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Comparative risk analysis

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10.1 ESSENTIALS OF RISK ASSESSMENT

Risk assessment is a systematic process to estimate the level of risk related to some specific action or activity. It is now commonly applied to a wide variety of human endeavours in which harm to people, the environment or economic interests might occur. In the context of public health, the process attempts to quantify the likelihood and severity of illness to individuals or populations from a specified hazard.

The primary purpose of risk assessment is to provide support for decisions about managing risks associated with those specific actions or activities. This is done by systematically synthesizing information about the factors that contribute to the risk in a coherent framework that enables risk, or relative risk, to be inferred from knowledge of those risk-contributing factors in specific circumstances. Depending on the needs of the risk manager, the risk assessment may attempt to assess the relative effectiveness of proposed risk mitigations, or to estimate the
magnitude of the risk under different circumstances, or the risk to different populations or sub-groups within the population.

There are many frameworks that describe the interaction between risk assessment and risk management, and a third component known as “risk communication” which involves understanding the interests, concerns and values of “stakeholders”, that is, those affected by the risk, so as to guide and optimize risk management decisions. One depiction of the interaction among these aspects of risk analysis is presented in Figure 10.1.

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**Figure 10.1** A depiction of the interaction between risk assessors, risk managers and those affected by the risk ("stakeholders") within the risk analysis framework developed by the World Health Organization and the Food and Agriculture Organisation of the United Nations. Arrows indicate lines of communication, while separate circles are intended to depict discrete roles of those responsible for risk assessment, those responsible for making decisions about risk, and stakeholders (based on: WHO/FAO 2009).

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While some organisations consider risk assessment to encompass risk management, risk communication and risk analysis, many organisations involved in environmental and public health risk assessment consider risk analysis to be the ‘umbrella’ activity that encompasses risk assessment, management and communication. For example, the Society for Risk Analysis (http://www.sra.org) broadly defines risk analysis “...to include risk assessment, risk characterization, risk communication, risk management, and policy relating to risk.” We choose to adopt this convention here.
Risk management involves a balance between the most effective risk mitigation action, based on cost or technical feasibility, and the interests and values of stakeholders. Therefore risk managers will also seek information on costs (compared to the benefits) of risk management options. The efficacy of various approaches and the factors that affect it may be considered in the risk assessment to support the decisions of risk managers. The combination of risk assessment, risk management and risk communication is, in some frameworks, collectively termed ‘risk analysis’. Microbiological risk assessment frameworks relevant to waterborne risk are discussed by Haas et al. (1999, Chapter 3); WHO (2003); Gale (2001a&b, 2003), Coffey et al. (2007) and Goss & Richards (2008).

10.1.1 Key elements

The first step in risk assessment usually is the development of a conceptual model or framework that combines knowledge of risk-affecting factors and how they could interact to cause harm. In many cases this information can be expressed mathematically as a series of equations that, provided sufficient quantitative data and knowledge exist, enable quantification of the risk or, at least, the relative risk. In assessing the public health risk from exposure to microbial pathogens, the “risk assessment” task is often broken down into four discrete components:

- **Hazard Identification** – which involves describing the hazard (pathogens), presenting the evidence that the hazard causes illness and that it can cause illness from the source of exposure being considered, and the type of illness caused.

- **Hazard Characterization** – which presents information about characteristics of the organism that affect its ability to cause illness, such as virulence factors, physiological traits that affect its survival in the environment and, importantly, the severity of disease caused, including consideration of differences in susceptibility of different members of the population exposed and the probability of illness as a function of the dose ingested. In the context of water and sanitation this corresponds to consideration and characterization of elements of “pathogen virulence” and “host susceptibility”. This includes consideration of dose-response relationships, which is sometimes considered as a further discrete component (e.g., Haas et al. 1999).

- **Exposure Assessment** – which attempts to estimate the exposure of the affected population(s) to the pathogen under scrutiny. In the context of public health risk from recreational/occupational exposure to water this could involve consideration of, for example: sources of contamination,
loads and composition of pathogens in sources of contamination, and factors that would increase or decrease the risk of exposure such as inactivation due to UV irradiation, predation, or human interventions. It should also consider factors that would alter exposure of different members of the population such as age (children may swim more often, or ingest more water when swimming), cultural and gender factors (e.g., women in developing nations may be more likely to be exposed due to their domestic responsibilities), type of water exposure (e.g., full immersion through swimming cf. accidental exposure through clothes-washing). In the context of water and sanitation this corresponds to consideration and characterization of elements of load, transport and exposure (see: Chapters 2–7).

- **Risk Characterization** – the systematic and scientific process of synthesizing all the relevant knowledge and information to produce estimates of risk, or relative risk.

### 10.1.2 Principles of risk assessment

Ideally, the model and the data and knowledge that it is based on will be clearly documented, or ‘transparent’. Usually, however, insufficient knowledge and data are available to enable unambiguous assessment of (comparative) risk, or decisions based on that risk, and a number of assumptions will need to be made in the development and application of the risk assessment model. These assumptions, and their potential consequences for the decisions based on them, should also be clearly articulated.

### 10.1.3 Pathogen selection

Many zoonotic pathogens have been reported in the literature as the causal agents of human infections or outbreaks of disease. The majority of these human infections were transmitted from animals to humans by food products (O’Brien 2005). Several outbreaks have been transmitted by drinking-water and only a small number have been transmitted by recreational water (Craun 2004). Outbreaks may, however, comprise only a minority of the actual cases suffered: “…probably the vast majority of waterborne disease burden arises outside of detected outbreaks” (Bartram 2003). The zoonotic bacterial pathogens mentioned most frequently are *E. coli* O157, *Campylobacter*, *Salmonella*, *Leptospira* and *Listeria*. Protozoan pathogens include *Cryptosporidium*, *Giardia* and *Toxoplasma* (Schlundt et al. 2004, Pell 1996, Rosen 2000, Bicudo & Goyal 2003). Although most of the zoonotic pathogens have been associated with sporadic and outbreak patterns of disease, a few have not been associated with
waterborne outbreaks or with faecal contamination of water. The following criteria have been adopted whereby pathogens from the list above will be selected for inclusion in our risk comparison process.

- The pathogens are known to be carried by animal or bird species;
- The pathogens are discharged in the faeces of animals or birds;
- The pathogens have been isolated from surface waters;
- The pathogens cause disease in humans.

The zoonotic pathogens that meet these criteria are:

*E. coli* O157 (more generally, EHEC) which are predominantly carried by ruminants including beef and dairy cattle and sheep (USEPA 2000a, Caprioli et al. 2005) and, to a lesser extent, monogastric animals (Chapman 2000, Chapman et al. 1997). This organism has been associated with outbreaks of disease that are related to recreational water (e.g. Keene et al. 1994) and has been isolated from surface waters. None of the outbreaks have been linked directly to direct contacts with animals. However, some have been associated with food and drinking-water.

*Campylobacter* species are frequently found in surface waters and this organism is carried by poultry, cattle and sheep (Jones 2001). *Campylobacter* has frequently been linked to outbreaks transmitted by food and water, but its occurrence is predominated by sporadic and endemic patterns, rather than outbreaks. In New Zealand, where campylobacteriosis is a reportable disease, about 300 to 400 cases per hundred thousand population have been reported in recent years (Till & McBride 2004, Till et al. 2008).² In western and northern Europe, in the late 1990s reported rates per 100,000 varied from ~20 (The Netherlands) to ~100 (England Wales) (Kist, 2002). Annual reported incidence in Australia is ~100/100,000 (NNDSS, 2010) and in Canada seems to be declining but at the time of writing is ~30/100,000 (PHAC, 2010). It is important to note that the actual incidence of this disease in the USA is estimated to be ~40 times higher than the reported rate (Mead et al., 1999), because many cases are not detected by a country’s health reporting system.³

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² This rate has approximately halved in recent years. This has been attributed to management interventions in the poultry industry (French et al. 2011, McBride et al. 2011).
³ The many reasons for this state of affairs are often described by the “reporting pyramid” (e.g., Lake et al. 2010). Layers in this pyramid depict all the necessary steps that must be taken before it is possible to report incidence. For example, an ill person must visit a doctor who must request that a stool sample be supplied and analysed for the presence of *Campylobacter*, an infected stool must be supplied and analysed correctly, and a positive result must be entered into the reporting system. If any one of these steps is not completed, the case will not be reported.
Salmonella species have been isolated from cattle, sheep, poultry and swine (Davies et al. 2004, Li et al. 2007). Although Salmonella has seldom been linked to outbreaks related to recreational waters, it is one of the most frequently reported foodborne diseases worldwide (Schlundt et al. 2004) and has been implicated in illness caused via contaminated drinking-water (Febriani et al. 2009). At least two swimming-associated outbreaks of salmonellosis have been reported in the literature (Moore 1954, Anon. 1961).

Cryptosporidium has been isolated from cattle faeces (USEPA 2000a) and from surface waters. Outbreaks of swimming-associated disease caused by Cryptosporidium have been reported in the literature (e.g. Hunter & Thompson 2005).

Giardia has been isolated mainly from cattle. In the United States more than 50% of dairy and cattle herds are infected with this organism (USEPA 2000a). It has frequently been isolated from surface waters. Although there is much evidence available that shows infection by the waterborne route is very frequent, little evidence is available about swimming-associated outbreaks. Craun et al. (2004) indicated that four outbreaks associated with untreated recreational water in USA lakes and ponds were detected between 1971 and 2000.

These five zoonotic pathogens of primary concern will be the subject of this risk comparison chapter. Notably, the organisms selected also correspond with those non-viral pathogens identified by Coffey et al. (2007) as being the greatest causes of waterborne disease outbreaks in USA and Europe during the late 20th century.

10.2 THREE RISK ASSESSMENT PARADIGMS

Microbial risk assessment is often categorised into three main forms: qualitative, semi-quantitative and quantitative (e.g., SA/SNZ 2004, FAO/WHO 2009) although, in practice, there is a spectrum of approaches particularly between the semi-quantitative and quantitative approaches. In the qualitative approach there is no attempt made to quantify risks. Instead, one essentially sets the context of the issues—especially identifying the pathogens of concern—accompanied by narrative statements of risk for different types and locations of sources and exposures, for example, risks are described in subjective, or relative terms, such as risk A is higher than risk B, or risk C is not significantly different to Risk D. In contrast, the semi-quantitative risk assessment approach, often called Comparative Risk Assessment (CRA), does attempt to quantify aspects of risk. It uses the context-setting information that
would be derived in a qualitative approach and uses risk ‘scores’ to construct a metric for comparing the magnitude of risks from different pathogens, sources and exposure locations. As such it calculates relative health risks and attempts to quantify the relative magnitude of risks. Typically, this uses the fundamental notion that “risk = likelihood of exposure × consequences”. Scores for “consequences” may be broken into sub-metrics for scale, magnitude of exposure and probability of illness, duration and severity of adverse health effects. The outcome can be somewhat dependent on the assignment of scores and the definition of boundaries between them.

