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# A quantitative risk assessment of microbial emissions from abattoirs

## PRMS.036

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# Final Report - PRMS.036
# A quantitative risk assessment of microbial emissions from abattoirs

Prepared for Meat & Livestock Australia

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# ABSTRACT

A quantitative microbiological risk assessment model was produced to estimate the risk of human illness from exposure to microorganisms released to the environment from abattoirs. The risk assessment model tracks pathogen transport through the air, water and solid waste streams associated with the critical stages in a primary meat processing operation. Six pathogens of greatest interest to the Australian meat processing industry and with potential to pose a risk to human health were identified and their routes of transmission to the environment established. The selected pathogens were *Campylobacter jejuni, Coxiella burnetii, Escherichia coli* (certain serotypes), *Salmonella* spp., *Cryptosporidium parvum* and *Listeria monocytogenes*. Quantitative risk assessments using Monte Carlo simulation were carried out for each pathogen to estimate the human health risks from five exposure pathways relating to airborne and waterborne exposure. Although there is considerable uncertainty associated with all of the risk estimates, the pathogens can be ranked with reasonable confidence. Site-specific characteristics of individual abattoirs will determine the actual magnitude of risk. For airborne exposure pathways, *Coxiella burnetii* appears to be of greatest concern. It is unclear whether the other pathogens pose any risk at all when inhaled. For waterborne pathways, *Cryptosporidium parvum* is ranked highest followed by *Campylobacter jejuni*, with the remaining pathogens several orders of magnitude lower. The quantitative risk assessment model provides an objective tool for the meat and livestock industry to assess the public health risks from abattoir emissions and direct future research activities.

# EXECUTIVE SUMMARY

Concerns about human illness associated with meat processing have focussed on the state of the final product and the maintenance of hygiene standards during meat processing and transport. However, pathogenic microorganisms may also be released to the environment through meat processing wastes in solid, liquid and gaseous forms. The objectives of this project were to:

- Identify pathogens of interest to the red meat industry and establish the routes by which they might be transmitted to the environment
- Quantitatively estimate the risk to human health for each selected pathogen
- Produce an overall ranking of risk for the selected pathogens.

Fifty-two pathogens were reviewed and six were selected for further study based on their relevance to the Australian meat processing industry and their potential to pose a risk to human health. They were *Campylobacter jejuni, Coxiella burnetii, Escherichia coli* (certain serotypes), *Salmonella* species, *Cryptosporidium parvum* and *Listeria monocytogenes.*

A quantitative microbiological risk assessment model was developed to track pathogen transport through the air, water and solid waste streams associated with the critical stages in a primary meat processing operation. The model considers a generic abattoir receiving cattle and sheep, with input sources of pathogens from the livestock yard, carcase processing, hide processing and offal handling. The total number of pathogens is calculated for each of the air, water and solid waste streams. These outputs are the inputs into the second part of the risk model which quantifies the risk of people becoming ill through five exposure pathways (three airborne, two waterborne).

The Monte Carlo simulation approach that was used allows uncertainty in the model inputs to be handled explicitly. The estimated risk is provided as a distribution thereby providing a more realistic impression of uncertainty than can be conveyed by just a mean or median value. Input data for the model was obtained from published literature, from unpublished information provided by MLA and from personal communications.

Although there is considerable uncertainty associated with all of the risk estimates, the pathogens can be ranked with reasonable confidence. For airborne exposure pathways, *Coxiella burnetii* appears to be of greatest concern. It is unclear whether the other pathogens pose any risk at all when inhaled. For waterborne pathways, *Cryptosporidium parvum* is ranked highest followed by *Campylobacter* jejuni, with the remaining pathogens several orders of magnitude lower.

Within the airborne pathways, contaminated irrigation water consistently poses the highest risk followed by air from the livestock yard and air released from the abattoir. For waterborne exposure, water ingested during recreation (swimming, boating etc) poses a higher risk than drinking water. This is not surprising since the model assumes that drinking water undergoes treatment. In all cases, actual risk of illness will depend on the number of people actually exposed. This quantity is not included in the assessment as it depends on abattoir specific details such as where the abattoir is located relative to centres of population and water supplies and how much time people spend in the vicinity.

The results of the assessment led to the following recommendations:

1. For the five pathogens other than *Coxiella burnetii*, seek expert opinion on:
    - whether the pathogen can be transmitted and survive in an airborne state
    - whether the pathogen can cause human illness via inhalation exposure

    If the answer is ‘no’ to either of these questions, then these pathogens can be dropped from further consideration with respect to the airborne pathway.

2. For the remaining pathogens of interest:
    - verify assumed concentration and prevalence data by sampling Australian livestock entering abattoirs
    - verify model outputs by monitoring the actual pathogen concentrations in abattoir emissions (air, soil, water)

   If the pathogens are not present in animals entering an abattoir, or not emitted from the abattoir, then there is no risk of human illness. If concentrations are lower or higher than those assumed in the model, the risk of human illness will be decreased or increased accordingly.

3. Investigate site-specific features (e.g. air dispersion, proximity of water bodies, waste treatment protocols, potential number of people exposed) of individual abattoirs and the degree to which they could influence absolute risk estimates.

4. Attempt to calibrate the model with actual statistics on human illness.

5. In prioritising further research, consider the seriousness of illness to the human population caused by exposure to each pathogen.

As far as we are aware, this is the first attempt to obtain quantitative estimates of the risk to human health of pathogens released to the environment from abattoirs. The advantage of a risk assessment based on a transparent, clearly documented model is that the model can be modified to explore the implications of new information or different assumptions. The model can continue to be used in the future as a tool for dissemination of information, responding to industry and regulatory enquiries, and deciding on the scope and direction of future risk assessment and risk management.

# INTRODUCTION

## Risks associated with animal production

One of the health risks posed by animal production facilities is the release of pathogens, or disease-causing agents, into waste streams and subsequently into the environment. Diseases and infections that are naturally transmitted between vertebrate animals and humans are known as zoonoses (WHO 1982). Pathogens commonly associated with zoonoses include various bacteria, viruses, and protozoan parasites. In the meat industry, employees working with live or slaughtered animals are at risk of contracting several zoonotic diseases. The main risk areas include abattoirs and slaughterhouses, knackeries and animal-by-products establishments (AMIEU and MATFA 1995).

This project was commissioned by Meat and Livestock Australia to explore the human health risks posed by zoonotic organisms released to the surrounding environment during abattoir processing. In particular, the project aims to establish the routes by which pathogens of relevance to the red meat industry may be transmitted to the environment and to estimate quantitatively the likelihood of risk in each case. In doing so, this work will rank selected human health risks from meat industry waste to allow the industry to set priorities for investment, research and necessary remedial activities in order to reduce potential public health risks to acceptable levels.

Zoonotic diseases may be transferred to humans by several routes, including direct animal contact, through vectors like mosquitos or ticks, through common vehicles like food, water and manure, and over long distances through aerosols (Weber and Rutala 1999). Animal production and meat processing give rise to several opportunities for spreading diseases from animals to humans. In this report, we do not differentiate between true zoonoses, where animals play an essential role in maintaining infection in nature, and other communicable diseases, where animals may contribute in varying degrees to the distribution and actual transmission of infections. The practical consequences are much the same from a meat production point of view.

Meat processing plants generate sizeable quantities of solid, liquid and gaseous wastes, some with high loads of microorganisms. Pathogenic microorganisms present in meat processing wastes, in any of their solid, liquid and gaseous forms, may present a potential human health hazard.

Environmental and health agencies both in Australia and overseas have raised concerns about reports of alleged and actual instances of human illness caused by exposure to meat industry or farm wastes. Examples of human disease concerns related to animal environmental contamination include:

- groundwater contamination with enterohaemorrhagic *Escherichia coli* in Walkerton, Canada,
- surface water contaminated with *Cryptosporidium* in Sydney, Australia and Milwaukee, USA,
- Salmonellae in abattoir waste water streams in Denmark and Scotland (Sogaard & Nielsen 1979),
- antibiotic resistant *Campylobacter* in poultry abattoir effluent in the Netherlands,
- *Listeria* spp. found in soil and fodder of animal paddocks adjacent to abattoirs in New Zealand (Pociecha *et al.* 1991); and
- the risk of the BSE agent being spread through environmental pollution from cattle abattoirs in Europe.

Meat processors, environmental and health regulatory authorities, as well as concerned community groups, are conscious of the unquantified threat posed by pathogens in meat-processing wastes. Since most plants are in close proximity to populated areas, there is a possibility that communities may be exposed to pathogens.

## Selected zoonotic diseases and pathogens

More than 200 infectious diseases are capable of being transmitted from animal to humans (Weber and Rutala 1999). New zoonotic pathogens continue to be recognised either because the microbial agent is newly isolated or because its potential to cause human disease is newly recognised. Understanding the epidemiology of animal-associated diseases requires a detailed understanding of the reservoir and source of the zoonotic infectious agents. The reservoir is defined as the niche that the organism normally inhabits. The source is the means by which the organism reaches the human. For most zoonotic infections, the normal life cycle does not involve humans. Rather, humans are accidental hosts and represent dead-end vectors. Some pathogens share maintenance of their life cycle with both animals and humans.

Factors affecting transmission of zoonotic diseases to humans include the geographical range of the animal host that serve as reservoir, the number and type of animal hosts, frequency of activities that bring people into contact with the infected source, the prevalence of infection in host animals, the means by which the infection can be transmitted from an infected animal to humans, and susceptibility of humans to infection. For a proper risk assessment to be performed, quantitative data on the aspects listed must be known. Unfortunately, this is not always the case. For vector-borne zoonotic diseases, the epidemiology of the disease is also critically dependent on the biology of the vector.

Fifty-two pathogens were reviewed (Appendix A) and six were selected for further study based on their relevance to the Australian meat processing industry and their potential to pose a risk to human health:

*Campylobacter jejuni*

*Coxiella burnetii*

*Escherichia coli* (certain serotypes)

*Salmonella* spp.

*Cryptosporidium parvum*

*Listeria monocytogenes*

The main body of the report provides a discussion of the pathways for pathogen transfer to the environment, a brief overview of the risk assessment process and the quantitative risk assessment model designed for this study, a comparison of the results across pathogens, and recommendations for further action. Technical details relating to the model are located in Appendices A-C, while individual risk assessments for the six pathogens are documented in Appendices D-I.

# ENVIRONMENTAL PATHWAYS

Untreated animal waste contains high concentrations of organic material, nutrients, and microorganisms that have the potential to negatively impact on the environment and health of communities surrounding animal production facilities.

Pathogens can be transferred to humans by direct or indirect transmission. Direct transmission involves direct contact between the source and host of the pathogen, while indirect transmission refers to processes that mediate contact between the source of infection and a susceptible host (Edmonds 1978). This section of the report is primarily concerned with examining the environmental pathways through which pathogens may be indirectly transferred from waste streams generated in the abattoir environment to the wider community, who are physically removed from the direct source of the infection.

The routes of transmission examined are through infective aerosols, liquids and solid materials. The risks of infection through vectors such as mites or ticks are not considered here. Fluid and solid routes generally refer to pathways for waterborne and soil borne pathogens, respectively. Importantly, the three main environmental pathways are closely linked and not independent of each other. For example, effluent containing pathogenic microorganisms, such as wastewater, may give rise to infective aerosols during spray irrigation. Solid wastes applied to land may lead to the transport of pathogens through soil and ultimately into surface and groundwater supplies. The survival and transmission of pathogens through multiple environmental pathways depends on numerous factors including temperature, humidity, wind speed, climate and the nature of the pathogen itself. Given the complexity and uncertainty surrounding the fate of pathogens when removed from their source, this discussion is limited to the most direct routes for human infection and not through feedback processes. For example, the potential risk to human health from consumption of contaminated drinking water will be examined, however, the less direct risk to humans through livestock grazed on pasture irrigated with contaminated water will not be addressed.

The risk of environmental spread of pathogens from an abattoir depends largely on factors such as number of livestock entering the abattoir environs, duration of holding prior to slaughter, the nature of waste management systems on site and the volume of waste generated. These factors influence the level of potentially pathogenic microorganisms and hence the risk of public exposure via the environmental pathways.

The potential risk to humans will also depend on the health status of the exposed population, as the elderly and immuno-compromised face a higher risk of infection. The extent to which animal waste constitutes a hazard for human health may vary from disease to disease. Similarly, the extent of risk involved in the handling, storage, land spreading and wider utilisation of animal manure will vary according to the agent involved, the numbers present, and the manner in which the population at risk may become exposed (Strauch and Ballarini 1994). Although animal wastes may cause health-related problems due to the release of emissions such as toxic gases or odours, the following discussion will be focussed on abattoir wastes from the perspective of causing infection by microorganisms.

## Airborne pathogens

### *Types of aerosols*

Pathogenic microorganisms such as bacteria and viruses can be introduced into the air from effluent generated in an abattoir. Once airborne, the infectious agents can be transported for considerable distances as aerosols (moist suspensions of airborne materials) or droplet nuclei (microbial-laden material that dries and remains airborne) (Edmonds 1978). Factors that are known to influence the rate of survival of airborne bacteria and viruses are temperature, humidity, ultraviolet solar radiation, availability of food sources, oxygenation, air pollution, distance of air transport and pressure.

Infective aerosols may be classified as either primary or secondary in type. Primary aerosols are those generated directly by, or in direct association with, an infected animal. For example, an infective aerosol may arise during parturition of an animal infected when the placenta and birth fluids are exposed. In contrast, secondary aerosols are produced indirectly by some factor, or incident that is temporally or spatially removed from those giving rise to primary aerosols. For instance, secondary aerosols may be produced during handling or processing of contaminated materials, such as wool and hides, at some

distance in time and/or space from the actual animal source responsible for the contamination (Welsh *et al.* 1958).

Secondary aerosols are considered a major factor in the dissemination of disease to new areas and populations and are considered to play a greater role in human illness than primary aerosols (Welsh *et al.* 1958). Although exposure to primary aerosols is a potential occupational health and safety issue for workers within an abattoir, secondary aerosols are possibly of greater concern to the environment external to the abattoir. There are numerous ways in which infective aerosols can be generated in an abattoir environment, enter the airborne pathway and potentially spread to the wider community. Examples of these are indicated below.

Microbes do not have to be infectious to cause a health hazard (Donham 2000). Many microbes contain toxins (e.g. endotoxins, glucans) that are potent inflammatory substances. These may not cause a health problem from drinking contaminated water, but aerosolised animal wastes could produce aerosol exposure to these substances, which could result in asthma-like symptoms, bronchitis, mucus membrane irritation, and organic dust toxic syndrome (a systemic influenza-like illness).

