmental risk management. Ambio 8, 260-264.

OSHA - Occupational Safety and Health Administration, U.S. Department of Labour (1985). OSHA Proposed Risk on Exposure to Formaldehyde. Chemical Regulation Reporter, Bureau of National Affairs Inc., Washington, D.C.

Otway, H. and Thomas, K. (1982). Reflections on risk perception and policy. Risk Analysis 2, 69-82.

Ozkaynak, H. (1986). Assessing the Health Impacts in Canada from Exposure to Transported Air Pollutants. Report prepared for Health and Welfare Canada, Ottawa.

Pest Management Advisory Board (1986). Working paper on the role of the Pest Management Advisory Board. Ottawa: Agriculture Canada, Ottawa.

Peto, R., Pike, M.C., Bernstein, L., Gold, L.S. and Ames, B.N. (1984). The TD50: a proposed general convention for the numerical description of the carcinogenic potency of chemicals in chronic-exposure animal experiments. Environmental Health Perspectives 58.

Pochin, E.E. (1981). A perspective on risk. In: Health Risk Analysis (C.R. Richmond, P.J. Walsh and E.D. Copenhaver, eds.). Franklin Institute Press. Philadelphia, pp. 15-27.

Purchase, I.F H. (1980). Inter-species comparison of carcinogenic potency. British Journal of Cancer 41, 454-468.

Purchase, I.F.H. Stafford, J.; and Paddle, G.M. (1985). Vinyl chloride: a cancer case study. In: Toxicological Risk Assessment, Vol. II, General Criteria and Case Studies (D.B. Clayson, D. Krewski, and I. Munro, eds.). CRC Press, Boca Raton, Florida, pp. 167-194.

Rader, J. (1974). Irritative Effects of Formaldehyde in Laboratory Halls. Dissertation, University of Würzberg.

Rasmussen, S. (1975). Reactor Safety Study: An Assessment of Accident Risks in U.S. Commercial Nuclear Power Plants. WASH-1400 (NUREG-75/014), U.S. Nuclear Regulatory Commission, Washington, D.C.

Rehn, L. (1985). Vebre blasentumoren bei buchsimarbeitern. Arch. Clin. Chur. 50, 588.

Remsen, I. and Fahlberg, C. (1879-1880). On the oxidation of substitution products of aromatic hydrocarbons IV: On the oxidation of orthotoluenesulfonamide. American Chemical Journal 1, 426-538.

Reschenthaler, G.B. (1979). Occupational Health and Safety in Canada: the Economics and Three Case Studies. Institute for Research on Public Policy, Montreal.

Rhein, R.W. and Marion, L. (1977). The Saccharin Controversy: A Guide for Consumers. Monarch Press, New York.

Richardson, A.W., and Gafni, A. (1983). Treatment of capital costs in evaluating health care programs. Cost and Management 57, 26-30.

Richmond, C.R., Walsh, P.J. and Copenhaver, E.D. (eds.) (1981). Health Risk Analysis. Franklin Institute Press, Philadelphia.

Roberts, R.S., Julian, J.A., Shannon, H.S. and Muir, D.C.F. (1980). Mortality studies in Ontario nickel workers: The INCO/JOHC Study. In: Nickel Toxicology (S.S. Brown and F.W. Sunderman, Jr., eds.). Academic Press. New York, pp. 27-30.

Rodricks, J.V., and Tardiff, R.G. (1984). Conceptual Basis for Risk Assessment. In: Assessment and Management of Chemical Risks. American Chemical Society, Washington, D.C., pp. 3-12.

Rosenbluth, R. (1980). Fair risk and technological change. Engineering Digest, February, pp. 35-38.

Rosenman, K. (1978). Benefits of saccharin: a review. Environmental Research 15, 70-81.

Ross, H.C. (1915). The danger of saccharin. British Medical Journal ii, 552-553.

Rowe, W.D. (1977). An Anatomy of Risk. Wiley-Interscience, New York.

Royal Society (1983). Risk Assessment: A Study Group Report. Royal Society London.

Ruckelshaus, W.D. (1983). Science, risk and public policy. Science 221: 1026-1028.

Sackett, D.L., and Torrance, G.W. (1978). The utility of different health states as perceived by the general public. Journal of Chronic Diseases 31, 697-704.

