Application of Health Risk Assessment Techniques to Microbial Monitoring Data

B Genthe • N Rodda

Report to the Water Research Commission
by the
Division of Water, Environment and Forestry Technology
CSIR

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EXECUTIVE SUMMARY

Background and motivation:

The task of formulating water quality criteria and guidelines for the protection of public health is complicated by the difficulties of relating levels of hazardous agents to health effects in the population. Similar difficulty is experienced in the interpretation of monitoring studies, which provide detailed surveys of water quality but which cannot easily be linked to health effects in a quantitative manner. Epidemiological studies are time- and labour-intensive and extremely costly. They are also limited to overt, statistically detectable results, hence have little predictive power and are limited in sensitivity by size of the study population and the extent of the health impact. The level of sensitivity which can be detected with the aid of epidemiological studies is often several orders of magnitude less than desired, especially with regard to guideline development for the protection of public health.

Quantitative health risk assessment provides a means of estimating the probability of adverse effects associated with measured or estimated levels of hazardous agents, and a tool for predicting the extent of potential or probable health effects. Levels of health effects which would not be detectable in an epidemiological study can be predicted by risk assessment. The level of health effect detectable need not be statistically significant, however the risk assessment process facilitates the quantification of health effects in an exposed population.

The use of microbial risk assessment in the development of water quality guidelines in South Africa holds many potential benefits. It would permit guide limits to be based on community perceptions of acceptable risk and on cost-benefit considerations, rather than on practicability of monitoring as at present. However, the application of risk assessment techniques to microbial water evaluation is still relatively new. All implementations of microbial risk assessment to date originate in the USA where different monitoring and detection methods, disease-reporting and cost factors apply. It is not clear whether microbial risk assessment techniques developed in the USA would be directly applicable to South Africa. The aim of present study was to address this question.

Aims and objectives as specified in contract:

The aim of the study was to investigate the usefulness of microbial risk assessment techniques in the South African context.

The objectives as stated in the proposal are:

* to assess the usefulness of data collected by current microbial monitoring programmes for quantitative analysis of the associated health risks;

* to estimate the minimum levels of risk detectable using the volumes of water currently analysed

* to evaluate the usefulness and implications of health risk assessment for the formulation of microbial water quality guidelines

Methodology used:

The applicability of microbial risk assessment techniques in a South African context was evaluated by means of a number of case studies based on microbial monitoring data drawn from surveys conducted in South Africa in recent years. The case studies were used to assess the usefulness of data collected by representative microbial monitoring programmes for quantitative analyses of the associated health risks; to estimate the minimum levels of risk detectable using the volumes of water typically analysed; and to evaluate the capability of risk assessment techniques to indicate desirable microbial water quality in the context of the monitoring methods used.

Case studies considered the following exposure scenarios: drinking water consumption; ingestion of water during swimming; and ingestion of polluted shellfish. Organisms considered in the case studies were enteric viruses, since these are pathogenic microorganisms as opposed to indicator organisms, and can therefore be used in risk assessment models.

Brief summary of results and conclusions reached:

With respect to the objectives of this study, microbial risk assessment was found to be of potential benefit in the South African context to the description of microbial water quality impacts and to the development of guidelines in the future. However, certain limitations were found, namely:

- the adoption of a risk assessment approach would hold implications for the manner in which monitoring data are collected. Detection of low levels of risk was hampered by high detection limits and relatively small volumes of water sampled. The sampling and concentration of large volumes of water (at least 100ℓ) would be the most important factor in facilitating the detection of low risks.

- the availability of better exposure data would decrease the uncertainty associated with risk assessments. A drawback of current microbial risk assessment models is that they apply only to exposure by ingestion. Possible future development of models which address other exposure routes explicitly, would allow microbial risk assessment to be implemented on a wider basis.

- the choice of pathogen for which risk is estimated when a more general group is monitored (*e.g.* enteric viruses) is an additional source of uncertainty which plays a significant role in the uncertainty in the risk calculation

- standardisation of methods used in estimations of longer term risks is needed to ensure that values can be compared across studies, both nationally and internationally.

Presently, the application of microbial risk assessment techniques in South Africa is hampered by inadequacies in the data available. Considerations of what constitutes an acceptable risk is necessary and desirable in the South African context. However, this would only provide a partial solution, as any reasonable acceptable risk guideline would still require more sensitive data.

Results represented here and reported by other investigators using a range of techniques indicate that much historical water quality monitoring aimed at protection of public health may have missed the mark. While monitoring has been extremely useful in controlling water-borne disease outbreaks, it is possible that insufficient protection is provided against low levels of relatively low grade gastrointestinal and other complaints associated with water-borne pathogens.

Extent to which objectives were reached:

All objectives of the study, as described above, were reached. The usefulness of data collected for risk assessment was assessed; minimum levels of risk detectable using volumes of water currently analysed were estimated; and the usefulness and implications of health risk assessment for the formulation of microbial water quality guidelines was evaluated.

a) *the usefulness of data collected by current microbial monitoring programmes was assessed for quantitative analysis of the associated health risks.*

Minimum risks detectable under each of the case studies presented were almost uniformly above acceptable average yearly risks recommended by the US EPA (Federal Register, 1989) and microbial risk experts. A consequence of this was that it was not possible to compare estimated risks with existing suggested guidelines for acceptable risk. This indicates that under monitoring practices represented in this investigation limitations were placed on detection of levels of risk which would be useful in interpreting monitoring water quality in terms of risk-based guidelines or in preparing water quality guidelines.

b) *to estimate the minimum levels of risk detectable using the volumes of water currently analysed*

One of the major limitations imposed by monitoring methods represented in this study was the inability to detect low levels of risk suitable for comparison to risk-based guidelines. Monitoring of relatively low volumes (10ℓ in the case of water samples) restricted the risk range which could be detected and resulted in numerous non-detect results. Monitoring of larger volumes, at least 100ℓ, is necessary to detect risk levels comparable to that recommended by the EPA. It may be possible to overcome the problem in

the short-term by exploiting the relatively high sampling frequency practised at present (approximately twice per month) to estimate risks over a shorter time frame (*e.g.* monthly). These risks may be compared with the EPA recommended yearly risk, or with risk recommendations which may be developed for South Africa. However, to gain maximum benefit from the application of risk assessment methods to microbial monitoring data, concentration of large volumes of water is imperative and of primary importance.

c) *to evaluate the usefulness and implications of health risk assessment for the formulation of microbial water quality guidelines*

The most significant restrictions in the application of risk-based guidelines identified in this study were:
insensitivity to low levels of risk;
uncertainty of assessments which limit the confidence of the estimated risks to guidelines, namely:

- choice of pathogen for which risk is calculated when a general group is monitored,
- lack of reliable exposure data,
- method of calculating longer term risks, and
- variability in monitoring data

As a result of the conflict between data requirements for risk assessment and the need for practical monitoring programmes, it is likely that microbial risk assessment will not be used in association with generalised monitoring studies. Rather, risk assessment may be utilised in studies targeted specifically for guideline development or at establishing quantitative risks associated with specific pathogens. The greatest benefit will be obtained from microbial risk assessment if such studies are planned rigorously in terms of exposure data, sample volumes and sample numbers to provide a balance between limits of detectable risk and a representative distribution of exposure estimates.

Technology transfer and recommendations for further research:

The findings from this study may be useful in assisting government departments such as the Department of Water Affairs and Forestry to apply risk assessment in the development of guidelines for microbial water quality.

Further research is required to address the numerous issues raised in this study. Risk assessment should be applied to additional available monitoring data including pathogens other than enteric viruses, in addition to examining other exposure routes. Calculated risk estimates need to be compared to water-related disease reporting in this country and it is recommended that the risk assessment procedure be repeated with new data available, examining larger volumes of water (*i.e.* 100ℓ).

1. AIM:

The aim of the study was to investigate the usefulness of microbial risk assessment techniques in the South African context.

2. OBJECTIVES:

* to assess the usefulness of data collected by current microbial monitoring programmes for quantitative analysis of the associated health risks;

* to estimate the minimum levels of risk detectable using the volumes of water currently analysed

* to evaluate the usefulness and implications of health risk assessment for the formulation of microbial water quality guidelines

3. INTRODUCTION:

The task of formulating water quality criteria and guidelines for the protection of public health is complicated by the difficulties of relating levels of hazardous agents to health effects in the population. Similar difficulty is experienced in the interpretation of monitoring studies, which provide detailed surveys of water quality but which cannot easily be linked to health effects in a quantitative manner. Such questions can be, and are, addressed by epidemiological studies. However, these studies are time- and labour-intensive and extremely costly. Results cannot be obtained rapidly and resources are generally insufficient to link monitoring surveys and epidemiological studies on a routine basis. Furthermore, epidemiological investigations are limited to overt, statistically detectable results, hence have little predictive power and are limited in sensitivity by size of the study population and the extent of the health impact. The level of sensitivity which can be detected in epidemiological studies is often several orders of magnitude less than desired, especially with regard to guideline development for the protection of public health.

Quantitative health risk assessment provides a means of estimating the probability of adverse effects associated with measured or estimated levels of hazardous agents, and a tool for predicting the extent of potential or probable health effects. Levels of health effects which would not be detectable in an epidemiological study can be predicted by risk assessment. The level of health effect detectable need not be statistically significant, however, the risk assessment process facilitates the quantification of health effects in an exposed population. A health risk assessment of a hazardous agent attempts to describe the agent and the associated health hazards, characterise the nature and extent of exposure of humans to the agent, and assess the response of exposed humans. Exposure and response are described by means of mathematical models, which are used to predict the response of the exposed human population to environmental concentrations of the same agent (NRC, 1986; Cotruvo, 1987).

Health risk assessment is widely used in the development of guidelines and standards for chemical water contaminants in the Unites States (US EPA, 1987). It is also used in Europe and by international organisations such as the World Health Organisation (WHO, 1984; IPCS, 1990). The application of risk assessment techniques to microbial water pollutants is, however, less well developed. It has been examined and developed in concept (Haas, 1983a,b; Cooper *et al.*, 1984; US EPA, 1988; US EPA, 1989), and implemented on a limited basis (Haas, 1983a; Gerba and Haas, 1988; Asano and Sakaji, 1990; Rose and Gerba, 1991a,b; Rose *et al.*, 1991), but is generally recognised as still being in the experimental stage.

The use of microbial risk assessment in the development of water quality guidelines in South Africa holds many potential benefits. It would permit guide limits to be based on community perceptions of acceptable risk and on cost-benefit considerations, rather than on practicability of monitoring as at present. However, all implementations of microbial risk assessment listed above originate in the USA where different monitoring and detection methods, disease-reporting and cost factors apply. It is not clear whether microbial risk assessment techniques developed in the USA would be directly applicable to South Africa. The aim of present study was to address this question.

The applicability of microbial risk assessment techniques in a South African context was evaluated by means of a number of case studies based on microbial monitoring data drawn from surveys conducted in South Africa in recent years. The case studies were used to assess the usefulness of data collected by representative microbial monitoring programmes for quantitative analyses of the associated health risks; to estimate the minimum levels of risk detectable using the volumes of water typically analysed; and to evaluate the capability of risk assessment techniques to indicate desirable microbial water quality in the context of the monitoring methods used. This information can be utilised by legislative authorities to define desirable water quality on a scientific basis and to protect public health.

3.1 RISK ASSESSMENT OF HUMAN HEALTH HAZARDS IN WATER

3.1.1 Risk assessment, risk management and water quality management

The transmission of disease by polluted water has long been recognised. Among the most common water-related infectious diseases are gastroenteritis, amoebiasis, salmonellosis, dysentery, cholera, typhoid fever and hepatitis A (Craun, 1986). It has been estimated that 50 000 people die daily worldwide as a result of water-related disease (Schalekamp, 1990). Improvements in wastewater disposal, protection of water sources, and the treatment of water supplies has reduced, but not eliminated, the incidence of these diseases in developed countries (Craun, 1986). At the same time, concern is mounting regarding the health effects of rapidly increasing chemical pollution of water bodies as a result of industrial development and agriculture (Craun, 1986; Williams, 1980). Health risks associated with polluted water include infectious diseases, acute or chronic chemical toxicity, and carcinogenicity.

Polluted water not only holds the potential to cause human suffering, but also results in economic loss. An assessment of the cost of water-related enteric illness in developing regions, based on Indian conditions, estimated an average cost of approximately $1 700 dollars per 100 people per annum, with an associated estimate of approximately $1 500 days lost per 100 people per annum (Verma and Srivastava, 1990). In developed areas, costs of illness may be considerably higher. This represents an under-estimate of current costs since figures have not been adjusted for inflation. Costs of illness resulting from chemical water pollution could be expected to be higher, due to the often long-term and irreversible nature of such diseases. The control of water pollution and the management of water quality is therefore both an economic and a social responsibility.

Decisions taken with respect to the health implications of water use are derived, either explicitly or implicitly, by three processes, *viz.* (1) the identification of a health risk to humans, (2) the assessment of the likelihood of the risk and of its health significance, and (3) the consideration of the feasibility, cost and effectiveness associated with available options for reducing or controlling the risk (Cotruvo, 1987; Deisler, 1987). This encompasses the functions of health risk assessment (1 and 2) and risk management (3).

As it is the task of water quality management to minimise the spread of disease as a result of polluted water, the heaviest users of health risk assessment techniques in relation to water quality are environmental and public health authorities, whose task it is to ensure the provision of safe water supplies for drinking and other purposes (Cotruvo, 1987). Safety relates to the probability of adverse effects. The protection of water supplies therefore requires some estimate of the probability, or risk, of adverse health effects associated with their use.

Health risk assessment attempts to define the most likely health effects under given conditions of exposure, and to estimate the probability of manifestation of these effects in the exposed population (Hutzler and Boyle, 1982). Fundamental questions addressed relate to the criteria which are used to identify substances which are likely to increase the risk of disease in the exposed population, to the mechanisms of action of such substances and the types of effects caused, and to the magnitude of the risk posed by episodic or chronic exposure (Hallenbeck and Cunningham, 1986; Cotruvo, 1987). In the context of water quality, the goal of health risk assessment is to define levels of microbiological and chemical pollutants in water which can be considered acceptable in terms of the human health hazard they pose.

Risk management, carried out by risk managers such as environmental and public health authorities, integrates the recommendations derived from risk assessment with an evaluation of cost-effectiveness, technical factors and policy, business and social considerations to identify management options which are both feasible and acceptable to the population. The interaction of risk assessment and risk management allows for the correlation of water quality with human health hazards, and for the selection of the most efficient methods for avoiding or minimising negative health effects which may be associated with various aspects of water use

(Deisler, 1987).

In the context of water quality management, decisions are the outcome of risk management, supported explicitly or implicitly by some form of risk assessment, which is conducted by the risk assessor. In conducting a risk assessment, the separation of the roles of the risk assessor and the risk manager is essential to avoid the possibility of risk management concerns jeopardising the objectivity of the risk assessment. The risk assessor supports the risk manager by presenting his findings with a clear statement of all assumptions and uncertainties. It is then the task of the risk manager to evaluate the health hazard in terms of the available control options and of the economic and social benefits gained or lost with each option (Cotruvo, 1987; Deisler, 1987; Lave, 1987). Thus risk assessment strives to determine the extent of hazard and the goals of controls imposed by the risk manager (Cotruvo, 1987).

Water quality management has often been unconsciously equated with water quality analysis. Levels of common water contaminants have been determined with little attempt at further evaluation of the trends and implications of analytical results. In such instances only the first of the three driving processes in policy formulation, identified above, has been carried through explicitly and systematically. Consequently, actual risks due to the presence of water contaminants may be over- or underestimated. This may result in risk management action which is not cost-effective, or more seriously, which does not provide adequate protection of public health.

3.1.2 Health hazards associated with polluted water

The safety of water sources is impacted by two clearly distinguishable classes of health hazards: microorganisms and chemicals. The former can cause infectious diseases, while the latter can have carcinogenic, mutagenic, teratogenic or acute and chronic toxic effects.

Microbiological contamination of water is the largest and most immediate health hazard. Among the most common water-related diseases are gastroenteritis, amoebiasis, salmonellosis, dysentery, cholera, typhoid fever, and hepatitis A, involving bacterial, viral and parasitic contamination of water (Craun, 1986). The primary goal of water quality management, from a health perspective, is therefore to ensure that consumers are not exposed to infective doses of pathogens along a route of exposure that is likely to cause infection. Improvements in wastewater disposal, protection of water sources and treatment of water supplies has greatly reduced the incidence of these diseases in developing countries (Craun, 1986). Microbiological health risks remain associated with many aspects of water use, from drinking water in developing countries (Craun, 1986; Lloyd *et al.*, 1989) to irrigation reuse of treated wastewater (Fattal *et al.*, 1986; Rose, 1986) and recreational water contact (Cheung *et al.*, 1991; Grabow, 1991; Van Olphen *et al.*, 1991). In many such situations water quality criteria are either lacking or inappropriate. Microbial water quality is predominantly managed on the basis of

levels of indicator organisms rather than pathogens due to the large range of potential pathogens which may be present in water and due to the expensive, time consuming and complex nature of most pathogen detection methods. Coliform (total and faecal) bacteria are the most frequently used indicators of the general sanitary condition of water.

The application of risk assessment to problems relating to the microbiological quality of water has been limited in the past, due to a lack of data on pathogen levels and of suitable microbial risk assessment techniques. Available work concentrates predominantly on wastewater and sludge disposal or reuse (Hutzler and Boyle, 1982; Haas, 1983a; Gerba and Goyal, 1988; US EPA, 1988; US EPA, 1989; Asano and Sakaji, 1990; Rose and Gerba, 1991a), and on drinking water (Cooper *et al.*, 1984; Gerba and Haas, 1988; Rose and Gerba, 1991b; Rose *et al.*, 1991).

The health effects caused by microbial contaminants of water, and those caused by chemical pollutants differ widely. Microbiological contamination usually results in immediate and often widespread ill health. Chemicals, particularly those which bioaccumulate, generally cause irreversible, long-term health effects after prolonged periods of exposure (WHO, 1987). This difference in the types of health effects caused is likely to mean that risk assessments of microbial and chemical water pollutants will be initiated as a result of concern for different aspects of public health.

All factors determining exposure, availability and type of health effect influence the hazards associated with chemical and microbiological pollutants. This is not reflected by merely monitoring levels of contaminants in water. Health risk assessment provides one tool for estimating the extent of such interactions and their outcome in terms of human health.

3.1.3 The health risk assessment process

The process of risk assessment has been variously described (Hutzler and Boyle, 1982; NRC, 1983; Deisler, 1986; Hallenbeck and Cunningham, 1986; Cotruvo, 1987; IPCS, 1990). The same fundamental concepts incorporated in most risk assessment protocols are included in that formulated by the United States Office of Science and Technology, and adopted by the United States Environmental Protection Agency (US EPA) (Deisler, 1987; Mentzel, 1987; US EPA, 1987). This protocol was originally intended to accommodate only carcinogen assessment. However, current trends favour the application of similar procedures to chemical toxicants as well as to carcinogens (Barnes and Dourson, 1988), and to the assessment of microbiological hazards (US EPA, 1989; Asano and Sakaji, 1990). The risk assessment process, as defined by the US EPA, consists of four distinguishable but interacting phases, generally referred to as hazard identification, exposure assessment, dose-response assessment and risk characterisation (US EPA, 1987). The interrelation of these phases is depicted in Figure 3.1.