Quantitative Microbial Risk Assessment (QMRA, Haas et al. 1999) attempts to calculate absolute health risks. Whilst ‘deterministic’ approaches are sometimes used, QMRA usually estimates risk by considering many possible combinations, via statistical or ‘stochastic’ modelling of exposures and dose-response. As such it is much more data-intensive than the comparative approach, particularly because it requires data on the variability over time of the degree of pathogen contamination at exposure locations.

In deterministic approaches to risk modelling, risk (or relative risk) is evaluated on the basis of representative values of the risk-affecting variables. The values may be the average, or mode, but to generate conservative estimates of risk, values that are deliberately conservative (e.g., the value that will not be exceeded in 95% of cases) may be used (use of a maximum value is not favoured, because it is always possible that it may be exceeded). This is done so that when the model is calculated to evaluate the risk, the estimate is inherently conservative. Decisions based on that estimate provide a high degree of public health protection, but these estimates may be unrealistically high and lead to poor decisions.4 Other potential consequences of employing a deterministic approach, compared to stochastic modelling, were exemplified in Nauta (2000) showing the failure to consider variability in risk assessment modelling could lead to erroneous risk management decisions.

For reasons such as those described above, a stochastic simulation modelling approach is often used to be able to better represent and assess the influence of variability in the various elements and circumstances known to influence

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4 A problem with this approach is that in making a series of conservative simplifying assumptions the conservatism compounds and decisions based on the approach represent a level of risk that only occurs in very rare sets of circumstances, rather than being more representative of the risk from the “normal” situation, but with allowance for rare events. This problem was explained, and its implications discussed, by Cassin et al. (1996) who coined the term “compounding conservatism” to describe it.
risk. The process involves the construction of a conceptual mathematical model of the way that the risk arises, and then, by systematically varying the inputs of the model and calculating the resultant risk in each of those circumstances, to learn how widely the risk can vary in different circumstances and for different people. By analysing all the results statistically, the risk can be characterised by a most likely value, as well as the extreme outcomes on both ends of the spectrum. Obviously, depending on the complexity of the model and the number of variations that should be investigated, this analysis can be very time-consuming.

Fortunately, the advent of powerful ‘user-friendly’ software, some of which runs in conjunction with common spreadsheet software, has made stochastic simulation modelling much more readily available. The software runs through the model time after time after time. Each time is called an iteration. At each iteration a value is selected from each variable’s range, more-or-less at random (according to the probability distribution describing that variable and accounting for any correlation with other variables), and the outcome is evaluated for that set of circumstances. Typically, tens of thousands or hundreds of thousands of iterations are calculated. All of those values are collated by the software and summary of the spread of risk and the most frequent, or most typical, result identified. This kind of software, and the approach of stochastic simulation modelling, implement ‘Monte Carlo’ methods, indicating their basis in random probability processes such as occur in games of chance.

In a detailed QMRA study of waterborne gastro-intestinal pathogens the variability in risk-affecting factors needs to be obtained from a combination of detailed monitoring and modelling of faecal indicators and pathogens. It should also include uncertainty analysis, especially with regard to dose-response information (Teunis 2009). For the purposes of this text, in its intended application to many types of pathogen sources and environments, that level of environmental data will not be available. Accordingly we present a prototype of a deterministic, comparative risk assessment procedure, based on the notion that risk to public health can be considered as the combination of the likelihood of exposure to a hazard and the severity of the consequences should that exposure occur.

Table 10.1 presents the range of data, in both numeric and narrative form, needed to run the procedure (which is presented as a Microsoft Excel® spreadsheet). The rationale for the entries given in the Table is given in the Appendix.

The model itself is described after first considering four determinants: the diseases selected; the possible sources of their agents; exposure risk factors; and, the population risk from exposure to waterborne microbial hazards.
Table 10.1  Risk affecting characteristics for selected zoonotic pathogens.

<table>
<thead>
<tr>
<th>Potential risk factors</th>
<th>Pathogen/hostr</th>
<th>Campylobacter jejuni</th>
<th>E. coli (EHEC)</th>
<th>Salmonella</th>
<th>Giardia lamblia</th>
<th>Cryptosporidium (parvum or hominis)</th>
</tr>
</thead>
<tbody>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Pathogen/host</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Infectivity for healthy adults (ID$_{50}$)$^a$</td>
<td></td>
<td>897</td>
<td>750</td>
<td>23,600</td>
<td>35</td>
<td>35</td>
</tr>
<tr>
<td>Severity of infection</td>
<td></td>
<td>Mild</td>
<td>Severe</td>
<td>Moderate</td>
<td>Mild</td>
<td>Moderate</td>
</tr>
<tr>
<td>Higher susceptibility for children?</td>
<td></td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>**Pathogen in excreta from individual animals (frequency, relative concentrations)$^{b,g}$</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Cattle (10–30 kg/day)$^c$</td>
<td></td>
<td>(H, H)</td>
<td>(L, H)$^d$</td>
<td>(L, L)</td>
<td>(M, M)</td>
<td>(L, H)</td>
</tr>
<tr>
<td>Swine (2.7–4.0 kg/day)$^c$</td>
<td></td>
<td>(H, M)</td>
<td>(L, M)</td>
<td>(M, M)</td>
<td>(M, L)</td>
<td>(L, L)</td>
</tr>
<tr>
<td>Sheep (0.7–1.5 kg/day)$^c$</td>
<td></td>
<td>(H, H)</td>
<td>(L, H)</td>
<td>(L, L)</td>
<td>(L, L)</td>
<td>(L, L)</td>
</tr>
<tr>
<td>Poultry (0.1–0.14 kg/day)$^c$</td>
<td></td>
<td>(H, H)</td>
<td>(L, L)</td>
<td>(M, M)</td>
<td>(L, L)</td>
<td>(L, L)</td>
</tr>
<tr>
<td><strong>Pathogen survival in environment</strong></td>
<td></td>
<td>(S, S)</td>
<td>(M, M)</td>
<td>(L, M)</td>
<td>(L, L)$^f$</td>
<td>(L, L)$^f$</td>
</tr>
<tr>
<td>Survival, days (faeces, water)$^e$</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

$^a$ ID$_{50}$ values for these pathogens are reviewed in the Appendix (ID$_{50}$ is the dose for which, on average, half of an exposed population will be infected).

$^b$ L, M, H = Low, Medium, High. These judgements have been made in the light of the data summarized in the Appendix.

$^c$ Typical faecal load from each animal group.

$^d$ Marked seasonal effect; highest in summer.

$^e$ S, M, L = Short, Medium, Long. The metric for “Survival” is the $T_{90}$: the time for 90% of the original population of pathogens to be inactivated.

$^f$ Marked seasonal effect, longer survival times in cooler conditions.

$^g$ The model also includes the category “supershedders”. The values for this are the same as for E. coli in cattle but with the relative concentration of enterohaemorrhagic E. coli given an extremely high value, that is, 1000 times greater than the “average” concentration.
10.2.1 The diseases

The severity of consequences of exposure depend on a number of factors related to the pathogen and the host including the infectiousness of the pathogen, the dose ingested, and the severity of disease that is the usual outcome. In turn, the severity of the disease depends on the susceptibility of the host to infection by the pathogen. As discussed above, there is variability in each of these risk-affecting factors but in the risk assessment tool developed here we adopt average values to characterise the relative risk. As noted above, if conservative or worst-case values are taken for each variable, the resulting risk estimate is characterized by an extremely improbable event. It should also be noted that point estimates based on measures of central tendencies, for example, average, or mode, will not necessarily lead to an answer that represents the most likely outcome and can lead to large errors (Cassin et al. 1996). Nonetheless, we have included different categories in the risk ranking tool where appropriate to be able to distinguish situations when risks are systematically higher or systematically lower. For example, children are often more susceptible to infection than adults and for this reason we have included options in the tool that can differentiate this risk, or if there is some correlation between sporadic contamination and the likelihood that people will be exposed to the recreational water. (Differential susceptibility is discussed in greater detail below.) In the case of differential exposure due to age, the population exposed can be selected from “general”, “children” only or “adults” only. More sub-categories could easily be included in the tool if there were data that showed that specific populations were physiologically more likely to become infected. Note, also, that some populations are more likely to be exposed due to custom, location, and so on, but this aspect is addressed in a separate question concerning frequency of exposure to the recreational water resource being assessed.

10.2.2 Assessing infectivity

The infectiousness of a pathogen is sometimes characterised as “the infectious dose”. This is inappropriate because there is variability in the number of pathogens required to cause infection or illness (depending on the pathogen itself and the susceptibility of the consumer). Recognising this, infectiousness is often characterised by the ID$_{50}$: the number of cells of the pathogen that results in 50% of the exposed population becoming infected. The relationship between probability of infection and dose ingested is described as the dose-response curve and can be described by a variety of mathematical equations. A detailed review of dose-response relationships for infection processes, both from a biological and mathematical perspective, is presented in FAO/WHO (2003).
For many diseases evaluation of ID$_{50}$ has come from clinical trials using healthy adult volunteers. It therefore ignores any elevated health risks that may be faced by children, by immuno-compromised people, and by the elderly (USEPA 2000b, Nwachuku & Gerba 2004, Wade 2008). The pattern for children may be of considerable importance for developing countries where it is known that children can exhibit campylobacteriosis rates many times higher than those for adults (Blaser 1997, Rao et al. 2001, Teunis et al. 2005). In developed countries such as New Zealand the reported illness rates for all five of the selected diseases exhibit higher rates among children (see www.nzpho.org.nz, Lake et al. 2011) and similar findings have been made for Scotland (Strachan et al. 2009) and in USA (Denno et al. 2009). Accordingly, differential rates between children and adults need careful attention. A good example of differential sensitivity of identifiable sub-populations has been demonstrated for listeriosis: the susceptibility to infection from this food-borne pathogen ranges over 1000-fold between the healthy, young adult, population and those who are immuno-compromised due to underlying illness (e.g. AIDS) or medical treatment such as organ transplant recipients (Marchetti 1996).