Salem, H. and Cullumbine, H. (1960). Inhalation Toxicities of Some Aldehydes. Toxicology and Applied Pharmacology 2, 183-187.

Salter, L. (1987). Mandated Science: Science and Scientists in the Making of Public Policy. D. Reidel, Amsterdam.

Salter, L. (1985). Observations on the politics of assessment: the captan case. Canadian Public Policy II, 64-76.

Salter, L. (1985). Science and peer review - The Canadian Standard-setting experience. Science, Technology and Human Values 53, 37-46.

Sardinas, A.V., Most, R.S., Giulietti, M.A. and Honchar, P. (1979). Health Effects Associated with Urea-Formaldehyde Foam Connecticut. Journal of Environmental Health

Sawyer, C., Peto, R., Bernstein, L. and Pike, M.C. (1984). Calculation of carcinogenic potency from long-term animal carcinogenesis experiments. Biometrics 40, 27-40.

Sax, N.I. (1981). Cancer Causing Chemicals. Van Nostrand Reinhold, New York.

Schimmel, H. and Greenburg, L. (1972). A study of the relation of pollution to mortality in New York City. Journal of the Air Pollution Control Association 22, 607-616.

Schmahl, D., Habs, M. and Ivankovic, S. (1978). Carcinogeneis of n-nitrosodiethyamine (DENA) in chickens and domestic cats. International Journal of Cancer 22, 552-557.

Schuck, E.A., Stephens, E.R., and Middleton, J.T. (1966). Eye irritation response at low concentrations of irritants. Archives of Environmental Health 13, 570-575.

Scott, J.A. (1963). The London fog of December, 1962. Medical Officer 109, 250.

Sellakumar, A.R., Snyder, C.A., Solomon, J.J., and Albert, R.E. (1985). Carcinogenicity of Formaldehyde and Hydrogen Chloride in Rats. Toxicology and Applied Pharmacology 81, 401-406.

Shannon, C.E., and Weaver, W. (1949). The Mathematical Theory of Communication. University of Illinois Press, Urbana.

Sharatt, M.T. and Cerny, F.J. (1979). Pulmonary function and health status of chilfren in two cities of different air quality: a pilot study. Archives of Environmental Health 34, 114-119.

Sharlin, H.I. (1986). EDB: a case study in communicating risk. Risk Analysis 6, 61-68.

Shenk, H.H., Heimann, H., Clayton, G.D. Gafafer, W.M. and Wexler, H. (1949). Air pollution in Donora, Pa.: Epidemiology of the unusual smog episode of October 1948 - Preliminary report. Public Health Bulletin No. 306, Washington, D.C.

Shrader-Frechette, K.S. (1985). Risk Analysis and Scientific Method. D. Reidel Publishing, Dordrecht.

Shubik, P. (1985). Saccharin/cyclamates: laboratory evidence. In Interpretation of Negative Epidemiological Evidence for Carcinogenicity (N.J. Wald and R. Doll, eds.). International Agency for Research on Cancer, Lyon, pp. 125-128.

Silverman, D.T., Hoover, R.N. and Swanson, G.M. (1983). Artificial sweeteners and lower urinary tract cancer: hospital vs. population controls. American Journal of Epidemiology 117, 326-334.

Slovic, P. (1986). Informing and educating the public about risk. Risk Analysis 6, 403-415.

Slovic, B., Fischhoff, B. and Lichtenstein, S. (1979). Rating the risks. Environment 21, 14-20.

Slovic, P., Fischhoff, B. and Lichtenstein, S. (1982). Why study risk perception? Risk Analysis 2, 83-94.

Society for Risk Analysis (1986). Committee on Definitions, "Draft Paper on Risk Definitions" and "Draft Glossary," June 1986.

Somers, E. (1984). Risk estimation for environmental chemicals as a basis for decision making. Regulatory Toxicology and Pharmacology 4, 99-106.

Somers, E., and MacDonald, G.E. (1983). Regulatory control of chemicals: the Canadian experience. In: Safety Evaluation and Regulation of Chemicals (F. Homberger, ed.). Karger, Basel. 16-25.

Spangler, M.B. (1987). Policy issues related to worst case risk analyses and the establishment of acceptable standards of de minimus risk. In:, Uncertainty in Risk Assessment, Risk Management and Decision Making (V.T. Covello, L.B. Lave, A. Moghizsi & V.R.R. Upputuri). Plenum Press, New York, pp. 1-26.