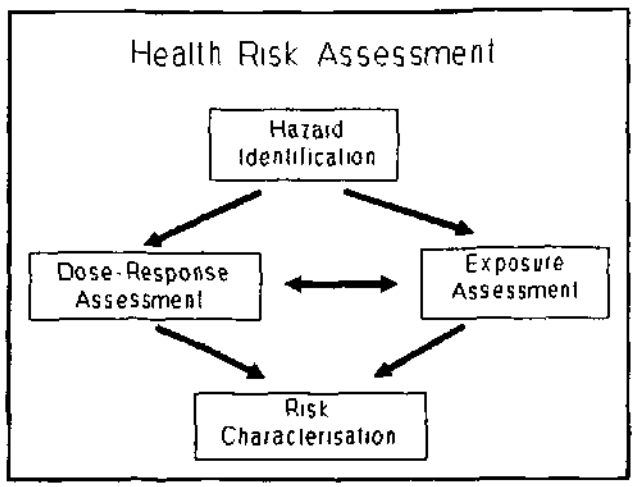

Figure 3.1 The Health Risk Assessment Process

3.1.3.1 Hazard Identification

The goal of hazard identification is to establish whether exposure to a chemical or microbiological agent can cause an increase in the incidence of illness or infection (Deisler, 1987). This is the first step in any risk assessment and determines whether the process should be continued or abandoned. Hazard identification itself holds an inherent risk since a hazardous substance may be erroneously declared safe, or a safe substance declared hazardous (Hutzler and Boyle, 1982).

A number of sources are used in identification of human health hazards, including historical disease statistics, epidemiological studies, animal studies, short-term screening with non-mammalian systems and analogy with known hazards. Acute effects and direct cause-effect relationships, frequently observed with microbiological agents, are relatively easily identified. Chronic or delayed responses, particularly cancer, are often difficult to link conclusively to a specific agent (Hutzler and Boyle, 1982; Wilson and Crouch, 1987).

Once a health hazard has been identified, the remainder of the process encompasses the description of the properties of the hazardous agent, and the identification of both acute and chronic health effects. Any benefits associated with the generation or use of the contaminant should also be described. The inclusion of a preliminary outline of the likely exposure conditions during the hazard identification aids in the selection of appropriate data for exposure and dose-response assessment (Hutzler and Boyle, 1982; Hallenbeck and Cunningham, 1986; Deisler, 1987; US EPA, 1987).

3.1.3.2 Exposure Assessment

Exposure assessment has been defined as the process of measuring or estimating the intensity, frequency and duration of human exposure to a contaminant which

is present in the environment. Estimation of hypothetical exposures which might arise from the release of new agents into the environment may also be required (Deisler, 1987). The task of exposure assessment is to provide the actual exposure conditions required to predict risk, and to identify and predict the effects of the proposed control options (Severn, 1987).

A broad range of questions is addressed in an exposure assessment, and this is often seen as the most resource-intensive phase of the risk assessment process (Severn, 1987). US EPA (1987) guidelines recommend that a complete assessment should deal with five major aspects of exposure. These are the source of the health hazard, exposure pathways via various media and routes, measured or estimated concentrations and exposure durations, the exposed populations, and an integrated exposure analysis. Depending on the scope of the exposure assessment, the level of detail in which each topic is addressed may be varied. Integrated exposure analysis combines the estimation of environmental concentrations of the health hazards with descriptions of the exposed population to yield one or more exposure profiles of the hazard (US EPA, 1987).

Since accuracy of exposure assessment is a primary determinant of the soundness of health risk assessment, it is advisable to use environmental data and actual population counts in preference to literature values or estimates. It has been recommended that upper limits of exposure and population size should be used to provide maximum protection of public health (Hallenbeck and Cunningham, 1986). Unfortunately, many situations in which estimates of exposure and risk are required are characterised by a lack of quantitative data. It then becomes necessary to construct conceptual and mathematical models of possible exposure pathways, and to use these to estimate exposure (Severn, 1987). A number of models are available to assist in the description of environmental fate and transport of organic chemicals, metals and, to a more limited extent, microorganisms (Ambrose and Barnwell, 1989).

Quality of the data supporting an exposure assessment and uncertainty of quantitative assessment of exposure are closely related. Characterisation of reliability and uncertainty of the data used, and specification of any assumptions made, are essential if exposure assessment is to be used correctly in support of risk management (Severn, 1987; US EPA, 1987).

3.1.3.3 Dose-Response Assessment

Dose-response assessment is the process of characterising the relationship between dose of a hazardous agent and incidence of an adverse effect in the exposed population (Cotruvo, 1987; Deisler, 1987). This is conducted simultaneously, and in interaction, with exposure assessment.

A study of the exposed population of interest, showing a well-defined dose-response relationship between the health hazard under consideration and the effects observed, provides the ideal basis for a quantitative estimate of the risk incurred by the population. Unfortunately, population studies of this type are often

crude or non-existent (Hutzler and Boyle, 1982; Cotruvo, 1987). Where possible, epidemiological studies are used as the basis of dose-response assessment. However, the design and implementation of such studies often renders them inappropriate for use in quantitative risk assessment (Hallenbeck and Cunningham, 1986; Lave, 1987). In the absence of adequate human data, well-quantified animal data may be used, particularly for chemical hazards. There is considerable precedent to indicate that substances which are toxic, particularly carcinogenic, to animals are likely to elicit a similar response in humans (Hallenbeck and Cunningham, 1986; Cotruvo, 1987).

The type of human or animal study chosen for dose-response assessment, and ultimately, the method of risk calculation used, are closely linked to mode of action of the health hazard. Two broad classes of chemical hazards have been defined: those causing acute or chronic systemic toxic effects and those displaying carcinogenic or similar effects. This distinction is based on the assumption that toxic effects are associated with some non-zero dose threshold below which no adverse health effects are expected, whereas no such dose threshold can be associated with carcinogenicity (Cotruvo, 1987; Hallenbeck and Cunningham, 1986). Microbiological hazards are further distinguished from chemical hazards by their mode of action, *viz.* infection. Models of dose-response relationships pertaining to micro-organisms have been evaluated (Haas, 1983a,b; Cooper *et al.*, 1984; US EPA, 1988; US EPA, 1989). Most recent work uses two models shown by Haas (1983b) to describe satisfactorily the relationship between microbial dose and probability of infection (Gerba and Haas, 1988; Asano and Sakaji, 1990; Rose *et al.*, 1991; Rose and Gerba, 1991a,b). These are an exponential model and a beta-distributed probability model and are explained in more detail in section 3.2.

3.1.3.4 Risk Characterisation

Risk characterisation has been defined as the process of calculating the incidence of the health effect under the conditions of exposure described in the exposure assessment, using the identified dose-response relationship (Deisler, 1987). Guidelines provided by the US EPA (1987) distinguish two components of risk characterisation, *viz.* presentation of a numerical estimate of risk and analysis of the significance of the risk estimate.

Risk estimates may be presented in a number of forms, depending on the context of the risk assessment. Cancer risks are frequently expressed as a unit risk, representing the excess lifetime risk due to constant lifetime exposure of one concentration unit of the carcinogen. It should be noted that this representation encompasses an assumption of a linear low-dose response. Risk may also be presented as excess individual or population risks. Examples of risks characterised in this format include individual lifetime risks, excess number of cancers per year in the exposed population, or excess number of infections per month. Finally, the risk may be fixed and the dose corresponding to the given risk level presented (Rose and Gerba, 1991b; US EPA, 1987).

Irrespective of methods used to extrapolate low-dose responses and to present

risks, a major component of risk assessment is an evaluation of all assumptions used and all sources of uncertainty. The effect of these methods on the confidence associated with risk estimation is evaluated and, wherever possible, expressed in a quantitative manner (*e.g.* probability distributions or confidence levels around exposure and risk estimates). Where there is no choice but to present a single number, this should be suitably qualified to indicate to the risk manager the confidence which may be attributed to the estimate (Severn, 1987; US EPA, 1987).

3.2 MICROBIAL RISK ASSESSMENT

A number of microbial risk assessment methodologies have been proposed in the past decade (Hutzler and Boyle, 1982; Cooper *et al.*, 1984; US EPA, 1989; Haas, 1983a,b). The following section reviews recent developments in microbial risk assessment, with the aim of identifying the most appropriate approach and models for this study.

3.2.1 *Microbial risk assessment models developed for specific applications*

A number of models have been developed to evaluate risks associated with microbial contamination in specific contexts. These frequently have the disadvantage of being strongly linked to the purpose for which they were designed. However, aspects of the methodology used may be more widely applicable.

The risk of illness due to hepatitis A virus during wastewater reuse was assessed by Hutzler and Boyle (1982). Each step in the exposure pathway was described quantitatively, in order to estimate the ingested dose of virus. This was compared to the infective dose to determine the expected number of excess cases of hepatitis A associated with wastewater reuse. By attaching a cost to illness, a cost-benefit evaluation could be conducted.

A computerised model for risk assessment of drinking water supplies for small population groups was developed by Cooper *et al.* (1984) for estimation of risks posed by polluted drinking water supplies to army troops. The model and accompanying data base were designed for a number of pathogenic bacteria, viruses and parasites. The possibility for different exposure scenarios was taken into account by defining a "low risk" and a "high risk" scenario for developed countries and a "low risk" scenario for developing countries. These were specified in terms of raw water quality and the degree of treatment water would receive. The endpoint of exposure considered was disease, with the exception of viruses for which infection was used. Four models were considered to describe dose-response data, *viz.* a logistic model, a log-normal model, an exponential model and a beta-distributed model. Risk within the population was considered to be beta-distributed. The most conservative model was identified for each pathogen evaluated and used to estimate low and high risk boundaries. Risks were presented graphically, plotted as the chance (or risk) that less than or equal to a specific proportion of a troop would fall ill against the proportion of the troop that

fell ill.

The US EPA (1988) evaluated available models for the microbial risks associated with sludge disposal options, and on this basis developed a computerised model to describe fate of microorganisms during sludge disposal and risks associated with human exposure to treated sludge. Initial evaluation of available information (US EPA, 1988) identified three models, two concerned with specific sludge disposal methods and one addressing discharge of and exposure to treated wastewater (Haas, 1983a). One of the identified sludge models was used as a basis for the computerised model developed (US EPA, 1989). A number of sludge disposal options were represented, with detailed descriptions of the fate and transport of microorganisms which could lead to possible human exposure. Default values based on the literature or estimates were provided, but users generally were given the option of entering site-specific data (*e.g.*, rainfall, wind speed) if this was available. Provision was made for modelling of bacteria, viruses and parasites. The endpoint of exposure considered in most instances was infection. Not everyone who may become infected with pathogens will necessarily become clinically ill (diseased) as asymptomatic infections are particularly common among some enteroviruses. The development of clinical illness depends on numerous factors such as the immune status of the host, age of host, virulence of the microorganism, type, strain of microorganism and route of infection. Risk of infection was modelled using an assumption of random distribution of microorganisms in the contaminated medium, described by a Poisson distribution. The model estimate of dose was compared to generalised minimum infective doses for the pathogen of interest to evaluate the risk of infection.

A qualitative assessment of microbial health risks posed by viruses during sewage sludge disposal was presented by Gerba and Goyal (1988). No quantitative model was considered. Human pathogens associated with sewage sludge were identified and the risks posed by them following the ocean dumping of sludge examined by scrutinising the possible fate of the pathogens and their persistence in marine environments. Attempts were made to identify different routes by which pathogens can reach man.

Haas (1983a) developed a model of the fate of microorganisms during wastewater treatment and the risk of illness associated with recreational exposure to water to which treated effluent was discharged. Equations for the removal or inactivation of viruses during wastewater treatment, reduction in virus numbers after discharge due to decay and dilution, probability of exposure to pathogenic viruses in water ingested during primary contact recreation, and probability of contracting disease as a result of infection were described. Random distribution of microorganisms in water was assumed, described by a Poisson distribution. The model was used to evaluate the increase in risk to public health which would result if disinfection of wastewater intended for discharge to water bodies used for recreation were discontinued.

3.2.2 Infectivity probability models

The ability of three infectivity probability models to describe dose-response relationships in human volunteer feeding studies was evaluated and compared by Haas (1983b) by fitting experimental data to the models being examined. Models were based on the assumption of random distribution of microorganisms in water, described by a Poisson distribution.

Log-normal Model

In the log-normal model each host organism is assumed to have an inherent minimal infective dose and if exposed to a dose in excess of this minimum amount then an observed response (either infection or disease) will result. The model is described as:

$$P' = \frac{1}{\sqrt{2\pi}} \int_{-\infty}^{z} \exp(-z^2/2)dz$$

$$Z = (1N - \mu)/\sigma$$

Where P = probability that a single individual exposed to a dose of N organisms will be infected or contract disease.
μ = average logarithm
σ = log standard deviation of the minimum infective doses

Single-hit exponential model

This model was developed based on the assumptions that the pathogenic organisms have uniform effectiveness and that the pathogen-host interactions are characterised by a discrete value(r). The single-hit exponential model can be summarised as:

$$P = 1 - \exp(-rN)$$

Where P = probability that a single exposure will result in disease/infection
N = average number of organisms ingested
r = constant

Beta-distribution infectivity probability model

Because there is some variation in the virulence of the individual pathogens and in the sensitivity of the hosts or both, the beta-distribution model was developed assuming that pathogen-host interactions are characterised not by a discrete value (r), but by a distribution of values (ß,α). Based on this assumption, the mathematical model was developed to be expressed as

$$P = 1 - (1 + (N/ß))^{-\alpha}$$

The predictive models described above were used to compare predictions with

regard to the development of infection/disease at any time subsequent to a single exposure. Nine data sets from eight sources were used to test the three models cited above. The pathogens were administered orally and include organisms of waterborne disease concern. Best fit values were determined for each data set and whether a given model was consistent with the data set was determined using the chi-squared goodness of fit test. Of the nine data sets, the beta-distribution model fit seven data sets. The log-normal model fit five and the simple exponential model fit three. In three cases all three models provided a satisfactory fit to the data. In cases where both the beta-distribution and exponential models were found to fit the data sets the agreement of the risk estimates between the two models is within a factor of 2.

It was concluded that of the log-normal model, single-hit exponential model and beta-distributed model, the last-mentioned proved superior in most instances.

The majority of subsequent microbial risk assessment studies are based on the work of Haas (1983b). Risks associated with exposure to treated wastewater were evaluated by Asano and Sakaji (1990) and by Rose and Gerba (1991a). Asano and Sakaji (1990) used all three models described by Haas (1983b) to assess risks of infection by *Shigella dysenteriae, S. flexneri, Entamoeba coli,* echovirus 12 and poliovirus 1, poliovirus 2 and poliovirus 3. Rose and Gerba (1991a) evaluated risks associated with enteric viruses, using the beta-distributed model, and with cysts of the protozoan parasite *Giardia,* using the exponential model. The exponential model was employed in the latter case since it gave similar results to the beta-distributed model for *Giardia* and was simpler.

Risks associated with enteric viruses in drinking water were evaluated by Gerba and Haas (1988), using the beta-distributed model. Rose *et al.* (1991) utilised the exponential model to assess risks associated with *Giardia* cysts. Risks posed by the presence of both *Giardia* cysts and enteric viruses in drinking water were determined by Rose and Gerba (1991b), using the exponential model for *Giardia* and the beta-distributed model for viruses. Results were utilised in a discussion of the use of microbial risk assessment in the development of microbial water quality guidelines.

4. METHODOLOGY

The development of methods for implementation of microbial risk assessment case studies was divided according to the processes of risk assessment identified above: hazard identification, exposure assessment, dose-response assessment and risk characterisation. Incorporated in the last process is the characterisation of uncertainty. No development of experimental methods was undertaken since the case studies made use of existing monitoring data.

4.1 Hazard identification

No specific methods were developed for hazard identification. Necessary information for review of pathogen properties was obtained from the literature.

4.2 Exposure assessment

Exposure assessment was based on monitoring data. No modelling of pathogen fate, transport or exposure was conducted. Description of environmental pathways and behaviour patterns leading to exposure were derived from literature and from reports on the monitoring surveys from which data were drawn.

Analysis of monitoring data was conducted in order to characterise the distribution of data. This allowed quantitative description of uncertainty in the risk estimates which could be associated with variability in exposure. Monitoring data were tested for goodness-of-fit to a number of distributions using the Statgraphics statistical software package (STSC, 1989). Goodness-of-fit was evaluated by the Kolmogorov-Smirnov test. This was used in preference to the chi-square test since the relatively small sample size typical of microbiological data sets is not suitable for the chi-square test.

4.3 Dose-response assessment and risk characterisation

The beta-distributed (Formula 1) and exponential (Formula 2) models were identified in the literature as the most appropriate models of pathogen dose-response relationships on the basis of results reported by Haas (1983b), Gerba and Haas (1988), Rose and Gerba (1991a,b) and Rose *et al.* (1991).

Formula 1: Beta-distributed model of daily risk of infection

$$P = 1 - (1 + N/\beta)^{-\alpha}$$

where
- P = probability (risk) of infection
- N = dose or exposure (number of organisms)
- α,β = parameters characterised by dose-response relationship

The above model estimates daily risks of infection. Longer term risks were determined by several methods. Average risks were calculated from daily risks

using a measure of central tendency over the period of interest, usually the geometric mean (Formula 2) (Haas, 1983b; Gerba and Haas, 1988). Where appropriate, a distribution of measure of central tendency over subsets of the period of interest could be substituted for a single measure of central tendency (*e.g.* substitution of a distribution of monthly geometric means for a single annual geometric mean).

Formula 2: Calculation of average risk over longer exposure periods

$$P_x = 1 - (1 - P(N))^x$$

where P_x = probability (risk) of one or more infections over period x
x = number of days of exposure
$P(N)$ = daily risk, using geometric mean for N
N = geometric mean number of organisms ingested on a daily basis over period x

In some instances, daily risk was sampled directly from a distribution of daily exposure and used to calculate longer term risk as above, with the daily exposure estimates used in place of the geometric mean. The expected value of risk arising from 1000 model iterations was used as an estimator of longer term risk. This yielded a value which estimated the most likely risk (modal risk) over the exposure period, rather than the average risk.

An alternative method for the calculation of longer term risk involved the product of estimates of daily risk for each day over the exposure period of interest. For example, yearly risk could be calculated as a product of all estimated daily risks over a year (Haas, personal communication) Calculation of yearly risks according to this approach is shown in Table 4.1. This method allowed the overall risk estimate to be influenced more strongly by outlying results than the average risk based on a measure of central tendency, and is more representative of the cumulative risk over the exposure period.