10.2.3 Assessing severity

The dose-response relationship also does not consider the severity of the illness. For example, Salmonella infections are usually self-limiting and of relatively short duration, while infections from enterohaemorrhagic E. coli often lead to life threatening illness which is clearly more severe. Similarly, reliance only on clinical trial data frequently ignores any sequelae that may arise. For example, Guillain-Barré Syndrome may affect about 0.03% of people who have contracted campylobacteriosis (McCarthy & Giesecke 2001). Infections with Salmonella or Campylobacter have been found to increase the short term risk of death and long term mortality (Helms et al. 2003).

One way of comparing disease severity is to use the metric of the “disability adjusted life years” (DALY) concept, originally developed by Murray and Lopez (1996) and adopted by the World Health Organization to inform global health planning (AIHW 2000, Kemmeren et al. 2006). The DALY is a measure of the years of “healthy” life lost due to illness or injury, that is, time lived in states of ‘less-than-full’ health. DALYs are calculated as the sum of years of life lost due to premature death (YLL) and the equivalent years of “healthy” life lost due to poor health or disability (YLD). The YLD considers the extent of the disability that is endured, that is, YLD is weighted according to the severity of the disability. The origin and application of the DALY concept, particularly in
relation to the establishment of disability estimates, and their relevance to undeveloped nations, was reviewed King & Bertino (2008).

10.2.4 The sources

The sources considered here will be confined to four major groups of domestic animals in the world: cattle, swine, sheep and poultry. The world-wide animal census developed by the Food and Agriculture Organization of the United Nations (http://faostat.fao.org) in 2009 lists cattle as the largest animal population at 1.34 billion. Sheep are the next most numerous at 1.09 billion (note that sheep tend to shed similar amounts of annual faecal material per unit area, compared with cattle—Wilcock 2006) and hogs follow at 0.92 billion population. Poultry outnumber all of the above three large animal populations by 5.3 to 1 with a world population of 17.86 billion. These high population numbers are mainly due to the great commercial value associated with these domestic birds and animals. They are the major groups related to food production around the world. This particular group of domestic livestock is also of special significance because in many countries they are held in Concentrated Animal Feeding Operations (CAFO) where many thousands of animals are confined in very small areas. Faecal wastes from CAFO’s are usually treated in septic lagoon systems before discharge to receiving waters. The risks of illness associated with exposure to the discharged animal wastes is, however, largely unknown (see: Chapter 11).

The world population of other birds and animals, such as geese, ducks, horses and goats, are very small relative to the above four species. Although urbanized geese and gulls are well recognized as major polluters of bathing beaches, they will not be considered because of their relatively small populations. Wild animals and wild birds are likewise not considered, even though their population densities in the world might be quite high and their faecal contribution to recreational waters is well recognized. Furthermore, good estimates of feral bird and animal populations in the world are not available and the linkage of human enteric illness attributable to zoonotic pathogens from feral animals and birds is not very strong.

10.3 THE EXPOSURES AND RISK FACTORS

We consider risk of human infection from recreational water contact by ingestion only, excluding any risk from inhalation. We do not consider the “knock-on” effects whereby food gets contaminated via water (e.g., irrigation, or processing
water), that is, whereas an illness was foodborne, the source was contaminated water.

### 10.3.1 Ingestion rates

The ingestion of water during swimming activities can be a significant factor affecting risk. There is a dearth of empirically collected data on ingestion of water by swimmers. In a study of divers wearing masks, estimates based on self-reported volumes of water that the divers believed they had swallowed were mainly in the range of 30 mL or less, but with some reporting much larger volumes (Schijven & de Roda Husman 2006). These self-assessed volumes of ingested water were not dissimilar from the amounts of water swallowed by recreational swimmers in a pilot study conducted in a swimming pool (Dufour et al. 2006). In that study, ingestion of water was estimated by the amount of cyanuric acid measured in a 24 hour urine sample collected after a one hour swimming activity in a pool disinfected with chlorine isocyanurate. The average amount of water swallowed by 53 participants was about 30 mL. Swimmers less than 16 years old swallowed about 37 mL of water, which was more than twice that swallowed by adults (average 16 mL). These systematic differences should be taken into account in risk assessments because they directly affect exposure.

### 10.3.2 Climate change

Risk of illness associated with exposure to non-human faecal pollution may be significantly affected by global warming and climate changes. Events similar to those which might occur under global warming conditions have been observed in recent years (Epstein 2005, Patz et al. 2005). Weather extremes related to atmospheric and ocean warming have resulted in heat waves and extensive flooding, and these phenomena have given a preview of what may be expected under full-scale global warming. In North America, weather extremes have resulted in drought and very high temperatures, and in unusually heavy rainstorms that have caused extensive flooding. Curriero et al. (2000) have documented an association between extreme rainfall and waterborne disease outbreaks in the United States. They showed that over 50% of the drinking-water outbreaks of disease in the United States were associated with rain events above the 90th percentile value of total monthly precipitation. Similarly, Thomas et al. (2006) have shown that in Canada “high impact” weather events are associated with waterborne disease outbreaks. The association between outbreaks of disease and extreme rainfall events described in these studies may be a harbinger of

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5 Similar results were obtained in a follow-up study (Evans et al. 2006).
increased risk of disease related to global warming and climate change. Conversely, high temperatures may also have the effect of decreasing risk associated with animal or faecal pollution in some regions. Higher temperatures and lack of rainfall may cause desiccation of faecal material in open areas and thereby enhance the die-off of pathogens that might otherwise survive and contaminate water resources (Sinton et al. 2007a, Meays et al. 2005). Risk modifying events of the type described above will have to be anticipated in future assessments of the relationship between animal and bird faecal pollution, and human health.

10.4 THE COMPARATIVE RISK MODEL

10.4.1 Background and objectives

Microbial risk assessment modelling is gaining importance in relation to water quality and protection. According to Coffey et al. (2007) risk assessment models are critical to protect human health from contaminated water sources.

A number of models that are intended for, or could be adapted to, microbial waterborne risk assessment do currently exist and were reviewed by Coffey et al. (2007). Some are qualitative while others are quantitative and complex in terms of data needs and computational structure. While qualitative approaches can provide an effective means of assessing risks with minimum resources and limited data, they lack the precision and predictive ability of fully quantitative approaches. Conversely, the quantitative models are complex and require large amounts of data, are variable in their accuracy and, in the evaluation of Coffey et al. (2007), no one model could account for all hydrological and geological factors of relevance and also model the physical transport of bacteria in surface run-off. The best performing models were of medium to high complexity in terms of user expertise and the quantity of data required for their implementation.

Coffey et al. (2007) observed that the most common bacterial model used to estimate bacterial loadings was HSPF (Hydrological Simulation Programme—Fortran), but that it was complex, requires large quantities of monitoring data, needs extensive calibration, and had a limited capacity to accurately represent diverse watershed topography and land uses. They further noted that some models were not full, qualitative models, but can give a good initial estimate of risk from pathogens and highlight requirements for a full quantitative assessment.

some uses in food safety risk assessment, in this Chapter we seek to translate that model into a format appropriate for use in microbial water quality risk assessment and present it for evaluation.

It is not intended nor inferred that the model presented can provide accurate estimates of recreational waterborne risks under all circumstances and scenarios. Rather, it can provide quick screening of relative risk, and the effects of multiple factors in combination on overall risk. It is also intended to illustrate an approach that could make risk assessments for recreational waters more accessible and also has great utility in teaching the principles and philosophy of quantitative risk assessment.

However, the model clearly has limitations. For example, while the model is superficially simple to use, it relies on a relatively high level of knowledge of the watershed being considered to be able to answer the questions appropriately. If answers to the questions posed are inappropriate, the relative risk estimates from the model will be unreliable (in other words: “garbage in – garbage out”). While the logic inherent in the model is essentially correct, the weightings used for responses to the answers may not be appropriate in all situations and this could lead to unrealistic or illogical conclusions in some circumstances. Furthermore, the model only considers one source of faecal contamination at a time when, in many circumstances, there will be multiple sources of contamination. Nonetheless, the model could be used to estimate which source represents the greatest risk by assessing each source separately, or assessing the combined risk from multiple sources.

Users should be aware of the uses and limitations of the model. Such limitations and caveats were discussed in detail by Ross and Sumner (2002) in relation to the food safety risk assessment model and most apply equally to the model presented here.

10.4.2 Model structure and interface

Evaluation of the health risk from a water source requires knowledge of the strength (“load”) of the hazard, and an understanding of the modification of pathogen numbers together with the characteristics of the “transport” (Goss & Richards, 2008) and routes of exposure. The structure of the decision tool corresponds to that generally accepted paradigm (i.e. load, transport, exposure) and can be considered as three banks of questions corresponding to those three aspects of risk.

The model attempts to consider the collective contribution of many factors to the overall risk to the public exposed to bodies of water, whether due to
recreation, domestic needs (e.g., clothes washing in developing nations; food gathering) or employment (e.g. irrigation farmers, fishermen).

The model is implemented in spreadsheet software and uses an approach similar to the microbial food safety risk model of Ross & Sumner (2002). The benefits of the use of spreadsheet software are that it:

- Allows automation of the calculations required to estimate the risk, facilitating a quick exploration of the effect of different assumptions by the user;
- Is widely available and used, that is, users do not need special training nor to have access to specialized software.

Users are presented with a series of sixteen questions and asked to select from a list of possible answers to those questions. Figure 10.2 shows the ‘user interface’ of the tool. Question 1 relates to the animals that are the source of the contamination to estimate the severity of the microbial hazard. Questions 2 to 5 are used to estimate the pathogen “load”. Questions 6 to 11 relate to mobilisation and transport of the pathogens to the recreational water body being assessed. Questions 12 to 16 relate to exposure to the water body being assessed.

The model requires users to provide answers based on ‘average’ situations, not extreme or unusual circumstances, for the purposes of estimating relative risk. However, users could potentially use the model to estimate the relative risk increase, or decrease, due to unusual circumstances that may be of interest or relevance for water safety management.6

In its current form the model is limited to consideration of faecal contamination of recreational water by farmed cattle, sheep, pigs or poultry.

10.4.3 Model logic

The overall principal of the tool is that the answers that are chosen by the user for each of the qualitative Questions 1 to 16 are assigned numeric values. The numeric values are assumptions about the relative risk contribution of the alternative answers offered for each question. Those values can then be used in calculations to generate estimates of relative risk.