Spence, A.M., and Weitzman, M.L. (1978). Regulatory Strategies for Pollution Control. In: Approaches to Controlling Air Pollution (A.F. Friedlaender, ed.). MIT Press, Cambridge, 199-219.

Spitzer, W.O., Shenker, S.C. and Hill, G.B (1982). Cancer in a Montreal suburb: The investigation of a nonepidemic. Canadian Medical Association Journal 127, 971-974.

Squire, R.A. (1981). Ranking animal carcinogens: A proposed regulatory approach. Science 214, 877-880.

Squire, R.A., and Cameron, L.L. (1984). An Analysis of Potential Carcinogenic Risk from Formaldehyde. Regulatory Toxicology and Pharmacology 4, 107-129.

Starr, C. (1985). Risk Management, Assessment, and Acceptability. Risk Analysis 5, 97-102.

Starr, C. (1969). Social benefit versus technological risk: what is our society willing to pay for safety? Science 165, 1232-1238.

Starr, T.B. (1986). Testimony Before OSHA on Proposed Formaldehyde Rules. CIIT Activities, 6, 3-4.

Starr, T.B. and Buck, R.D. (1984). The Importance of Delivered Dose in Estimating Low-Dose Cancer Risk from Inhalation Exposure to Formaldehyde. Fundamental and Applied Toxicology 4, 740-753.

Starr, T.B., Gibson, J.E., Barrow, C.S Boreiko, C.J., Heck, H.d'A., Levine, R.J. Morgan, K.T., and Swenberg, J.A. (1985). Estimating Human Cancer Risk from Formaldehyde: Critical Issues. Formaldehyde: Analytical Chemistry and Toxicology. V. Turoski (ed.), American Chemical Society, Wahington, D.C. 299-333.

Statistics Canada (1980). Directory of Health Division Information. Supply & Services Canada, Ottawa.

Statistics Canada (1983). Hospital Annual Statistics. Cat. No. 83-232. Supply and Services Canada, Ottawa.

Statistics Canada (1979). Life Tables, Canada and Provinces, 1975-77. Statistics Canada Ottawa.

Stayner, L. Smith, A B., Reeve, G., Blade, L., Elliott, L., Keenlyside, R., and Halperin, W., (1985). Proportionate Mortality Study of Workers in the Garment Industry Exposed to Formaldehyde. American Journal of Industrial Medicine 1, 229-240.

Sumner, M.T. (1980a). Benefit-cost analysis in Canadian practice. Canadian Public Policy 6, 389-393.

Sumner, M.T. (1980b). Comments on the public-sector discount rate: response to Jenkins. Canadian Public Policy 6, 648-650.

Swenberg, J.A., Gross, E.A., Morgan, K.T., Chang, J.C.F. and Barrow, C.S. (1982). Effects of Formaldehyde Concentration on Deposition and Cell Proliferation (Abstract. Proceedings of the American Association for Cancer Research 23, 55.

Swenberg, J.A., Barrow, C.S., Boreiko, C.J., Heck, H.d'A., Levine, R.J., Morgan, K.T., and Starr, T.B. (1983). Non-linear Biological Responses to Formaldehyde and their Implications for Carcinogenic Risk Assessment. Carcinogenesis 4, 945-952.

Swenberg, J.A., Heck, H.d'A., Morgan, K.T., and Starr, T.B. (1985). A Scientific Approach to Formaldehyde Risk Assessment. Banbury Report 19: Risk Quantitation and Regulatory Policy, Cold Spring Harbor Laboratory, 255-267.

Symposium on Public Policy Towards Risk (1982). Policy Studies Review. Hadden, S., ed., 1, 651-747.

Tannenbaum, S.R. & Skipper, P.L. (1984). Biological aspects to the evaluation of risk: dosimetry of carcinogens in man. Fundamental and Applied Toxicology 4. S367-S373.

Tegeris, A.S. (ed.)(1984). Toxicology Laboratory Design and Management for the 80's and Beyond. S. Karger, Basel.

Thomson, E.J., Shackleton, S., and Harrington, J.M. (1984). Chromosome aberrations and sister-chromatid exchange frequencies in pathology staff occupationally exposed to formaldehyde. Mutation Research 141, 89-93.

Thompson, M.S. (1982). Decision Analysis for Program Evaluation. Ballinger Publishing, Cambridge, Massachusetts.