Table 4.1: Calculation of yearly risk from individual estimates of risk

Day	Daily risk (P)	1 - P	Product
1	P_1	$1 - P_1$	$(1-P_1)$
2	P_2	$1 - P_{12}$	$(1-P_1)(1-P_2)$
•	•	•	•
•	•	•	•
365	P_{365}	$1 - P_{365}$	$(1-P_1)(1-P_2)...(1-P_{365})$
	Yearly risk = 1 - Product = $1 - (1-P_1)(1-P_2)...(1-P_{365})$		

A number of assumptions, previously described by Haas (1983b), Rose and Gerba (1991b), Rose *et al.* (1991) and others, were also accepted in application of microbial risk models in this investigation. Random distribution of microorganisms in the contaminated medium, described by a Poisson distribution, was assumed. All members of the exposed population were considered to be equally susceptible to a single exposure. In addition, a number of assumptions are commonly employed in the use of data sets including non-detect values. In this study, the effects of substituting non-detect results with the detection limit, half the detection limit and zero were compared.

Dose-response assessment and risk characterisation using the single-hit exponential and beta-distributed models were implemented using a Lotus 123 Spreadsheet (Lotus Development Corporation, 1988). Data were specified as probability distributions using a risk assessment add-in (@Risk, Palisade Corporation, 1988). The add-in facilitated characterisation of uncertainty associated with risk estimates as a consequence of variability in the input data. The spreadsheet permitted estimation of risk (probability of infection) in experimental human volunteer feeding studies and use of these risk calculations to determine fit of experimental data to infectivity models. This allowed checking of published values of model parameters to be conducted. Calculation of exposure (dose) and of weekly, monthly and yearly risk using environmental monitoring data were supported by the spreadsheet.

Uncertainty surrounding risk estimates as a consequence of variability in exposure was evaluated quantitatively by entering exposure data as probability distributions, and is explained in detail in section 5. If exposure data did not display significant fit to any of the distributions tested, a general distribution was defined. Analysis of uncertainty was conducted by executing infectivity models from within the risk assessment add-in. Exposure levels were sampled from defined distributions by Latin hypercube sampling. The distribution of risk estimates was calculated by 1000 model iterations.

5. CASE STUDIES

5.1 CASE STUDY 1: DRINKING WATER ENTERIC VIRUSES IN RAW AND TREATED DRINKING WATER

5.1.1 Hazard identification

Viruses are submicroscopic inert particles of protein and nucleic acid which are unable to replicate or adapt to environmental conditions outside a living host (Thomann and Mueller, 1987). Human enteric viruses are associated with the digestive tract of humans. These viruses are excreted in faecal matter and may occur in water, including water used as a source for drinking water supply (Gerba and Rose, 1990).

More than 110 serotypes of human enteric viruses are known, and additional ones are discovered almost annually. Among the best known enteric viruses which have been isolated from the environment are enterovirus, including poliovirus, coxsackievirus and echovirus. These cause diseases such as paralysis, meningitis, respiratory illness and diarrhoea. Other enteric viruses which may occur in water include adenovirus, reovirus, rotavirus, Hepatitis A virus and Norwalk virus and "small round structured viruses" (Craun, 1986; Gerba and Rose, 1990).

Waterborne viral diseases remain a cause for concern in public health protection, even in developed countries. Drinking water may become contaminated by failure of water treatment systems, or by sewage. Viruses have been recovered from treated drinking water with ≈53% of reported isolations coming from water with complete treatment (Gerba and Rose, 1990). Sewage contamination may occur directly by seepage into the water supply. Runoff from land treated with sewage sludge or irrigated with wastewater, or disposal of sewage to surface water may cause indirect contamination of water sources for drinking water supply. Difficulties associated with the isolation of viruses and under-reporting of gastrointestinal illness make estimation of the extent of viral waterborne disease complex. However, viruses are known to have been responsible for 12% of reported waterborne disease outbreaks in the USA in the period 1946-1980, and it has been suggested that as much as 64% of the total number of outbreaks may be attributable to viruses (Craun, 1986; Gerba and Rose, 1990).

Although viruses cannot multiply outside a living host they can survive in the environment for extended periods of time, hence levels tend to only decrease gradually after discharge to the aquatic environment. A number of physical, chemical and biological factors determine the rate of decline in virus levels. Physical factors include light, temperature, hydrostatic pressure, sedimentation, adsorption and aggregation. Chemical factors include pH, ionicity, cation concentrations, heavy metals, organic molecules and levels of dissolved oxygen. Biological factors incorporate type of virus, bacterial and algal activity, and predation by protozoa (Block, 1983; Thomann and Mueller, 1987). Adsorption, aggregation and sedimentation of viruses are among the most important factors

increasing virus survival, while temperature and microbial activity appear to be the dominant factors in decreasing virus survival (Block, 1983). Decline in virus levels in the aquatic environment as a result of dilution and inactivation is an important element in avoidance of waterborne viral disease and management of water quality.

Quantitative data on occurrence of enteric viruses in surface and treated drinking water are relatively limited due to the complexity of viral recovery and detection methods. Most data relate to enteroviruses, since techniques for these viruses are relatively well established. Methods most widely used encompass adsorption/ elution techniques for the concentration of viruses from environmental water samples. Other methods include ultrafiltration and filtration using glass powder or wool. Viruses are eluted from filters either with a proteinaceous substance, such as beef extract or mechanically. After concentration, viruses are detected by the ability to infect cells. The measure of infection generally used is the tissue culture infective dose (TCID), often the dose required to infect 50% of culture tubes in a quantitative assay ($TCID_{50}$). Laboratory studies have shown recovery efficiencies of approximately 50% using these methods, but it is likely to be lower for environmental samples (Gerba and Rose, 1990). South African laboratories using ultrafiltration methods have reported higher recovery, but analyze smaller volumes of water.

A number of viruses cannot be detected by cell culture techniques. Methods such as immunofluorescence, radio-immunoassay (RIA) and enzyme-linked immunosorbent assay (ELISA) have been applied for the detection of such viruses. With the advent of recombinant DNA technology, the use of gene probes and molecular-based techniques such as the polymerase chain reaction (PCR) for sensitive and rapid detection of viruses has been successfully applied (Gerba and Rose, 1990; Kfir and Genthe, 1993).

The presence of enteric viruses in drinking water represents a sufficiently pressing public health problem to warrant evaluation of risks of infection associated with their presence in raw water supplies and treated drinking water. The methodological difficulties associated with recovery, detection and enumeration of viruses in environmental samples should be taken into consideration in the development and interpretation of risk estimates.

5.1.2 Exposure assessment

5.1.2.1 Source of hazard

This study considered drinking water exposure which could have resulted from the presence of enteric viruses in the raw surface water supply to one of three water treatment plants supplying treated drinking water to the Pretoria-Witwatersrand-Vereeniging (PWV) region. The raw water source considered was the most microbially polluted of three possible surface water sources to the plant in question. Under normal circumstances, this source contributed 5% or less of the total intake at the water treatment plant. However, under extremely dry climatic conditions, this source could on rare occasions comprise a larger proportion of the intake

(M. Steynberg, Rand Water, personal communication).

Raw intake water was treated by a high pH, lime coagulation process, followed by sand filtration and breakpoint chlorination. Lime (calcium hydroxide, $Ca(OH)_2$) was added to raw water to form a floc, which settled and was removed. Lime raised the pH of the water, creating conditions unfavourable for biological life. Bacteria and viruses were also removed by sedimentation. Sand filtration also removes microorganisms and breakpoint chlorination serves to inactivate microorganisms not removed during coagulation and filtration (M. Steynberg, personal communication).

5.1.2.2 Exposed population

Approximately 8 million consumers in the PWV area are provided with treated water. One third are supplied by the above plant, hence the maximum size of the exposed population was approximately 2.3 million (J. Geldenhuys, Rand Water, personal communication). It was not possible to characterise the exposed population more accurately since mixing of treated water in the distribution system precluded identification of sub-populations which are served exclusively or predominantly by this plant.

5.1.2.3 Exposure routes

The extent of enteric virus removal during this specific water treatment process was not known. Water was considered to be of satisfactory virological quality if no enteric viruses were detected in 10 ℓ samples of treated water. Removal of the bacterial indicator organisms, the faecal coliforms was approximately 6 log (6 orders of magnitude), and that of bacteriophages (viral indicators of faecal pollution) was between 5 log and 6 log (J. Geldenhuys, Rand Water Board, personal communication) This may be compared with the US EPA recommendation that all drinking water treatment plants should be capable of reducing virus levels by at least 4 log (Federal Register, 1989).

Enteric viruses are infective on ingestion, hence ingestion in drinking water was the most significant route of exposure for the majority of urban consumers. Risks associated with ingestion of untreated water were not considered. Inhalation and dermal contact were assumed to be of minimal significance. This assumption is likely to be valid for dermal contact, but some chance of disease transmission by inhalation of aerosols, *e.g.* during showering, may exist.

5.1.2.4 Exposure monitoring

Quality of raw water and treated water was monitored for viruses and other microbiological variables from April 1981. Enteric virus analyses were carried out by the Division of Water Technology (formerly National Institute for Water Research), CSIR, Pretoria. Sample volumes of 10 ℓ raw and treated water were concentrated by ultrafiltration and analyzed by cell culture methods. Enteric viruses were reported as the most probable number (MPN) 50% tissue culture infective dose ($TCID_{50}$) per 10 ℓ (Grabow and Nupen, 1981). The detection limit was

63 MPN $TCID_{50}$ per 10 ℓ. Results accumulated since 1981 indicated water from the river source in question to be the greatest source of viral pollution, hence this was selected as the raw water source to gain an indication of "worst case" risks (Bateman *et al.*, 1991). Monitoring data for the period April 1981 to March 1991 were evaluated.

5.1.2.5 *Analysis of monitoring data*

Geometric mean enteric virus levels in raw water over the monitoring period, with data aggregated by month, are presented in Figure 5.1. The highest enteric virus levels were recorded in July, which corresponds to mid-winter. During this period the raw water source was typically dominated by low flow conditions and virus survival periods are increased due to low water temperatures.

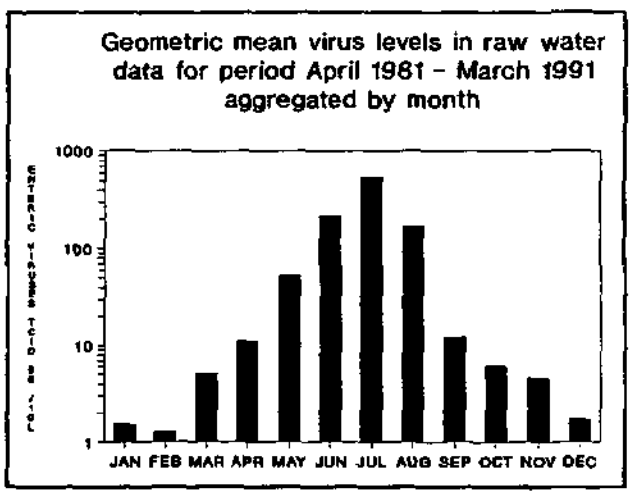

Fig. 5.1 Geometric mean virus levels in raw water (aggregated by month)

All enteric virus levels were treated as enteroviruses, as represented by poliovirus 1, poliovirus 3 and echovirus 12. Non-detect results were replaced with the detection limit, half the detection limit or zero (1 MPN $TCID_{50}$/10 ℓ was used in place of zero for calculation of geometric means).

Distributions of data aggregated by month were tested for goodness of fit to a log-normal distribution and several other distributions represented in the Distribution Fitting Function of Statgraphics ver. 5.0 (STSC, 1991). The Kolmogorov-Smirnov test was used, which is a one-sample test which allows one to test the overall goodness of fit of a data set to determine if the data set follows a specified distribution. This test measures how much the empirical cumulative distribution function differs from that of the fitted hypothesized distribution. None of the tested distributions showed consistently significant fit to the data, although data for some months showed reasonable fit to a log-normal distribution. In the interests of consistency, data for all months were represented as general distributions. This facilitated the use of @Risk Palisade corporation, 1988) to assess uncertainty in risk estimates associated with variability in monitoring data. A general distribution of monitoring data per month was therefore defined to

facilitate the use of @Risk ver. 1.5 (Palisade Corporation, 1988) in assessing uncertainty due to the distribution of monitoring data. The form of the general distribution specified by @Risk was @GENERAL (*minimum, maximum,* x_1, p_1, x_2, p_2, ...,x_n,p_n,*n*) and represented a general density function for a probability distribution ranging between *minimum* and *maximum* with *n* (x,p) pairs with value *x* and probability weight *p* for each point (Palisade Corporation, 1988). Aggregated monitoring data for each month were entered as a general distribution in the Lotus 123 ver. 2.01 (Lotus Development Corporation, 1988) spreadsheet used in dose-response assessment and risk characterisation, as described in section 4.

5.1.3 Dose-response assessment

5.1.3.1 Infectious dose

An important factor in the estimation of probability of adverse effects associated with waterborne viruses is the quantity of enteric viruses which must be ingested to initiate infection, but satisfactory estimates of such minimum infective doses are difficult to establish. Ideally, these levels would be established by epidemiological studies. However, while epidemiology can provide valuable information regarding patterns of microbial health risks and establishing statistically significant associations between the presence of an infectious agent and a population health effect, it cannot provide quantitative estimates of minimum infective doses (Gerba and Haas, 1988). Controlled dose-response studies conducted on animals are of questionable significance, and similar studies on humans are fraught with ethical problems (Ward and Akin, 1984; Gerba and Haas, 1988). Prediction of the probability of infection by a low dose of enteric viruses, similar to those encountered in environmental samples, would require large groups of volunteers while not providing certainty that the distribution of susceptibility within the volunteer group would adequately reflect that of the population (Gerba and Haas, 1998).

A review of minimum infective doses of various respiratory and enteric viruses revealed that doses as low as 1 to 2 pfu may cause infection in man (Ward and Akin, 1984). Studies on enteric viruses, primarily vaccine strains, have shown poliovirus 1 to be infective at doses of 2 pfu, poliovirus 3 at 10 $TCID_{50}$ and 1 $TCID_{50}$, and echovirus 12 at 17 pfu (Gerba and Haas, 1988).

Several additional factors apply to the transmission of water-borne infectious diseases. Illness may be spread by secondary contact, either person-to-person or by contamination of other materials with which non-infected individuals may come into contact. Furthermore, all infections do not necessarily result in the appearance of clinical symptoms. Development of clinical disease depends on numerous factors, including the immune status and age of the host, and the type, strain and virulence of the microbial agent. Observed frequencies of symptomatic infection for enteroviruses range from 1% for poliovirus to greater than 75% for some coxsackie B viruses. Modern medicine has greatly reduced the mortality rate of infectious diseases, and in the developed countries of Europe and North America enteric virus infections have low mortality rates. Mortality for enterovirus

infections has been reported to range from less than 0.1% to 1.8% (Gerba and Haas, 1988).

5.1.3.2 Dose-response model

The risk to which a population is exposed to known levels of a microbial agent can be estimated by dose-response extrapolation models, as outlined in the section 3.2., describing Microbial Risk Assessment. The beta-distributed infectivity probability model was selected for this study since review of probability models of infection indicated that this provided the best description of viral infection (Haas, 1983b; Gerba and Haas, 1988). The model for daily risk, the modified format for calculation of longer term risks from average pathogen levels or from daily risk estimates, and assumptions accepted in implementation of the model are presented in section 4 describing microbial risk assessment.

All data were treated as representing the enteric virus group as a whole. Risk model parameters were available for the enteroviruses echovirus 12, poliovirus 1 and poliovirus 3 (Haas, 1983b). Echovirus 12, poliovirus 1 and poliovirus 3 were assumed to be representative enteroviruses, and enteroviruses were assumed to representative of enteric viruses, in risk calculation. Parameter values are presented in Table 5.1.1.

Table 5.1.1 Representative values of parameters for the beta-distributed model for enteroviruses

Host-pathogen Parameter values	Echovirus 12	Poliovirus 1	Poliovirus 3
α	1.3	15	0.5
β	75	1000	1.14

Daily probability of infection in each month was calculated by sampling from the specified general distributions and calculating the risk corresponding to sampled virus exposures. A Latin hypercube sampling method was employed in a simulation run of 1000 model iterations. This produced tabulated expected values of risk in the Lotus spreadsheet and a graphical presentation of simulation results showing trend in the mean, one standard deviation around the mean, and 5th and 95th percentiles of risk. The maximum expected value of risk was obtained from the distribution of simulated risk in the month showing the highest risk (July).

Yearly risk was estimated by two methods. In the first, average yearly risk was estimated from a general distribution of monthly geometric mean virus levels, similar to the monthly distributions of monitoring data specified for estimation of daily risks. Exposures were sampled from this distribution, the corresponding daily risk calculated and the average yearly risk derived from the daily risk as described

in section 4. For purposes of comparison, average yearly risk was also calculated using the overall geometric mean of all data for a limited sub-set of results. In the second method, daily risk was estimated for each day of the year by sampling from the general distribution of monitoring data for each month. The yearly risk was calculated as a product of the daily risk, as described in section 4.

Simulation of yearly risk calculated by both above methods, using @Risk, produced tabulated results in the Lotus spreadsheet and a distribution of simulated risk values with an indicated expected value.

5.1.4 Risk characterisation

5.1.4.1 Estimates of daily and yearly risk of infection

Treated drinking water

No enteric viruses were detected in treated drinking water. Daily risks were estimated at the minimum measurable limit, *i.e.* the detection limit, and at one non-detect surrogate value, (half the detection limit), for all three representative enteric viruses. Results are shown in Table 5.2. Based on analysis of treated water, it may be concluded that daily risks of enteric virus infection did not exceed $2x10^{-1}$ to $7x10^{-1}$, if echovirus 12 and polioviruses 1 and 3 were considered as representative enteric viruses. This corresponds to the minimum measurable risk. If it is assumed that non-detect results can be safely substituted by half the detection limit, then it is likely that the maximum daily risk did not exceed $9x10^{-2}$ to $6x10^{-1}$. This is considerably higher than the mean yearly risk of 10^{-4} recommended by the EPA for treated drinking water (Federal Register, 1989). Detection of low risk levels in drinking water was hampered by small sample volumes and a high detection limit, causing many non-detect results to be recorded.

Table 5.1.2 Daily risks of infection, based on analysis of treated drinking water

	Echovirus 12	Poliovirus 1	Poliovirus 3
At detection limit (63 MPN $TCID_{50}/10\ell$)	$2x10^{-1}$	$2x10^{-1}$	$7x10^{-1}$
At half detection limit (31.5 MPN $TCID_{50}/10\ell$)	$1x10^{-1}$	$9x10^{-2}$	$6x10^{-1}$

Extrapolation from raw water

In an attempt to circumvent some of the difficulties associated with non-detect results in drinking water, virus levels in treated water were estimated from raw water levels, as described by Rose and Gerba (1991b) and Rose *et al.* (1991). Enteric virus reductions of 4 log, 5 log and 6 log were assumed on the basis of approximate bacteriophage removal by the water treatment plant and of US EPA recommended virus removal (Federal Register, 1989). Expected values of maximum daily risk, yearly risk and average yearly risk are shown in Table 5.3.