### Airborne pathogens from animal houses

Animal houses or holding pens are a rich source of airborne particles that vary in size and chemical composition. Infectious dust particles may arise by agitation of contaminated surfaces such as the ground and soil of areas housing infected animals—the large airborne particles frequently carry bacteria and viruses. Bacteria levels in the air are influenced by the density of stocking, animal age, ventilation system, and the microclimate of the animal house (Strauch and Ballarini 1994). Infective aerosols may be ducted from the identified risk areas via air conditioning and ventilation systems to expose workers in other buildings (away from the source), those working in proximity to the outlet, and even those walking through the plant grounds (MRC 1997).

Contaminated water droplets or fine mists dispersed when using high pressure hoses to wash infected material (including stock, building structures, animal transport vehicles and clothing) may also generate an infectious aerosol that can freely disperse in the air and eventually settle to form an infectious dust, away from the initial site of contamination (MRC 1997).

With reference to a study on swine production, Donham (2000) commented that although there is discharge of airborne particulates and vapours from swine barns to the exterior environment, the aerosols downwind differ considerably in composition and concentration of specific agents. Some substances may be deposited on the ground with rain, and the surrounding buildings, trees and crops may also influence the dispersion of effluents. Donham (2000) concluded that extrapolating occupational health risks from inside swine facilities to community health risks outside swine production is of limited use. This suggests there is a degree of uncertainty in quantifying the risks of pathogen spread via the airborne route. However, there is strong evidence that points towards an airborne route of transport for some pathogens from animal production facilities to the general community. Contaminated aerosols, whether infected dust or droplets, are considered extremely infectious (MRC 1997), and maintain the potential to infect neighbouring communities located downwind of the source.

A study of modelled survival distances of airborne bacteria from battery hen found that the maximum concentration of bacteria was reached at 25-50 m from the source. The highest bacterial count was associated with the lowest air speed (Muller and Wiesner 1987). Other studies suggest that bacteria can be found at distances of up to 100 m; a survival distance of 200 m is only reached under specific conditions such as at greater pressure from the source, a modest but uniform pointed air stream and at high humidity (Hilliger 1991).

### Airborne pathogens from solid waste application

Since many pathogenic microorganisms may be excreted in the faeces of infected animals, the application of animal waste to land is another potential source of infective aerosols. In terms of microbial airborne contamination, little is known about the hazards to humans arising from sludge (the undigested solid residue from sewage-treatment processes) application (Boutin *et al.* 1988). However there are public concerns regarding the potential exposure to airborne pathogens within population centres surrounding the application sites (Pillai *et al.* 1996).

The spray generated as a result of sewage treatment methods is known to inject large numbers of potentially pathogenic bacteria into the atmosphere. However, Akers *et al.* (1978) noted that the viable cells downwind are reduced dramatically as a function of wind velocity, distance from source, time in the air, relative humidity, and species. Similarly, an investigation on microbial and endotoxin levels in the vicinity of a composting plant suggested that people living more than 150 m away from composting facilities do not bear a significant risk due to exposure to endotoxin emitted from the composting plant. In addition, the study found that microbial concentrations calculated at a distance of about 500 m from the plant are comparable to the concentration for ambient air (Danneberg *et al.* 1997).

Boutin *et al.* (1988) showed that the intensity and extent of airborne contamination from land spreading of faecally contaminated agricultural waste (from cattle and pigs) depends mainly on the initial bacterial load of the slurry and on the spreading conditions (projection height and droplet size). From a study on municipal waste, Pillai *et al.* (1996) concluded that land application of sludges poses little risk of airborne transmission of bacterial pathogens, although physical agitation of sludge material could generate a large number of diverse bacterial populations in the immediate vicinity. Similarly, Cliver (1980) suggested that sewage aerosols are probably a relatively inefficient means of transmitting enteric infections. Contrary to this, Nicholson *et al.* (2000) noted that spray drift from aerosols created during the spreading of liquid manures has been widely documented as a route for the dissemination and direct infection of humans by pathogens. In addition, according to Strauch and Ballarini (1994), the spraying of slurry on pasture is a potential hazard for aerosol spread of small parasites such as *Cryptosporidium parvum*.

### Airborne pathogens from wastewater applications

Recycling of wastewater generated in an abattoir is another possible route by which pathogenic microorganisms may enter the airborne environment and expose humans to infection. Cole *et al.* (1999) note that aerosols created during spray irrigation of animal waste may contain human pathogens, though sufficient research has not been performed to fully characterise the level of risk associated with animal waste aerosols. It has been suggested that direct contact with municipal wastewater and its sprayed mist, and downwind exposure to the aerosol, are the most probable environmental pathways for exposure to viruses (Sorber *et al.* 1974) and pathogenic bacteria (Guntzel 1978). Cole *et al.* (1999) remark that exposure to aerosolised municipal wastewater may be associated with an excess risk of infection, so the exposure to aerosolised animal waste may pose similar potential risks. Therefore, depending on the type of water treatment processes and the mechanisms for recycling water at an individual abattoir, spray drift from an irrigation site could be a source of infectious microbial aerosols.

A study by Camann *et al.* (1988) on municipal wastewater found a pattern of reduced microorganism density in the air with increasing downwind distance from the irrigation site. Spray irrigation of poor-quality, direct pipeline effluent, was found to significantly elevate the air densities of enteric bacteria and viruses for at least 200 m downwind relative to ambient background levels near homes and in fields. However, storage of wastewater in reservoirs prior to spraying resulted in a large reduction in downwind air densities of indicator organisms, equivalent to a buffer zone of about 300 m for the hardier indicator organisms. Enteric indicator organisms have also been isolated from aerosols during spray irrigation of animal waste at cattle and swine farms, at distances of up to 130 m (Cole *et al.* 1999).

Exposures to potentially harmful animal waste constituents during spray irrigation can be minimised by avoiding irrigation sites during spraying, not spraying during windy days, improving wastewater treatment prior to land application or using alternative land application techniques such as low pressure spray irrigation and liquid injection into the soil (Cole *et al.* 1999).

### Examples of airborne pathogens

Outside of the abattoir environment, outbreaks of Q fever have occurred at distances of kilometres away from the suspected abattoir or farmland source, associated with windborne spread of the rickettsia *Coxiella burnetii*. In addition to aerosol spread of bacteria, viruses have also been reported to travel over long distances. For example during several outbreaks in the United Kingdom it was shown that transmission of the Foot-and-Mouth-Disease (FMD)-virus from farm to farm corresponded with the wind direction. A transport distance of 120-150 km is considered possible for the FMD-virus (Strauch and Ballarini 1994).

**Box 1: Windborne spread of Q-fever**

A large outbreak of Q fever was reported in Birmingham in the UK in 1989. This outbreak was considered unusual in that it predominantly affected residents of a large urban centre. The probable cause of the outbreak was the windborne spread of *C. burnetii* spores by unusually high winds of 130 km/h, from either farmland or an abattoir (with outdoor facilities for holding stock) situated 6-8 km away from the affected area (Hawker *et al.* 1998).

Windborne spread from a rendering plant believed to be dealing with infected carcasses was held responsible for a large Q fever outbreak in California during the 1950's, which infected individuals as far away as 16 km from the source of origin (Wellock 1960).

# Soil and waterborne pathogens

## *Source & transport of effluent pathogens*

The previous section discussed possible routes for human exposure to airborne pathogens derived from abattoir waste. In addition to the airborne route, potentially pathogenic microorganisms in solid and liquid animal wastes may negatively impact on the health of communities via contaminated soil and water.

Pathogenic microorganisms are found in all sewage effluents. Where the same watercourses are used for domestic, recreational, agricultural, industrial and sewage disposal purposes, the population is at risk unless the water supplies are appropriately treated and monitored. According to Bitton and Gerba (1984), land disposal of sewage effluents is an important contributor to groundwater pollution by biological agents. Slurry disposal may also have implications for human health where crops are sprayed with liquid slurry or are grown on land fertilised with solid non-composted waste (Strauch and Ballarini 1994). Human health and environmental impacts associated with animal waste can be minimised through appropriate waste management techniques, treatment systems and land application practices (Cole *et al.* 1999). Although waste management procedures will differ across abattoirs, there are similar potential routes for pathogenic transfer between the abattoir environment and humans through the solid and fluid pathways. Soil and water contamination are inter-related and will thus be addressed together.

Animal waste generated in an abattoir is generally comprised of faeces, urine and wastewater (slurry) as well as blood, offal and carcass material. During the slaughter of animals, large quantities of water are used for processing, cleaning, disinfection and transport of slaughter by-products. This wastewater is polluted with organic matter (protein and fat) of animal origin (Fransen *et al.* 1996). Untreated (solid) animal waste also contains high concentrations of organics, nutrients, and microorganisms—animal manure is considered a potent source of pathogenic organisms (Keswick 1984). Drain effluent arising from cold rooms in an abattoir has been found to be a source of the pathogen *Listeria monocytogenes* (Pociecha *et al.* 1991). Depending on the systems operating at an individual abattoir, manure effluent and processing effluent may be separated into different waste streams. Solid wastes are generally removed from the liquid effluent streams to generate solids of dominantly faecal origin and solids that are associated with paunch content (MLA 1991). Most studies on the human and environmental impacts of animal waste disposal relate to animal faecal matter and not to the other constituents of effluent such as offal. From the perspective of potential pathogenic exposure, the pathogens in animal manure are considered a satisfactory analogue for abattoir effluent on the whole.

Bacteria are the most common pathogens in faecal matter and sewage (Keswick, 1984). The human pathogenic bacteria which have been isolated from cattle manure are *Salmonella*, *Listeria*, *Escherichia coli* and *Campylobacter*. The protozoan parasites *Cryptosporidium* and *Giardia* are also found in cattle manure (Nicholson *et al.* 2000). Salmonellosis is regarded as the most important disease spread by slurry (Kearney *et al.* 1993). Soil is an important reservoir of the bacterium *Listeria monocytogenes* and can be expected to occur in animal paddocks adjacent to abattoirs (Pociecha *et al.* 1991). The levels of pathogens in cattle excreta depend on animal age, diet and management, as well as regional and seasonal factors. Replication of viral pathogens without their usual host range is rare, and thus viral pathogens in animal wastes are unlikely to pose a significant health risk to humans—the exception is a class of viruses termed the rotaviruses (Nicholson *et al.* 2000).

Waste disposal at abattoirs can take several forms including dumping or burying of solid wastes; land spreading of yard solids and paunch content; disposal to lagoons; or composting (either aerobic or anaerobic). Irrigation is widely practised by meat processing plants in several states of Australia and is considered a useful means of recycling both the nutrient and water components of effluent (MLA 1991).

Both irrigated effluent and disposal of solid animal wastes have the potential to contaminate groundwater resources as well as impact on surface waters through run off (MLA 1991; Keswick 1984). Although treatment of solid and liquid animal wastes can significantly reduce the concentrations of microorganisms, pollution of water supplies is a concern where animal waste is applied to land at rates above which soil and vegetation can absorb and utilise waste constituents (Cole *et al.* 1999). The health risks associated with the land application of slurry depend on the species of pathogen in the waste, population size, ability to survive storage and/or treatment, and the ability of the pathogen to remain virulent (Kearney *et al.* 1993).

Despite the wide variety of infective agents that can be present in slurry, there are few published records of disease transmission to animals or humans through slurry, treated or untreated. However, a risk does exist, but it can be reduced to proportions that are acceptable if care is taken to treat and use slurry according to recommended guidelines (Strauch and Ballarini 1994).

## Soil contamination

Pathogenic bacteria are able to survive and multiply in the environment provided that the proper nutrient conditions are present. Similarly, viruses are able to survive for long periods outside their hosts, although they do not replicate in the environment (Keswick 1984).

Pathogen survival in soils is mainly influenced by climatic conditions, soil properties, and by the nature of the microorganism (Table 1). Survival of bacteria and viruses is generally highest under low temperature and high soil moisture content; the abundance of organic matter may also allow growth of certain bacteria (Bitton and Harvey 1992). It is suggested that 2-3 months is a sufficient time for the reduction of pathogens to negligible numbers once they have been applied to the soil, although survival times of 5 years have been reported for some pathogens (Gerba and Bitton 1984).

**Table 1.** Factors affecting survival of enteric bacteria in soil (after Gerba and Bitton 1984).

| Factor | Comments |
|---|---|
| Moisture content | Greater survival time in moist soils & during times of high rainfall |
| Moisture holding capacity | Survival time is less in sandy soils with lower water-holding capacity |
| Temperature (T) | Longer survival time at low T; longer survival in winter than in summer |
| pH | Shorter survival time in acid soils (pH 3-5) than in alkaline soils |
| Sunlight | Shorter survival time at soil surface |
| Organic matter | Increased survival & possible regrowth when sufficient organic matter present |
| Antagonism from soil microflora | Increased survival time in sterile soil |

In addition to pathogen survival, the degree of movement of pathogens in soils, both vertically and horizontally, will affect the risk of pathogens reaching aquifers or surface waters. The processes influencing the transport of microorganisms through the subsurface are complicated by geohydrological, chemical and biological factors (Matthess *et al.* 1988). Factors that control the retention of microbial pathogens in soils are cations, pH, soluble organic materials, flow rate, soil type and the nature of water flow through the soil. Bacterial pathogens are retained in soils mainly through straining or filtration, whereas the smaller enteric viruses are retained mainly by adsorption. Parasites are large enough to be removed by filtration through the upper soil profiles (Keswick 1984). According to Nicholson *et al.* (2000), pathogen survival is generally favoured in aqueous environments and thus water availability and movement are the single most important factors in determining how far pathogens are likely to move through or across soils.

Studies on sludge-associated bacteria and viruses indicate that the microorganisms do not migrate significantly through sludge-amended soils. It is suggested that bacteria and viruses derived from on-land sludge disposal are mostly retained at the soil surface and are unlikely to cause groundwater contamination (Bitton and Harvey 1992). According to Keswick (1984), contamination of groundwater by microorganisms is unlikely unless the sludge comes into direct contact with groundwater.

### Groundwater contamination

The hidden nature of groundwater renders it vulnerable to both point and non-point source pollution. Pathogens found in abattoir effluent have the potential to penetrate the groundwater system directly or via soil to water transmission. Surface waters are also at risk of pathogenic contamination. Studies indicate that bacteria and viruses persist longer in groundwater than in surface waters—virus survival in groundwater is influenced by water temperature, dissolved oxygen, and other microorganisms in the groundwater (Bitton and Harvey 1992).

