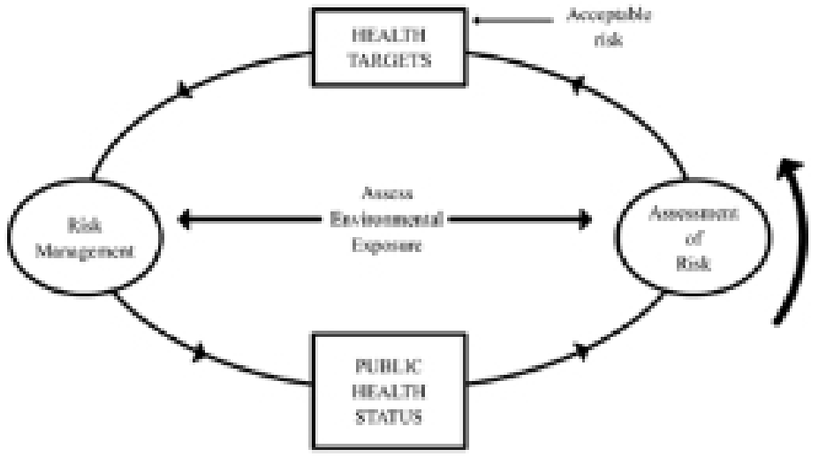

8

Risk assessment

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This chapter introduces the technique of microbial risk assessment and outlines its development from a simple approach based upon a chemical risk model to an epidemiologically-based model that accounts for, among other things, secondary transmission and protective immunity. Two case studies are presented to highlight the different approaches.

8.1 BACKGROUND

Quantifiable risk assessment was initially developed, largely, to assess human health risks associated with exposure to chemicals (NAS 1983) and, in its simplest form, consists of four steps, namely:

- hazard assessment
- exposure assessment
- dose–response analysis
- risk characterisation.

The output from these steps feeds into a risk management process. As will be seen in later sections this basic model (often referred to as the chemical risk paradigm) has been extended to account for the dynamic and epidemiologic characteristics of infectious disease processes. The following sub-sections elaborate on the chemical risk paradigm as outlined above.

8.2 CHEMICAL RISK PARADIGM

8.2.1 Hazard assessment

For micro-organisms, hazard assessment (i.e. the identification of a pathogen as an agent of potential significance) is generally a straightforward task. The major tasks of Quantitative Microbiological Risk Assessment (QMRA) are, therefore, focused on exposure assessment, dose–response analysis and risk characterisation. The task of risk management is one of deciding the necessity of any action based upon the risk characterisation outputs, and incorporates significant policy and trans-scientific concerns.

One outcome of the hazard analysis is a decision as to the principal consequence(s) to be quantified in the formal risk assessment. With micro-organisms, consequences may include infection (without apparent illness), morbidity or mortality; furthermore, these events may occur in the general population, or at higher frequency in susceptible sub-populations. Although mortality from infectious agents, even in the general population, cannot be regarded as negligible (Haas *et al.* 1993), the general tendency (in water microbiology) has been to regard infection in the general population as the particular hazard for which protection is required. This has been justified based on a balance between the degree of conservatism inherent in using infection as an endpoint and the (current) inability to quantify the risks to more susceptible sub-populations (Macler and Regli 1993).

8.2.2 Exposure assessment

The purpose of an exposure assessment is to determine the microbial doses typically consumed by the direct user of a water (or food). In the case of water microbiology, this may necessitate the estimation of raw water micro-organism levels followed by estimation of the likely changes in microbial concentrations with treatment, storage and distribution to the end-user (Regli *et al.* 1991; Rose *et al.* 1991). A second issue arising in exposure assessment is the amount of ingested material per 'exposure'. As a default number, two litres/person-day is used to estimate drinking water exposure (Macler and Regli 1993), although this may be conservative (Roseberry and Burmaster 1992). For contact recreational

exposure, 100 ml/day has often been assumed as an exposure measure (Haas 1983a), but actual data to validate this number are lacking.

8.2.3 Dose–response analysis

It is generally necessary to fit a parametric dose–response relationship to experimental data since the desired risk (and dose) which will serve to protect public health is often far lower than can be directly measured in experimental subjects (at practical numbers of subjects). Hence it is necessary to extrapolate a fitted dose–response curve into the low-dose region.

In QMRA, for many micro-organisms, human dose–response studies are available which can be used to estimate the effects of low level exposure to micro-organisms. In prior work, it has been found that these studies may be adequately described by one of two semi-mechanistic models of the infection process. In the exponential model, which may be derived from the assumption of random occurrence of micro-organisms along with a constant probability of initiation of infection by a single organism (r), the probability of infection (P_I) is given as a function of the ingested dose (d) by:

$$Pr = 1 - \exp(-rd) \tag{8.1}$$

For many micro-organisms, the dose–response relationship is shallower than reflected by Equation 8.1, suggesting some degree of heterogeneity in the micro-organism-host interaction. This can be successfully described by the beta-Poisson model, which can be developed from Equation 8.1 if the infection probability is itself distributed according to a beta distribution (Furumoto and Mickey 1967a,b; Haas 1983b). This model is described by two parameters, a median infectious dose (N_{50}) and a slope parameter (α):

$$P_I = 1 - \left[1 + \frac{d}{N_{50}} \left(2^{1/\alpha} - 1\right) \right]^{-\alpha} \tag{8.2}$$

Figure 8.1 depicts the effect of the slope parameter on the dose–response relationship; in the limit of $\alpha \rightarrow \infty$, Equation 8.2 approaches Equation 8.1.