Thun, M.J., Lakat, M.F., and Altman, R. (1982). New Jersey Urea-Formaldehyde Foam Insulation Study. Mimeographed Report, New Jersey State Health Department.

Tobe, M. et al. Studies of the Inhalation Toxicity of Formaldehyde. National Sanitary and Medical Laboratory Service, Japan, in press.

Tobin, R.S. (1984). Water treatment for the home or cottage. Canadian Journal of Public Health 75: 79-82.

Torrance, G.W , Thomas, W.H. and Sackett D.L. (1972). A utility maximization model for evaluation of health care programs. Health Services Research 7, 118-133.

Torrance, G. and Krewski, D. (1986). Economic evaluation of toxic chemical control programs. Toxic Substances Journal 7, 53-71.

Torrance, G.W. and Oxman, A.D. (1986). Cross-sector information needs for environmental risk management. In: Information Needs for Environmental Risk Management in Canada (C.D. Fowles. ed.). Environmental Monograph No. 5, Institute for Environmental Studies, University of Toronto.

Treasury Board of Canada (1979). Administrative Policy Manual (Chapter 490 Appendix E). Supply and Services Canada, Ottawa.

Truhaut, R. (1980). The problem of thresholds for chemical carcinogens - its importance in industrial hygiene, especially in the field of permissible limits for occupational exposure. American Industrial Hygiene Association Journal 41, 685-692.

Tuazon, E.C., Graham, R.A. Winer, A.M., Easton, R.R., Pitts, J.N., and Hanst, P.L. (1978). A Kilometer Pathlength Fourier-Transform Infrared System for the Study of Trace Pollutants in Ambient and Synthetic Atmospheres. Atmospheric Environment 12, 865-875.

Turner, H.E. (1988). Case study on acid rain. In: Information Needs for Risk Assessment. (A.P. Grima, C.D. Fowle & R.E. Munn, eds.). Institute for Environmental Studies, Univ. Toronto, EM-8.

Tversky, A. and Kahneman, D. (1981). The framing of decisions and the psychology of choice. Science 211, 453-458.

Twort, C.C. & Twort, J.M (1933). Suggested methods for the standardization of the carcinogenic activity of different agents for the skin of mice. American Journal of Cancer 17, 293-320.

Twort, C.C. and Twort, J.M. (1930). The relative potency of carcinogenic tars and oils. Journal of Hygiene 29, 373-379.

United States Department of Health and Human Services, Committee to Coordinate Environmental and elated Programs (1985). Risk Assessment and Risk Management of Toxic Substances. Department of Health and Human Services, Washington, D.C.

United States Environmental Protection Agency (1984). Risk Assessment and Management: Framework for Decision Making. Environmental Protection Agency, Washington, D.C.

United States Food and Drug Administration (1977). Annual Report. U.S. FDA, Washington, D.C., pp. 40-43.

Vanderlende, R., Kok, T.J., Reig, R.P., Quanjer, P.H. and chouten, J.P. (1981). Decreases in VC and FEB1 with time-indicators for effects f of smoking and air pollution. Clinical Respiratory Physiology Bulletin 17, 775-792.

Van Horn, A.J. & Wilson, R. (1977). Factors Influencing the Public Perception of Risks to Health and Safety: A Brief Summary. Department of Energy, Washington, D.C.

Vertinsky, I. and Vertinsky, P. (1982) Communicating environmental health risk assessment and other risk information: analysis of strategies. In: Risk: A Seminar Series (H. Kunreuther, ed.). International Institute for Applied Systems Analysis Laxenburg, Austria, pp. 421-482.

Volkman, Von R. (1874). Ueber theer-and ruskrebs. Klin. Wschr. 11, 218.

Wald, N.J. and Doll, R. (eds.)(1988). Interpretation of Negative Epidemiological Evidence for Carcinogenicity. International Agency for Research on Cancer, Lyon.

Waller, R.E. and Commin, B.T. (1966). Episodes of high pollution in London, 1952-1966. Proc. Intern. Clean Air Conf., Part I. National Society for Clean Air, p. 288.

Walrath, J. and Fraumeni, J.F. (1983). Mortality Patterns Among Embalmers. International Journal of Cancer 31, 407-411.