Despite the soil barrier for microbial retention, some pathogens may reach groundwater and survive for long periods in the subsurface environment (Bitton and Gerba 1984). Studies have shown that rainfall mobilises previously retained bacteria and viruses in the soil and promotes their transport to groundwater (Keswick 1984). According to Bitton and Harvey (1992), the greatest degree of drinking water well contamination has been found to occur after periods of heavy rainfall. In addition to affecting groundwater supplies, rainfall can also contribute to the pathogen loading in surface waters, including streams and rivers.

According to Dumontet *et al.* (2001), the risk of microbial contamination of ground and surface waters is enhanced by land application of inadequately treated sewage sludge. In addition to land spread waste material, spray-irrigated wastewater may similarly contain pathogens that can penetrate the groundwater system and potentially infect individuals who come in contact with the contaminated water. Viruses have been recovered from groundwater after spray irrigation of secondary sewage effluent on land and studies have also found that viruses can move over long distances laterally such that the source of pollution is located at some distance from the site of groundwater extraction (Keswick 1984).
The ability of bacteria to penetrate the groundwater system is clearly demonstrated by the contamination of Walkerton's (Ontario) drinking water supply with *E. coli* in May 2000.

**Box 2. Pathogens contaminate Walkerton drinking well**

In May 2000, Walkerton's drinking water system became contaminated with deadly bacteria. Seven people died and more than 2,300 became ill. The community was devastated. The losses were enormous. There were widespread feelings of frustration, anger, and insecurity. The tragedy triggered alarm about the safety of drinking water across the province.

The vast majority of the deaths and illnesses in Walkerton were caused by two bacteria, *Escherichia coli* O157:H7 and *Campylobacter jejuni*. The primary, if not the only, source of the contaminants was cattle manure that had been spread on a farm near a supply well during late April 2000. The owner of the farm followed best management practices in spreading the manure and was not considered to be at fault—the outbreak would have been prevented by the use of continuous chlorine residual and turbidity monitors at Well 5.

Extraordinary rainfall in early May 2000 greatly assisted the transport of the bacterial contaminants to the entry point for Well 5. The most likely pathway identified in the investigation was the channelling of contaminated surface water directly into a conduit in the fractured bedrock and into the well through a break in the shallow soil overburden (O'Connor 2000).

### Surface water contamination

The waterborne transmission of the intestinal protozoan parasites *Giardia duodenalis* and *Cryptosporidium parvum* has been associated with activities related to cattle farming such as waste spreading, slurry spraying and runoff from contaminated grazing land (Slifko *et al.* 2000). Runoff from agricultural lands is considered one of the most important routes of surface water contamination with *Cryptosporidium* (Dumontet *et al.* 2001). The cysts and oocysts of *Giardia* and *Cryptosporidium* are insensitive to the disinfectants commonly used in water treatment and thus pose a risk to humans via domestic water, as illustrated by the contamination of Sydney's drinking water supply in 1998 with these parasites. *Giardia* and *Cryptosporidium* are also considered the most commonly recognised cause of recreational waterborne disease, although contamination of recreational waters by animal wastes is not well documented (Slifko *et al.* 2000).

**Box 3. *Cryptosporidium* in Sydney's water supply**

Evidence of contamination in Sydney's water supply by the organisms *Cryptosporidium* and *Giardia* was first detected on 21 July 1998. The levels did not raise health concerns at the time. However, by 30 July, high readings were obtained from water sampled in the distribution system and a Sydney-wide boil water alert was declared. The water supply was declared safe on 4 August. This was but the first of three contamination events. Further contamination was identified on 24 August, leading to an extended boil water alert. The alert was progressively being lifted when further contamination was reported on 5 September. A two-week boil alert was instituted and not lifted until 19 September 1998.

The Sydney Water Inquiry noted that it is impossible to be definitive about the specific sources of the parasites that contaminated the raw water in July-September 1998. The Inquiry concluded that the catchment waters for much of Sydney's water supply contain multiple and significant sources of *Cryptosporidium* and *Giardia*, such as sewage treatment plants, unsewered and sewered urban areas, and cattle grazing. Tests indicate that the parasites found in the contaminated waters were derived from both human and animal waste—in the second and third events the bulk of the parasites detected were most likely derived from the faeces of animals in the catchment. There is agreement that a combination of drought and heavy rainfall in July and August during all events mobilised pathogens accumulated in the catchment and transported them into watercourses and dams (McClellan, 1998).

## Food contamination

Most animal manures are recycled to agricultural land, providing an important source of plant nutrients and organic matter. However, the disposal of these animal wastes can have implications for human health via the food chain. Pathogenic microorganisms in abattoir effluent have the potential to enter the human food chain through numerous possible routes. These pathways include; direct contamination of growing crops with waste material, contamination of crops with soil where waste was previously applied; ingestion of contaminated pasture/fodder crops and soil by grazing livestock following waste spreading; ingestion by livestock through contaminated drinking water; contamination of crops from irrigation water; or contamination of livestock and crops via airborne pathogens (Nicholson *et al.* 2000).

The most likely, and important, potential routes for pathogen transfer into the food chain are through the application of manures directly to growing crops, and through growing crops on land that has previously been grazed by livestock (where plant surfaces may subsequently become contaminated with soil pathogens). Crops irrigated with contaminated water may also pose a risk of pathogen transfer (Nicholson *et al.* (2000).

**Box 4. Lettuce vehicle for cattle pathogen spread**

In 1996, a mass outbreak of *E. coli* O157:H7 sickened 61 people in Illinois, Connecticut and New York. At least 21 people were hospitalized, including three children who were severely harmed. One was a 3-year-old Connecticut girl, who was almost blinded from eating pre-washed lettuce.

According to health authorities, the lettuce-producing company was rinsing lettuce in dirty, bacteria-laden water in a shed 100 feet away from a cattle pen, right in the path of dust-borne manure. The wash water came from wells near cattle pastures and no chlorine was added to the water to kill bacteria. In addition, the lettuce was grown in the same field where cows grazed and deposited their manure (Hilborn *et al.* 1999).

It has been found that some pathogens such as *E. coli* are able to survive for longer than 3 weeks on a variety of human food crops, including salad vegetables, while *Listeria* can survive on plant materials for a number of years. UV irradiation in bright sunshine enhances pathogen decline on plant surfaces and the drying effects of wind and high temperatures also lower the viability of pathogens. Rainfall heavy enough to produce splash on leaf surfaces may cause the spread of pathogens to other plants, the soil and to surface waters (Nicholson *et al.* 2000).

Studies indicate that pathogen survival times are longer in soils than on the surface of crops, with some pathogens still being viable in the soil several months after manure spreading onto land. Therefore, the risk of human infection through crops grown on, and in contact with, contaminated soil, is likely to be greater than for crops grown away from the soil surface. Nicholson *et al.* (2000) suggest that salad crops, which may be eaten raw, present the highest risk of pathogenic transmission and that manures should never be applied directly to growing plants. Furthermore, a six-month interval between manure spreading and crop harvest is recommended to ensure effective pathogen destruction.

# A QUANTITATIVE RISK ASSESSMENT MODEL FOR MICROBIAL EMISSIONS FROM ABATTOIRS

## Quantitative risk assessment

The risk of human illness from pathogens released to the environment through meat processing wastes can be quantified using risk assessment methodology. Risk assessment is the process through which information on risks is identified, organised and analysed in a systematic way to get a clear, consistent presentation of the data available for practical decision-making (Rodricks 1999). Quantitative risk assessment was initially applied to chemical hazards in the early 1970's as a discipline using scientific data to quantitatively evaluate human cancer risk (Potter 1996). The methodology has been refined and adapted for application in other fields including microbial hazards (*Anon*. 1999). Data assembled during the risk-assessment process is often modelled, and uncertainty and variability scenarios are tested through mathematical simulations. A common method is Monte Carlo analysis. Simulation modelling can include a sensitivity analysis whereby the influence of assumptions can be tested and the effect of corrective actions assessed. Critical knowledge gaps can also be identified and used to direct future research efforts.

## Modelling microbial emissions

The operational steps in meat processing plants have been reviewed in detail to identify sources of contaminated waste streams (Appendix B). Based on this, a quantitative microbiological risk assessment model has been developed to track pathogen transport through the air, water and solid waste streams associated with the critical stages in a primary meat processing operation. The model considers a generic abattoir receiving cattle and sheep, with input sources of pathogens from the livestock yard, carcase processing, hide processing and offal handling. The carcase processing line is treated as one entity, while hide and offal processing are treated separately. In terms of pathogen contribution, offal handling and the livestock yard are considered the most significant contamination sources. The model assumes that other than particles carrying airborne pathogens, all waste generated in the abattoir is partitioned between the effluent treatment, rendering, and composting waste streams. It is assumed that contaminated air does not pass through a filter in the abattoir and thus the concentration of airborne pathogens is not reduced by any treatment step.

The total number of pathogens derived from the processing and treatment steps in the abattoir are calculated for each of the air, water and solid waste streams. These outputs are the inputs into the second part of the risk model, which simulates the public health risks associated with waste disposal. The model quantifies the risk of people becoming ill from pathogens reaching drinking or recreational waters via spray irrigated wastewater and landspread solid waste. The risk of exposure to groundwater was not modelled. In addition, the model simulates the risk of illness from airborne pathogens reaching the surrounding community. A flow diagram illustrating how the model tracks pathogens and the fate of the air, fluid and solid waste products is shown in Figure 1.

Inputs in the model fall into 2 categories, fixed and variable, which are sourced either from the literature or based on estimated values. Fixed values are defined by the user and may be based on a particular abattoir, such as throughput of animals. The majority of inputs are entered as a range of values to allow for variability and uncertainty. For example, literature values for pathogen prevalence in cattle fall into a range of values due to factors such as age of the animal, feeding practices, geographical effects and seasonal fluctuations, as well as variability due to sample size and detection method. These variable inputs are incorporated into the model by assigning a distribution function that best describes the variability. A PERT distribution has been applied to the majority of variable inputs in the model, where statistical data or expert opinion indicated that a value was most likely to fall in between a minimum and maximum, and may be skewed towards one end of this range. The exception is the dose response parameter, which is simulated by a uniform distribution (see Appendix C).

The quantitative risk assessment model is a simplification of a complex environmental-microbiological system. The waste treatment processes are also likely to vary between abattoirs and thus the model is for a generic abattoir situation. However, the model has the flexibility to be customised for the characteristics of a particular abattoir. The major underlying assumptions of the model relate to:

- *Time scale of 24 hours*
  - no change in pathogen population during that period e.g. no multiplication/reproduction of pathogens
  - pathogen load spread uniformly over the 24 hour period then reset to zero for the next day e.g. no cumulative build-up of pathogens
  - the model looks at the risk of exposure per day being equally likely, rather than non-constant exposure e.g. exposure 7 days out of a year, with the risk on other days being zero
- *Air contamination*
  - treated as 3 point sources spreading to a variable volume based on wind speed
  - no overlap of the 3 air contamination volumes e.g. nobody receives 2 or 3 times the dose from exposure to multiple sources of infected air
  - air is assumed to be untreated by any filtration system
- *Water contamination*
  - treated as a single catchment with all pathogens reaching either a single surface water body or a single groundwater body, with a uniform distribution of pathogens
- *Composted waste*
  - in reality, composting fluids may not end up in the effluent stream as manure derived waste is generally kept separate to other waste material
- *Dose response*
  - the model assumes that humans can be infected by a pathogen through ingestion or inhalation and that the dose response is the same for both modes of infection
  - dose response curves derived from high doses can be extrapolated to low doses

## Modelling dose response

In the hazard characterisation phase of a microbial risk assessment, a dose-response analysis is undertaken to characterise the relationship between dose, infectivity and the likelihood and severity of adverse health effects associated with the hazard in an exposed population. Infective dose is typically determined by human feeding trials, but for many microorganisms, such trials carry an unacceptably high risk due to their high mortality rate or association with long-term effects. Even where human feeding trial data are available, these typically represent healthy adults, rather than more susceptible populations. Therefore, for most microorganisms, dose-response relationships are weak points because reliable and accurate data on infective dose are either scarce or do not exist. While some animal models have been used to develop dose-response models, their utility depends on how well the data can be correlated with the human response to the pathogen. There is not necessarily a clear relation between dose and occurrence of symptoms; therefore dose-response data may not allow assessment of the disease risk.

Dose-response models are generally derived from data in the high dose region. Hence extrapolation from high doses to the low dose region of clinical exposure may result in inaccurate estimates of the dose response. To reduce the uncertainty in the dose-response estimate, additional dose-response data at low doses are needed. In addition, models may not take account of strain differences in the dose-response, which creates another level of uncertainty. Most dose-response models look at ingestion of a pathogen rather than inhalation. The overall risk from inhalation has thus been calculated in our model from dietary data and should be interpreted with caution. However, it is thought that some airborne organisms could be swallowed as well; hence a digestive model is partly justified.