6 Note, however, that in the model if only a single value is changed, the predicted change in risk will in nearly all cases simply reflect the difference in “weight” applied to the subjective answers offered to the user. The weights are a very simplified measure of relative risk contribution from each factor and changing the answer to one question will not usually generate a reliable estimate of the increased relative risk, because the weights used are, for most questions, arbitrary. The benefit of the model is to assess the influence on relative risk of simultaneous changes in multiple risk-affecting factors.
Figure 10.2 Image of the ‘user-interface’ of the recreational waterborne microbial risk assessment model, showing the questions, alternative answers and risk outputs. Users select answers from the ‘pull down’ menus, which are translated into numerical values used to calculate the risk indices.
In some cases the values ascribed are similar to relevant absolute values (e.g., the ID$_{50}$ values used in Question 1; in Question 15 weekly exposure is weighted four times as heavily as monthly exposure, etc). In other cases the values are relative to the most extreme value. For example, for Question 5: “continuous contamination” has a weight of ‘1’, and other frequencies of input/contamination are weighted relative to that value, for example, “frequent” contamination has arbitrarily been assigned a value of 0.3, “rare” has been assigned a value 0.001.

The relative risk of different scenarios is calculated, essentially, as the simple product of the relative risk from each of the risk-contributing factors explicitly addressed in Questions 1 to 16. There are three exceptions, however, where a logical test is also applied. The first relates to assessment of the efficacy estimates of actions taken to reduce contamination of the water resource being considered, and aims to jointly assess the ability of the action to reduce contamination as well as the reliability of the process. The logic involved is discussed in detail later, in the sections describing those questions.

The second case relates to the possibility that human exposure is, in some way, correlated with contamination events. Question 16 asks the user to comment on whether correlations, either positive or negative, between contamination events and human exposure are possible and to rank these as “possible” or “probable”. If, however, contamination is “continuous” or “frequent” or if exposure is “daily” or more frequent, it is assumed any such correlation is irrelevant because exposure frequency and likelihood of contamination are such that there is near certainty of exposure to contaminated water and that the relative risk cannot be increased nor decreased. However when exposure to the water is low and contamination is rare, the risk will be under-predicted if there is a correlation between exposure to the recreational water and contamination, for example, if contamination, while rare, were more likely in summer, when people are more likely to swim. Conversely, if contamination events are rare but are detected in sufficient time to alert swimmers prior to the contamination reaching the recreational water, there would be a negative correlation between contamination events and exposure.

The third use of a logical test, as explained in the next section, is to determine the greatest hazard potential from pathogens in different animal sources of contamination.

10.4.4 The questions

The following section provides advice on interpretation of the sixteen questions as well as describing the relative risk weights applied to each of the possible responses.
10.4.4.1 Identifying the source

Question 1 asks the user to select the type of farmed animal population that contributes most to the source of contamination of the recreational/working water body. Doing so determines both the pathogen considered to present the greatest risk from that animal species and also the relative risk, based on information presented in Table 10.1, as described below. The overall hazard presented by the pathogen is considered to arise from:

- its relative prevalence among herds/flocks of the animal selected;
- the concentration of the pathogen in faeces of the species selected;
- the relative survival of the pathogen in the environment (expressed as $T_{90}$);
- the relative severity of the disease caused by the pathogen;
- the infectiousness of the pathogen as expressed by the $ID_{50}$.

For each animal group a simplified index of “hazardousness” for each of the five pathogens considered is calculated by the following formula:

$$Relative\ pathogen\ risk = (relative\ prevalence \times relative\ concentration \times relative\ survival\ (or\ T_{90}) \times relative\ disease\ severity)/ID_{50}$$

The maximum of the values generated for each pathogen for the animal species selected is the relative risk value assigned as the answer to Question 1 and is used in further calculation of relative risk. It should be noted that this approach is based on several subjective decisions and assumptions. The most apparent is the translation of qualitative assessment of pathogen prevalence and concentration (see Table 10.1) into relative quantities. Table 10.2 indicates the values, or relative weights, that were applied. The weightings are based on factors of ten for simplicity but could be altered if reliable, representative, quantitative data were available.

To allow the estimation of the consequences of the effects of more extreme hazards, for example, a “supershedder”, or an epidemic level of pathogen excretion within a herd/flock, an additional choice reflecting a higher level of pathogen excretion is added to the range of responses to Question 1. Currently, selection of this option only has the effect of increasing the modelled concentration of $E.\ coli$ in the faeces of cattle by 1000-fold, but other options could be included.

In practice, the combination of the above weights and $ID_{50}$ values results in $Cryptosporidium$ representing the greatest level of hazard when cattle or poultry are selected, $Giardia$ and $Cryptosporidium$ when pigs are selected, and EHEC
for sheep except when the “supershedder” option is selected. In that case, enteropathogenic *E. coli* is evaluated to be the greatest source of risk overall. Modification of the weightings (see: Table 10.2) could change the relative risk estimates and predicted relative importance of each pathogen in each animal species. Epidemiologically, *Cryptosporidium* scores the highest as cause of detected waterborne infections (Coffey *et al.* 2007). The relative severity of illness might also be made less subjective by deriving estimates of DALYs lost for each pathogen but was not undertaken at this time. The qualitative descriptions of severity for the five pathogens applied in this example are in accordance with those presented by Goss and Richards (2008).

Table 10.2 Weighting factors applied when assessing hazard relative importance.

<table>
<thead>
<tr>
<th>Hazard Characteristic</th>
<th>Qualitative Description</th>
<th>Numerical weight assigned</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Severity of infection</strong></td>
<td>Severe</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Moderate</td>
<td>0.1</td>
</tr>
<tr>
<td></td>
<td>Mild</td>
<td>0.01</td>
</tr>
<tr>
<td><strong>Survival (in faeces, water)</strong></td>
<td>Long (L)</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Medium (M)</td>
<td>0.1</td>
</tr>
<tr>
<td></td>
<td>Short (S)</td>
<td>0.01</td>
</tr>
<tr>
<td><strong>Prevalence of pathogen in faeces</strong></td>
<td>High (H)</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Medium (M)</td>
<td>0.1</td>
</tr>
<tr>
<td></td>
<td>Low (L)</td>
<td>0.01</td>
</tr>
<tr>
<td><strong>Concentration of pathogen in faeces</strong></td>
<td>High (H)</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Medium (M)</td>
<td>0.1</td>
</tr>
<tr>
<td></td>
<td>Low (L)</td>
<td>0.01</td>
</tr>
</tbody>
</table>

The estimation of risk also relies on consideration of the dose ingested and the likelihood that the dose will lead to a symptomatic infection. The relative risk calculations in the model assume that there is a direct proportionality between the dose ingested and the probability of illness. This is generally in accord with the predictions of the dose response models considered herein (single-parameter simple exponential or two-parameter beta-Poisson, as used in the discussion of ID$_{50}$ values in the appendix to this chapter). If the dose is rather lower than the ID$_{50}$, the risk can be considered to be directly proportional to the dose (Haas 1996, Gale 2001a&b, 2003), because the dose-response relationship is linear at low doses. The assumption of proportionality is incorrect, however, if the dose in significantly greater than the ID$_{50}$ for the pathogen of interest. This is because the dose-response curve for probability of infection is asymptotic and non-linear.
at higher doses, with the asymptote being approached at doses that are at least an order-of-magnitude higher than the $\text{ID}_{50}$.\footnote{The assumption will also be incorrect if the pathogens act co-operatively to cause infection and disease, requiring more complex dose-response models (FAO/WHO, 2003).} However, due to the effects of dilution, inactivation and the volume of water ingested, it is assumed that, in most practical situations, the dose ingested will be below the $\text{ID}_{50}$ for most pathogens. In cases where this assumption is not valid (e.g., direct contamination adjacent to a point where people are exposed) the consequence will be an underestimation of the relative risk of other situations compared to that most extreme situation.

The selection of the animal source of contamination in Question 1 is also used to assign a relative weight of faeces produced per animal type, which will also affect the load. The following relative quantities of faeces per animal are assumed and used in the relative risk calculations:

- cattle: relative quantity (= relative risk) = 1
- pigs: relative quantity (= relative risk) = 0.17
- sheep: relative quantity (= relative risk) = 0.06
- poultry: relative quantity (= relative risk) = 0.006

### 10.4.4.2 Estimating load

Questions 2 to 5 relate to the load of pathogens expected to arise from the herd or flock considered as the source of contamination. The questions, and alternative answers provided, are relatively self-explanatory from the discussion presented earlier, but the weightings applied are presented below for transparency.\footnote{These weightings greatly simplify the complex set of “pathogen delivery processes” operating in the environment, such as modeled by Collins & Rutherford (2004) for *E. coli*.}

They aim to estimate the load of pathogen entering the water body by estimating the level of pathogens based on the rate and scale of faecal contamination entering the water body on the basis of animal type, herd size, herd density and frequency of contamination, and the manner in which contamination of the water occurs. More detailed discussions of factors affecting load, and approaches to minimizing load in animal faeces, are presented in Chapters 3 and 4.


The question is phrased as a statement to be completed, that is, “The density of the animal population causing contamination is ...” with four possible responses offered. Those responses, and the relative risk assigned to them are:

- high (>1000 per hectare) relative risk = 1
- medium (100 to 1000 per hectare) relative risk = 0.1
low (10 to 100 per hectare) relative risk = 0.01
very low (<10 hectare) relative risk = 0.001

Question 3: Size of Herd or Flock. The question is phrased as a statement to be completed, that is, “The size of the herd or flock causing contamination is ...” with four possible responses offered. Those responses, and the relative risk assigned to them are:

- large (1000s of animals) relative risk = 1
- medium (100s of animals) relative risk = 0.1
- small (scores of animals) relative risk = 0.01
- very small (a few animals) relative risk = 0.001

Question 4: Mode of Contamination. The question is phrased as a statement to be completed, that is, “The mode of contamination is ...” with five possible responses offered. Those responses, and the relative risk assigned to them are:

- direct – untreated relative risk = 1
- diffuse – for example, agricultural run-off grazing relative risk = 0.01
- diffuse – for example, manure spreading occurs relative risk = 0.5
- direct – primary treated effluent relative risk = 0.1
- direct – secondary treated effluent relative risk = 0.001

Other factors affecting the risk from the mode of contamination are considered in Questions 6–11, relating to mobilisation.