If a 4 log reduction during treatment was assumed, expected daily risks associated with drinking water were estimated in the range $4x10^{-2}$ to $4x10^{-1}$, considering echovirus 12 and polioviruses 1 and 3 as representative enteric viruses. If a 5 log reduction was assumed, expected maximum daily risk estimates were in the range $5x10^{-3}$ to $1x10^{-1}$. An assumed 6 log reduction yielded maximum daily risk estimates in the range $5x10^{-4}$ to $1x10^{-2}$. Risk estimates for poliovirus 3 were noticeably higher, overall, than those for echovirus 12 and poliovirus 1.

Table 5.1.3 Daily and average yearly risks of infection from treated drinking water, based on extrapolation from raw water quality

Assumed reduction during treatment	Expected value of risk, based on distributions of monitoring data			Average yearly risk based on geometric mean of whole data set (Selected data only)
	Maximum daily risk	Average yearly risk based on distribution of monthly geometric means	Average yearly risk based on individual daily risk estimates	
Echovirus 12				
Non-detect value: 63 MPN $TCID_{50}$/10 ℓ				
4 log	5×10^{-2}	6×10^{-2}	9×10^{-1}	
5 log	5×10^{-3}	7×10^{-3}	2×10^{-1}	
6 log	5×10^{-4}	7×10^{-4}	2×10^{-2}	
Non-detect value: 31.5 MPN $TCID_{50}$/10 ℓ				
4 log	5×10^{-2}	6×10^{-2}	9×10^{-1}	9×10^{-3}
5 log	5×10^{-3}	6×10^{-3}	2×10^{-1}	9×10^{-4}
6 log	5×10^{-4}	6×10^{-4}	2×10^{-2}	9×10^{-5}
Non-detect value: 0 MPN $TCID_{50}$/10 ℓ				
4 log	5×10^{-2}	3×10^{-2}	9×10^{-1}	
5 log	5×10^{-3}	3×10^{-3}	2×10^{-1}	
6 log	5×10^{-4}	3×10^{-4}	2×10^{-2}	
Poliovirus 1				
Non-detect value: 63 MPN $TCID_{50}$/10 ℓ				
4 log	4×10^{-2}	6×10^{-2}	8×10^{-1}	
5 log	5×10^{-3}	6×10^{-3}	2×10^{-1}	
6 log	5×10^{-4}	6×10^{-4}	2×10^{-2}	
Non-detect value: 31.5 MPN $TCID_{50}$/10 ℓ				
4 log	4×10^{-2}	5×10^{-2}	8×10^{-1}	8×10^{-3}
5 log	5×10^{-3}	5×10^{-3}	2×10^{-1}	8×10^{-4}
6 log	5×10^{-4}	5×10^{-4}	2×10^{-2}	8×10^{-5}
Non-detect value: 0 MPN $TCID_{50}$/10 ℓ				
4 log	4×10^{-2}	3×10^{-2}	8×10^{-1}	
5 log	5×10^{-3}	3×10^{-3}	2×10^{-1}	
6 log	5×10^{-4}	3×10^{-4}	2×10^{-2}	

Assumed reduction during treatment	Expected value of risk, based on distributions of monitoring data			Average yearly risk based on geometric mean of whole data set (Selected data only)
	Maximum daily risk	Average yearly risk based on distribution of monthly geometric means	Average yearly risk based on individual daily risk estimates	
Poliovirus 3				
Non-detect value: 63 MPN $TCID_{50}$/10 ℓ				
4 log	4×10^{-1}	7×10^{-1}	1	
5 log	1×10^{-1}	2×10^{-1}	1	
6 log	1×10^{-2}	2×10^{-2}	4×10^{-1}	
Non-detect value: 31.5 MPN $TCID_{50}$/10 ℓ				
4 log	4×10^{-1}	7×10^{-1}	1	3×10^{-2}
5 log	1×10^{-1}	1×10^{-1}	1	3×10^{-3}
6 log	1×10^{-2}	1×10^{-2}	4×10^{-1}	3×10^{-4}
Non-detect value: 0 MPN $TCID_{50}$/10 ℓ				
4 log	4×10^{-1}	5×10^{-1}	1	
5 log	1×10^{-1}	8×10^{-2}	1	
6 log	1×10^{-2}	8×10^{-3}	4×10^{-1}	

Risk values are calculated assuming either 4, 5 or 6 log reduction in viral concentrations from raw water in the treatment plant and assuming consumption of 2 ℓ water per day

Discussion of risk estimates

Expected values of maximum daily risks calculated assuming 4-6 log reductions in enteric virus levels during treatment were lower than daily risks estimated on the basis of analysis of treated water. This is to be expected since the latter risk estimates were dependent on sample volumes and assumptions regarding non-detect results.

All estimates of maximum daily risks were higher than the EPA recommended yearly risk of 10^{-4}. However, daily risks estimated in months characterised by lower virus levels (*e.g.* summer months) were markedly reduced, with the lowest estimates of the order 10^{-7}. This indicates that the calculated maximum risks represent extreme events. Detection of low risks was hindered predominantly by the small sample volumes analysed.

Average yearly risks were similar to maximum daily risks when calculated from the distribution of monthly geometric mean virus levels or on an overall geometric mean, but were considerably higher when based on daily risk estimates. All yearly

risks were markedly higher than the yearly risk of 10^{-4} recommended by the US EPA (Federal Register, 1989), even when 6 log reduction in virus levels during treatment was assumed.

Risk calculations were relatively insensitive to the treatment of non-detect results, since substitution of non-detects with the detection limit, half the detection limit or zero yielded the same or similar risk estimates. This indicates that the high detection limit of techniques used in South Africa was not the dominant factor restricting detection of low risk levels, although it did contribute heavily to the proportion of non-detect results.

5.1.4.2 Characterisation of uncertainty

A number of sources of uncertainty can be identified in the risk estimates presented here. Some of these are quantifiable to a limited extent, while others can be considered in a qualitative sense only.

One source of uncertainty is variability in the exposed population, related both to water supply pattern and to population behaviour patterns which determine the amount of water consumed. It was not possible to describe the fate of water once it left the treatment plant, hence no indication of the uncertainty associated with distribution of treated water to consumers could be given. Behavioral patterns regarding water consumption are generally dealt with by assuming an adult water consumption of 2 ℓ per day. This approach is adopted in many countries and by a number of international organisations. In some cases, *e.g.* in the development of Canadian drinking water guidelines, surveys have been conducted to describe water consumption patterns in greater detail (Canadian Council of Resource and Environmental Ministers, 1987). A similar survey conducted in the greater Cape Town area (analysed by sex, age, population group, income and season) found differences in water consumption between population groups (WRC report 74/2/87, 1987). The average water consumption was highest for "whites" at 2,19 ℓ per day, compared to 1,26 ℓ per day for "coloureds". The assumption of 2 ℓ per day adopted by other agencies was therefore continued as it represents the higher consumption rate of the population groups and will therefore not underestimate risks.

Removal of viruses during treatment was dealt with by assumption. The validity of the assumptions is uncertain, although available estimates indicate that the ranges selected were probably representative. However, there is no indication of variability in treatment and the effect thereof on final water quality.

Uncertainties may also arise from the recovery and detection methods used, particularly since low recovery is often reported for viruses (Rose and Gerba, 1990). Methods used in South Africa generally show high recoveries, even from environmental samples, and it is expected that uncertainties from this source were less than from others, such as the volume of water sampled.

Considerable uncertainty was associated with values assigned to non-detect

results. As a consequence of the relatively small volumes of water analyzed (10 ℓ) and the relatively high detection limit (63 MPN $TCID_{50}$), an extremely large number of non-detect results was recorded. The use of values such as the detection limit and half the detection limit may have resulted in gross overestimation of the actual value of non-detect results. It is felt that this source of uncertainty was the primary cause for the high risk estimates obtained in the study. The extent of this overestimation cannot be evaluated until methods are implemented in South Africa for monitoring of large volumes of water.

A further uncertainty is associated with the use of echovirus 12, poliovirus 1 and poliovirus 3 as representative enteric viruses. Results obtained indicate a variability of up to one order of magnitude due to virus-specific characteristics. This may be greater if the selected viruses are not truly representative of the enteric virus group.

Uncertainty in risk estimates derived by extrapolation from raw water virus concentrations was also due to variability in virus levels in water. This uncertainty was quantified by specifying monitoring data as probability distributions. Probability distributions of risk estimates indicate the range in risk estimates attributable to variability in monitoring data. Representative distributions are presented in Figures 5.2 and 5.3 (a-c). These show the distribution of risk for each of the three representative enteric viruses, respectively (a-c per figure), assuming 4 log (Fig 5.2) and 6 log (Fig. 5.3) reduction in virus levels during treatment, and using half the detection limit as surrogate for non-detect results. The extreme treatment assumptions were selected to provide an indication of the extent of spread in risk estimates.

Distributions of daily risk estimates, calculated per month - indicated on graphs in Fig 5.2 and 5.3 as Daily Risk, with months indicated as cells 1 to 12 along the horizontal axis - showed that the spread of risk estimates was small for months in which risks were low, but increased markedly in months during which daily risks peaked. The maximum spread between the 5th and 95th percentiles appeared to be approximately one order of magnitude.

Representations of average yearly risk calculated by sampling from a distribution of monthly geometric mean exposures - indicated on graphs as Mn Yr Risk - show the spread of values sampled from a single cell in the spreadsheet in which this value was calculated. The resultant distributions were usually approximately uniform for assumptions of both 4 log (Fig 5.2) and 6 log (Fig 5.3) reduction in virus levels during treatment, and for all three viruses, respectively (a-c per figure). One exception was observed. The distribution of sampled values for poliovirus 3, assuming a 4 log reduction during treatment, resembled an exponential distribution (Fig 5.2c, Mn Yr Risk). This effect was suppressed under an assumption of 6 log reduction (Fig 5.3c, Mn Yr Risk).

Distributions of yearly risk calculated from daily risk estimates - indicated as Yearly Risk on graphs in Fig 5.2 and 5.3 - generally took the form of approximately normal distributions. These risks were based on values sampled from monthly distributions of exposure, rather than from aggregated exposure data such as the distributions

of geometric means, discussed above. Yearly risks calculated assuming 4 log (Fig 5.2) reduction during treatment tended to be skewed slightly to the right. This effect was less noticeable when 6 log reduction was assumed (Figure 5.3). The normal distribution of yearly risks estimated from daily exposure and daily risks was in marked contrast to the uniform distributions obtained when risk was calculated by sampling from distributions of monthly geometric mean exposure, and supports the argument that calculation of yearly risk based on daily risk estimates is more representative of the full spread of risks occurring throughout the year.

The ability of dose-response models, developed on the basis of controlled volunteer human feeding studies, to predict infectivity of viruses in the environment to a heterogenous exposed population is a further source of uncertainty. Lack of suitable data to validate microbial dose-response models under environmental conditions precludes further description of uncertainty attributable to this source. It is unlikely that this question will be satisfactorily resolved in the foreseeable future.

A large source of uncertainty is the method used to calculate yearly risk since the methods employed in this study yielded widely disparate results. Calculation of yearly risk as the product of daily risks (Haas, personal communication) typically resulted in risk estimates up to 2.5 orders of magnitude greater than those obtained from a distribution of monthly geometric mean virus levels using the conversion described by Gerba and Haas (1988). These results in turn exceeded those obtained when an overall geometric mean was used instead of a distribution of monthly geometric means. It is not clear which method is preferable. In addition, international standardisation of the method for calculating yearly risks will be required.

It is clear that considerable uncertainty surrounds estimates of risk of infection associated with treated drinking water. These uncertainties must be borne in mind when interpreting risk estimates.

Fig. 5.2(a) Echovirus 12 - Daily risk

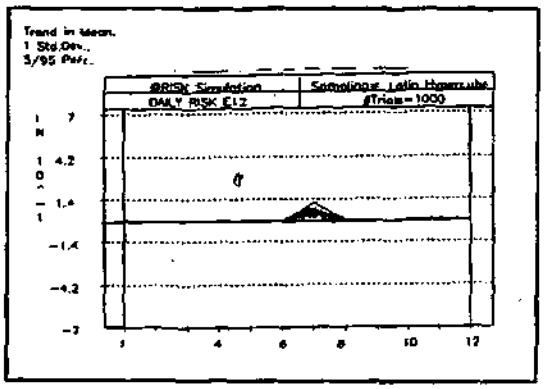

Fig. 5.2 (a, cont.) Echovirus 12 - Average yearly risk, calculated from distribution of monthly geometric mean data

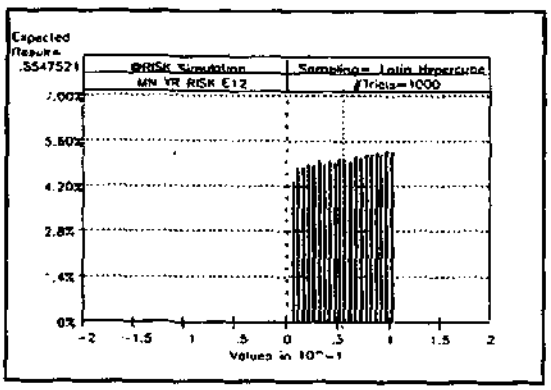

Fig. 5.2 (a, cont.) Echovirus 12 - Yearly risk, calculated directly from daily risk estimates

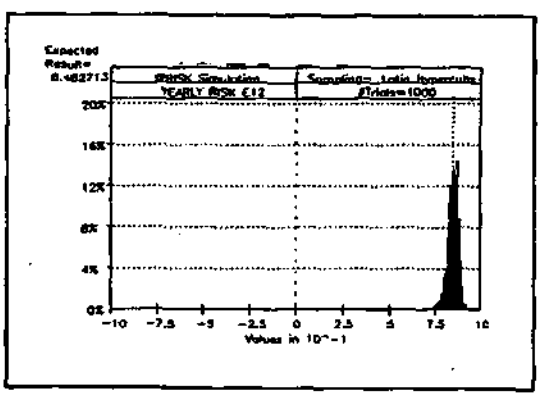

Fig. 5.2 Results of risk simulation with characterisation of uncertainty due to variability in monitoring data, assuming 4 log reduction during treatment, replacing non-detect results with 1/2 detection limit

Fig. 5.2 (b) Poliovirus 1 - Daily risk

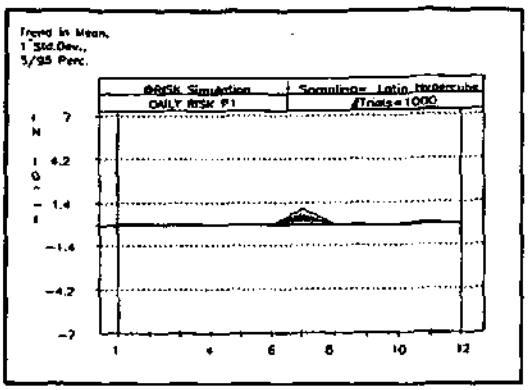

Fig. 5.2 (b, cont.) Poliovirus 1 - Average yearly risk, calculated from distribution of monthly geometric mean monitoring data

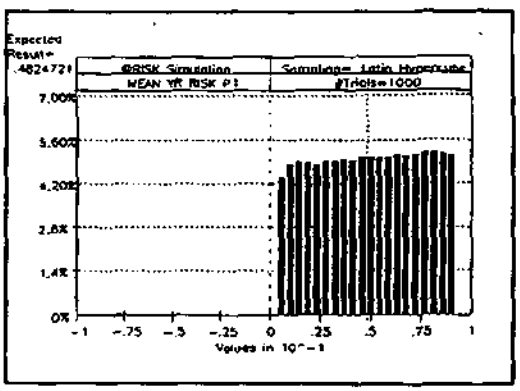

Fig. 5.2 (b, cont.) Poliovirus 1 - Yearly risk, calculated directly from daily risk estimates

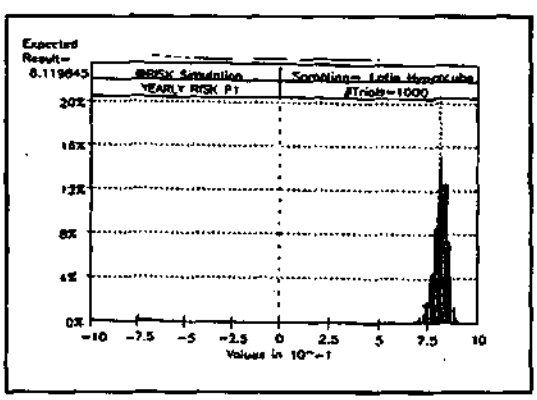

Fig. 5.2 cont. Results of risk simulation with characterisation of uncertainty due to variability in monitoring data, assuming 4 log reduction during treatment, replacing non-detect results with 1/2 detection limit

Fig. 5.2 (c) Poliovirus 3 - Daily risk

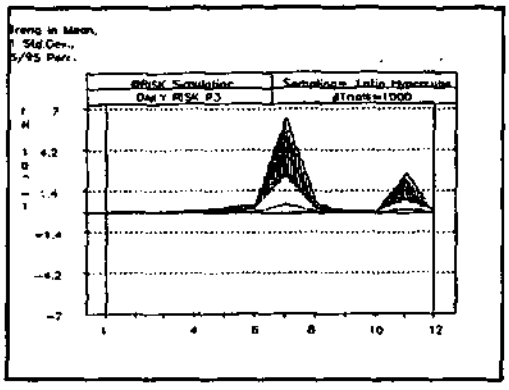

Fig. 5.2 (c, cont.) Poliovirus 3 - Mean yearly risk, calculated from distribution of monthly geometric mean monitoring data

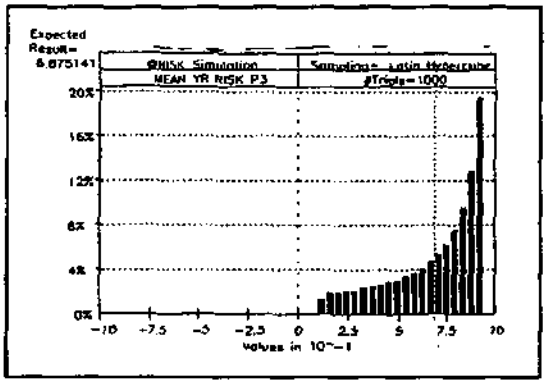

Fig. 5.2 *cont.* *Results of risk simulation with characterisation of uncertainty due to* variability in monitoring data, assuming 4 log reduction during treatment, replacing non-detect results with 1/2 detection limit

Fig. 5.3 (a) Echovirus 12 - Daily risk

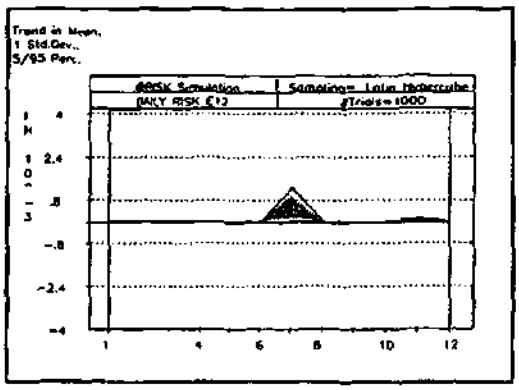

Fig. 5.3 (a, cont.) Echovirus 12 - Mean yearly risk, calculated from distribution of monthly geometric mean monitoring data