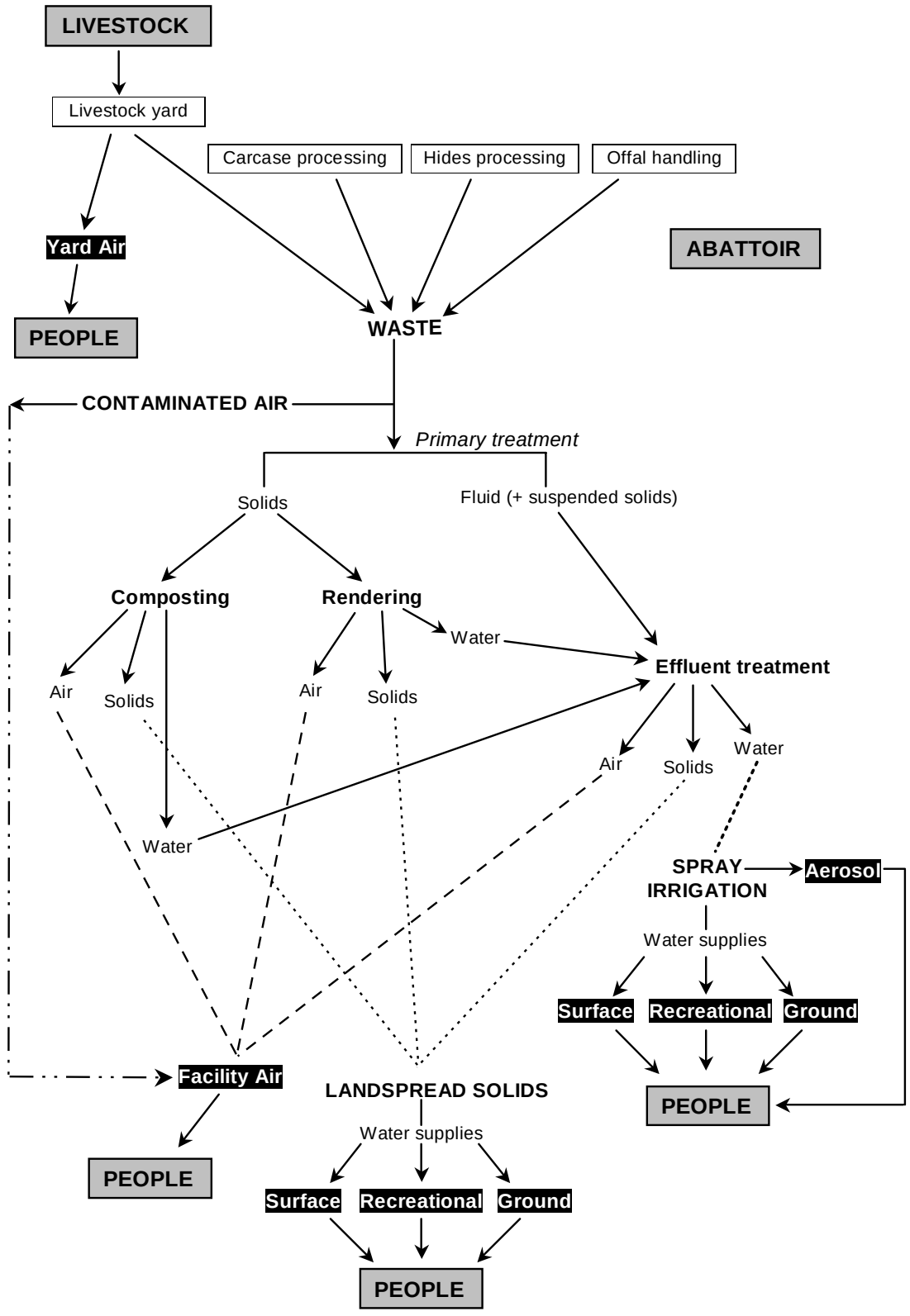


**Figure 1.** Flow diagram illustrating how the developed model tracks pathogens through the processing and treatment streams of an abattoir and the fate of air, fluid and solid waste in the environment.

# RESULTS AND RECOMMENDATIONS

## Summary results

The quantitative risk assessments for the 6 pathogens: *Escherichia coli*, *Salmonella* spp., *Campylobacter jejuni*, *Listeria monocytogenes*, *Coxiella burnetii* and *Cryptosporidium parvum* are provided at Appendices D-I for a 1000 cattle/day abattoir and a 3000 sheep/day abattoir. The median results for the airborne and waterborne routes of exposure to each pathogen are summarised in Figures 2-4 and Table 2 for a cattle abattoir scenario. The relative rankings of pathogens and the exposure pathways are similar for a sheep abattoir and hence are not included in summary form. There is considerable uncertainty associated with each risk estimate, as shown in Figures 3 and 4. We have more confidence in the relative positions of the pathogens than in the absolute risk estimates, which will depend on site-specific features of individual abattoirs. Estimates for airborne pathways relative to waterborne pathways will also depend on abattoir-specific characteristics, as depicted by sliding arrows in Figure 2.

We attach an additional level of uncertainty to the airborne pathway results for the pathogens *E. coli*, *Salmonella* spp., *C. jejuni*, *L. monocytogenes* and *C. parvum* because the inhalation route of exposure for these pathogens is not well documented in the literature. In the absence of dose response information for inhalation, we assume the same dose response as for ingestion. Although a digestive model is partly justified if some airborne organisms are swallowed, it seems likely that this assumption will be a gross overestimate. For this reason, these pathogens for each airborne pathway have been represented in smaller font in Figure 2. Assuming inhalation is not a significant pathway for the other pathogens, *C. burnetii* emerges as the most important airborne pathogen.

For the waterborne pathways of contaminated recreational and drinking water, the results indicate that *Cryptosporidium parvum* represents the most important waterborne pathogen. Although pathogen prevalence is relatively low compared to the other pathogens (Table 3), the concentration in cattle faeces is much greater. Sensitivity analyses indicate that the model outputs (number of ill from exposure to the pathogen) are highly sensitive to the concentration of *C. parvum* in cattle faeces. Compared to the other pathogens, the median pathogen intake calculated by the model for *C. parvum* is also several orders of magnitude larger than for the other pathogens, for both recreational and drinking water. The dose response curve is much steeper for *C. parvum* and thus results in the highest relative number of ill / million exposed / days of exposure. *Campylobacter jejuni* ranks as the next important pathogen in relation to exposure from waterborne pathways. As in the case of *C. parvum*, the concentration of the pathogen in cattle faeces is the most sensitive parameter in influencing the number of ill from exposure to *C. jejuni*. *Coxiella burnetii* is unlikely to cause human illness through ingestion of contaminated water. Therefore, the waterborne pathway was not considered for this pathogen.

For the three pathways of airborne exposure, contaminated irrigation water (spray drift) consistently shows the greatest relative risk for individual pathogens, followed by air from the livestock yard (yard air) and air released from the abattoir (facility air). This trend would be expected as the wastewater stream (after processing and treatment) has a higher pathogen load than the contaminated air released from the livestock yard or the air stream after processing and treatment in the abattoir. Although contaminated spray drift emerges as the highest risk airborne pathway, the ultimate risk will depend on the number of people that actually come in contact with the various types of contaminated air that have been modelled. For example, a greater number of people may be exposed to contaminated yard air or facility air than spray drift. For waterborne exposure, contaminated drinking water shows a lower risk than recreational water. This is plausible based on the assumption in the model that drinking water, unlike recreational water, undergoes a treatment step before possible ingestion.

## Recommendations

Based on the quantitative risk assessments for the six pathogens, we make the following recommendations for future work:

1. For the five pathogens other than *Coxiella burnetii*, seek expert opinion on:
    - whether the pathogen can be transmitted and survive in an airborne state
    - whether the pathogen can cause human illness via inhalation exposure

   If the answer is 'no' to either of these questions, then these pathogens can be dropped from further consideration with respect to the airborne pathway. This is especially relevant for *C. parvum* because of its relatively high risk estimate.

2. For the remaining pathogens of interest,
    - verify assumed concentration and prevalence data by sampling Australian livestock entering abattoirs
    - verify model outputs by monitoring the actual pathogen concentrations in abattoir emissions (air, soil, water).

   If the pathogens are not present in animals entering an abattoir, or not emitted from the abattoir, then there is no risk of human illness. If concentrations are lower or higher than those assumed in the model, the risk of human illness will be decreased or increased accordingly.

3. Investigate site-specific features (e.g. air dispersion, proximity of water bodies, waste treatment protocols, potential number of people exposed) of individual abattoirs and the degree to which they could influence absolute risk estimates.

4. Attempt to calibrate the model with actual statistics on human illness.

5. In prioritising further research, consider the seriousness of illness to the human population caused by exposure to each pathogen.

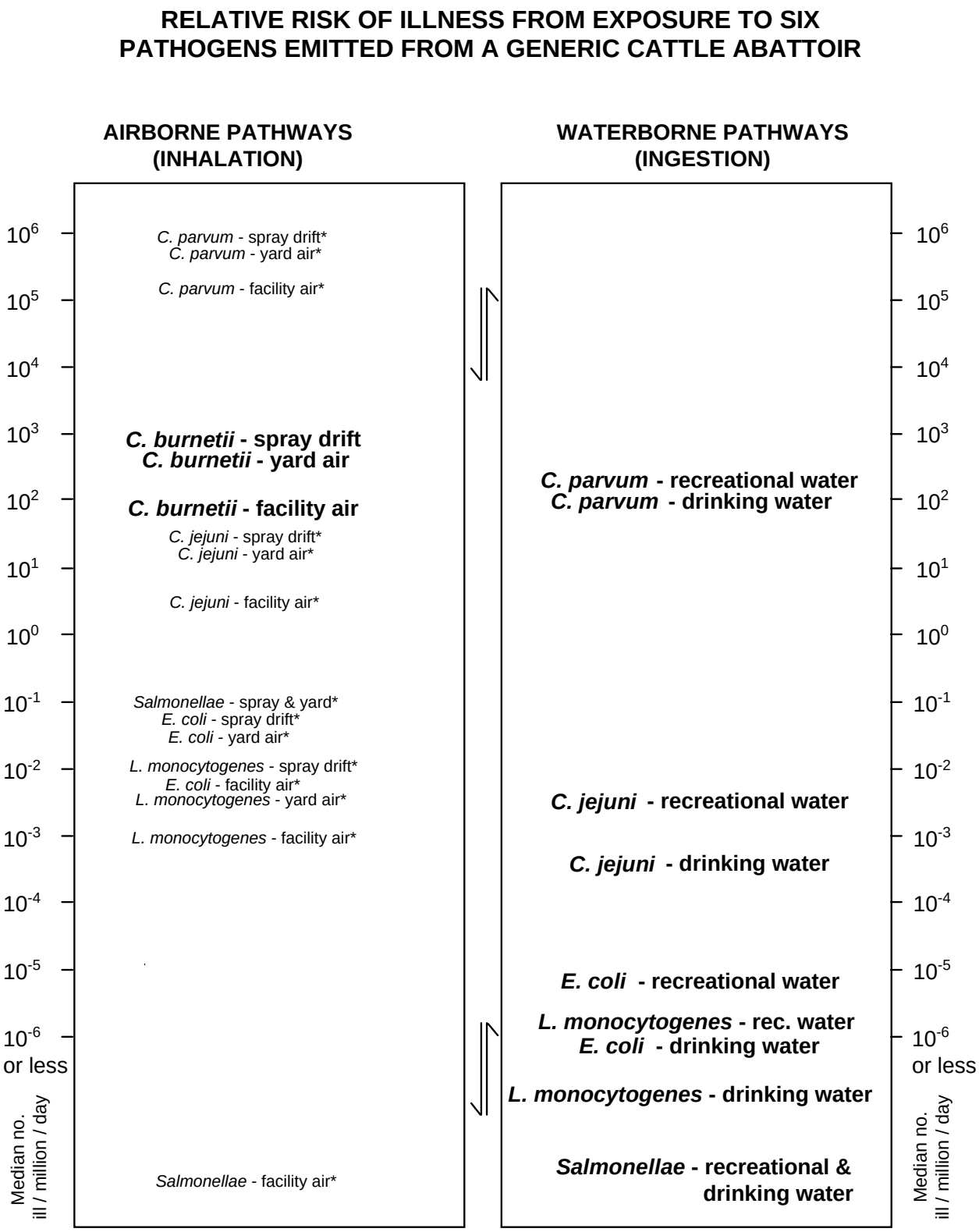


**Figure 2.** Estimated median number of ill / million exposed / days of exposure to the 6 pathogens: *Escherichia coli*, *Salmonellae*, *Campylobacter jejuni*, *Listeria monocytogenes*, *Coxiella burnetii* and *Cryptosporidium parvum*. Estimates for airborne pathways relative to waterborne pathways will depend on abattoir-specific characteristics. This is depicted by sliding arrows in the diagram.

* Relevance of airborne pathway uncertain

**Table 2.** Summary of results: median number of people ill / million exposed / days of exposure to airborne and waterborne pathogens from a generic cattle abattoir, as depicted in Figure 1.

| *50th percentile* | **CATTLE** | | | | |
|---|---|---|---|---|---|
| **Exposure route** | **Yard air** | **Facility air** | **Spray drift** | **Drinking water** | **Recreational water** |
| ***C. jejuni*** | 21 | 3 | 27 | 0.0004 | 0.0034 |
| ***E. coli*** | 0.035 | 0.005 | 0.044 | 6.50E-07 | 5.87E-06 |
| ***Salmonella* spp.** | 0.079 | 0.000 | 0.079 | 0.000 | 0.000 |
| ***C. burnetii*** | 468 | 70 | 594 | - | - |
| ***L. monocytogenes*** | 0.005 | 0.001 | 0.007 | 9.77E-08 | 8.62E-07 |
| ***C. parvum*** | 583085 | 138627 | 803407 | 114 | 168 |

**Table 3.** Simulated median inputs for individual pathogen models.

| *50th percentile* | **Prevalence of pathogen (%)** | | **Concentration of pathogen in faeces (log/g)** | | **Concentration of pathogen in placenta (log/g)** | |
|---|---|---|---|---|---|---|
| | Cattle | Sheep | Cattle | Sheep | Cattle | Sheep |
| ***C. jejuni*** | 37.56 | 9.51 | 3.06 | 4.06 | - | - |
| ***E. coli*** | 34.09 | 27.78 | 3.04 | 3.89 | - | - |
| ***Salmonella* spp.** | 56.58 | 2.88 | 2.57 | 2.57 | - | - |
| ***C. burnetii*** | 12.62 | 19.81 | 4.12 | 4.11 | 4.31 | 4.24 |
| ***L. monocytogenes*** | 66.13 | 30.91 | 1.22 | 1.22 | - | - |
| ***C. parvum*** | 9.46 | 9.12 | 8.71 | 6.82 | - | - |