Question 5: Frequency of contamination. The question is phrased as a statement to be completed, that is, “The frequency of contamination is ...” with five possible responses offered. Those responses, and the relative risk assigned to them are:

- rare relative risk = 0.001
- sporadic relative risk = 0.01
- intermittent relative risk = 0.05
- frequent relative risk = 0.3
- continuous relative risk = 1

10.4.4.3 Estimating contamination of the recreational water

Pathogen concentrations in the water body to which people will be exposed will affect the probability of illness, that is, the greater the contamination level and the dose ingested, the greater the probability of illness. Questions 6 to 11 relate to mobilisation and transport of pathogens from the source to the water body of concern to estimate the reduction in pathogen load due:

- Die-off in the environment due to time and distance;
Dilution, and

The effectiveness and reliability of implementation of actions taken to minimize contamination of the water from the source considered.

More detailed discussion of mobilisation and transport, and means of assessing and minimising this, are presented in Chapters 6, 7 and 8. Growth of pathogens in the environment is assumed not to occur under scenarios relevant to this comparative risk model.

**Question 6: Proximity of Source to Exposure Site.** In addition to the effect of time on the extent of pathogen inactivation (see Question 7, below), the likelihood that the pathogen will reach the water body is reduced due to absorption onto soil particles, predation, and so on. Accordingly, risk to recreational water users will be reduced the further the contamination source (i.e., the animals that are the source of the faeces) is from the water body, or water supplying the water body. The question is phrased as a statement to be completed, that is, “Proximity of source to the exposure site is ...” with four possible responses offered. Those responses, and the relative risk assigned to them are:

- immediate (direct deposition) relative risk = 1
- close (<10 m) relative risk = 0.1
- intermediate (10–100 m) relative risk = 0.01
- distant relative risk = 0.001

**Question 7: Temporal proximity to source of exposure.** Pathogen die-off in the environment will be greater, under a given set of inimical conditions, the longer they are exposed to those conditions. As such, greater time between the point of contamination and the moment of exposure will reduce the level of pathogen in the water and, consequently, the risk of illness. Question 7 is phrased as a statement to be completed, that is, “Temporal proximity of source to the exposure site is ...” with four possible responses offered. Those responses, and the relative risk assigned to them are:

- short (up to a few hours) relative risk = 1
- intermediate (hours to days) relative risk = 0.1
- long (several days) relative risk = 0.05
- very long (several weeks) relative risk = 0.005

The weighting factors used reflect that microbial inactivation is usually characterised as a log-linear decline over time.

**Question 8: Dilution from Source to Exposure Site.** As noted above, dilution of pathogens would be expected to decrease risk because the dose ingested from a
given mode of exposure (see Question 15) will be less, leading to decreased probability of infection. Question 8 is phrased as a statement to be completed, that is, “Dilution from Source to Exposure Site is…” with four possible responses offered. Those responses, and the relative risk assigned to them are:

- slight (<5-fold) relative risk = 0.4
- medium (5 to 50-fold) relative risk = 0.04
- high (50 to 500-fold) relative risk = 0.004
- extreme (> 500-fold) relative risk = 0.002

The weights used are proportional to the means of the ranges of dilution specified.

**Question 9: Likelihood of mobilisation.** Increased mobilisation of contaminants will increase the load reaching the water body of concern. Mobilisation can be affected by agricultural practices, for example, tile-drain systems are the most frequently reported route by which liquid manure can contaminate surface water courses, but a range of other factors and practices affect mobilisation, for example, the slope and direction of land and how it affects run-off, irrigation practices, vegetation and so on. as discussed by Goss & Richards (2008). Question 9 is framed as a phrase to be completed: “The likelihood of mobilisation is …” with four possible responses offered. Those responses, and the relative risk arbitrarily assigned to them, are:

- high relative risk = 1
- medium relative risk = 0.1
- low relative risk = 0.01
- very unlikely relative risk = 0.001

**Questions 10 and 11: Effectiveness, and reliability of risk mitigating actions** are included in recognition that sufficient knowledge exists to be able to reduce risk of contamination of surface water by various practices and actions, but only if they are reliably implemented. Such actions include “streambank retirement”, fences near waterways to prevent animal ingress, bridges over water courses to allow animals to cross without entering the water. The answers to both questions are based on subjective assessments. Question 10 is framed as a statement to be completed: “The effectiveness of risk mitigating factors is … “ with five possible responses, as follows:

- absolute relative risk = 0
- extremely high relative risk = 0.0001
- high relative risk = 0.01
- medium relative risk = 0.1
- low relative risk = 1
Question 11 is also framed as a statement to be completed: “The reliability of risk mitigating factors is …” with five possible responses, as follows:

- completely reliable relative risk = 0
- virtually “fail-safe” relative risk = 0.01
- usually reliable relative risk = 0.1
- sometimes effective relative risk = 0.5
- unreliable relative risk = 0.9

The first response to both questions are unusual compared to all other responses in the risk model because both have the potential to reduce the risk estimate to zero, that is, no risk. However, a process that completely eliminates pathogens, but is unreliable still has a risk associated with it. Thus, the degree to which a process is unreliable reduces the effective risk reduction. Conversely, a completely unreliable process that has little effect on pathogen numbers cannot increase the relative risk and the relative risk score must remain as “1”. To model these logical considerations of the interplay between process efficacy and process operational reliability the two scores are combined and a logical test applied. Thus, the relative risk due to both of these factors in combination is taken as the sum of the two relative risk scores. However, to prevent the model from predicting an increase in risk from a low efficacy process operated unreliably, the “MIN” function in Microsoft Excel is used so that if the combined relative risk score is greater than “1”, the model substitutes a value of “1”. The net effect of this on the relative risk score for mitigations is shown in Table 10.3, below.

It is noted that the answers to the above questions may be subjective and can have a profound affect on the risk estimate. Accordingly, it is advised that users make careful considerations and seek guidance as needed, when assessing reliability.

10.4.4.4 Exposure

The risk to people from contaminated recreational waters depends not only on the level of contamination but also the magnitude of the exposure to that contaminated water. This depends on how frequently people are exposed and the manner in which exposure occurs. For the sake of this example of the approach to relative risk estimation we have limited the scope to the risk due to ingestion of contaminated water.

Risk can be expressed as risk to an individual or risk to an entire population, and can be affected by the susceptibility of individuals or sub-groups within the population, for example, young children may be more susceptible to pathogens because they have not yet experienced the organism nor developed immunity.
Table 10.3  Combined relative risk scores from responses to Questions 10 and 11.

<table>
<thead>
<tr>
<th>Question 10 response</th>
<th>Question 11 response</th>
<th>Completely reliable</th>
<th>Virtually “fail-safe”</th>
<th>Usually reliable</th>
<th>Sometimes reliable</th>
<th>Unreliable</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absolute</td>
<td></td>
<td>0.0001</td>
<td>0.01</td>
<td>0.1</td>
<td>0.5</td>
<td>0.9</td>
</tr>
<tr>
<td>Extremely high</td>
<td>0.0001</td>
<td>0.0001</td>
<td>0.0101</td>
<td>0.1001</td>
<td>0.5001</td>
<td>0.9001</td>
</tr>
<tr>
<td>High</td>
<td>0.01</td>
<td>0.01</td>
<td>0.02</td>
<td>0.11</td>
<td>0.51</td>
<td>0.91</td>
</tr>
<tr>
<td>Medium</td>
<td>0.1</td>
<td>0.1</td>
<td>0.11</td>
<td>0.2</td>
<td>0.6</td>
<td>1</td>
</tr>
<tr>
<td>Low</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>
The elderly can be more susceptible to infectious disease because immune function begins to diminish with age. The magnitude of risk is also affected by the time period considered, that is, a greater time period usually increases the risk of exposure. The questions in this section enable estimation and discrimination of risk on the basis of these factors.

**Question 12: Size of Affected Population.** This question is included to enable overall public health risk to be estimated, in addition to risk to individuals. In risk assessment, risk encompasses elements of probability and severity of outcomes. Severity can be considered to include both the severity and magnitude of the consequences of exposure to the hazard, that is, the number of people affected by the hazard. Question 12 simply requests the user to indicate the size of the population exposed to the microbiological hazard in the recreational water being considered. An option is included for the user to nominate the size of the population exposed, rather than to use one of the options presented. The same approach could have been used with other question as well, that is, to allow the user to specify their own estimate of relative risk for any other factor specifically considered in the model but was not implemented in this example to maintain simplicity for the sake of demonstrating the approach.

**Question 13: Composition of Population.** As noted elsewhere, susceptibility to infection varies with medical condition, age and other factors. In this question, susceptibility on the basis of age only is considered. The choices presented, and the relative risk rating assigned to those sub-population, is shown below, where the highest relative risk is assumed to apply to young children.

- General relative risk = 0.7
- Predominantly young children relative risk = 1
- Predominantly adults relative risk = 0.1

**Question 14: Frequency of Exposure.** Frequency of exposure is self-evidently a factor that contributes to the risk from hazard in a contaminated body of water. The response choices are based on common units of time and the weights applied based on the actual relationships of those times, as shown below:

- Several times a day relative risk = 1
- Daily relative risk = 0.365
- Weekly relative risk = 0.052
- Monthly relative risk = 0.013
- A few times a year relative risk = 0.003
- Once every few years relative risk = 0.0003

Note that the risk is ranked relative to a person who is exposed to the potentially contaminated water body several times a day.
Question 15: Type of Exposure. There are various ways in which people can be exposed to pathogens in contaminated recreational waters. We extend the scope here slightly to include occupational exposures as well, for example people harvesting food from such waters or washing clothes, or involved in religious or other custom. Exposure to contaminated irrigation waters, for example, from applying water or exposure to spray from overhead irrigations, is also considered. The options presented and relative risk weightings are:

- Swimming relative risk = 1
- Other primary (wading, working, etc.) relative risk = 0.3
- Secondary (splashing, wet equipment) relative risk = 0.1
- Irrigation relative risk = 0.1

Question 16: Is Time of Exposure Likely Correlated with Contamination Events. As discussed earlier, there may be situations in which contamination events and exposure are correlated. Where correlations may significantly alter risk, the following relative risk factors are applied:

- Correlation unlikely relative risk = 1
- Possible increased likelihood relative risk = 10
- Possible decreased likelihood relative risk = 0.1
- Probable increased likelihood relative risk = 100
- Probable decreased likelihood relative risk = 0.01

These factors are only included in the risk calculations where both the exposure frequency is “weekly” or less and the contamination frequency is “intermittent” or less. An additional question is included to enable users to model the effect of scenarios and factors not easily assessed via the other questions in the model. For this question, the user enters a value to represent how much better, or worse, the risk to human health from exposure to that recreational water would be with the additional factor considered. Thus, if the situation, due to some other factor is ten times worse, then the user should enter “10” in the space provided and select “increase by this factor” in the list provided. If the situation is only half as bad due to some intervention or other factor not specifically included in the model, the user would enter “2” in the space provided and select “decrease by this factor” in the list provided. If there is no effect the user can enter “1”, or “0”, or leave the box empty.