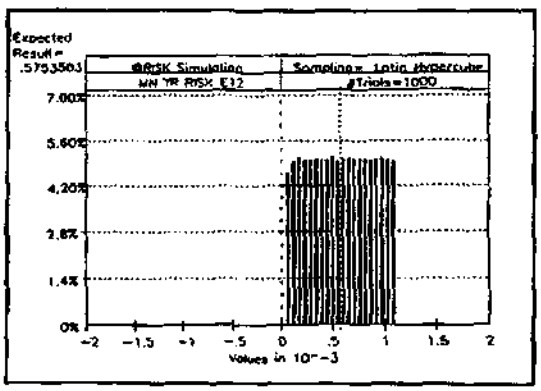

Fig. 5.3 (a, cont.) Echovirus 12 - Yearly risk calculated directly from daily risk estimates

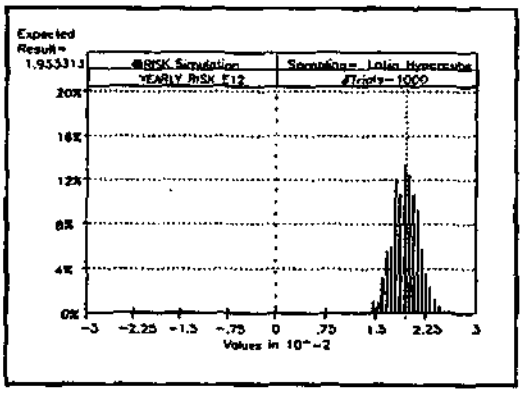

Fig. 5.3 Results of risk simulation with characterisation of uncertainty due to variability in monitoring data, assuming 6 log reduction during treatment, replacing non-detect results with 1/2 detection limit

Fig. 5.3 (b) Poliovirus 1 - Daily risk

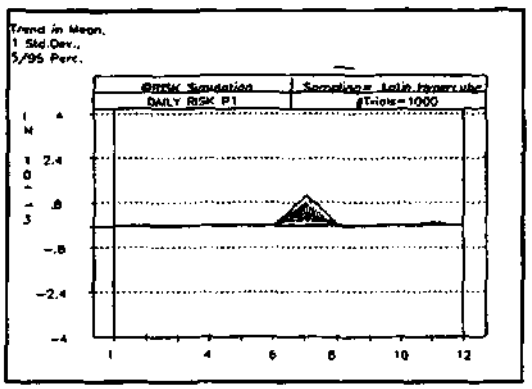

Fig. 5.3 (b, cont.) Poliovirus 1 - Mean yearly risk calculated from distribution of monthly geometric mean monitoring data

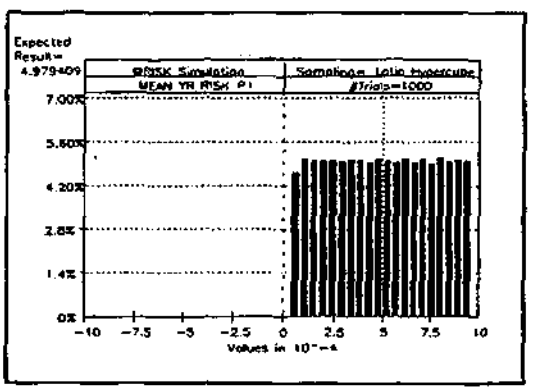

Fig. 5.3 (b, cont.) Poliovirus 1 - Yearly risk calculated directly from daily risk estimates

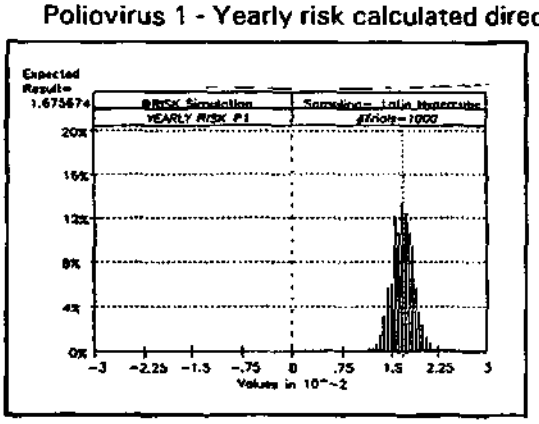

Fig. 5.3 cont. Results of risk simulation with characterisation of uncertainty due to variability in monitoring data, assuming 6 log reduction during treatment, replacing non-detect results with 1/2 detection limit

Fig. 5.3 (c) Poliovirus 3 - Daily risk

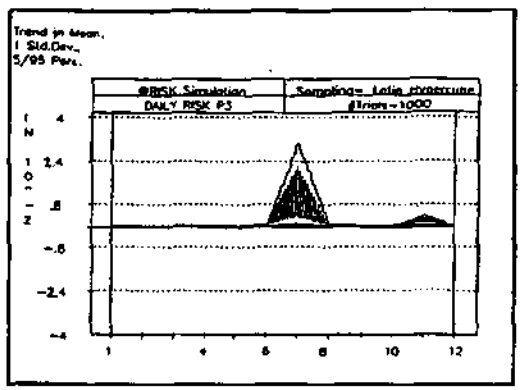

Fig. 5.3 (c, cont.) Poliovirus 3 - Mean yearly risk calculated from distribution of monthly geometric mean monitoring data

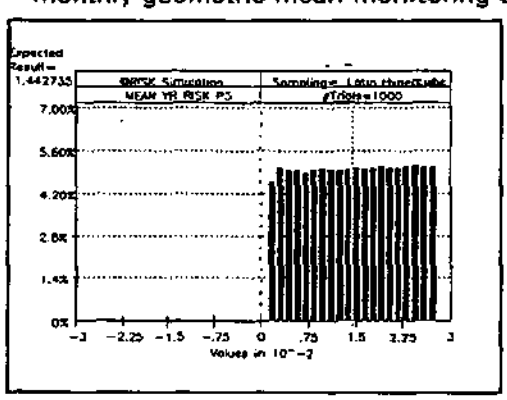

Fig. 5.3 (c, cont.) Poliovirus 3 - Yearly risk calculated directly from daily risk estimates

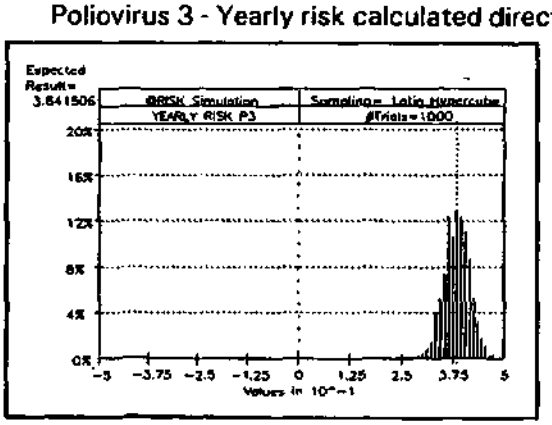

Fig. 5.3 cont. Results of risk simulation with characterisation of uncertainty due to variability in monitoring data, assuming 6 log reduction during treatment, replacing non-detect results with 1/2 detection limit

5.2. CASE STUDY 2: RECREATIONAL WATERS ENTERIC VIRUSES IN MARINE BATHING WATERS

5.2.1 Hazard identification

Transmission of infectious diseases through water used for recreational purposes has gained considerable attention in recent years. Epidemiological surveys have been conducted at marine and freshwater recreational sites in several countries, including the USA, Canada, UK, Egypt, Israel, Australia, Hong Kong and South Africa (Cabelli, 1983; Dufour, 1984; Holmes, 1989; Cheung *et al.*, 1990; Kay and Jones, 1992; Wheeler and Alexander, 1992; Grabow *et al.*, 1993; Harrington *et al.*, 1993; Von Schirnding *et al.*, 1993). Particular concern has centred around the marine environment because of the practice of disposing treated - and sometimes untreated - wastewater to sea, and because of the increasing popularity of bathing beaches. Despite ongoing debate surrounding the design of such studies and the interpretation of results, a probable association between microbiological water quality and swimming-associated illness was demonstrated in virtually all except the Australian study. Potentially contractible diseases include gastroenteritis; respiratory disease; eye, ear and skin infections; hepatitis A, cholera and typhoid. A number of these may be associated with enteric viruses. Viruses have been suggested as possibly the major aetiological agents of recreationally transmitted waterborne gastroenteritis (Jones *et al.*, 1990; Grabow *et al.*, 1993). Enteric viruses and their role as agents of waterborne disease have been described in section 5.1.

Survival of viruses in the marine environment is affected by the same factors as in freshwater environments, described in section 5.1. A number of additional factors also impact on viral persistence, particularly in relation to that of common indicator microorganisms. Both chemical and biological mechanisms appear important in influencing virus survival in the marine environment. However, a pure thermal effect is probably of less significance than in fresh water. Association of viruses with colloidal and particulate matter greatly prolongs survival, holding implications for the role of marine sediments as possible reservoirs of infective viruses (Kapuscinski and Mitchell, 1980). Members of the enteric virus group have been shown to respond to all the above factors, although the interaction of factors affected viruses differently (Chung and Sobsey, 1993). Since bacterial decay is accelerated in sea water, the disparity between levels of bacterial indicator organisms and viral pathogens may be exaggerated in marine waters (Kapuscinski and Mitchell, 1980).

Detection of viruses in seawater is conducted by the same methods as those employed for freshwater, and is subject to the same limitations. These have been described in section 5.1.

Both the existence of measurable, although relatively low, levels of viral pollution of South African coastal waters (Idema and Kfir, 1990) and the occurrence of swimming-associated health effects correlated with microbiological water quality

(Von Schirnding *et al.*, 1993) have been reported. It is unrealistic to expect these conditions to improve in the foreseeable future, largely due to rapid urban growth and the development of informal settlements along the coast and along water courses which discharge into the sea. Water pollution problems associated with such settlements have been described by Wright (1993). Epidemiological studies are valuable in identifying the existence of adverse health impacts and linking these to water quality. However, they cannot detect low levels of illness, or recreational exposure-associated illness associated with a specific etiological agent. It is in these respects that the application of risk assessment techniques to water quality monitoring data finds potential application. This is particularly true of agents such as viruses, which do not show adequate correlation with classical bacterial indicators in the marine environment and therefore are not included in epidemiological studies which evaluate water quality in terms of such indicator systems.

5.2.2 Exposure assessment

5.2.2.1 Source of hazard

This case study evaluated the probability of infection of recreational water users by enteric viruses present in marine water at a number of recreational sites along the Natal coast. These included two recognised bathing beaches and a river mouth which was not a recognised recreational site, but was used for various recreational pursuits. Discharge of domestic and industrial effluents from treatment works, and of stormwater and surface run-off from formal and informal urban developments, occurred along the coast. Upstream of the river mouth, effluents and surface run-off entered the river and was washed out to sea.

5.2.2.2 Exposed population

Large numbers of residents use the beaches and the river mouth throughout the year. During peak months, such as midsummer, numbers are considerably supplemented by inland visitors. Estimates of numbers of beach visitors are not available.

5.2.2.3 Exposure routes

All three sites are utilised for numerous recreational purposes requiring bodily contact with sea water, including paddling, swimming, angling and surfing. Dermal, inhalation and ingestion exposure occurs as a consequence of these activities. The major route of exposure investigated was ingestion, since this is the route explicitly evaluated by available risk models. However, it is likely that particularly inhalation exposure is of significance since several enteric viruses cause respiratory diseases via the inhalation route. In this instance, it is necessary to assume that the mode of infection, as modelled, is the same for such viruses as for enteric viruses which are infective by ingestion. The extent to which this assumption holds is not clear at present.

5.2.2.4 Exposure assumptions

Exposure was divided into a spring/summer season, comprising the seven months September through March and representing the main swimming season, and an autumn/winter season, comprising the months April through August and representing the off-peak period for recreational water contact. During these periods, two frequencies of exposure were considered. Firstly, a typical frequency for holiday makers of 7 exposure events per year was assumed, based on estimates of swimming events used in risk assessment software developed for the US EPA (Hampshire Research Institute, 1991). Secondly, an exposure frequency of 250 events per year, corresponding to five days per week, was assumed as a worst case exposure representative of local residents engaging in activities such as surfing and lifesaving.

Two exposure volumes were considered. The estimate of ingestion of 100 mℓ water per swimming event, used in the development of Australian recreational water quality guidelines (National Health and Medical Research Council, 1990) and accepted in the development of guidelines by several other bodies, was accepted. An alternative exposure of 10 mℓ per swimming event, which may be more realistic of accidental ingestion of saline water, was also considered.

5.2.2.5 Exposure monitoring

Sea water at sites along the Natal coast were monitored for enteric viruses and other microbiological water quality variables from March 1980 to September 1991. Sample concentration and detection of viruses was performed as described in section 5.1.

5.2.2.6 Analysis of monitoring data

No enteric viruses were detected in water collected at the two recognised bathing beaches. Viruses were detected in 5 out of 24 samples collected near the river mouth. results were divided into a spring/summer season and an autumn/winter season, as described, and are summarised in Table 5.2.1. The occurrence of one outlying result in the autumn/winter data set made it difficult to determine from aggregated data in which season the greater incidence of viruses was detected. However, this positive result was the only instance of virus detection in the autumn/winter season. All other positive results occurred in the spring/summer season.

Table 5.2.1 Enteric virus levels in marine water at a river mouth used for recreation

Season	Enteric virus MPN $TCID_{50}$ per 10 ℓ		
	Spring/summer*	Autumn/winter	both seasons combined
Mean	45.8	39.7	40.6
Geometric mean	41.4	65.0	54.6
Minimum	31.5 (ND**)	31.5 (ND)	31.5 (ND)
Maximum	39.0	400***	400***

* Spring/summer season includes months September through March
Autumn/winter season includes months April through August
** ND = Surrogate for non-detect result
*** Maximum value is an outlier

As in case study 1, all enteric viruses were treated as enteroviruses, as represented by poliovirus 1, poliovirus 3 and echovirus 12. Non-detect results were replaced with the detection limit, half the detection limit or zero, with 1 MPN $TCID_{50}/10\ell$ used in place of zero for calculation of geometric means.

Distributions of data for the spring/summer season, autumn/winter season and combined over both seasons were tested for goodness of fit to a log-normal and other distributions, as described in case study 1. No distribution with significant fit to the data was identified, hence monitoring data were represented as general distributions in the spreadsheet used in dose-response assessment and risk characterisation.

5.2.3 Dose-response assessment

5.2.3.1 Infectious dose

The difficulties in determining the infectious dose of enteric viruses and likely ranges of infectious dose were discussed in case study 1. The same considerations apply for exposure resulting from recreational activities in marine water polluted with viruses.

5.2.3.2 Dose-response model

The beta-distributed infectivity probability model was employed, as outlined in section 4. Echovirus 12, poliovirus 1 and poliovirus 3 were again used as representative enteric viruses. The same parameter values as presented in section

4 were used.

Maximum daily risks were estimated from distributions of monitoring data, as described, for the spring/summer season, the autumn/winter season and for both seasons combined. Seasonal and yearly (both seasons combined) risk was estimated by three methods. Firstly, the seasonal or overall geometric mean was used as an estimator of average exposure to calculate average seasonal or average yearly risk. Secondly, yearly risk was calculated by sampling once from the defined general distributions of seasonal or total monitoring data and calculating the longer term risk according to Formula 2 in section 4. Finally, risk was calculated by sampling exposure from the distribution of monitoring data for each day of the postulated exposure period and calculating the longer term risk as the product of the daily risk estimates, as illustrated in Table 4.1. in section 4.

Uncertainty in risk estimates arising from variations in detected virus levels was simulated using the @Risk function in the microbial risk assessment spreadsheet.

5.2.4 Risk characterisation

5.2.4.1 Estimates of daily, seasonal and yearly risk of infection

Recognised bathing beaches

Since no enteric viruses were detected at either of the two monitored bathing beaches, daily and yearly risk was estimated at the detection limit, representing the lowest directly detectable risk. Daily and seasonal or yearly risk estimates based on two exposure volumes for the three representative viruses are presented in Table 5.2.2. Assuming ingestion of a maximum of 100mℓ contaminated water per exposure event, the minimum daily risk detectable at the two beaches ranged from $9x10^{-3}$ to $2x10^{-1}$, where echovirus 12 and polioviruses 1 and 3 are considered representative enteric viruses. If ingestion exposure is limited to 10mℓ per recreational event, the risks decrease to $1x10^{-3}$ to $1x10^{-1}$. If it is assumed that the detection limit can be safely replaced by half the detection limit as a surrogate for non-detect results, then risk estimates at the two bathing beaches decrease to $5x10^{-3}$ to $1x10^{-1}$ for ingestion of 100mℓ water per recreational event, and to $5x10^{-4}$ to $1x10^{-2}$ for ingestion of 10mℓ per recreational event.

Longer term risks were estimated, assuming a total of 7 exposures per year or 250 exposures per year. In considering the lower exposure frequency, exposures were considered as occurring in the spring/summer season, the autumn winter season or throughout the year. Assuming ingestion of 100 mℓ contaminated water per recreational event, seasonal or annual risks of infection were in the range $6x10^{-2}$ to $8x10^{-1}$ at the detection limit. If ingestion of 10mℓ water was assumed, risks were $7x10^{-3}$ to $2x10^{-1}$. At half the detection limit, risks were $3x10^{-2}$ to $6x10^{-1}$ for ingestion of 100mℓ and $3x10^{-3}$ to $9x10^{-1}$ for ingestion of 100mℓ.

For the higher exposure frequency, it was assumed that 104 of the 250 exposure

events occurred in autumn/winter and 146 occurred in spring/summer. Spring/summer, autumn/winter and yearly risks at the detection limit, and assuming ingestion of 100 mℓ, were all at or close to 1. Assuming ingestion of 10mℓ, estimated risks ranged from $1x10^{-1}$ to 1 in spring/summer, $9x10^{-2}$ to $9x10^{-1}$ in autumn/winter and $2x10^{-1}$ to 1 over both seasons. At half the detection limit, assuming ingestion of 100 mℓ per recreational event, risks were $5x10^{-1}$ to 1 in spring/summer, $4x10^{-1}$ to 1 in autumn/winter and $7x10^{-1}$ to 1 over both seasons. Assuming ingestion of 10mℓ, these estimates dropped to $7x10^{-2}$ to $9x10^{-1}$ in spring/summer, $5x10^{-2}$ to $8x10^{-1}$ in autumn/winter and $1x10^{-1}$ to 1 over both seasons. Risk estimates are presented in Table 5.2.2.