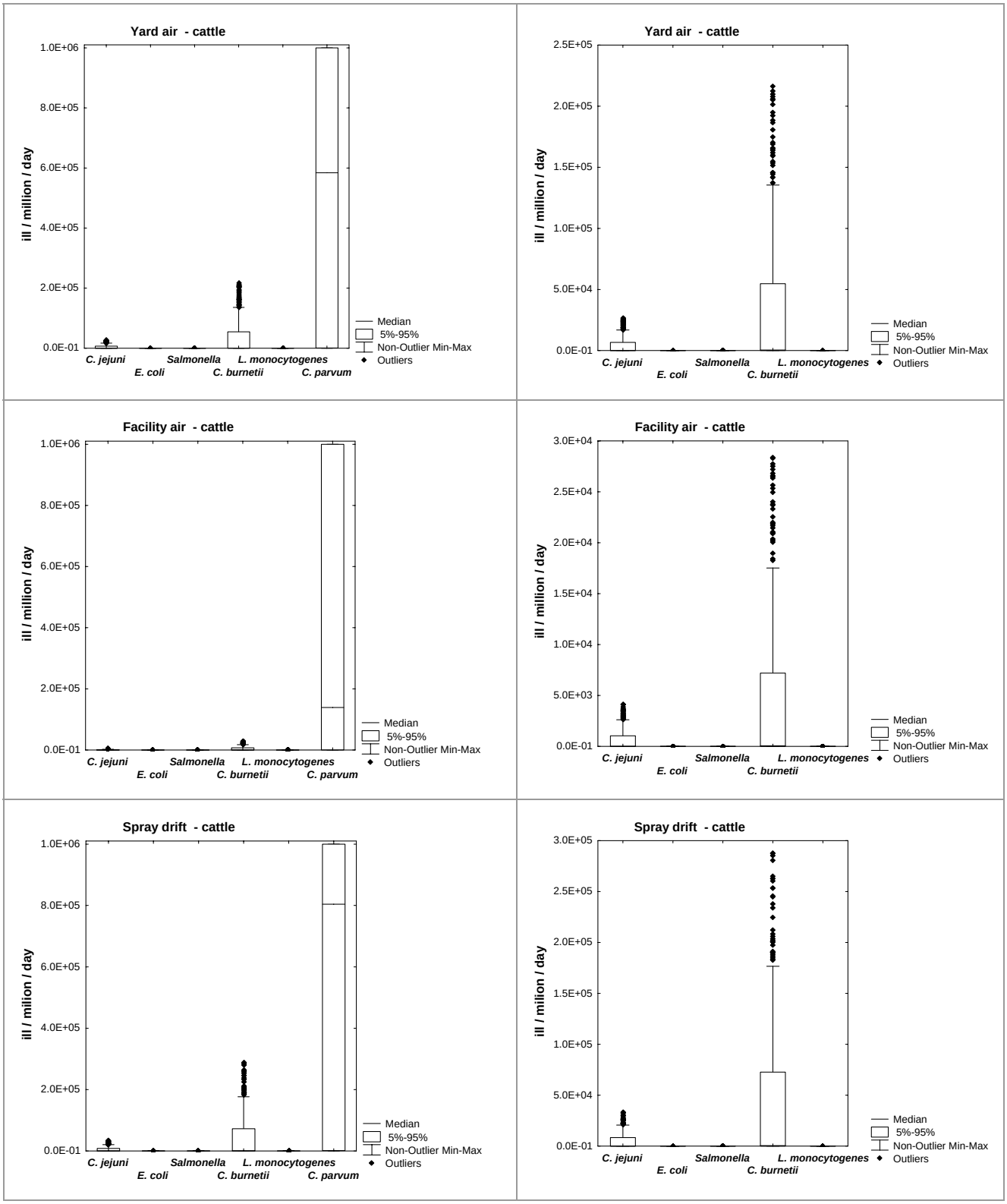


**Figure 3.** Number of people ill / million exposed / days of exposure to pathogens via the 3 airborne pathways: yard air, facility air, spray drift for a generic cattle abattoir. The left column shows the spread of results for all pathogens, while the figures on the right exclude *C. parvum*.

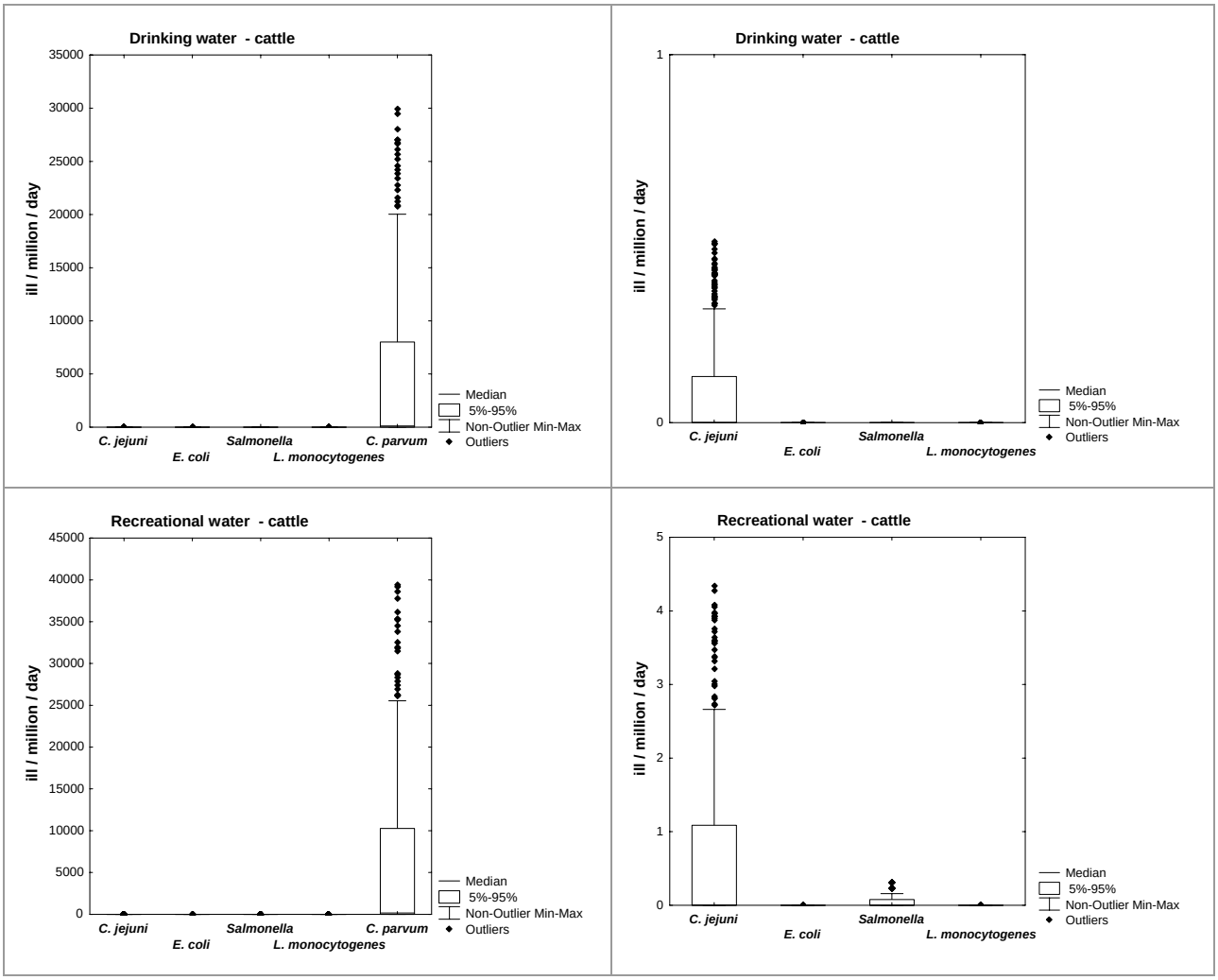


**Figure 4.** Number of people ill / million exposed / days of exposure to pathogens via the 2 waterborne pathways: drinking and recreational water for a generic cattle abattoir. The left column shows the spread of results for all pathogens, while the figures on the right exclude *C. parvum*.

# RELEVANCE OF THIS WORK

## Success in achieving objectives

The objectives of this project were to:

- Identify pathogens of interest to the red meat industry and establish the routes by which they might be transmitted to the environment
- Quantitatively estimate the risk to human health for each selected pathogen
- Produce an overall ranking of risk for the selected pathogens.

Each of these objectives has been achieved. Fifty-two pathogens were reviewed and six were selected for further study based on their relevance to the Australian meat processing industry and their potential risk to human health. Exposure routes by which they might be transmitted to the environment were investigated. A quantitative risk assessment was carried out for each of the six pathogens and an overall ranking provided both in terms of pathogen and exposure pathway. In addition we have made recommendations for future work.

Quantitative microbial risk assessments have previously been published in relation to food production (Fabiansson 2001). Risk assessment methodology has also been used in environmental studies in related fields to quantify such risks. To date however, risks posed to human health by meat industry waste streams have not been estimated or published. Previous studies have produced qualitative risk assessments for some of the pathogens studied in this project, but have concluded that the uncertainty was too great to allow quantitative assessments. By dealing with uncertainty explicitly through a simulation model, we have been able to derive quantitative results including characterising the uncertainty around them. As in all risk assessment work, we have had to make many assumptions in our modelling approach. However, the quantitative risk assessment model that we have established has the ability and flexibility to explore and test different assumptions as new information becomes available. Therefore, this project has provided the industry with an objective tool for assessing the public health risks from abattoir emissions.

## Impact on the meat and livestock industry

Recently, environmental and health agencies both in Australia and overseas have raised concerns about reports of alleged and actual instances of human illness caused by exposure to industry or farm wastes. The current ad-hoc nature of this process is costly and industry risk management is not based on scientifically validated facts. This project has provided an objective means of assessing risk and thereby improving risk management. By ranking the potential human health risks from meat industry waste the outcomes of this project will allow industry to take a pro-active approach to targeting investment, research and necessary remedial activities, while preventing unnecessary investment in areas of low to negligible risk. In the short term, this work will provide a better basis for deciding where to invest in research.

In 5 years down the track there should be a greater understanding and awareness in the industry of the risks to human health from microbial emissions from abattoirs. This should lead to the implementation, where necessary, of measures to reduce or eliminate those risks.

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# APPENDIX A. REVIEW OF PATHOGENS CAPABLE OF BEING SPREAD FROM AUSTRALIAN MEAT PROCESSING ESTABLISHMENTS WHICH POTENTIALLY COULD POSE A RISK TO HUMAN HEALTH

Medically important zoonotic diseases have been divided according to the pathogen's position in biological classification, and specifically marked when involving farm animals with incidence of the disease quantified (Table 1).

**Table 1.** Ranking of zoonotic disease agents and their importance to human disease in Australia (based on information in Weber and Rutala 1999; AHA 2002; Stevenson and Hughes 1988).

| Disease | Pathogen | Incidence in farm Animals | Importance in Australia |
|---|---|---|---|
| **Bacterial diseases** | | | |
| Aeromonas | *Aeromonas spp.* | +++ | A |
| Anthrax | *Bacillus anthracis* | +++ | A? |
| Brucellosis | *Brucella spp.* | +++ | A |
| Campylobacteriosis | *Campylobacter jejuni* | ++ | A |
| Caseous lymphadenitis | *Corynebacterium pseudotuberculosis* | ++ | A |
| Dermatophilosis | *Dermatophilus congolensis* | ++ | A |
| Erysipeloid | *Erysipelothrix rhusiopathiae* | +++ | A |
| Escherichia coli | *Escherichia coli* | +++ | A |
| Leptospirosis | *Leptospira interrogans spp.* | +++ | A |
| Listeriosis | *Listeria monocytogenes* | | |
| Lyme disease | *Borrelia burgdorferi* | ++ | A |
| Melioidosis | *Pseudomonas pseudomallei* | ++ | A |
| Pasteurellosis | *Pasteurella multocida* | + | A |
| Psittacosis | *Chlamydia psittaci* | +++ | A |
| Q fever | *Coxiella burnetti* | | |
| Relapsing fever | *Borrelia spp.* | + | A |
| Rhodococcus infection | *Rhodococcus equi* | +++ | A |
| Salmonellosis | *Salmonella spp.* | ++ | A |
| Staphylococcal infection | *Staphylococcus aureus* | + | A |
| Streptococcosis | *Streptococcus suis* | | |
| Tetanus | *Clostridium tetani* | ++ | A? |
| Tuberculosis | *Mycobacterium bovis/tuberculosis* | ++ | |
| Tularemia | *Francisella tularensis* | | |
| Vibriosis | *Vibrio parahemolyticus* | ++ | A |
| Yersiniosis | *Yersinia enterocilitica* | | |
| | | | |
| **Viral Diseases** | | | |
| Bovine papular stomatitis | Parapoxvirus | + | A |
| California encephalitis | Bunyavirus | | |
| Eastern equine encephalitis | Alphavirus | | |
| Herpesvirus simiae | Vessiculovirus | | |
| Lymphocytic choriomeningitis | Arenavirus | | |
| Milker's nodule | Parapoxvirus | + | A |
| Newcastle disease | Paramyxovirus | ++ | |
| Orf (contagious ecthyma) | Parapoxvirus | + | A |
| Rabies | Lyssavirus | | |
| Rotavirus | Rotavirus | +? | |
| St. Louis encephalitis | Flavivirus | | |
| Venezuelan equine encephalitis | Alphavirus | | |

| | | | |
|---|---|---|---|
| **Mycotic** | | | |
| Blastomycosis | *Blastomyces dermatitidis* | | |
| Cryptococcosis | *Cryptococcus neoformans* | | |
| Histoplasmosis | *Histoplasma capsulatum* | | |
| Ringworm | *Dermatophytes* | ++ | A |
| Sporotrichosis | *Sporothrix schenckii* | | |
| | | | |
| **Parasites** | | | |
| Acanthamoebiasis | *Acanthamoeba spp.* | | |
| Cryptosporidiosis | *Cryptosporidia spp.* | +++ | |
| Dypylidiasis | *Dipylidium caninum* | | |
| Dirofilariasis | *Dirofilaria immitis* | | |
| Echinococcosis | *Echinococcus granulosus* | ++ | |
| Giardiasis | *Giardia lamblia* | | |
| Naegleriasis | *Naegleria fowleri* | | |
| Toxocariasis | *Toxocara canis, cati* | | |
| Toxoplasmosis | *Toxoplasma gondii* | | |
| | | | |
| **Other** | | | |
| BSE/vCJD | Prion | + | |

A brief description of each of the above organisms is provided in the following section with comments related to their relevance for this study.

# Bacterial diseases

## *Aeromonas*

**Facts**: *Aeromonas* species are associated with gastroenteritis and with wound infections, particularly wounds incurred in outdoor settings. *Aeromonas hydrophila* is present in all freshwater environments and in brackish water. Not as much is known about the other *Aeromonas* spp., but they too are aquatic microorganisms and have been implicated in human disease. *A. hydrophila* has frequently been found in fish and shellfish.

**Importance:** Although it has also been found in market samples of red meats (beef, pork, lamb) and poultry, farm animals are not considered an important vehicle.

## *Anthrax*

**Facts:** Anthrax is an acute infectious disease caused by the spore-forming bacterium *Bacillus anthracis*. Anthrax most commonly occurs in wild and domestic animals (cattle, sheep, goats, camels, antelopes, and other herbivores), but it can also occur in humans when they are exposed to infected animals or tissue from infected animals. Human anthrax infection can occur in three forms: cutaneous (skin), inhalation, and gastrointestinal. *B. anthracis* spores can live in the soil for many years, and humans can become infected with anthrax by handling products from infected animals or by inhaling anthrax spores from contaminated animal products.

**Importance:** An example of possible airborne infection. Established in Australia with rare outbreaks. Not likely to ever be common in abattoir environmental contamination.