10.4.4.4 Relative risk calculations

The answers to the above questions are translated into the relative risk values shown above. These values are then used in calculations to establish how the relative risk from each factor affects the relative risk overall. In general the
answer is simply calculated as the product of the relative risk factors. Thus, the “Individual Annual Relative Risk” is based on the following calculation:

\[
\text{Individual Annual Relative Risk} = \text{source relative risk (based on the calculation described under “Question 1”, above)} \times \text{relative amount of excrement produced per animal of the species selected in Question 1 (as described above under “Question 1”, above)} \times \text{relative risk due to density of animal population (Question 2)} \times \text{relative risk due to size of herd or flock causing contamination (Question 3)} \times \text{relative risk due to mode of contamination (Question 4)} \times \text{relative frequency of contamination (Question 5)} \times \text{relative risk due to proximity of faecal contamination to recreational water (Question 6)} \times \text{dilution between source and recreational water (Question 7)} \times \text{relative risk reduction due to time between source and recreational water (Question 8)} \times \text{the likelihood of mobilisation (Question 9)} \times \text{relative risk reduction due to reliability and efficacy of mitigation actions (combination of Questions 10 and 11 as described in Table 10.3, above)} \times \text{relative risk due to composition of affected population (Question 13)} \times \text{relative frequency of exposure (Question 14)} \times \text{relative risk due to type of exposure (Question 15)} \times \text{relative risk adjustment for possible correlation between infrequent contamination and infrequent exposure (Question 16) and, if included by the user} \times \text{relative risk adjustment due to other factors not explicitly considered in the model.}
\]

The above calculation leads to a number, on an arbitrary scale, based on risk over a year of potential exposure for an individual. The higher the number, the greater the relative risk.

Assuming the most extreme relative risk (i.e., ‘1’) for each factor, and combining this with the relative risk estimate based on the animal species considered to represent the greatest hazard, generates a maximum score of \(5.33 \times 10^{-4}\). Conversely, assuming the lowest relative risk for each factor, leads to a prediction of “No Risk”. The next lowest predicted relative risk is \(2.25 \times 10^{-4}\), obtained if all answers are selected to represent the lowest relative risk, but with Questions 10 and 11 answered as “Very High” and “Completely Reliable” respectively, or as “Absolute” and “Virtually fail-safe” respectively. These extremes set the scale of relative risk for the model presented. To make the scale more ‘natural’ to users, the logarithm of the above calculation is taken
and 41 added to avoid generating negative values under some other scenarios. Similarly, the calculated value is rounded to the nearest integer. This results in a scale of relative risk from 1 to 38. (Note that the upper value can be increased if the effect of other risk-increasing factors is included using the additional question.) Because the scale is logarithmic, every unit increase in the relative risk score corresponds to a ten-fold increase in risk, that is, due to the combined effect of probability of infection and the expected severity of infection.

To calculate the relative population health risk, the individual risk is multiplied by the population size (Question 12). To set the risk calculation on a similar scale, the logarithm of the population size is added to the individual risk index.


10.5 CONCLUSIONS

Comparative risk assessment is an approach for evaluating and quantifying risks without resorting to the complex, time-intensive quantitative microbial risk assessment process. It also provides a relative quantitative aspect not available in the qualitative risk assessment process, relying instead on some narrative to describe risk when dealing with different types or sources of exposure (e.g. low, medium, high for animal excretion rates, as in Table 10.1). The comparative risk model in this chapter makes use of an interactive spreadsheet programme that can be applied in a form that is readily understood and easy to use.

While the model presented here has been developed for very specific zoonotic pathogens, it might have other applications which may be very attractive for evaluating risk under various situations. For instance, risk differences between local, regional or larger areas can be evaluated using the comparative risk model, thereby providing water resource managers with a means to prioritize where they should apply the greatest risk reduction efforts and in what order. The model could also provide risk managers with a means to determine the most effective treatment or management options regarding public health risks associated with recreational activities. Furthermore, it may provide a tool for risk managers, wherein various scenarios might be developed and evaluated to determine which approach provides the greatest public health protection. Lastly, the spreadsheet approach for applying the model may be very useful as a training tool for those not entirely familiar with the risk assessment process.

Although the comparative risk model sacrifices some of the detailed aspects of the quantitative microbial risk assessment paradigm, the relative nature of this approach is valuable for examining many of the issues associated with risk assessment. The model presented here should be considered a prototype for determining risk posed
only by specific waterborne zoonotic pathogens. It has however proved to be effective in the foods area where it has been used to evaluate risk associated with meat and fish products. The true value of the model for estimating risks to recreationists posed by waterborne zoonotic pathogens, however, will be known only after it has been evaluated under actual conditions in the field.

ACKNOWLEDGEMENTS

Jeff Soller and Nigel French provided useful information on animal studies. The first author thanks the New Zealand Foundation for Research, Science and Technology for the research grant C01X0307: “Effects-based management of aquatic ecosystems”. Desmond Till and Dr Andrew Ball reviewed the manuscript.
APPENDIX: BASIS OF VALUES PRESENTED IN TABLE 10.1

This appendix provides an explanation and reference to published literature for values presented in Table A10.1, which describes characteristics of the selected microbial hazards relevant to the risk they pose to people exposed to recreational waters contaminated by them.

**ID$_{50}$ VALUES**

ID$_{50}$ values are taken from best-estimate dose-response relationships as reported in the literature. No explicit account is taken of uncertainty, though that is often desirable in particular quantitative risk assessments (Teunis 2009).

**CAMPYLOBACTERIOSIS**

The two parameters for the beta-Poisson dose-response curve were derived by Medema et al. (1996), using data for healthy urban adult volunteers reported by Black et al. (1988). This curve, for probability of infection given an average dose, is given generally by $Pr_{infection} = 1 - (1 + d/\beta)^{-\alpha}$, where $d$ is the average dose given to each group of volunteers, $\alpha$ is a shape parameter and $\beta$ is a scale parameter. They obtained the parameter values as $\alpha = 0.145$, $\beta = 7.589$, from which ID$_{50}$ (for infection) $\approx 897$ (see also Teunis & Havelaar 2000). Teunis et al. (2005) later analysed campylobacteriosis rates among two sets of children at school camps, which indicated an ID$_{50}$ (for illness) $< 10$. In other words, even in developed countries children exhibit markedly higher rates of campylobacteriosis than is the case for adults (see also Rao et al. 2001).

**E. COLI O157:H7 INFECTION**

Teunis et al. (2008) analysed several outbreaks for illness using the two-parameter beta-Poisson dose-response model and obtained prediction parameters for the heterogeneous case ($\alpha = 0.248$, $\beta = 48.80$), which results in ID$_{50}$ (for illness) $\approx 750$.

**SALMONELLOSIS**

Haas et al. (1999) analysed infectivity of Salmonella (non-typhoid strains) in human volunteers in studies reported by McCullough and Eisele (1951a&b), obtaining ID$_{50}$ = 23,600. A more recent study (Bollaerts et al. 2008) has analysed a larger set of data which generally suggests lower ID$_{50}$ values, particularly for the “susceptible” component of a population (see also Blaser & Newman 1982 and Rose & Gerba 1991).
GIARDIASIS

Rose et al. (1991) fitted the “simple exponential model” to infections exhibited by volunteers in studies reported by Rendtorff (1954) and Rendtorff & Holt (1954), using the exponential dose response model in which 

\[ P_{\text{infection}} = 1 - e^{-rd} \]

where \( d \) is again the average dose given to each group of volunteers and \( r \) is the probability that a single \( Giardia \) cyst could cause infection. They obtained \( r = 0.01982 \). Therefore the \( ID_{50} = -\ln(\frac{1}{2})/r = 0.693/r \approx 35 \).

CRYPTOSPORIDIOSIS

Clinical trials for infectivity of oocysts of \( Cryptosporidium parvum \) were done as part of a set of three studies in the Medical School of the University of Texas. Individual analyses for each set have generally indicated that the appropriate dose-response curve is the single-parameter “simple exponential model”. But a meta-analysis has identified different infectivity levels when fitting a number of candidate curves to each trial’s dataset, such that the differences depend on the particular isolate used and on the method of “passaging” the \( Cryptosporidium \) in the laboratory (Teunis et al. 2002a, 2002b). Having regard to all these studies USEPA (2003), in developing its “Long Term 2 Enhanced Surface Water Treatment Rule” for drinking water, concluded that the dose-response function (for infection, cf. illness) should indeed be of “simple exponential” form, with a particular value of its single parameter (\( r = 0.09 \)). This gives rise to \( ID_{50} \approx 8 \). However, two further studies have since been reported. Teunis (2009) has interpreted all five studies together, together with a sixth, and this (omitting the infectious TU502 Crypto. hominis data, because it has a human source) leads to a conclusion that on average the \( ID_{50} \) for \( Cryptosporidium \) can be taken as approximately the same as is inferred for \( Giardia \) (i.e., about 35).

PATHOGENS IN ANIMAL EXCRETA

The following material has been particularly guided by information presented by Soller et al. (2010) and USEPA (2010), along with some other published literature. Chapter 3 of this text gives further detailed information.