Table 5.2.2 Daily and seasonal/yearly risks of enteric virus infection calculated at two recognised bathing beaches

	Daily risk of infection	Seasonal or yearly risk of infection			
		7 day exposure Spring/ summer or Autumn/ winter or both seasons	146 day exposure Spring/ summer season of total yearly exposure of 250 days	104 day exposure Autumn/ winter season of total yearly exposure of 250 days	250 day exposure Both seasons combined
Echovirus 12					
At detection limit:					
100 mℓ	1×10^{-2}	7×10^{-2}	8×10^{-1}	7×10^{-1}	9×10^{-1}
10 mℓ	1×10^{-3}	8×10^{-3}	2×10^{-1}	1×10^{-1}	2×10^{-1}
At half detection limit:					
100 mℓ	5×10^{-3}	4×10^{-2}	6×10^{-1}	4×10^{-1}	7×10^{-1}
10 mℓ	6×10^{-4}	4×10^{-3}	8×10^{-2}	5×10^{-2}	1×10^{-1}
Poliovirus 1					
At detection limit:					
100 mℓ	9×10^{-3}	6×10^{-2}	8×10^{-1}	6×10^{-1}	9×10^{-1}
10 mℓ	1×10^{-3}	7×10^{-3}	1×10^{-1}	9×10^{-2}	2×10^{-1}
At half detection limit:					
100 mℓ	5×10^{-3}	3×10^{-2}	5×10^{-1}	4×10^{-1}	7×10^{-1}
10 mℓ	5×10^{-4}	3×10^{-3}	7×10^{-2}	5×10^{-2}	1×10^{-1}
Poliovirus 3					
At detection limit:					
100 mℓ	2×10^{-1}	8×10^{-1}	1	1	1
10 mℓ	3×10^{-2}	2×10^{-1}	1	9×10^{-1}	1
At half detection limit:					
100 mℓ	1×10^{-1}	6×10^{-1}	1	1	1
10 mℓ	1×10^{-2}	9×10^{-2}	9×10^{-1}	8×10^{-1}	1

Beach at river mouth

Maximum daily risks of enteric virus infection at the beach situated at the river mouth were calculated in spring/summer, in autumn/winter and for both seasons combined. Results are shown in Table 5.2.3. Risks were of the order of 10^{-2} for echovirus 12 and poliovirus 1 at an assumed ingestion of 100 mℓ, and approximately an order of magnitude lower if ingestion of 10 mℓ per recreational event was assumed. Estimates of daily risk for poliovirus 3 were in the vicinity of 10^{-1} at an assumed ingestion of 100 mℓ and 10^{-2} at an assumed ingestion of 10 mℓ. Assumptions regarding the value substitute for non-detect results caused a variation of up to half an order of magnitude in estimated risks.

Table 5.2.3 *Maximum daily risk of enteric virus infection estimated at a river mouth used for recreation*

Assumed volume ingested	Expected value of daily risk		
	Spring/summer season	*Autumn/winter season*	*Both seasons combined*
Echovirus 12			
Non-detect value: 63 MPN $TCID_{50}$/10ℓ			
100 mℓ	$1x10^{-2}$	$3x10^{-2}$	$4x10^{-2}$
10 mℓ	$1x10^{-3}$	$3x10^{-3}$	$4x10^{-3}$
Non-detect value: 31.5 MPN $TCID_{50}$/10ℓ			
100 mℓ	$9x10^{-3}$	$3x10^{-2}$	$2x10^{-2}$
10 mℓ	$9x10^{-4}$	$3x10^{-3}$	$3x10^{-3}$
Non-detect value: 0 MPN $TCID_{50}$/10ℓ			
100 mℓ	$6x10^{-3}$	$2x10^{-2}$	$2x10^{-2}$
10 mℓ	$5x10^{-4}$	$3x10^{-3}$	$2x10^{-3}$
Poliovirus 1			
Non-detect value: 63 MPN $TCID_{50}$/10ℓ			
100 mℓ	$1x10^{-2}$	$3x10^{-2}$	$3x10^{-2}$
10 mℓ	$1x10^{-3}$	$3x10^{-3}$	$3x10^{-3}$
Non-detect value: 31.5 MPN $TCID_{50}$/10ℓ			
100 mℓ	$8x10^{-3}$	$2x10^{-2}$	$2x10^{-2}$
10 mℓ	$8x10^{-4}$	$2x10^{-3}$	$2x10^{-3}$
Non-detect value: 0 MPN $TCID_{50}$/10ℓ			
100 mℓ	$5x10^{-3}$	$2x10^{-2}$	$2x10^{-2}$
10 mℓ	$4x10^{-4}$	$2x10^{-3}$	$2x10^{-3}$
Poliovirus 3			
Non-detect value: 63 MPN $TCID_{50}$/10ℓ			
100 mℓ	$2x10^{-1}$	$4x10^{-1}$	$4x10^{-1}$
10 mℓ	$3x10^{-2}$	$7x10^{-2}$	$8x10^{-2}$
Non-detect value: 31.5 MPN $TCID_{50}$/10ℓ			
100 mℓ	$2x10^{-1}$	$3x10^{-1}$	$3x10^{-1}$
10 mℓ	$2x10^{-2}$	$6x10^{-2}$	$6x10^{-2}$
Non-detect value: 0 MPN $TCID_{50}$/10ℓ			
100 mℓ	$1x10^{-1}$	$3x10^{-1}$	$2x10^{-1}$
10 mℓ	$1x10^{-2}$	$6x10^{-2}$	$4x10^{-2}7$

Average seasonal or yearly risks, at 7 days exposure per season or per year, ranged from $2x10^{-2}$ to $8x10^{-2}$ for echovirus 12 and poliovirus 1, assuming ingestion of 100 mℓ and an order of magnitude lower assuming ingestion of 10 mℓ. Risk estimates were more than an order of magnitude higher for poliovirus 3, increasing up to 1. Average risks assuming 250 days exposure per year (146 days in spring/summer and 104 days in autumn/winter) ranged between $5x10^{-1}$ and 1 at an assumed ingestion of 100 mℓ and replacing non-detect results with either the detection limit or half the detection limit in calculation of the geometric mean. Replacement of non-detect results with a value of 1 MPN $TCID_{50}$ per 10 ℓ yielded risk estimates almost an order of magnitude lower. Assumption of ingestion of 10 mℓ per recreational event resulted in estimates of average seasonal and yearly risks an order of magnitude lower than at the higher volume. Average risks calculated for poliovirus 3 were, in general, approximately 1.5 orders of magnitude higher than the corresponding results calculated for poliovirus 1 and echovirus 12. Estimates of average seasonal and yearly risks, based on geometric mean monitoring data, are presented in Table 5.2.4.

Table 5.2.4 Average seasonal and yearly risks of enteric virus infection associated with marine water at a river mouth used for recreation, calculated from seasonal or yearly geometric mean virus levels

	Expected value of risk					
	Spring/summer season		Autumn/winter season		Both seasons	
Assumed volume of water ingested	7 days exposure	146 days exposure	7 days exposure	104 days exposure	7 days exposure	250 days exposure
Echovirus 12						
Non detect value: 63 MPN $TCID_{50}$/10 ℓ						
100 mℓ	$8x10^{-2}$	$8x10^{-1}$	$9x10^{-2}$	$7x10^{-1}$	$8x10^{-2}$	1
10 mℓ	$8x10^{-3}$	$2x10^{-1}$	$9x10^{-3}$	$1x10^{-1}$	$9x10^{-3}$	$2x10^{-1}$
Non detect value: 31.5 MPN $TCID_{50}$/10 ℓ						
100 mℓ	$5x10^{-2}$	$7x10^{-1}$	$5x10^{-2}$	$5x10^{-1}$	$5x10^{-2}$	$8x10^{-1}$
10 mℓ	$5x10^{-3}$	$1x10^{-1}$	$5x10^{-3}$	$7x10^{-2}$	$5x10^{-3}$	$2x10^{-1}$
Non detect value: 0 MPN $TCID_{50}$/10 ℓ						
100 mℓ	$5x10^{-3}$	$9x10^{-2}$	$2x10^{-3}$	$3x10^{-2}$	$3x10^{-3}$	$1x10^{-1}$
10 mℓ	$5x10^{-4}$	$1x10^{-2}$	$2x10^{-4}$	$3x10^{-3}$	$3x10^{-4}$	$1x10^{-2}$
Poliovirus 1						
Non detect value: 63 MPN $TCID_{50}$/10 ℓ						
100 mℓ	$7x10^{-2}$	$8x10^{-1}$	$6x10^{-2}$	$7x10^{-1}$	$7x10^{-2}$	$9x10^{-1}$
10 mℓ	$7x10^{-3}$	$1x10^{-1}$	$8x10^{-3}$	$1x10^{-1}$	$7x10^{-3}$	$2x10^{-1}$

Assumed volume of water ingested	Expected value of risk					
	Spring/summer season		Autumn/winter season		Both seasons	
	7 days exposure	146 days exposure	7 days exposure	104 days exposure	7 days exposure	250 days exposure
Non detect value: 31.5 MPN $TCID_{50}/10\ell$						
100 mℓ	$4x10^{-2}$	$6x10^{-1}$	$4x10^{-2}$	$5x10^{-1}$	$4x10^{-2}$	$8x10^{-1}$
10 mℓ	$4x10^{-3}$	$9x10^{-2}$	$4x10^{-3}$	$6x10^{-2}$	$4x10^{-3}$	$1x10^{-1}$
Non detect value: 0 MPN $TCID_{50}/10\ell$						
100 mℓ	$4x10^{-3}$	$8x10^{-2}$	$2x10^{-3}$	$3x10^{-2}$	$3x10^{-3}$	$1x10^{-1}$
10 mℓ	$4x10^{-4}$	$8x10^{-3}$	$2x10^{-4}$	$3x10^{-3}$	$3x10^{-4}$	$1x10^{-2}$
Poliovirus 3						
Non detect value: 63 MPN $TCID_{50}/10\ell$						
100 mℓ	$8x10^{-1}$	1	$8x10^{-1}$	1	$8x10^{-1}$	1
10 mℓ	$2x10^{-1}$	1	$2x10^{-1}$	1	$2x10^{-1}$	1
Non detect value: 31.5 MPN $TCID_{50}/10\ell$						
100 mℓ	$6x10^{-1}$	1	$7x10^{-1}$	1	$7x10^{-1}$	1
10 mℓ	$1x10^{-1}$	$9x10^{-1}$	$1x10^{-1}$	$8x10^{-1}$	$1x10^{-1}$	1
Non detect value: 0 MPN $TCID_{50}/10\ell$						
100 mℓ	$1x10^{-1}$	$9x10^{-1}$	$5x10^{-2}$	$5x10^{-1}$	$8x10^{-2}$	$9x10^{-1}$
10 mℓ	$1x10^{-2}$	$2x10^{-1}$	$5x10^{-3}$	$8x10^{-2}$	$8x10^{-3}$	$2x10^{-1}$

When seasonal and yearly risks were calculated iteratively by sampling exposure from a distribution of monitoring data (most likely risk), risks for a 7-day seasonal or yearly exposure were in the range $3x10^{-2}$ to $2x10^{-1}$ at an ingested volume of 100 mℓ, and an order of magnitude lower than ingested volume of 10 mℓ, for echovirus 12 and poliovirus 1. Estimates using zero as a surrogate for non-detect result were up to half an order of magnitude lower than those using the detection limit or half the detection limit, which were generally similar. Estimates of seasonal and yearly risk assuming 250 days exposure per year (146 days in summer/spring and 104 days in autumn/winter) were generally close to 1 at an assumed ingestion of 100 mℓ for echovirus 12 and poliovirus 1 and using the detection limit or half the detection limit in place of non-detect values. They were a little lower, but still $4x10^{-1}$ or greater, when zero was substituted for non-detect values. Estimated risks at an ingested volume of 10 mℓ were up to one order of magnitude lower than the corresponding value of 100 mℓ. Risks estimated for poliovirus 3 were generally 1 or closely approached 1. Risk estimates are shown in Table 5.2.5.

Estimation of seasonal and yearly risks by sampling daily exposures from

distributions of monitoring data and calculating longer term risk by the product of daily risk estimates (cumulative risk estimates) yielded results similar to those obtained when longer term risk was calculated as a power function of a single daily risk estimate. Estimation of longer term risks from distributions of monitoring data segregated by season yielded comparable results to those obtained when all monitoring data were pooled in a single distribution from which exposures were sampled. Risks calculated by the product of daily risk estimates are shown in Table 5.2.6.

Table 5.2.5 Average seasonal and yearly risks of enteric virus infection associated with marine water at a river mouth used for recreation, calculated by sampling once from a distribution of monitoring data and determining seasonal or yearly risk as a power function.

	Expected value of risk					
	Spring/summer season		Autumn/winter season		Both seasons	
	7 days	*146 days*	*7 days*	*104 days*	*7 days*	*250 days*
Echovirus 12						
Non detect value: 63 MPN $TCID_{50}/10\ell$						
100 mℓ	9×10^{-2}	9×10^{-1}	2×10^{-1}	1	9×10^{-2}	1
10 mℓ	9×10^{-3}	2×10^{-1}	2×10^{-2}	3×10^{-1}	9×10^{-3}	6×10^{-1}
Non detect value: 31.5 MPN $TCID_{50}/10\ell$						
100 mℓ	6×10^{-2}	7×10^{-1}	2×10^{-1}	9×10^{-1}	6×10^{-2}	9×10^{-1}
10 mℓ	6×10^{-3}	1×10^{-1}	2×10^{-2}	3×10^{-1}	6×10^{-3}	4×10^{-1}
Non detect value: 0 MPN $TCID_{50}/10\ell$						
100 mℓ	4×10^{-2}	5×10^{-1}	2×10^{-1}	8×10^{-1}	3×10^{-2}	7×10^{-1}
10 mℓ	4×10^{-3}	7×10^{-2}	2×10^{-2}	2×10^{-1}	3×10^{-3}	3×10^{-1}
Poliovirus 1						
Non detect value: 63 MPN $TCID_{50}/10\ell$						
100 mℓ	8×10^{-2}	8×10^{-1}	2×10^{-1}	9×10^{-1}	8×10^{-2}	1
10 mℓ	8×10^{-3}	2×10^{-1}	2×10^{-2}	2×10^{-1}	8×10^{-3}	5×10^{-1}
Non detect value: 31.5 MPN $TCID_{50}/10\ell$						
100 mℓ	5×10^{-2}	7×10^{-1}	2×10^{-1}	8×10^{-1}	5×10^{-2}	9×10^{-1}
10 mℓ	6×1^{-3}	1×10^{-1}	2×10^{-2}	2×10^{-1}	5×10^{-3}	4×10^{-1}
Non detect value: 0 MPN $TCID_{50}/10\ell$						
100 mℓ	3×10^{-2}	4×10^{-1}	1×10^{-1}	8×10^{-1}	3×10^{-2}	7×10^{-1}
10 mℓ	3×10^{-3}	6×10^{-2}	2×10^{-2}	2×10^{-1}	3×10^{-3}	3×10^{-1}
Poliovirus 3						
Non detect value: 63 MPN $TCID_{50}/10\ell$						
100 mℓ	8×10^{-1}	1	1	1	8×10^{-1}	1
10 mℓ	2×10^{-1}	1	4×10^{-1}	1	2×10^{-1}	1

	Expected value of risk					
	Spring/summer season		Autumn/winter season		Both seasons	
	7 days	146 days	7 days	104 days	7 days	250 days
Non detect value: 31.5 MPN $TCID_{50}/10\ell$						
100 mℓ	7×10^{-1}	1	9×10^{-1}	1	7×10^{-1}	1
10 mℓ	2×10^{-1}	9×10^{-1}	4×10^{-1}	1	1×10^{-1}	1
Non detect value: 0 MPN $TCID_{50}/10\ell$						
100 mℓ	5×10^{-1}	1	8×10^{-1}	1	5×10^{-1}	1
10 mℓ	8×10^{-2}	6×10^{-1}	3×10^{-1}	9×10^{-1}	7×10^{-2}	8×10^{-1}

Table 5.2.6 Average seasonal and yearly risks of enteric virus infection associated with marine water at a river mouth used for recreation, calculated from daily risk estimates based on sampling from a distribution of exposures for each day of the exposure period

	Expected value of risk				
	Spring/ summer season	Autumn/ winter season	Both seasons		
	7 days	7 days	7 days	146 days spring/ summer + 104 days autumn/ winter*	250 days**
Echovirus 12					
Non detect value: 63 MPN $TCID_{50}/10\ell$					
100 mℓ	9×10^{-2}	2×10^{-1}	2×10^{-1}	1	1
10 mℓ	9×10^{-3}	2×10^{-2}	3×10^{-2}	4×10^{-1}	6×10^{-1}
Non detect value: 31.5 MPN $TCID_{50}/10\ell$					
100 mℓ	6×10^{-2}	2×10^{-1}	2×10^{-1}	1	1
10 mℓ	6×10^{-3}	2×10^{-2}	2×10^{-2}	4×10^{-1}	5×10^{-1}
Non detect value: 0 MPN $TCID_{50}/10\ell$					
100 mℓ					
10 mℓ	4×10^{-2}	2×10^{-1}	1×10^{-1}	1	1
	4×10^{-3}	2×10^{-2}	2×10^{-2}	3×10^{-1}	4×10^{-1}
Poliovirus 1					
Non detect value: 63 MPN $TCID_{50}/10\ell$					
100 mℓ	8×10^{-2}	2×10^{-1}	2×10^{-1}	1	1
10 mℓ	8×10^{-3}	2×10^{-2}	2×10^{-2}	4×10^{-1}	4×10^{-1}
Non detect value: 31.5 MPN $TCID_{50}/10\ell$					
100 mℓ	5×10^{-2}	2×10^{-1}	1×10^{-1}	1	1
10 mℓ	6×10^{-3}	2×10^{-2}	2×10^{-2}	3×10^{-1}	3×10^{-1}
Non detect value: 0 MPN $TCID_{50}/10\ell$					
100 mℓ					
10 mℓ	3×10^{-2}	1×10^{-1}	1×10^{-1}	1	1
	3×10^{-3}	2×10^{-2}	1×10^{-2}	3×10^{-1}	3×10^{-1}

	Expected value of risk				
	Spring/ summer season	Autumn/ winter season	Both seasons		
	7 days	7 days	7 days	146 days spring/ summer + 104 days autumn/ winter*	250 days**
Poliovirus 3					
Non detect value: 63 MPN $TCID_{50}/10\ell$					
100 mℓ	$8x10^{-1}$	1	1	1	1
10 mℓ	$2x10^{-1}$	$4x10^{-1}$	$4x10^{-1}$	1	1
Non detect value: 31.5 MPN $TCID_{50}/10\ell$					
100 mℓ	$7x10^{-1}$	$9x10^{-1}$	$9x10^{-1}$	1	1
10 mℓ	$2x10^{-1}$	$4x10^{-1}$	$3x10^{-1}$	1	1
Non detect value: 0 MPN $TCID_{50}/10\ell$					
100 mℓ	$6x10^{-1}$	$9x10^{-1}$	$8x10^{-1}$	1	1
10 mℓ	$8x10^{-2}$	$3x10^{-1}$	$2x10^{-1}$	1	1

* Exposure sampled from spring/summer distribution for 146 days of the 250 day exposure period, and from the autumn/winter distribution for 104 days of the 250 day exposure period.