## *Brucellosis*

**Facts:** Brucellosis is an infectious disease caused by the bacteria of the genus *Brucella*. These bacteria are primarily passed among animals, and they cause disease in many different vertebrates. Various *Brucella* species affect sheep, goats, cattle, deer, elk, pigs and dogs. Humans are generally infected in one of three ways: eating or drinking something that is contaminated with *Brucella*, breathing in the organism (inhalation), or through skin wounds. Inhalation is often responsible for a significant percentage of cases in abattoir employees. In humans brucellosis can cause a range of symptoms that are similar to the flu and may include fever, sweats, headaches, back pains, and physical weakness.

**Importance:** Eradicated from the Australian cattle population, the ovine strain is still around but does not infect humans. Although a possible airborne infection, it is not considered a risk in Australia.

### *Campylobacteriosis*

**Facts:** *Campylobacter jejuni* is now recognized as an important enteric pathogen. Before 1972, when methods were developed for its isolation from faeces, it was believed to be primarily an animal pathogen causing abortion and enteritis in sheep and cattle. Surveys in several countries have shown that *C. jejuni* is the leading cause of bacterial diarrheal illness. It is often isolated from particularly chickens, but also from healthy cattle, birds and even flies. It is sometimes present in non-chlorinated water sources such as streams and ponds. The infective dose of *C. jejuni* is considered to be small. Human feeding studies suggest that about 400-500 bacteria may cause illness in some individuals

**Importance:** This is an important pathogen in Australia and should be further considered since it can also be spread through the environment.

### *Caseous lymphadenitis*

**Facts:** An infection involving lymph nodes widespread in sheep and goats and caused by *Corynebacterium pseudotuberculosis*. Reported human cases are uncommon and always associated with handling sheep and their products. The organism can survive for two months in soil.

**Importance:** No reported human environmental cases.

### *Dermatophilosis*

**Facts:** Mycotic dermatitis caused by *Dermatophilus congolensis* occurs in temperate regions worldwide. Natural disease described in a wide range of wild, captive and domestic animals including horses, cattle, sheep and goats. It is an obligate parasite that has been isolated only from lesions in animals. Human cases have arisen from direct contact with infected animals. The most common means of transmission between animals is mechanical thru arthropod vectors.

**Importance:** Although the infection is relatively common in sheep in Australia, particularly in higher rainfall areas, reported human disease is rare.

### *Erysipeloid*

**Facts:** Erysipelas in swine caused by *Erysipelothrix rhusiopathiae* can spread to man through animal contact and cause erysipeloid, cutaneous swellings on fingers and hands. The organism can also be spread through fish. It is usually an occupational disease affecting persons engaged in handling animal and fish products. The organism can survive for long times in the environment but this is not considered a common route of infection.

**Importance:** Usually a mild disease in man spread through direct contact with animals or animal products.

### *Escherichia coli*

**Facts:** There are many pathogenic strains of *Escherichia coli* causing human disease. Emerging strains belong to the enterohaemorrhagic *E. coli* (EHEC) group of which the O157:H7 serotype is a typical example. EHEC can spread through meat, other foods contaminated with animal faeces, and through environmental contamination. It can cause serious disease and death in particularly the young and elderly.

**Importance:** An organism of environmental concern also in Australia.

### *Leptospirosis*

**Facts:** Leptospirosis is caused by many different serovars of *Leptospira interrogans* and is spread by infected animal urine. In Australia, cattle comprise the major source of infection for man, with dairy

farmers and abattoir workers being the major occupational groups at risk. Recreational water activities have been shown to be capable of spreading the organism.

**Importance:** Leptospirosis is not uncommon in Australia with the highest prevalence in Queensland. Leptospires can remain viable in soil for months under suitable conditions.

### *Listeriosis*

**Facts:** Sporadic cases of human listeriosis are reported in Australia caused by *Listeria monocytogenes*. The source of the organism can be domestic and wild animals, poultry, soil, fish, crustaceans, vegetables, water, sewage and mud. In Australia, the disease in animals occurs largely in ruminants and small marsupials.

**Importance:** Particularly dangerous for neonatals and immunocompromised individuals. Will be looked at in more detail.

### *Lyme disease*

**Facts:** *Borrelia burgdorferi* is a vector-spread spirochaete causing Lyme disease reported in New South Wales and Queensland. The host animal in Australia has not been identified.

**Importance:** The abattoir environment is not considered important.

### *Melioidosis*

**Facts:** Melioidosis is a disease of the tropics and subtropics. It is an uncommon disease caused by *Pseudomonas pseudomallei* and usually spread through contaminated soil, mud and water. Most sporadic cases occur during the wet season from February to May.

**Importance:** The organism has been associated with pig production and isolated from pig manure heaps over three years old. Although sheep and goat can carry the disease it is not considered a big risk for abattoirs.

### *Pasteurellosis*

**Facts:** *Pasteurella multocida* and *P. haemolytica* are present in the nasopharynx of a wide range of animals including ruminants. In Australia, *P. multocida* occurs commonly but will only sporadically be a primary or secondary cause of respiratory illness in a wide range of livestock. Spread to humans only through direct animal contact.

**Importance:** Not spread through the abattoir environment.

### *Psittacosis*

**Facts:** *Chlamydia psittaci* is associated with a wide variety of birds and infection has been detected in livestock.

**Importance:** Livestock has not been identified as a source of human infection.

### *Q fever*

**Facts:** Q fever is caused by a rickettsia called *Coxiella burnetii* that can be found Australia wide. Common host animals are cattle, sheep, goats, and bandicoots. It is most commonly spread through inhalation of aerosols or dust contaminated with rickettsiae from infected ruminants in or near abattoirs or animal by-products establishments.

**Importance:** Will be considered as a potential aerosol contaminant.

### *Relapsing fever*

**Facts:** *Borrelia recurrentis* causing relapsing fever can be louse or tick spread. Is considered a zoonoses if spread by ticks from wild rodents

**Importance:** Not a problem for the abattoir environment.

### *Rhodococcus infection*

**Facts:** *Rhodococcus equi* is an opportunistic pathogen present in soil and faeces from a wide range of animals, including man. Only reported in immunocompromised individuals.

**Importance:** Not believed to be important in abattoir environments.

### *Salmonellosis*

**Facts:** Over 2000 serotypes identified on a global basis of Salmonellae. Can affect a wide range of domestic livestock, poultry, birds, wild and pet animals, and man. Salmonellae are ubiquitous in the animal and human populations. Can often be found in sewerage sludge.

**Importance:** An important pathogen for the abattoir environment.

### *Staphylococcal infection*

**Facts:** Certain strains of *Staphylococcus aureus* can cause food poisoning through contaminated food. However, human carriers are the most common source for staphylococcal infections and intoxications.

**Importance:** Not important in the abattoir environment.

### *Streptococcosis*

**Facts:** Caused by various streptococci species, including *Streptococcus suis* and *S. zooepidemicus*. Humans have been infected with *S. suis* by handling infected pork and with *S. zooepidemicus* after direct contact with animals and from drinking raw milk.

**Importance:** Not important in the abattoir environment.

### *Tetanus*

**Facts:** Tetanus is caused by *Clostridium tetani*. Clostridia are part of the normal intestinal flora and can also survive as spores in the soil.

**Importance:** The abattoir environment is not considered an important source.

### *Tuberculosis*

**Facts:** Tuberculosis caused by *Mycobacterium bovis* with cattle as the host animal was previously common in man but is now rarely seen.

**Importance:** *M. tuberculosis* infection is rarely acquired from animal sources.

### *Tularaemia*

**Facts:** *Francisella tularensis*, the cause of tularaemia, can survive several weeks in the external environment.

**Importance:** Never reported in Australia.

### *Vibriosis*

**Facts:** Vibrios other than *Vibrio cholerae* that cause human disease are *V. parahaemolyticus*, *V. vulnificus* and *V. alginolyticus*.

**Importance:** All are halophilic marine organisms.

### *Yersiniosis*

**Facts:** Although *Yersinia pseudotuberculosis* and *Y. enterocolitica* are ubiquitous in nature, isolated from dust, soil, water and milk. Normally spread through the faecal-oral route, human cases have been reported after animal contact and epidemics after consumption of contaminated food or water.

**Importance:** Human cases are rare.

## Viral diseases

### *Zoonotic viral diseases*

**Facts:** Several viral diseases are associated with wild or domesticated animals and spread to humans by arthropod vectors. These include California encephalitis, Eastern equine encephalitis, St. Louis encephalitis, and Venezuelan equine encephalitis and are not relevant to this review. Parapoxvirus or Paravaccinia infections like Bovine papular stomatitis, Milker's nodule, Orf (contagious ecthyma) and Scabby Mouth infect humans through direct contact and are again no threat in the abattoir environment. Replication of viral pathogens outside their usual host range is rare, and thus viral pathogens in animal wastes are unlikely to pose a significant health risk to humans. The single exception to this rule may be a class of viruses termed the rotaviruses, which are the causative agent of scour in calves. The exact relationship between animal and human rotaviruses remains unclear, as does the ability of animal rotaviruses to cause disease in humans

**Importance:** Spread of animal viruses to man through the abattoir environment is considered unlikely.

### *Rotavirus*

**Facts:** Rotaviruses cause acute gastroenteritis. Infantile diarrhea, winter diarrhea, acute nonbacterial infectious gastroenteritis, and acute viral gastroenteritis are names applied to the infection caused by the most common and widespread group A rotavirus. The infective dose is presumed to be 10-100 infectious viral particles. Rotaviruses are transmitted by the faecal-oral route. Person-to-person spread through contaminated hands is probably the most important means by which rotaviruses are transmitted in close communities such as pediatric and geriatric wards, day care centers and family homes. Infected food handlers may contaminate foods that require handling and no further cooking, such as salads, fruits, and hors d'oeuvres. Rotaviruses are quite stable in the environment and have been found in estuary samples at levels as high as 1-5 infectious particles/gal. Sanitary measures adequate for bacteria and parasites seem to be ineffective in endemic control of rotavirus, as similar incidence of rotavirus infection is observed in countries with both high and low health standards.

**Importance:** Difficult to assess, common in young calves.

### *Norwalk virus*

**Facts:** Common names of the illness caused by the Norwalk and Norwalk-like viruses are viral gastroenteritis, acute nonbacterial gastroenteritis, food poisoning, and food infection. The disease is self-limiting, mild, and characterized by nausea, vomiting, diarrhoea, and abdominal pain. Headache and low-grade fever may occur. The infectious dose is unknown but presumed to be low. Norwalk gastroenteritis is transmitted by the faecal-oral route via contaminated water and foods. Secondary person-to-person transmission has been documented. Water is the most common source of outbreaks and may include water from municipal supplies, well, recreational lakes, swimming pools, and water stored aboard cruise ships. Shellfish and salad ingredients are the foods most often implicated in Norwalk outbreaks. Ingestion of raw or insufficiently steamed clams and oysters poses a high risk for infection with Norwalk virus. Foods other than shellfish are contaminated by ill food handlers. Only the common cold is reported more

frequently than viral gastroenteritis as a cause of illness in the U.S. Although viral gastroenteritis is caused by a number of viruses, it is estimated that Norwalk viruses are responsible for about 1/3 of the cases not involving the 6-to-24-month age group.

**Importance:** Probably not spread through domesticated animals.

# Parasitic diseases

## *Acanthamoebiasis*

**Facts:** *Acanthamoeba* are microscopic amoeba commonly found in the environment. *Acanthamoeba spp.* are found worldwide, most commonly in soil and dust, in fresh water sources such as lakes, rivers, and hot springs and in hot tubs. The organism is often associated with contact lenses.

**Importance:** No specific animal association.

## *Cryptosporidiosis*

**Facts:** *Cryptosporidium* infects many animal species, causing symptomatic illnesses mainly in young animals, and is thought to be readily passed from animals to humans through the faecal-oral route. Classification of *Cryptosporidium parvum* is currently undergoing rapid changes. There are reports of at least two different genotypes of *C. parvum*, one of which is exclusively isolated from humans, and one of which can be isolated from both humans and cattle. It was previously assumed that *Cryptosporidium* infections in humans were zoonotic. This assumption has now been questioned and the contribution made by the human and bovine forms needs further clarification. *Cryptosporidium* spores can remain viable for about 18 months in a cool or wet environment.

**Importance:** Will be further considered.

## *Giardiasis*

**Facts:** Giardiasis is a diarrheal illness caused by *Giardia intestinalis* (also known as *Giardia lamblia*), a one-celled, microscopic parasite that lives in the intestine of people and animals. The parasite is passed in the stool of an infected person or animal. The parasite is protected by an outer shell that allows it to survive outside the body and in the environment for long periods of time. During the past 2 decades, *Giardia* has become recognized as one of the most common causes of waterborne disease (drinking and recreational) in humans and is found throughout the world. Firm evidence that *Giardia lamblia* of animal origin can infect humans has been provided. The organism has not until recently been considered a threat to human health because of its mild symptoms and susceptibility. *Giardia* cysts can survive for long periods in effluent at low temperatures but decline rapidly at higher temperatures.

**Importance:** Uncertain.

## *Toxoplasmosis*

**Facts:** Infection with *Toxoplasma gondii* is widespread in both humans and animals. Domestic cats predominate as reservoir for domestic transmission although sheep and cattle can act as intermediary hosts and spread the disease to humans through meat.

**Importance:** Abattoir environment not an important route for infection.

# Other organisms

## *Prion*

**Facts:** The prion (proteinaceous infectious agent). Prions are proteins thought to originate as regular components of neurological tissues in animals: they are not cellular organisms or viruses. In their normal non-infectious state, these proteins may be involved in cell-to-cell communication. When these proteins become abnormally shaped i.e., prions, they are able to transform molecules of the normally shaped protein with which they come into contact to the abnormal prion configuration. This process is repeated

numerous times until the number of abnormally shaped molecules causes overt illness. When consumed in the diet, prions are thought to be absorbed into the body where, again, they begin the process of changing their normal protein counterparts into prions. The specific prions of interest in disease and their normally configured proteins are those found in mammals; however, similar proteins occur in other organisms, from chickens to yeasts. While the "prion theory" of Transmissible Spongiform Encephalopathies (TSEs) is widely accepted and explains the occurrence of TSEs, there are other theories of the cause of these illnesses. Prions are associated with a group of diseases called Transmissible Spongiform Encephalopathies (TSEs). In humans, the illness suspected of being foodborne is variant Creutzfeldt-Jakob disease (vCJD). The human disease vCJD and the cattle disease, bovine spongiform encephalopathy (BSE), also known as "mad cow" disease, appear to be caused by the same agent. Other similar but not identical TSE diseases exist in animals, but there is no known transmission of these to humans. Included among these is chronic wasting disease (CWD) of deer and elk, and the oldest known of these diseases - scrapie - which occurs in sheep and goats. No early acute clinical indications for TSEs have been described. After an extended incubation period of years, these diseases result in irreversible neurodegeneration that becomes the cause of death.