Tables A10.1–A10.5 present summaries of studies of prevalence and concentration of the five pathogens considered in this chapter, each including the four animal groups

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9 These studies were conducted for the TAMU, Iowa and ICP isolates (Okhuysen et al. 1999).
10 The Moredun Crypto. parvum isolate (Okhuysen et al. 2002) and the TU502 Crypto. hominis isolate (Chappell et al. 2006).
11 The 16W (Crypto. parvum) isolate.
<table>
<thead>
<tr>
<th>Reference</th>
<th>Prevalence (%)</th>
<th>Concentration</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cattle</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Berry <em>et al.</em> (2007)</td>
<td>2.2–14.9</td>
<td>–</td>
<td>Beef cattle feedlots</td>
</tr>
<tr>
<td>Besser <em>et al.</em> (2005)</td>
<td>1.6–62.2</td>
<td>–</td>
<td>Beef cattle feedlots</td>
</tr>
<tr>
<td>Devane <em>et al.</em> (2005)</td>
<td>97.8</td>
<td>–</td>
<td>New Zealand dairy cattle (all positive for <em>C. jejuni</em>)</td>
</tr>
<tr>
<td>Hakkinen &amp; Hänninen (2009)</td>
<td>49.7</td>
<td>–</td>
<td>Substantial differences between herds</td>
</tr>
<tr>
<td>Hutchison <em>et al.</em> (2004/5)</td>
<td>12.8</td>
<td>320 cfu/g (g.m.)</td>
<td>Fresh composite farm manure, UK. max. = 1.5 × 10^5 cfu/g</td>
</tr>
<tr>
<td>Kwan <em>et al.</em> (2008)</td>
<td>35.9</td>
<td>–</td>
<td>Five NW England farms, prevalence range = 26.4% (winter) to 50.8% (summer)</td>
</tr>
<tr>
<td>McAllister <em>et al.</em> (2005)</td>
<td>30–47</td>
<td>–</td>
<td>Cows (Ontario, Canada)</td>
</tr>
<tr>
<td>McAllister <em>et al.</em> (2005)</td>
<td>41.7</td>
<td>–</td>
<td>Calves (British Columbia, Canada)</td>
</tr>
<tr>
<td>Moriarty <em>et al.</em> (2008)</td>
<td>41.7</td>
<td>430 cfu/g (med.)</td>
<td>New Zealand: Concentration range 15–1.8 × 10^7 cfu/g</td>
</tr>
<tr>
<td>Stanley <em>et al.</em> (1998a)</td>
<td>–</td>
<td>610 MPN/g (ave.)</td>
<td>UK beef cattle at slaughter</td>
</tr>
<tr>
<td></td>
<td>–</td>
<td>69.9 MPN/g (ave.)</td>
<td>UK cows</td>
</tr>
<tr>
<td></td>
<td>–</td>
<td>33,000 MPN/g (ave.)</td>
<td>UK calves</td>
</tr>
<tr>
<td><strong>Swine</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dorner <em>et al.</em> (2004)</td>
<td>45.9, 79.7</td>
<td>–</td>
<td>Canadian sows and gilts (females, not yet mated)</td>
</tr>
<tr>
<td>Hutchison <em>et al.</em> (2004/5)</td>
<td>13.5</td>
<td>310 cfu/g (g.m.)</td>
<td>Fresh composite farm manure, UK. max. = 1.5 × 10^4 cfu/g</td>
</tr>
</tbody>
</table>

(Continued)
<table>
<thead>
<tr>
<th>Reference</th>
<th>Prevalence (%)</th>
<th>Concentration</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weijtens et al. (1997)</td>
<td>–</td>
<td>$10^{3.6}$–$10^5$ cfu/g</td>
<td>Five samples. Shedding dominated by <em>C. coli</em>, less infectious to humans cf. <em>C. jejuni</em></td>
</tr>
<tr>
<td><strong>Sheep</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Akik &amp; Cetinkay (2006)</td>
<td>49.5</td>
<td>–</td>
<td>Intestinal contents, gall bladders and faeces from 610 healthy sheep</td>
</tr>
<tr>
<td>Brown et al. (2004)</td>
<td>25</td>
<td>–</td>
<td>Rural Cheshire, UK, for <em>C. jejuni</em>; 21% positive for <em>C. coli</em></td>
</tr>
<tr>
<td>Devane et al. (2005)</td>
<td>59.8</td>
<td>–</td>
<td>New Zealand dairy cattle (52/66 positive for <em>C. jejuni</em>)</td>
</tr>
<tr>
<td>Hutchison et al. (2004/5)</td>
<td>20.8</td>
<td>390 cfu/g (g.m.)</td>
<td>Fresh composite farm manure, UK. Max. = 2100 cfu/g</td>
</tr>
<tr>
<td>Rotariu et al. (2009)</td>
<td>22</td>
<td>$2.7 \times 10^4$ cfu/g</td>
<td>Cattle vs. sheep. No statistically significant difference in prevalence or average concentrations for cattle or sheep between hosts or regions in Scotland.</td>
</tr>
<tr>
<td></td>
<td>25</td>
<td>$2.0 \times 10^5$ cfu/g</td>
<td></td>
</tr>
<tr>
<td>Stanley et al. (1998b)</td>
<td>91.7</td>
<td>$10^7$–$10^7$ MPN/g</td>
<td>Thermophilic <em>Campylobacter</em> in lambs. See also Skelly &amp; Weinstein (2003)</td>
</tr>
<tr>
<td></td>
<td>29.3</td>
<td>–</td>
<td>Adult sheep</td>
</tr>
<tr>
<td><strong>Poultry</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cox et al. (2002)</td>
<td>–</td>
<td>$10^{2.8}$–$10^{3.9}$ cfu/g</td>
<td>Breeders: composite samples from 35 farms</td>
</tr>
<tr>
<td></td>
<td>–</td>
<td>$10^{3.5}$–$10^{6.5}$ cfu/g</td>
<td>Broilers: composite samples from 35 farms</td>
</tr>
<tr>
<td>Reference</td>
<td>Prevalence (%)</td>
<td>Concentration</td>
<td>Notes</td>
</tr>
<tr>
<td>--------------------------</td>
<td>----------------</td>
<td>---------------</td>
<td>----------------------------------------------------------------------</td>
</tr>
<tr>
<td>El-Shibiny et al. (2005)</td>
<td>–</td>
<td>$10^6$–$10^9$ cfu/g</td>
<td></td>
</tr>
<tr>
<td>Hutchison et al. (2004/5)</td>
<td>19.4</td>
<td>260 cfu/g (g.m.)</td>
<td>Fresh composite farm manure, UK. max. = $2.9 \times 10^4$ cfu/g</td>
</tr>
<tr>
<td>Stanley &amp; Jones (2003)</td>
<td>Up to 100%</td>
<td>–</td>
<td>Varies between flocks</td>
</tr>
</tbody>
</table>

“g.m.” = geometric mean, “ave.” = arithmetic mean, “max.” = maximum
Table A10.2  Pathogenic *E. coli*.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Prevalence (%)</th>
<th>Concentration</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cattle/cows/calves</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Besser <em>et al.</em> (2001)</td>
<td>–</td>
<td>≤30 to ≥10^7/g</td>
<td>Calves, Washington State</td>
</tr>
<tr>
<td>Chase-Topping <em>et al.</em> (2006/7)</td>
<td>–</td>
<td>–</td>
<td>Demonstrated presence of “super-shedders” within herds</td>
</tr>
<tr>
<td>Donkersgoed <em>et al.</em> (1999)</td>
<td>19.7, 0.7</td>
<td>–</td>
<td>Summer, winter</td>
</tr>
<tr>
<td>Duffy (2003)</td>
<td>0.1–62</td>
<td>&lt;3 to 2.4 × 10^4</td>
<td>Irish stock (review of 26 studies)</td>
</tr>
<tr>
<td>Fegen <em>et al.</em> (2003)</td>
<td>–</td>
<td>&lt;3 to 2.4 × 10^4</td>
<td>Australia</td>
</tr>
<tr>
<td>Hancock <em>et al.</em> (1997)</td>
<td>1.8</td>
<td>–</td>
<td>Cattle feedlots</td>
</tr>
<tr>
<td>Hutchison <em>et al.</em> (2004/5)</td>
<td>13.2</td>
<td>1200 cfu/g (g.m.)</td>
<td>Fresh composite farm manure, UK. max. = 2.6 × 10^8 cfu/g</td>
</tr>
<tr>
<td>Meyer-Broseta <em>et al.</em> (2001)</td>
<td>0–100</td>
<td>–</td>
<td>France</td>
</tr>
<tr>
<td>Reinstein <em>et al.</em> (2009)</td>
<td>7.2</td>
<td>–</td>
<td>Kansas, organically fed: range = 0–24%.</td>
</tr>
<tr>
<td>Robinson <em>et al.</em> (2004)</td>
<td>–</td>
<td>up to 10^6 cfu/g</td>
<td>Kansas, naturally raised: range = 0–20.3%</td>
</tr>
<tr>
<td><strong>Swine</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chapman <em>et al.</em> (1997)</td>
<td>0.4</td>
<td>–</td>
<td>Fresh composite farm manure, UK. max. = 7.5 × 10^5 cfu/g</td>
</tr>
<tr>
<td>Hutchison <em>et al.</em> (2004/5)</td>
<td>11.9</td>
<td>3900 cfu/g (g.m.)</td>
<td>Fresh composite farm manure, UK. max. = 7.5 × 10^5 cfu/g</td>
</tr>
<tr>
<td><strong>Sheep</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hutchison <em>et al.</em> (2004/5)</td>
<td>20.8</td>
<td>780 cfu/g (g.m.)</td>
<td>Fresh composite farm manure, UK. max. = 4.9 × 10^4 cfu/g</td>
</tr>
<tr>
<td>Kudva <em>et al.</em> (1998)</td>
<td>&lt;10^2–10^6 cfu/g</td>
<td>–</td>
<td>United Kingdom</td>
</tr>
<tr>
<td>Ogden <em>et al.</em> (2005)</td>
<td>6.5</td>
<td>&lt;10^2 to &gt;10^6 cfu/g</td>
<td>Negligible presence/excretion: Chapman <em>et al.</em> (1997), Hutchison <em>et al.</em> (2004/5)</td>
</tr>
<tr>
<td><strong>Poultry</strong></td>
<td>0</td>
<td>–</td>
<td></td>
</tr>
</tbody>
</table>