** Exposure sampled from a combined distribution of all monitoring data over spring/summer and autumn/winter seasons.

Discussion of risk estimates

The US EPA has recommended an acceptable yearly risk of infection of 10^{-4} for drinking water. On the basis of different exposure patterns, a higher acceptable risk of 10^{-3} has been suggested for recreational water contact (Rose, personal communication). Compliance was not achieved with either value in any longer term risk estimates, under any of the exposure or calculation assumptions tested. Daily risk estimates only occasionally met these recommended levels, and even risk estimates at the detection limit or half the detection limit frequently failed to comply. This strongly indicates that monitoring methods based on current sample volumes, are lacking in sensitivity for detecting sufficiently low risk levels.

Risk estimates attained using an assumed intake of 10 mℓ water and a seasonal or yearly exposure of 7 days occasionally met the recommended 10^{-3} risk guideline suggested by Rose. It should be borne in mind that current assumptions regarding non-detect results are likely to grossly over-estimate actual virus levels in these

samples. Hence the achievement of the guideline values given these extreme assumptions is a promising indication that better knowledge of actual levels in samples presently reported as non-detect would yield risk estimates which met the guideline.

For both daily and longer term risk estimates, risk in the autumn/winter season closely approached those for both seasons combined. However, when the single outlying result was removed from the autumn/winter data set, risk estimates for the combined season more closely approached that of the spring/summer season, with those of the autumn/winter season markedly lower. This latter scenario is probably the more realistic.

Under all exposure scenarios, risk estimates were similar when non-detect results were substituted with either the detection limit or half the detection limit. A noticeable decrease occurred when a value of 0 (1/10 ℓ for determination of geometric means) was used. However, this effect was not substantial, causing a maximum of half an order of magnitude reduction in risk estimates.

Differences in assumptions regarding the volume of water ingested resulted in changes of between half and one order of magnitude in the risk estimates.

Assumptions in exposure frequency markedly affected estimates of longer term risks of infection. At an assumed exposure of 7 days per season or per year, risks rarely approached 1, while most risks at 250 days exposure per year, spread over both seasons, in general closely approached or were 1.

Risk estimates for echovirus 12 and poliovirus 1 were up to one and a half orders of magnitude below those for poliovirus 3. The same observation was made for the drinking water case study. This appears to indicate differing mechanisms of infection.

Unlike risks estimated in the drinking water case study, longer term risk estimates calculated as a power of a single daily risk estimate were not markedly lower than risks calculated as the product of daily risk estimates for each day of the assumed exposure period. Three factors may account for this. In this case study the distributions on which determination of the single daily risk estimated was based for the former method was the same as the distribution from which daily exposures were sampled and daily risks calculated over the entire exposure period. In the previous case study, a distribution of monthly geometric means was used in the former method. Secondly, in the recreational exposure case study, longer term risk were already generally approaching the upper limits of risk which could be calculated and were therefore less strongly influenced than in the case of lower risks in the drinking water case study. Finally, the exposure period of 7 days was sufficiently low that the probability of extreme exposure values being sampled from the distribution of monitoring data on one of the days of the exposure period was relatively low.

From the above, the greatest limitation on detection of low risk levels was the

volume of water sampled. This, combined with the relatively high detection limit, resulted in large numbers of non-detect results for which results had to be assumed. The value used in place of non-detect results made little difference to the risk estimate, while in the range of half the detection limit to the detection limit were noticeable, but not marked, reduction in risk estimate occurred when zero was used as surrogate. Exposure frequency affected longer term risk estimates strongly. This may be expected since the frequency considered (250 days per year) represented an extreme situation. Data on the marine bathing behaviour of South African recreational water users is required. Methods of calculation of longer term risks did not, in this case, strongly affect the estimates obtained.

5.2.4.2 Characterisation of uncertainty

A number of sources of uncertainty in this estimation of risks of enteric virus infection associated with marine recreational water exposure have already been discussed in section 5.1. These include uncertainties arising from virus recovery and detection methods, surrogate values used for non-detect results occurring due to the volume of water sampled and the detection limit, the degree to which the three enteroviruses considered are representative of the enteric virus group and uncertainties inherent in the dose-response model used. Uncertainties relating to the method used for the calculation of longer term risks apply to this study as well as to the preceding case study, but the effect was less marked than in the drinking water case study.

The extent of uncertainty associated with the choice of surrogate values for non-detect results, assumptions regarding exposure frequency and volume and calculation method have been indicated in the discussion of risk estimates above.

A great deal of uncertainty is introduced into the risk assessment by the lack of exposure information. All aspects of exposure were dealt with by assumption. Information specific to marine bathing behaviours in South Africa, and preferably to the locality being evaluated are necessary to reduce such uncertainty. This would include data such as bathing frequency, preferred seasons, extent of immersion during bathing, estimates of volumes of water ingested, and concurrent exposures which could make bathers more or less susceptible to infection. Data available on an age-specific basis would be especially useful since adults and children differ markedly in the frequency at which they enter the water, the extent of immersion and the degree of accidental ingestion of water. Another significant difference is that adults will tend to swim only if healthy, while children will often swim while suffering from minor infections such as colds and coughs. Such children would naturally be more susceptible to infection.

The above sources of uncertainty can all be recognised and described to a greater or lesser extent, but only limited quantitation was possible. Uncertainty associated with variability in monitoring data and hence in at least the water quality aspect of exposure, could be modelled to some extent by specifying exposures as distributions of monitoring data and evaluating the distribution of risks determined by sampling from these distributions of postulated exposure.

5.3 CASE STUDY 3: SHELLFISH CONSUMPTION ENTERIC VIRUSES IN SHELLFISH

5.3.1. *Hazard Identification*

Enteric virus transmission due to consumption of faecally contaminated shellfish is a significant public health concern. Microbial pollution in shellfish-growing waters is a common phenomenon on most coasts (Martinez-Manzanares *et al.*, 1993). Pollution from human activities can lead to the potential risk of diseases transmitted by the consumption of raw or lightly cooked shellfish.

A wide range of pathogens, including enteric viruses may accumulate in shellfish grown in polluted water and consumption of polluted shellfish has been associated with a range of infectious diseases. One of the largest recorded outbreaks of shellfish-associated disease was of hepatitis A in China and was epidemiologically linked to the consumption of uncooked clams contaminated with hepatitis A virus (Halliday, 1991). Uncontrolled urbanisation along the South African coastline is often accompanied by limited sanitary facilities, resulting in an increased discharge of polluted stormwater run-off and effluent into the marine environment. This increased pollution may subsequently pollute shellfish growing in the marine environment with pathogenic organisms such as enteric viruses.

Bivalves such as mussels and oysters are filter feeders, filtering large volumes of seawater. In doing so, they may accumulate pathogenic organisms of human and animal origin.

In South Africa, guidelines for marine water quality refer to the water quality of the area where shellfish are collected and cultured. Faecal coliforms are used as an index of quality of shellfish growing waters, with the percentage of water samples that fulfil a certain microbiological level being established. However, pathogens are frequently detected in shellfish cultured in water of "acceptable" quality (Martinez-Manzanares *et al.*, 1993), *i.e.* there is a lack of correlation between the concentrations of indicators in seawater and the presence of pathogens in shellfish.

The problem of viral pollution of South African coastal waters has been described in the preceding section. Similarly, viruses have been detected in shellfish collected in areas of coastal pollution (Idema and Kfir, 1990). Although no epidemiological studies of shellfish-associated disease outbreaks are available in South Africa, such examples do exist in other countries. There is reason therefore to consider the possibility of health risks to consumers associated with viral pollution of shellfish. Comments in preceding sections regarding the limitations of epidemiological studies for predictive purposes and of prediction of health effects on the basis of classical indicators apply equally here. The evaluation of the usefulness of health risk assessment techniques in assessing potential health impacts associated with contaminated shellfish is therefore justified. In this context, both formal harvesting at designated commercial shellfish beds and informal harvesting of shellfish by

coastal communities need to be considered. Limitations imposed by shortcomings of detection methods should also be borne in mind.

5.3.2. Exposure Assessment

5.3.2.1 Source of hazard

This case study evaluated the risk of infection by enteric viruses as a result of ingestion of contaminated shellfish. Data on quality of shellfish from three shellfish beds along the Eastern Cape coast were obtained for the period June 1985 to October 1989. Two were recognised shellfish culturing sites where shellfish are cultivated for commercial purposes. One was a shellfish bed situated near the outlet of a canal discharging effluent, and from which shellfish are harvested informally. The effluent has been shown to be microbiologically polluted.

5.3.2.2 Exposed population

The exposed population is likely to comprise two groups. Shellfish cultivated for commercial purposes would be offered for sale in restaurants and supermarkets. The exposed population would be likely to be middle to upper class suburban residents. This group could be expected to have relatively low resistance to microbial diseases transmitted by contaminated shellfish, but this may be expected to be of lesser importance as a result of quality control practices in the food industry.

Shellfish harvested informally would be likely to be consumed directly by the people collecting them. These would be likely to comprise poorer residents of coastal residential areas in the vicinity of the beds and visitors to the area. Of this group, visitors are likely to be at higher risk since local residents exposed to polluted shellfish and the polluted water in which they have developed are likely to have higher levels of resistance to microbial diseases.

No quantitative or qualitative investigations of the exposed populations were undertaken.

5.3.2.3 Exposure route

Exposure in all instances would be by ingestion of shellfish. In the case of informal shellfish harvesting, further inhalation, dermal contact and possible ingestion exposure would occur during harvesting. These routes are not considered in this case study since any infections transmitted would not be directly related to the level of microbial contamination of harvested shellfish.

It is assumed that ingestion of infective virus particles in shellfish produces infection by the same mechanism as free virus particles in water. The extent to which this assumption is valid is not currently known.

5.3.2.4 Exposure assumptions

No subdivision of exposure period over the year was undertaken in this case study since none was indicated by either the available monitoring data or probable behaviour patterns of the exposed population.

Two exposure frequencies were considered. An estimate of 250 exposure events per year was adopted from risk assessment software developed for environmental management in the USA (Risk Assistant, Hampshire Research Institute, 1991). This was considered a worst case scenario under South African conditions since the cost of shellfish makes it unlikely that such frequent consumption represents the population norm. A more realistic "typical" scenario was taken as being 12 exposure events per year, representing shellfish consumption on an approximately once per month basis.

Three exposure masses were considered: 1 g, 10 g and 100 g shellfish flesh consumed per exposure event. Consumption of 1 g shellfish is acknowledged to be unrealistic, but is included since this represents the mass to which all monitoring data were reduced and forms the basis of the non-detect level. A mass of 10 g may be expected to be consistent with the consumption of shellfish in a starter dish (*hors d'ouvre*), while 100 g may be a slightly conservative estimate for consumption of shellfish as part of a main dish.

Monitoring data of raw shellfish were used, *i.e.* it is assumed that shellfish were consumed raw. This represents a worst case scenario, but a realistic one since shellfish are often cooked lightly or not at all. No data were available on shellfish inactivation during cooking, therefore it was not feasible to estimate corrected exposures associated with consumption of cooked shellfish.

5.3.2.5 Exposure monitoring

Shellfish collected at the three monitoring sites were analysed for enteric viruses from June 1985 to October 1989. Shellfish samples, adjusted to 1 g shellfish flesh, were treated with buffers to release viral particles. Viruses were separated by centrifugation and the supernatant analysed by cell culture methods. Enteric viruses were reported as the most probable number 50% tissue culture infective dose (MPN $TCID_{50}$). The detection limit was considered to be 1 MPN $TCID_{50}$ /g shellfish flesh.

5.3.2.6 Analysis of monitoring data

No enteric viruses were detected in shellfish cultivated in recognised commercial shellfish beds. Viruses were detected in 12 of 24 samples of shellfish growing in the vicinity of the canal discharging polluted effluent. Summarised results are shown in Table 5.3.1. The maximum detected value was an outlier, all other values being below half this.

Table 5.3.1: Summarised results of monitoring enteric viruses in shellfish from a shellfish bed near a canal discharging polluted effluent. (Monitoring period June 1985 - October 1989)

	Enteric virus MPN $TCID_{50}$/g
Mean	2.06
Geometric mean	1.15
Minimum	0.5 (ND [1])
Maximum	14.8 [2]

[1] ND = Surrogate for non-detect value
[2] Maximum value is an outlier

As in previous case studies, all enteric viruses were treated as enteroviruses, represented by poliovirus 1, poliovirus 3 and echovirus 12. Non-detect values were substituted with the detection limit, half the detection limit and zero for purposes of risk estimation. A value of 1 MPN $TCID_{50}$ /g was used in place of zero in the calculation of geometric means.

The annual distribution of monitoring data was evaluated for fit to a log-normal and other distributions, as described previously. No distribution showing significant fit to the data was identified, therefore, a general distribution of monitoring data was defined for the evaluation of uncertainty associated with variability in the data.

5.3.3 Dose-response assessment

5.3.3.1 Infectious dose

Infectious doses of viruses have been discussed in previous case studies. The same considerations probably apply to exposure resulting from consumption of virally contaminated shellfish. The exact implications of ingestion of viruses adsorbed to a matrix such as shellfish on the infectious dose is not presently clear. It is possible that ingestion of an infectious dose of virus in a single exposure becomes more likely since filter-feeders such as shellfish are extremely efficient bio-concentrators of small particulate matter, including viruses. Contaminated shellfish would, therefore, present a far more concentrated inoculum than contaminated drinking or bathing water.

5.3.3.2 Dose-response model

Infection was modelled by the beta-distributed, infectivity, probability model, as described previously. Echovirus 12, poliovirus 1 and poliovirus 3 were used as representative viruses, with the same parameter values as previously described.

Maximum daily risk was estimated by sampling from a distribution of monitoring

data. Three estimates of yearly risk were developed. The first was the average yearly risk, calculated using the yearly geometric mean exposure as determined from the monitoring data. The most likely risk was estimated by sampling - using Latin hypercube sampling - the exposure used to calculate yearly risk from the distribution of monitoring data. The most probable estimate from a model run of 1000 iterations served as the yearly risk estimate. Finally, the yearly risk was calculated as the product of daily risk estimates for each day of the exposure period.

5.3.4. Risk characterisation

5.3.4.1 Estimates of daily and yearly risk of infection

Shellfish originating from commercial shellfish beds

Daily and yearly risks of enteric virus infection associated with consumption of commercially cultivated shellfish were assessed at the detection limit and half the detection limit as substitute for non-detect values, since no viruses were detected in these samples. Results are tabulated in Table 5.3.2. Yearly risks are shown only for an assumed 12 exposure events per year since at 250 exposure events per year all risks were at 1.0 or closely approached this value. These risk estimates represent the minimum risks detectable under the described monitoring procedure. As is evident from Table 5.3.2., all risks are well above the recommended acceptable yearly risks of 10^{-4} suggested for drinking water and 10^{-3} for recreational exposure. At the detection limit and assuming ingestion of only 1 g of contaminated shellfish, daily risks of infection ranged from 2×10^{-2} to 3×10^{-1}, and yearly risks from 2×10^{-1} to 1. These increased further as the assumed exposure mass was increased to 10 g or 100 g. If it was assumed that half the detection limit could reasonably be used as surrogate for the uniformly non-detect results recorded for these samples, then daily risks at a consumption of 1 g shellfish per exposure event dropped slightly to 8×10^{-3} to 2×10^{-1}, and yearly risk assuming 12 exposure events to 9×10^{-2} to 9×10^{-1}. These remain above recommended acceptable risk levels.

Table 5.3.2: Daily and yearly risks of enteric virus infection as a result of shellfish consumption, calculated for shellfish originating from two commercial shellfish beds.

	Daily risk of infection	Yearly risk of infection, assuming 12 exposure events per year
Echovirus 12		
At detection limit:		
1 g	2×10^{-2}	2×10^{-1}
10 g	2×10^{-1}	9×10^{-1}
100 g	7×10^{-1}	1.0
At half detection limit:		
1 g	9×10^{-3}	1×10^{-1}
10 g	8×10^{-2}	6×10^{-1}
100 g	5×10^{-1}	1.0
Poliovirus 1		
At detection limit:		
1 g	2×10^{-2}	2×10^{-2}
10 g	1×10^{-1}	8×10^{-1}
100 g	8×10^{-1}	1.0
At half detection limit:		
1 g	8×10^{-3}	9×10^{-2}
10 g	7×10^{-2}	6×10^{-1}
100 g	5×10^{-1}	1.0
Poliovirus 3		
At detection limit:		
1 g	3×10^{-1}	1.0
10 g	7×10^{-1}	1.0
100 g	9×10^{-1}	1.0
At half detection limit:		
1 g	2×10^{-1}	9×10^{-1}
10 g	6×10^{-1}	1.0
100 g	9×10^{-1}	1.0

Shellfish originating from beds near canal discharging polluted effluent

Estimates of maximum daily risks and of yearly risks associated with consumption of shellfish growing in contaminated water are presented in Table 5.3.3. Only yearly risks associated with an assumed exposure frequency of 12 exposure events per year are presented. All risks calculated for an exposure frequency of 250 events per year were 1.0 or extremely close to 1.0.

Maximum daily risks associated with echovirus 12 and poliovirus 1 at an assumed consumption of 1 g per exposure event were close to 10^{-1} ; for poliovirus 3 this risk increased to approximately 5×10^{-1}. Changing the non-detect surrogate value from the detection to half the detection limit or to zero made very little difference to the risk estimates: a change from detection limit to half the detection limit produced an associated change of 0.1 order of magnitude in the risk, with a similar effect if the surrogate value was changed from half the detection limit to zero. Increasing the assumed ingestion mass from 1 g to 10 g produced a change of approximately half an order of magnitude, with a similar increase associated with increasing the mass from 10 g to 100 g. The magnitude of the increase declined as the upper limit of risk was approached, as seen for poliovirus 3.

Average yearly risks of infection based on geometric mean exposure and at an assumed consumption of 1 g were between 1×10^{-1} to 3×10^{-1} for echovirus 12; 9×10^{-2} to 3×10^{-2} for poliovirus 1 and 9×10^{-1} to 1.0 for poliovirus 3. As is evident from the ranges, assumptions regarding non-detect values made little difference to the risk estimates. Increasing the assumed consumption mass to 10 g increased the average yearly risk to close to or 1.0 in most instances. The lowest values were 6×10^{-1} for echovirus 12 and poliovirus 1, using zero as the non-detect surrogate value. At an assumed consumption of 100 g per exposure event, all average yearly risks were 1.0.

Estimates of most likely yearly risk, calculated by iterative sampling from distributions of monitoring data and statistical prediction of the likely result, were approximately half and order of magnitude greater than the corresponding values for average yearly risk. Cumulative risk estimates, calculated as the product of daily risk estimates, were close to the corresponding most likely risk estimates, being up to 0.1 order of magnitude greater. Most values of cumulative risk were at or near 1.0.

Table 5.3.3: Maximum daily and yearly risks of enteric virus infection associated with shellfish consumption, calculated for shellfish originating from a bed near a canal discharging polluted effluent (Monitoring period June 1985 to October 1989).