**Importance:** Not detected in Australia and will not be considered further. A separate risk assessment has been completed earlier by the Bureau of Rural Sciences (Quinn & Fabiansson 2001).

# APPENDIX B. ABATTOIR PROCESSES

The risk assessment model is based on tracking pathogen numbers through a series of processes which together result in microbial emissions. The abattoir process is illustrated below.

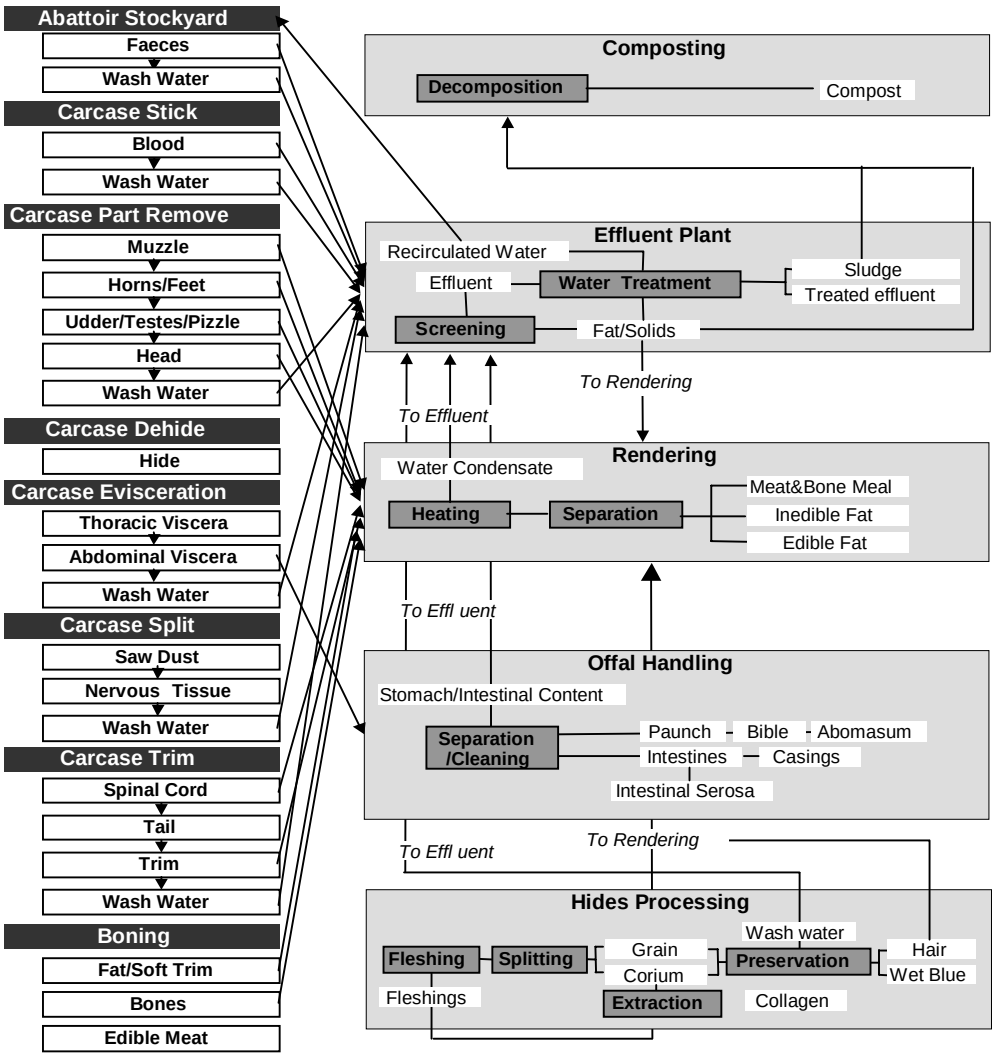



**Figure 1.** Typical operational steps in a meat processing plant and resulting waste streams.

# APPENDIX C. THE QUANTITATIVE RISK ASSESSMENT MODEL

This appendix describes the generic risk assessment model which was applied to selected pathogens in the main report. Where input parameters varied between pathogens, they are described in the main report for that pathogen. Where abattoir processes were modelled in the same way for each pathogen, the data is described here.

## Use of @*RISK* to model uncertainty

@*RISK* (Palisade Corporation 2002) is a risk assessment 'Add-In' to Microsoft Excel. The main function it adds to Excel is the ability to assign a probability distribution to any cell in a spreadsheet. A number of utilities are included to statistically analyse and summarise the results generated by running a simulation.

@*RISK* was used in this quantitative risk assessment to generate values for the variable inputs for a particular model run. @*RISK* uses a technique known as Monte Carlo simulation to recalculate the model inputs and hence outputs hundreds of times over, each time selecting random numbers within the specified distribution functions of the variable inputs. The result is the distribution of all possible outcomes and the probabilities of getting those results. In this way, the @*RISK* model was able to generate the probability of human illness from exposure to particular pathogens.

Two types of probability distributions were used in this case to model the uncertainty associated with the data on particular pathogens and on general abattoir processes. The PERT distribution (Program Evaluation and Review Technique) was developed by the consulting firm of Booz, Allen & Hamilton in conjunction with the United States Navy in 1958 as a tool for coordinating the activities of over 11,00 contractors involved with the Polaris missile program. The input parameters are the *minimum*, *most likely* and *maximum* values. This has been applied to most of the parameters where statistical data or expert opinion indicated that a value was most likely to fall in between a minimum and maximum, and may be skewed towards one end of this range. A PERT distribution (a special form of the beta distribution) is similar to a Triangular distribution in that it uses estimates of minimum, most likely and maximum values, thus allowing uncertainty to be incorporated into the model but with a more realistic equation to produce a smoother curve. The shape parameter for a PERT distribution is calculated from the defined *most likely* value (http://www.edecisiontools.com/help/rdk4/htm/pert.htm).

The model uses PERT distributions for the majority of variable inputs, except for the dose response parameter, which is simulated by a uniform distribution. Published data on dose response is so variable that there is no reason to expect that intermediate dose response curves are more likely than the extremes. In a uniform distribution, any one of the ranges of possible values is equally likely.

Other probability distributions may be applied in future applications of this model where data is available for specific abattoirs, for example a discrete distribution would apply where an uncertain value could be one of a number of known values, but nothing in between.

## Set up of the @*RISK* spreadsheet for risk assessment of microbial emissions from abattoirs

### *Data entry*

The risk assessment model was set up in an Excel spreadsheet with the @*RISK* Add-in. The model is described here as a series of modules which calculate the distribution of incoming pathogens in different waste streams. Attached is a printout of the spreadsheet for the *Campylobacter jejuni* model. It was used as the basis for each pathogen specific model.

To operate the model, the user enters data into the spreadsheet, which is colour-coded to indicate the types of inputs and outputs that are entered or calculated. Outputs in the model, such as number of people ill per million exposed per day of exposure, are defined by the user by assigning the *RiskOutput* function to the appropriate cells in the model spreadsheet. Only white fields require data entry by the user:

*White fields.* User defined inputs, numeric values and sources of the values (typically scientific literature or expert opinion). Where values are simulated the range of inputs are required to model the uncertainty of the value. In the current model, values are used to generate a probability distribution which is either PERT or uniform. These are reported as either PERT (x, y, z) or uniform (x, y).

*Yellow fields.* Numbers derived directly from a Monte Carlo simulation based on user-defined parameters entered in the white fields.

*Blue fields.* Values derived from other inputs and/or outputs, fixed or simulated.

*Pink fields.* Key model outputs derived from white, yellow and blue fields.

### *Simulation settings*

Once the user has entered the required parameters and uncertain values into the model spreadsheet and identified the outputs of the analysis, the spreadsheet is ready for @*RISK* to run a simulation. The first step is to set the *simulation settings* which determine the type of simulation @*RISK* performs. In the *Iterations Tab*, we set the *number of simulations* to *1*, and the *number of iterations* to *auto*, with each iteration to *update display* in the model spreadsheet so the user can see the recalculated results. The *auto-stop convergence percentage* was set to *1.5*. This setting directs @*RISK* to stop the simulation when all output distribution statistics change by less than the convergence threshold entered. A convergence monitor window is displayed in the @*RISK-Results* window while a simulation is running; as each monitored statistic converges (i.e. it is changing less than the entered convergence threshold) it is highlighted. An output distribution with all statistics converged is marked with a smile.

In the *Sampling Tab* we specified the:

- *sampling type* to be *Monte Carlo*
- the default *standard recalc* to be *expected value* (the expected or mean value is displayed in the model spreadsheet).
- *random generator seed* to be *chosen randomly* (@*RISK* randomly picks a new seed value each simulation).
- *collect distribution samples* to be *all* ( samples are collected for all input distribution functions)

In the *Monitor Tab* we set the:

- *@RISK Results Window* to update every *100 iterations* and to *monitor convergence* to track the stability of the output distributions being generated. By monitoring convergence this ensures that a sufficient, but not excessive, number of iterations have been run.
- *@RISK Statistics Functions* to *update every 1 iteration* and to *update during simulation*

## Running the @*RISK* model simulations

### *Dose response parameter*

The next step is to determine the appropriate dose response range, which is set by the number of ingested/inhaled pathogens in the water and air contamination pathways: surface drinking water, ground water, recreational water, yard air, facility air and spray drift (modules L-Q). The cells in the model spreadsheet, which correspond to the number of inhaled/ingested pathogens in each pathway, are set as the (only) model outputs using the *RiskOutput* function. The list of outputs in the generic model (excluding the groundwater pathway) should read:

- pathogen intake/person/day surface drinking water
- pathogen intake/person/day recreational drinking water
- pathogen intake/person/day yard air contamination
- pathogen intake/person/day facility air contamination
- pathogen intake/person/day spray drift contamination

A simulation is then run to determine the largest 95$^{th}$ percentile pathogen dose across the various contamination pathways. In the generic model, this maximum corresponds to the 95$^{th}$ percentile for the spray drift pathway, which is used to set the variable parameter of the dose response curve. The range of the dose response parameter is chosen to capture the full range of reported dose response models in the literature, within the range of pathogen ingestion/inhalation calculated by the model.

### *Number of people ill*

Having set the dose response parameter, the model outputs are also reset, using the *Riskoutput* function, to the cells in the spreadsheet corresponding to the number of ill per million per day from each contamination pathway. The list of outputs in the generic model should now read:

- ill per million per day surface water ingestion
- ill per million per day groundwater ingestion
- ill per million per day recreational water ingestion
- ill per million per day yard air inhalation
- ill per million per day facility air inhalation
- ill per million per day spray drift inhalation

A simulation is run based on the new list of outputs to estimate the relative risks of the air and water contamination pathways included in the model.

## Display of results from the @*RISK* model

The results generated from a simulation are displayed in the @*RISK-Results* window. The user has the choice of viewing the results in various ways. We chose the *Detailed Statistics* window to tabulate the minimum, maximum and percentiles (5$^{th}$, 50$^{th}$, 95$^{th}$) results for the important input variables and outputs. Results from the *Data* window, which shows the simulated outputs for each iteration, were exported to the graphing software *Statistica 6.0* to generate box plots for each contamination pathway. The plots were separated into air contamination and water contamination pathways for cattle and sheep due to differences in the magnitude of the results.

Graphs of the distributions for the inputs of pathogen prevalence and pathogen concentration in the faeces were generated in @*RISK* (and exported to *Excel*) to compare the distributions in cattle and sheep.

In addition, sensitivity analyses were performed in @*RISK* for each variable output in order to determine which input parameters have the greatest influence on the estimated number of people ill. The *Sensitivity* window allows the user to graph the sensitivity of the inputs for each output as a Tornado Graph (exported to *Excel*). We chose rank order correlation as the method for calculating sensitivity analysis results. This is a quantitative measurement of the strength of a relationship between two variables. The most common type of correlation is linear correlation, which measures the linear relationship between two variables.

The rank order correlation returned by *@RISK* can vary between –1 and 1. A value of 0 indicates there is no correlation between variables; they are independent. A value of 1 indicates a complete positive correlation between the two variables; when the input value samples 'high', the output value will sample 'high'. A value of –1 indicates a negative correlation between the two variables; when the input value samples 'high', the output value will sample 'low'. Other correlation values indicate a partial correlation; the output is affected by changes in the selected input, but may be affected by other variables as well (Palisade 2002).

# Pathogen input

## *A. Pathogen input calculations*

### Number & species slaughtered per day

The daily number of cattle and sheep processed is the starting point for the model. These numbers are entered as fixed values, i.e. they are not subject to uncertainty. The generic model considers a pure cattle abattoir as receiving 1000 cattle/day and a pure sheep abattoir as 3000 sheep/day (Johns, pers.comm.). The number of stock is automatically converted to tonnes of Hot Standard Carcase Weight (tHSCW). The cattle tHSCW is built into the spreadsheet as 0.25 per beast (i.e. an average of 250 kg carcase dressed weight) and the sheep tHSCW as 0.02 per sheep (i.e. an average of 20 kg carcase dressed weight). Both of these tHSCW values are fixed inputs in the model, i.e. there is no field for the user to enter a different value. However, they can be changed in the model by altering the formula in the cell which calculates the tHSCW.

### Prevalence of a pathogen

There are many technical challenges in surveillance for food borne pathogens in livestock and livestock products. In practice, the link between the *apparent prevalence* of a pathogen reported in the literature and the *actual prevalence* of a pathogen depends on a number of factors including; the amount of variation in the population; the intensity of sampling; the degree to which sampling is unbiased and the extent of bias in test performance as measured by test sensitivity and test specificity. One of the ways to improve the interpretation of animal health survey statistics is through stochastic modelling based on custom built Monte Carlo simulation models (Jordan 2002).

The prevalence of carriers for the pathogen in cattle and/or sheep is entered as a PERT distribution, i.e. minimum, most likely and maximum prevalence expressed as a percentage of the population carrying the pathogen.

### Pathogens in carrier faeces

The estimated level of pathogen contamination ($log_{10}$/g) of faeces is entered as a PERT distribution. This is multiplied by the estimated volume of material in the gastro-intestinal system (also entered as a PERT distribution) to calculate the total number of pathogens in the model, expressed as the daily pathogen load ($log_{10}$) that will be introduced into the process.