“g.m.” = geometric mean, “ave.” = arithmetic mean, “max.” = maximum
Table A10.3  *Salmonella*.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Prevalence (%)</th>
<th>Concentration</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cattle/cows/calves</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hutchison <em>et al.</em> (2004/5)</td>
<td>7.7</td>
<td>2100 cfu/g (g.m.)</td>
<td>Fresh composite farm manure, UK. max. = $5.8 \times 10^5$ cfu/g</td>
</tr>
<tr>
<td>Rodriguez <em>et al.</em> (2006)</td>
<td>0.4</td>
<td>–</td>
<td>From five USA states; rectal swabs</td>
</tr>
<tr>
<td><strong>Swine</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Davies (1998)</td>
<td>4, 60</td>
<td>–</td>
<td>In two North Caroline herds</td>
</tr>
<tr>
<td>Hutchison <em>et al.</em> (2004/5)</td>
<td>7.9</td>
<td>600 cfu/g (g.m.)</td>
<td>Fresh composite farm manure, UK. max. = $7.8 \times 10^4$ cfu/g</td>
</tr>
<tr>
<td>Sanchez <em>et al.</em> (2007)</td>
<td>17</td>
<td>–</td>
<td>Literature survey on subclinical infections in swine</td>
</tr>
<tr>
<td>Rodriguez <em>et al.</em> (2006)</td>
<td>6.0</td>
<td>–</td>
<td>From five USA states; rectal swabs</td>
</tr>
<tr>
<td><strong>Sheep</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hutchison <em>et al.</em> (2004/5)</td>
<td>8.3</td>
<td>710 cfu/g (g.m.)</td>
<td>Fresh composite farm manure, UK. max. = $2.0 \times 10^3$ cfu/g</td>
</tr>
<tr>
<td><strong>Poultry</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Li <em>et al.</em> (2007)</td>
<td>30.8</td>
<td>16.2 /g</td>
<td>North Carolina; average over all bird ages</td>
</tr>
<tr>
<td>Hutchison <em>et al.</em> (2004/5)</td>
<td>17.9</td>
<td>220 cfu/g (g.m.)</td>
<td>Fresh composite farm manure, UK. max. = $2.2 \times 10^4$ cfu/g</td>
</tr>
<tr>
<td>Rodriguez <em>et al.</em> (2006)</td>
<td>0.2</td>
<td>–</td>
<td>From five USA states; rectal swabs</td>
</tr>
</tbody>
</table>

“g.m.” = geometric mean, “ave.” = arithmetic mean, “max.” = maximum
Table A10.4  *Giardia.*

<table>
<thead>
<tr>
<th>Reference</th>
<th>Prevalence (%)</th>
<th>Concentration</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cattle/cows/calves</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heitman <em>et al.</em> (2002)</td>
<td>&lt;10–60</td>
<td>5800 cysts/g (ave.)</td>
<td>North Saskatchewan River Basin, Alberta</td>
</tr>
<tr>
<td>Hutchison <em>et al.</em> (2004)</td>
<td>3.6</td>
<td>10 /g</td>
<td>Fresh composite farm manure, UK.</td>
</tr>
<tr>
<td>McAllister <em>et al.</em> (2005)</td>
<td>8.7</td>
<td>85.9 cysts/g (ave.)</td>
<td>Cows (Ontario, Canada)</td>
</tr>
<tr>
<td></td>
<td>36</td>
<td>–</td>
<td>Calves (British Columbia, Canada), max. = 113,000 cysts/g</td>
</tr>
<tr>
<td>Nydam <em>et al.</em> (2001)</td>
<td>–</td>
<td>–</td>
<td>An infected calf could produce $3.8 \times 10^7$ oocysts in 6 days</td>
</tr>
<tr>
<td><strong>Swine</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hutchison <em>et al.</em> (2004)</td>
<td>2.4</td>
<td>68 /g (g.m.)</td>
<td>Fresh composite farm manure, UK.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>max. = $2.95 \times 10^4$ cfu/g</td>
</tr>
<tr>
<td><strong>Sheep</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hutchison <em>et al.</em> (2004)</td>
<td>20.8</td>
<td>$3.8 \times 10^2$ /g (g.m.)</td>
<td>Fresh composite farm manure, UK.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>max. = $1.6 \times 10^5$ cfu/g</td>
</tr>
<tr>
<td>Ryan <em>et al.</em> (2005)</td>
<td>8.7</td>
<td>–</td>
<td>Australia</td>
</tr>
<tr>
<td><strong>Poultry</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

“g.m.” = geometric mean, “ave.” = arithmetic mean, “max.” = maximum
Table A10.5 Cryptosporidium.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Prevalence (%)</th>
<th>Concentration</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cattle/cows/calves</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Davies et al. (2005a)</td>
<td>–</td>
<td>331 / gdw, (ave.)</td>
<td>Adult cattle; represents about $10^7$ oocysts per animal per day</td>
</tr>
<tr>
<td>Heitman et al. (2002)</td>
<td>0–12</td>
<td>249 oocysts / g (ave.)</td>
<td></td>
</tr>
<tr>
<td>Hoar et al. (2001)</td>
<td>1.1</td>
<td>–</td>
<td>Canadian adult beef cattle</td>
</tr>
<tr>
<td>Hutchison et al. (2004/5)</td>
<td>5.4</td>
<td>19 cfu/g (g.m.)</td>
<td>C. parvum in fresh composite farm manure, UK. max. = $3.5 \times 10^3$ cfu/g</td>
</tr>
<tr>
<td>McAllister et al. (2005)</td>
<td>18.4</td>
<td>–</td>
<td>Cows (Ontario, Canada)</td>
</tr>
<tr>
<td></td>
<td>13</td>
<td>–</td>
<td>Calves (British Columbia, Canada), max. = 132,000 oocysts/g</td>
</tr>
<tr>
<td>McAllister et al. (2005)</td>
<td>1.1</td>
<td>12,323 / g (max.)</td>
<td>California</td>
</tr>
<tr>
<td>Nydam et al. (2001)</td>
<td>–</td>
<td>–</td>
<td>An infected calf could produce $3.89 \times 10^{10}$ oocysts in 6 days</td>
</tr>
<tr>
<td>Santín et al. (2008)</td>
<td>8.7, 36</td>
<td>–</td>
<td>Cows, calves</td>
</tr>
<tr>
<td>Starkey et al. (2005)</td>
<td>–</td>
<td>$1.3 \times 10^5$ / g</td>
<td></td>
</tr>
<tr>
<td>Swine</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ferguson et al. (2009)</td>
<td>–</td>
<td>–</td>
<td>Australia. Prevalence highly variable; average shedding rates from prior studies = 14.3 oocysts/g for adults and 472 oocysts/g for juveniles (6–8 weeks old)</td>
</tr>
<tr>
<td>Heitman et al. (2002)</td>
<td>0</td>
<td>–</td>
<td>Canada</td>
</tr>
</tbody>
</table>

(Continued)
<table>
<thead>
<tr>
<th>Reference</th>
<th>Prevalence (%)</th>
<th>Concentration</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hutchison et al. (2004/5)</td>
<td>13.5</td>
<td>58 cfu/g (g.m.)</td>
<td>C. parvum in fresh composite farm manure, UK. max. = $3.6 \times 10^3$ cfu/g</td>
</tr>
<tr>
<td>Hutchison et al. (2004/5)</td>
<td>29.2</td>
<td>10 cfu/g (g.m.)</td>
<td>C. parvum in fresh composite farm manure, UK. max. = $2.5 \times 10^2$ cfu/g</td>
</tr>
<tr>
<td>Ryan et al. (2005)</td>
<td>2.6</td>
<td>–</td>
<td>Australia</td>
</tr>
<tr>
<td>Ferguson et al. (2009)</td>
<td>–</td>
<td>–</td>
<td>Two studies: (i) prevalence in US 6% (broilers) and 27% (layer chickens); (ii) prevalence = 27% (16 samples) in The Netherlands. Average shedding rate = 2100 oocysts/g faeces for Netherlands layers.</td>
</tr>
<tr>
<td>Hutchison et al. (2004/5)</td>
<td>0</td>
<td>–</td>
<td>C. parvum in fresh composite farm manure, UK.</td>
</tr>
</tbody>
</table>

“g.m.” = geometric mean, “ave.” = arithmetic mean, “max.” = maximum
(full descriptions of these studies are of course given in the cited references in these tables). Note that these studies typically analyse composite samples, rather than faecal material from individual animals. Therefore the variation between individual animals is smoothed somewhat. This is appropriate for risk studies, because faecal contamination of environmental water typically arises from groups of animals, not from an individual animal.

**PATHOGEN SURVIVAL ($T_{90}$) AND EFFECT OF SUNLIGHT**

**Campylobacter, E. coli and Salmonella**

Sinton et al. (2007a) reported inactivation rates of Campy. jejuni, E. coli, and Salm. enterica (inter alia) in bovine faeces on pasture. In the first one to three weeks, there were increases (up to 1.5 orders of magnitude) in the counts of E. coli (three seasons) and Salm. enterica (two seasons), but there was none for Campy. jejuni. Thereafter, the counts decreased, giving an average ranking of the times necessary for 90% inactivation of Campy. jejuni (6.2 days from deposition) $<$ S. enterica (38 days) $<$ E. coli (48 days). Sinton et al. (2007b) studied the inactivation of the same microorganisms in river water and seawater. All sunlight inactivation rates, as a function of were far higher than the corresponding dark rates. All the $T_{90}$ values were higher in winter than in summer. Seasonal values for Salm. enterica and E. coli were similar and both were considerably larger than those for Campy. jejuni. The rapid inactivation of Campy. jejuni was attributed to a high susceptibility to photo-oxidative damage.

**Giardia and Cryptosporidium**

Olson et al. (1999) reported that Giardia cysts were noninfective in water, faeces, and soil following one week of freezing to $-4^\circ$C and within two weeks at $25^\circ$C. At $4^\circ$C Giardia cysts were infective for 11 weeks in water, seven weeks in soil, and one week in cattle faeces. Cryptosporidium oocysts were more environmentally resistant. At $-4^\circ$C and $4^\circ$C, the oocysts could survive in water and soil for $>$12 weeks but degradation was accelerated at $25^\circ$C. Cryptosporidium oocysts also were degraded more rapidly in faeces and in soil containing natural microorganisms. Davies et al. (2005b) reported $T_{90}$ for closed non-irradiated soil microcosms ranging from 13–24 days at $35^\circ$C to 45–66 days at $20^\circ$C depending on soil type. In laboratory studies of Crypto. parvum, Ives et al. (2007) reported first-order log inactivation coefficients ranging from $k = 0.0088$ per day at $5^\circ$C to 0.20 per day at $30^\circ$C. These correspond to $T_{90}$ values of approximately 114 and 5 days respectively [using the first-order
relationship $T_{90} = -\log_{10}(0.1)/k = 1/k$. Similar studies for *Giardia* have not been cited, but it seems plausible to assume that a similar situation applies.

REFERENCES