	Maximum daily risk	Yearly risk (12 exposure events per year)		
		Average risk (Calculated from geometric mean)	Most likely risk (Calculated by iterative sampling of monitoring data)	Cumulative risk (Calculated as the product of daily risks)
Echovirus 12				
Non-detect value: 1 MPN $TCID_{50}$/g				
1 g	1×10^{-1}	3×10^{-1}	7×10^{-1}	7×10^{-1}
10 g	5×10^{-1}	1.0	1.0	1.0
100 g	9×10^{-1}	1.0	1.0	1.0
Non-detect value: 0.5 MPN $TCID_{50}$/g				
1 g	9×10^{-2}	2×10^{-1}	6×10^{-1}	7×10^{-1}
10 g	5×10^{-1}	9×10^{-1}	1.0	1.0
100 g	9×10^{-1}	1.0	1.0	1.0
Non-detect value: 0 MPN $TCID_{50}$/g				
1 g	8×10^{-2}	1×10^{-1}	5×10^{-1}	6×10^{-1}
10 g	5×10^{-1}	6×10^{-1}	8×10^{-1}	1.0
100 g	9×10^{-1}	1.0	1.0	1.0
Poliovirus 1				
Non-detect value: 1 MPN $TCID_{50}$/g				
1 g	1×10^{-1}	3×10^{-1}	6×10^{-1}	7×10^{-1}
10 g	6×10^{-1}	1.0	1.0	1.0
100 g	1.0	1.0	1.0	1.0
Non-detect value: 0.5 MPN $TCID_{50}$/g				
1 g	8×10^{-2}	2×10^{-1}	6×10^{-1}	7×10^{-1}
10 g	5×10^{-1}	9×10^{-1}	9×10^{-1}	1.0
100 g	9×10^{-1}	1.0	1.0	1.0

	Maximum daily risk	Yearly risk (12 exposure events per year)		
		Average risk (Calculated from geometric mean)	Most likely risk (Calculated by iterative sampling of monitoring data)	Cumulative risk (Calculated as the product of daily risks)
Non-detect value: 0 MPN $TCID_{50}$/g				
1 g	7×10^{-2}	9×10^{-2}	5×10^{-1}	6×10^{-1}
10 g	4×10^{-1}	6×10^{-1}	8×10^{-1}	1.0
100 g	8×10^{-1}	1.0	1.0	1.0
Poliovirus 3				
Non-detect value: 1 MPN $TCID_{50}$/g				
1 g	6×10^{-1}	1.0	1.0	1.0
10 g	9×10^{-1}	1.0	1.0	1.0
100 g	1.0	1.0	1.0	1.0
Non-detect value: 0.5 MPN $TCID_{50}$/g				
1 g	5×10^{-1}	1.0	1.0	1.0
10 g	8×10^{-1}	1.0	1.0	1.0
100 g	9×10^{-1}	1.0	1.0	1.0
Non-detect value: 0 MPN $TCID_{50}$/g				
1 g	4×10^{-1}	9×10^{-1}	9×10^{-1}	1.0
10 g	7×10^{-1}	1.0	1.0	1.0
100 g	9×10^{-1}	1.0	1.0	1.0

Discussion of risk estimates

None of the risks estimated for shellfish originating from beds in the vicinity of a polluted effluent discharge met recommended maximum acceptable risk limits of 10^{-3} or 10^{-4}, even under the least stringent assumptions regarding exposure. However, this is to be expected since estimates of the minimum risks detectable under current shellfish monitoring practices were all above these limits. This indicates that the monitoring regime represented in this case study is inadequate to detect levels of risk sufficiently low to be useful in formulating risk-based water quality guidelines or providing meaningful quantitative descriptions of health risks. This aspect dominated risk estimates under all assumed exposures tested. Risks were always near the maximum which could be calculated and this tended to

suppress differences caused by changing exposure assumptions.

The value substituted for non-detect results resulted in differences of up to 0.7 orders of magnitude when estimating the minimum risks detectable (Table 5.3.2). However, very little difference was observed between risks estimated at the tested non-detect surrogates at detectable levels of viruses (Table 5.3.3).

Changes in the assumed mass of shellfish ingested resulted in changes of approximately one order of magnitude when risks estimated were not close to the maximum possible (1 g and 10 g exposures in Table 5.3.2). However, as risk estimates approached 1.0, differences in risks associated with changing the exposure mass decreased to half an order of magnitude or less (Table 5.3.3).

Assumptions regarding exposure frequency did influence risk estimates, as may be expected. However, it was not possible to evaluate the impact of changes in this variable since risk estimates, even at the lower assumed exposure frequency (12 exposure events per year) reached the maximum value and all risks at the higher assumed exposure frequency (250 events per year) were at or near 1.0.

Risks estimated for echovirus 12 and poliovirus 1 were once again consistently lower than those estimated for poliovirus 3, further supporting observations from previous case studies. At its greatest, the difference was one order of magnitude. Once again, this difference decreased steadily as risk estimates approached 1.0.

Differences were noticeable between the measures of longer term risk. Average yearly risks were consistently lower than maximum likely values or cumulative risks, the difference being approximately half an order of magnitude at its greatest. Most likely risks were consistently lower than cumulative risks, but the difference was slight, amounting to 0.1-0.2 orders of magnitude.

It is clear that by far the greatest limitation in this case study was the ability to detect risks in the vicinity of the acceptable limit. This was due primarily to the low mass of shellfish analysed in the detection methodology. It is unfortunately difficult to suggest a solution to this dilemma since practical limitations in the analysis of shellfish samples make it difficult to envisage analysis of larger sample masses.

Aside from this consideration, it appeared that the choice of pathogen considered had the greatest impact on risk estimates, followed by assumptions regarding exposure mass and frequency. However, the implications of operating near the maximum risk calculable make any evaluation of the sensitivity of risk estimation to changes in assumptions and variables extremely imprecise.

5.3.4.2 Characterisation of uncertainty

Certain sources of uncertainty have been discussed in previous case studies. These include uncertainties inherent in the dose-response model used, the degree to which the three enteroviruses considered are representative of the enteric virus

group and uncertainty arising from the methods used for calculation of longer term risk, although the impact of the last-mentioned was less than in the drinking water case study. Other issues previously addressed but which deserve further consideration in the context of this case study include uncertainties associated with virus recovery and detection methods, detection limits, applicability of the model used to the route of exposure and the lack of exposure information.

The recovery and detection of viruses from shellfish presents certain methodological difficulties which impact the use of such monitoring data in quantitative risk assessment. Recovery of viruses requires their desorption from shellfish, accomplished by manipulation of the pH of a proteinaceous medium. The recovery efficiency of such methods determines how well monitoring data reflect actual exposure. Recovery efficiencies in the order of 90% were found for poliovirus 1 in seeded laboratory experiments (Idema *et al.*, 1991). This indicates that uncertainty in risk estimates in this case study is not greatly affected by viral recovery efficiencies. Shellfish samples are difficult to handle in the laboratory, particularly to reduce to volumes small enough to be inoculated in tissue culture. This places a limit on the mass of sample which can feasibly be analysed and makes the detection limit for such samples high. The sensitivity of risk assessment is greatly reduced at high detection limits: in this case, sensitivity was reduced to the extent that even minimum detectable risks were above acceptable limits. Sensitivity to the surrogate used for non-detect values in the data set, and the impact thereon of the high detection limit resulting from a small sample mass, has been discussed above with the risk estimates.

Infectivity probability models, such as the beta-distributed probability model used in this study, currently assume ingestion exposure in a water medium. While exposure considered here was indeed by ingestion, it is not clear whether the mode of infection of viruses ingested in a solid medium equates to that of viruses ingested in water. One difference which springs readily to mind is the greater protection offered by the solid medium against attack by stomach acid and digestive enzymes. A possible consequence of this could be that exposure to viruses in food, such as shellfish, results in a lower infectious dose since a greater proportion of ingested viruses survive the acidic stomach environment to infect the gastrointestinal region.

As in the case of the marine recreational case study, all aspects of exposure in this study were treated by means of assumption, including exposure mass, exposure frequency and nature of the exposed population. Virtually no data describing the shellfish-gathering and -consumption behaviour patterns or demographics of likely exposed populations were available. This introduces considerable uncertainty into the risk assessment, since a large number of scenarios must be considered to maximise the possibility of addressing all significant exposures, and since there is no objective indication of the most likely exposure. An additional source of uncertainty in exposure data in this case study was the degree of cooking which shellfish received prior to consumption. Shellfish consumed raw present a higher risk of infection than do lightly cooked shellfish. In this study shellfish quality data were treated as though shellfish were consumed raw. If shellfish were cooked,

additional data on the extent of pathogen inactivation as a function of cooking conditions and time would be required. It is probable that the sensitivity of the risk assessment could be improved with such information since the mass of cooked shellfish representing an equivalent viral exposure would be larger than the corresponding mass of uncooked, and hence more infectious, shellfish. This would assist in mitigating the limitations of sample mass and detection limit.

6. DISCUSSION AND CONCLUSIONS

Quantitative health risk assessment provides a means of estimating the probability of adverse effects with measured or estimated levels of hazardous agents and a tool for predicting the extent of potential or probable health effects. The use of microbial risk assessment techniques in a South African context was assessed in this study by means of three case studies based on available microbial monitoring data. The intention in selecting data sets was to identify data which represented a number of possible exposures and which were typical of data collected in water quality monitoring studies. It was anticipated that this would provide samples of the type of data to which microbial risk assessment techniques could be applied. The use of risk assessment was intended to add meaning to investigation of water quality and development of water quality guidelines.

This study identified a number of factors that need to be considered when applying microbial risk assessment techniques.

General

This study illustrated that the successful application of microbial risk assessment in South Africa requires that attention be paid to a number of issues pertaining to monitoring of viral quality of raw water and treated drinking water. The risk estimates presented in this assessment were high when compared to EPA recommendations (Federal Register, 1989) and results reported by other authors (Rose and Gerba, 1991b; Rose *et al.*, 1991). As a result, they do not provide information suitable as a basis for guideline development and are of limited assistance in the interpretation of monitoring surveys. Detection of low risk levels was dominated by methodological problems in the case studies, giving an impression of drinking water quality which is highly likely to be inaccurate.

Sample Volume

The greatest restriction on detection of low risk levels was the sample volume analysed, which were not sufficient to detect low risks. Monitoring of larger volumes, at least 100ℓ, is necessary to detect risk levels comparable to that recommended by the EPA. This has been implemented in our laboratory and evaluation of the improvement in risk assessment efforts using these data would be valuable. It may be possible to overcome the problem in the short-term by exploiting the relatively high sampling frequency practised at present (approximately twice per month) to estimate risks over a shorter time frame (*e.g.* monthly). These risks may be compared with the EPA recommended yearly risk, or with risk recommendations which may be developed for South Africa. However, to gain maximum benefit from the application of risk assessment methods to microbial monitoring data, concentration of large volumes of water is imperative and of primary importance.

Method of Longer Term Risk Calculation

A major source of uncertainty in the estimation of longer term risks rests in the method of risk calculation employed. Methods concentrating on central tendency of exposure over a period of interest yielded results which varied considerably relative to methods which were more representative of the spread of possible exposures. A policy decision on the type of estimate to be preferred would be required before risk assessment can be applied successfully and reproducibly in guideline development and data interpretation. The level of acceptable risk needs to be defined in relation to the method employed to develop the risk estimate. Methods based on central tendency may be more suited to defining acceptable average risks over time, while methods based on direct estimations from daily exposures may be better suited to defining acceptable upper limits to risk.

Other Factors

Advances in other spheres would further improve the use of risk assessment for evaluation of drinking water quality. Improved data describing removal of viruses and other pathogens during water treatment would allow better estimates of levels in treated water to be developed from raw water levels. Although the detection limit did not appear to be a major limiting factor in the estimation of low risk levels, more sensitive detection methods with a more sensitive detection limit would reduce the number of non-detects results, and hence the extent of uncertainty introduced by assumptions regarding substitute values for non-detect results. More comprehensive exposure data would permit meaningful interpretation of the significance of risk estimates to community health.

Marine Recreation and Shellfish Consumption

In addition to the factors described above, the case studies examining marine recreational activity and shellfish consumption highlighted additional factors requiring research. Exposure data for recreational activities in the marine environment and exposure to shellfish remains undefined. To date, exposure data with regard to recreational activities in water are extremely limited and assumptions are made with no supporting data. Information specific to marine bathing behaviours in South Africa, and preferably to the locality being evaluated are necessary to reduce such uncertainty. This would include data such as bathing frequency, preferred seasons, extent of immersion during bathing, estimates of volumes of water ingested, and concurrent exposures which could make bathers more or less susceptible to infection. With regard to exposure to contaminated shellfish; no exposure data is available, such as exposure volume, frequency and nature of exposed population. No data describing the shellfish-gathering and -consumption behaviour patterns are available.

Acceptable Risks for South Africa

Ultimately, acceptable risks for South Africa must be selected. The USA-EPA have

recommended risks acceptable for the USA, but whether these are applicable to South Africa is uncertain. A less (or more) stringent approach may be more appropriate.

Extent to which objectives were reached

The objectives of the study, as specified in the proposal, were achieved.

a) *the usefulness of data collected by current microbial monitoring programmes was assessed for quantitative analysis of the associated health risks.*

Minimum risks detectable under each of the case studies presented were almost uniformly above acceptable average yearly risks recommended by the US EPA (Federal Register, 1989) and microbial risk experts. A consequence of this was that it was not possible to compare estimated risks with existing suggested guidelines for acceptable risk. This indicates that under monitoring practices represented in this investigation limitations were placed on detection of levels of risk which would be useful in interpreting monitoring water quality in terms of risk-based guidelines or in preparing water quality guidelines.

b) *to estimate the minimum levels of risk detectable using the volumes of water currently analysed*

One of the major limitations imposed by monitoring methods represented in this study was the inability to detect low levels of risk suitable for comparison to risk-based guidelines. Monitoring of relatively low volumes (10ℓ in the case of water samples) restricted the risk range which could be detected and resulted in numerous non-detect results. Monitoring of larger volumes, at least 100ℓ, is necessary to detect risk levels comparable to that recommended by the EPA. It may be possible to overcome the problem in the short-term by exploiting the relatively high sampling frequency practised at present (approximately twice per month) to estimate risks over a shorter time frame (*e.g.* monthly). These risks may be compared with the EPA recommended yearly risk, or with risk recommendations which may be developed for South Africa. However, to gain maximum benefit from the application of risk assessment methods to microbial monitoring data, concentration of large volumes of water is imperative and of primary importance.

c) *to evaluate the usefulness and implications of health risk assessment for the formulation of microbial water quality guidelines*

The most significant restrictions in the application of risk-based guidelines identified in this study were:

* insensitivity to low levels of risk

* uncertainty of assessments which limit the confidence of the estimated risks to guidelines, namely,
  - choice of pathogen for which risk is calculated when a general group is monitored,
  - lack of reliable exposure data,
  - method of calculating longer term risks, and
  - variability in monitoring data

As a result of the conflict between data requirements for risk assessment and the need for practical monitoring programmes, it is likely that microbial risk assessment will not be used in association with generalised monitoring studies. Rather, risk assessment may be utilised in studies targeted specifically for guideline development or at establishing quantitative risks associated with specific pathogens. The greatest benefit will be obtained from microbial risk assessment if such studies are planned rigorously in terms of exposure data, sample volumes and sample numbers to provide a balance between limits of detectable risk and a representative distribution of exposure estimates.

One of the important benefits of the risk assessment approach to setting guidelines and evaluation water quality data is that it makes explicit the extrapolation from water quality to health impact. This avoids the questionable assumption, often made in setting water quality guidelines on the basis of indicator organisms and in evaluation of data gathered to monitor these guidelines, that the potential for health impacts can be inferred directly from such monitoring data. The case studies presented here suggest this might be a misconception, even where potential pathogens such as enteroviruses are monitored instead of indicator organisms. All data sets used in the case studies were generated with the implicit assumption that the water quality monitoring programmes served to protect water quality and hence human health. Despite this, water quality monitoring did not provide data which was sufficiently sensitive for inferring potential impacts on health relative to current guidelines for acceptable health risk. This shows that health effects are potentially associated with significantly lower levels of pathogens than can be detected in traditional water quality monitoring programmes. Similar observations were made by other investigators applying risk assessment techniques to microbiological water quality (Gerba and Haas, 1988) and in epidemiological investigations (Payment *et al.*, 1993).

Development of microbial water quality guidelines represents a conflict between the need to consider the practicalities of monitoring and the desire to relate data to health impacts. The former is served by use of indicator organisms which can be detected rapidly and easily in relatively small volumes of water. The latter requires a demonstrable cause and effect relationship between microbial contaminant and health effect. Guidelines which are not based on such a relationship run the risk of being arbitrary rather than meaningful in terms of public health. Payment *et al.* (1993)

detected drinking water-related health effects in a population receiving water conforming to all microbial water quality criteria. They conclude that current microbiological indicators serve as indicators of faecal pollution and treatment efficiency, but are not reliable as predictors of health effects.

Indications are that risk assessment has a role to play in the setting of appropriate health-centred guidelines, provided suitable data for meaningful risk assessments can be generated.

Conclusion

In conclusion, with respect to the objectives of this study, microbial risk assessment was found to be of potential benefit in the South African context to the description of microbial water quality impacts and to the development of guidelines in the future. However, a number of limitations were found:

- the adoption of a risk assessment approach would hold implications for the manner in which monitoring data are collected. Detection of low levels of risk was hampered by high detection limits and the relatively small volumes of water sampled. The sampling and concentration of large volumes of water (at least 100ℓ) would be the most important factor in facilitating the detection of low risks.

- the availability of better exposure data would decrease the uncertainty associated with risk assessments. A drawback of current microbial risk assessment models is that they apply only to exposure by ingestion. Possible future development of models which explicitly address other exposure routes would allow microbial risk assessment to be implemented on a wider basis.

- the choice of pathogen for which risk is estimated when a more general group is monitored (*e.g.* enteric viruses) is an additional source of uncertainty which plays a significant role in the uncertainty in the risk calculation

- standardisation of methods used in estimations of longer term risks is needed to ensure that values can be compared across studies, both nationally and internationally.

Presently, the application of microbial risk assessment techniques in South Africa is hampered by inadequacies in the data available. Considerations of what constitutes an acceptable risk is necessary and desirable in the South African context. However, this would only provide a partial solution, as any reasonable acceptable risk guideline would still require more sensitive data.

Results represented here and reported by other investigators using a range of techniques indicate that much historical water quality monitoring aimed at protection of public health may have missed the mark. While monitoring has been extremely useful in controlling water-borne disease outbreaks, it is possible that insufficient protection is provided against low levels of relatively low grade gastrointestinal and other complaints associated with water-borne pathogens.

Further research is required to address the numerous issues raised in this study. Risk assessment should be applied to available monitoring data including other pathogens, for example, the protozoan parasites *Giardia* and *Cryptosporidium*, other water-borne viruses such as rotavirus and hepatitis A virus, and pathogenic bacteria such as *Salmonella*. Additional exposure routes, namely, exposure *via* wastewater, need to be examined. To test the risk assessment procedure, calculated risk estimates need to be compared to water-related disease reporting in this country. It is also recommended that the procedure of using risk assessment techniques is repeated with data now available examining larger volumes of water.

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