Ware, J.H., Ferris, B.G., Jr., Dockery, D.W., Spengler, J.D., Stram, D.O. and Speizer (1986). Effects of ambient sulfur oxides and suspended particulates on rjespiratory health of preadolescent children. American Review of Respiratory Disease 133, 834-842.

Warner, K.E. and Luce, B.R. (1982). Cost-Benefit and Cost-Effectiveness Analysis in Health Care: Principles, Practice and Potential. Health Administration Press, Ann Arbor, Michigan.

Watannbe, F. Matsunage, T. Soejima, T. and Iwata, Y. (1954). Study on aldehyde carcinogenicity. Communication I. Experimentally induced rat sarcomas by repeated injection of formalin. Gann 45, 451-452.

Weber-Tschopp, A., Fischer, T., and Grandjean, E. (1977). Reizwirkungen des Formaldehyd auf den Menschin. International Archives of Occupational and Environmental Health 39, 207-218.

Weil, C.S. (1972). Statistics vs. safety factors and scientific judgement in the evaluation of safety for man. Toxicology and Applied Pharmacology 21, 454-463.

Weinstein, M.C. and Stason, W.B. (1977). Foundations of cost-effectiveness analysis for health and medical practices. New England Journal of Medicine 296, 716-721.

Weinstein, M.C. (1981). Economic assessments of medical practices and technologies. Medical Decision Making 1, 309-330.

Weisburger, J.H. and Williams, G.M. (1981). Carcinogen testing: Current problems and new approaches. Science 214, 401-407.

Whyte, A (1984). Background Notes in Risk Perception, Man and the Biosphere Programme. United National Educational, Scientific and Cultural Organization, Paris.

Whyte, A.V., and Burton, I. (eds) (1980). Environmental Risk Assessment. SCOPE Report No. 15. Wiley, Chichester.

Whyte, A. and Burton, I. (1982). Perception of risks in Canada. In: Living with Risk: Environmental Risk Management in Canada (I. Bruton, C.D. Fowle and R.S. McCullough, eds.). Institute for Environmental Studies. Toronto, pp. 39-69.

WHO: World Health Organization (1976). Environmental Health Criteria I, Mercury. World Health Organization, Geneva.

WHO: World Health Organization (1984). Health Promotion: A Discussion Document on the Concept and Principles. World Health Organization. Regional Office for Europe, Geneva.

Wigle, D.T., Mao, Y., Semenciw, R. and Morrison, H.I. (1986). Cancer patterns in Canada. Canadian Medical Association Journal 134, 231-235.

Wilkins, R., and Adams, O.B. (1978). Heathfulness of Life. Institute for Research on Public Policy, Montreal.

Williamson, O.E. (1981). Saccharin: an economist's view. In: The Scientific Basis of Health and Safety Regulations (R.W. Crandall and L.B. Lave, eds.). Brookings Institute, Washington, D.C., pp. 131-151.

Wilson, R. (1979). Analyzing the daily risks of life. Technology Review 81, 41-46.

Wilson, R. (1984). Risks and their acceptability - commentary. Science, Technology and Human Values 47, 11-22.

Withey, J. (1978). The role of pharmacokinetics in the design and conduct of chronic exposure studies. In: Industrial and Environmental Xenobiotics. (J.R. Fouts & I. Gut, eds.). Excerpta Medica, Amsterdam, pp. 190-195.

Wong, O. (1983). An Epidemiologic Mortality Study of a Cohort of Chemical Workers Potentially Exposed to Formaldehyde, with a Discussion on SMR and PMR. In: Formaldehyde Toxicology. (J. Gibson, ed.). Hemisphere, Washington, 256-272.

Woodbury, M.A., and Zenz. C. (1983). Formaldehyde in the Home Environment: Prenatal and Infant Exposures. In: Formaldehyde Toxicity (J E. Gibson. ed.), Hemisphere, Washington, pp. 203-211.

Workmen's Compensation Act. Revised Statutes of Ontario 1980. Chapter 539 pp. 1203-1266.

World Health Organization (1985). Risk Management in Chemical Safety. World Health Organization, European Regional Program on Chemical Safety, Geneva.

Wrage, P. (1985). Economic Incentives and Pollution Control Strategy. Report prepared for External Strategies Directorate, Environment Canada, Ottawa.

Wylie, E. (1984) Environment Canada's Mandate. Minister of Supply and Services Ottawa.