The estimated level of pathogen contamination is typically derived from the scientific literature on specific pathogens. For the generic model, the volume of material in the gastro-intestinal system is set at PERT (40, 50, 70) kg/cattle and PERT (4.0, 5.0, 7.0) kg/sheep (Ockerman & Hansen 2000).

### Pathogens in other sources of contamination

Sources of contamination other than faeces can be more or less significant, depending on the pathogen. These can be modelled in the same way as pathogens in faeces. The estimated level of pathogen contamination ($log_{10}$/g) of the other source is entered as a PERT distribution. This is multiplied by the estimated volume of material in the other source (also entered as a PERT distribution) to calculate the total number of pathogens in the model, expressed as the daily pathogen load ($log_{10}$) that will be introduced into the process.

The level of contamination and volume of other sources is set at PERT (0, 0, 0) in the generic model. For some pathogen localised in specific organs there will be non-zero values for these fields.

The output of Module A is the total daily pathogen load ($log_{10}$) that will be introduced into the process, the sum of the pathogens in faeces and other sources in both cattle and sheep.

### *B. Livestock yard data & calculations*

Initially, an estimate of the proportion of the pathogen load released in the livestock yard is entered, with a number between 0 and 1 indicating 0 to 100% (the sum of this number and subsequent numbers for carcase, hide and offal processing should add up to 1 to account for all contaminated product, assuming only 'clean' product is shipped to customers). For the generic model, PERT (0.20, 0.25, 0.30) of the pathogen load is released in the yard (Johns, pers.comm.).

The user inputs values to model the proportion of the pathogen load partitioned into solids, water and air components. In the generic model, we estimate that PERT (0.02, 0.04, 0.05) of the pathogen load goes to water, PERT (0.0025, 0.01, 0.02) goes to air and the remainder goes to solid wastes.

The proportion of contaminated air leaking outside the livestock yard is entered as a PERT distribution, in the generic model PERT (0.95, 0.99, 1.00) (estimate for an open outdoor livestock yard). It is assumed that pathogens in the remaining contaminated air are washed down and contribute to the water component of the pathogen load in the livestock yard.

The model calculates the total daily number of pathogens ($log_{10}$/day) spread to solids, water and air from the livestock yard. The pathogen load in each stream is recorded in columns D, E and F respectively.

## Processing

### *C. Pathogen output: Carcase processing*

The carcase processing module is similar to the livestock yard module and has the same inputs. After the data have been provided, the model calculates the total daily number of pathogens ($log_{10}$/day) spread to solids, water and air.

In the generic model, PERT (0.05, 0.10, 0.15) of the pathogen load is released in carcass processing (Johns, pers.comm.). We estimate that PERT (0.44, 0.48, 0.58) of the pathogen load goes to water, PERT (0.0001, 0.004, 0.01) goes to air and the remainder goes to solid wastes. The proportion of air leaking outside the carcase processing room is estimated as PERT (0.01, 0.02, 0.05). It is assumed that pathogens in the remaining contaminated air are washed down and contribute to the water component of the pathogen load in carcase processing.

### *D. Pathogen output: Hide processing*

The hide processing module is similar to the livestock yard and carcase processing modules and contains the same inputs. After the data have been entered, the model calculates the total daily number of pathogens (log/day) spread to solids, water and air.

In the generic model, PERT (0.01, 0.03, 0.05) of the pathogen load is released in hide processing (Johns, pers.comm.). We estimate that PERT (0.18, 0.28, 0.37) of the pathogen load goes to water, PERT (0.00001, 0.01, 0.02) goes to air and the remainder goes to solid wastes. The proportion of air leaking outside the hide processing room is estimated as PERT (0.02, 0.06, 0.10). It is assumed that pathogens in the remaining contaminated air are washed down and contribute to the water component of the pathogen load in hide processing.

### *E. Pathogen output: Offal handling*

The offal handling module is similar to the livestock yard, carcase and hide processing modules and contains the same inputs with the exception of pathogen load released. In the generic model, the fraction of the total pathogen load not released in the livestock yard, carcase and hide processing is released in offal handling. After the data have been entered, the model calculates the total daily number of pathogens (log/day) spread to solids, water and air.

In the generic model, we estimate that PERT (0.10, 0.30, 0.70) of the pathogen load goes to water, PERT (0.00001, 0.01, 0.02) goes to air and the remainder goes to solid wastes. The proportion of air leaking outside the offal handling room is estimated as PERT (0.02, 0.06, 0.10). It is assumed that pathogens in the remaining contaminated air are washed down and contribute to the water component of the pathogen load in offal handling.

### *F. Total pathogens per stream from processing*

The total pathogen load in the three different streams (solids, water and air) is calculated by the model as the sum of the load in each stream from carcase processing, hide processing and offal handling. Pathogens from the water and solids components of the livestock yard are also added to the respective pathogen streams from processing. However, pathogens from livestock yard air are not combined with the air stream from processing.

The model assumes that the total pathogen load in the solids and water streams after processing undergo a treatment step. The solids stream is split between rendering, manure and effluent treatment. All wastewater is assumed to pass through the effluent treatment process. Pathogens in the air stream are not treated but are added to during rendering, manure and effluent treatment.

## Treatment

### *G. Pathogen output: Rendering*

A proportion of the solids will be rendered. Rendering will kill most pathogens, although there is a slight risk of recontamination of solids and also some pathogen leakage to the water stream through stickwater and to air from the rendering operation.

The generic model estimates that the proportion of solids that are rendered is PERT (0.03, 0.50, 0.60) (estimate). Pathogen die-off is estimated as PERT (0.96, 0.98, 0.99) (estimate).

PERT (0.005, 0.008, 0.012) of the surviving pathogens is apportioned to the water stream, and PERT (0.00001, 0.00005, 0.00008) to air leakage. The remaining pathogens go to the solid waste stream.

### *H. Pathogen output: Composting - manure & paunch content*

Manure, mainly from the stockyard but also material from the offal room including paunch content, will most often be composted before being used as fertiliser. The model assumes that manure and paunch content are treated in a different effluent stream to all other processing wastes (MLA 1998) and that manure from the livestock yard is collected and not left on the soil substrate. The user input proportion specifies the fraction of solid waste derived from manure & paunch opening content.

The generic model estimates that the proportion of solids that are manure and paunch content and that are composted is PERT (0.20, 0.30, 0.40). Pathogen die-off is estimated as PERT (0.40, 0.60, 0.80).

PERT (0.005, 0.008, 0.012) of the surviving pathogens is apportioned to the water stream, and PERT (0.00001, 0.00005, 0.00008) to air leakage. The remaining pathogens go to the solid waste stream.

### *I. Pathogen output: Effluent treatment*

Water from all parts of the plant, including suspended solids, will go through the effluent treatment facility. The model adds pathogen loads from the processing plant to water leakage in rendering and composting.

In the generic model, the fraction of the total solid wastes not treated by rendering or composting goes to the effluent stream as suspended solids. Pathogen die-off is estimated as PERT (0.92, 0.95, 0.98).

PERT (0.40, 0.50, 0.60) of the surviving pathogens is apportioned to the water stream, and PERT (0.00001, 0.00005, 0.00008) to air leakage. The remaining pathogens go to the solid waste stream.

### *J. Pathogen output: Total*

The total pathogen load in the three different streams (solids, water and air) is calculated by the model as follows:

Air – pathogens from the air streams in the processing (carcase, hide and offal handling) and treatment (rendering, composting, effluent) streams are added together. This is referred to as facility air, distinct from air from the livestock yard.

Water – pathogens from the water streams of all parts of the abattoir are assumed to pass through the effluent treatment plant. The total pathogen load in the water stream is assumed to be the residual pathogens in the water stream after effluent treatment.

Solids – all pathogens in the solid streams after processing are assumed to pass through one of three treatment steps. The total pathogen load is the sum of the pathogens from rendering, manure treatment and effluent treatment.

## Water contamination

People can be exposed to water from the abattoir in one of four ways (note, the model does not consider exposure to more than one of these at a time):

- Ingestion of contaminated surface drinking water
- Ingestion of contaminated groundwater used as drinking water
- Ingestion of surface water used for recreational purposes
- Inhalation of aerosols from water used for irrigation

Each is described separately below.

### *K. Water in general*

The volume of water leaving the abattoir is modelled by multiplying slaughter volume (in terms of tHSCW) by PERT (2000, 8000, 16000) L/tHSCW (MLA 1996).

In the model, treated water leaves the abattoir and is used entirely for irrigating pastures. Before contacting the soil, some of the water is lost through evaporation or dispersal as aerosols. This fraction of the water, and the pathogens contained in it, is estimated as PERT (0.04, 0.33, 0.50) of the total (Calder 2000). The model also assumes a maximum pathogen survival time of 24 hours such that there is no cumulative build up of pathogens in the water supply over time.

Excess water in the soil, i.e. water which is not used by the plants, goes to groundwater and surface water run-off. At 100% irrigation efficiency, no water leaks from the pasture to groundwater or run-off. In reality, this is unlikely so the model parameters for the fraction which leaks are set to PERT (0.00, 0.10, 0.40). The model apportions half of this leaked water to groundwater and half to surface water run-off. The actual behaviour of irrigation water will vary from site to site.

### *L. Public health risk: surface drinking water*

Drinking and swimming water may be sourced from a large body of water such as a dam or lake, a river or groundwater. The model treats pathogens as being uniformly dispersed in a water volume which is determined by a figure set to a realistic order of magnitude and then subjected to uncertainty through a Monte Carlo simulation.

For drinking water, it is assumed that the only source is a body of surface water, hence the groundwater parameters are set to zero. A dam was chosen since it is difficult to quantify the volume of groundwater and river water. Wyangala Dam near Cowra was selected as a medium sized water storage. With a capacity of 1220 GL, the level of this reservoir steadily declined from 80 to 40% between August 2001

and August 2002 (DLWC 2002). Hence the parameters were set as PERT (488, 732, 976) GL (i.e. 40, 60 and 80% of full capacity).

Pathogens entering surface water are assumed to be derived from two sources: irrigation water and a fraction of the pathogens in landspread solid waste which are leached out by irrigation and/or rainwater. The pathogens leaking to surface water from irrigation water (module K) are added to the fraction of the pathogens from landspread solid wastes, estimated at PERT (0.01, 0.10, 0.20). The pathogen concentration is then calculated based on the number of pathogens from these sources entering the simulated surface water volume.

The number of pathogens surviving in the water body after UV radiation and other means of reduction is estimated as PERT (0.05, 0.08, 0.12) of the total. This figure is then reduced by a measure of drinking water treatment efficacy, estimated as PERT (0.980, 0.990, 0.995).

The total number of pathogens entering the water body is divided by the volume to give a concentration of pathogens. This is then multiplied by the daily amount of water drunk by a person, estimated as PERT (0.50, 1.00, 2.00) L, to simulate the number of pathogens a person might be exposed to.

The number of ill per million per day is calculated on the basis of a dose response equation described below (module R).

To make this part of the model more realistic would require dispersion modelling of the kind that could be applied to air pollution. The number of people exposed to each source of water could be set using data specific to the region surrounding the abattoir. The model doesn't account for different survival rates of pathogens in surface versus ground water supplies, although there is some evidence to suggest greater survival rates of bacteria in groundwater (Bitton and Harvey 1992). Therefore, an estimate of the number of days the water source will remain contaminated could also be included in the model.

### *M. Public health risk: aquifer (groundwater) as drinking water*

Exposure to pathogens through drinking of groundwater is simulated in the same way as for surface drinking water with one exception. The fraction of the pathogens from landspread solid wastes is estimated at PERT (0.01, 0.05, 0.10). In the generic model, the volume of the aquifer is set to zero, i.e. no groundwater is used as drinking water in this scenario.

### *N. Public health risk: recreational swimming*

Recreational (swimming) water is the same body of water as surface drinking water in the model. The pathogen load is higher because there is no drinking water treatment such as chlorination. The intake volume is lower; an assumption of 100 ml water ingested per person per day of swimming exposure has been used previously in recreational water risk assessment work (Haas 1983b). In the model, this water ingestion is estimated as PERT (0.05, 0.10, 0.15) L.

## Air contamination

The risk posed by air contamination spreading to the surrounding environment is the most difficult to quantify. Most airborne pathogens would be bound to particles and spread only to the immediate environment. A gradient is expected further away from the source, with resultant diminishing risk. However, in 2002 it was reported that sand from Sahara was transported to the snowfields of Switzerland and thus airborne particles seem capable of spreading randomly and at long distances.

The model assumes that airborne pathogens are derived from the livestock yard, the processing and waste treatment stages, solid waste application and via spray irrigation of treated wastewater. Three distinct types of air are modelled:

- *yard air* – air/dust from the livestock yard
- *facility air* - air from processing & treatment
- *spray drift* - aerosols (droplets) from spray irrigation

Each of these has different pathogen numbers.

It is assumed that all treated wastewater is dispensed of through spray irrigation and not recycled through the abattoir and that a fraction of the irrigation water is airborne (the rest settles on the ground). A proportion of pathogens in landspread waste to become airborne was not included in the model, as the amount of airborne particles generated would depend on the spreading rate, agitation etc.

According to Pillai *et al.* (1996), land application of sludges poses little risk of airborne transmission of bacterial pathogens, although physical agitation of sludge material could result in the generation of a large number of diverse bacterial populations in the immediate vicinity. Boutin *et al.* (1988) also reported that the intensity and extent of airborne contamination from landspreading of animal wastes depended mainly on the initial bacterial load of the slurries and the spreading conditions (projection height and droplet size). The model does not consider a dispersion model for solid waste application, but rather assumes waste is left in a pile and could expose humans to pathogens via runoff to water supplies. An extension to the model could consider different application techniques, if relevant to Australia, as in Boutin *et al.* (1988)

## *O. Public health risk: air contamination from livestock yard (yard air)*

The generic model includes a crude measure of dispersion of air from the abattoir. The total volume of air containing pathogens was estimated by multiplying simulated wind speed by time to give a maximum range for pathogens to move. This was converted to a volume by considering the dispersal to be a cylinder with a radius of half the maximum range. Wind speed was based on average monthly 9 am wind speed data for Wodonga over an 11.3-year period ending in 1984 (BOM 2001). The parameters of the Pert distribution were set using the minimum, maximum and average monthly averages, i.e. PERT (1.2, 4.9, 7.7) m/s. The height of the cylinder was simulated based on a 'best guess' of PERT (1, 4, 6) m.

The concentration of pathogens is calculated by multiplying the number of pathogens leaking to the air, simulated in module B, by this simulated volume of air.

Average daily breathing volumes were estimated as PERT (8, 10, 12) $m^3$/day/person (http://www.nhlbi.nih.gov/health/public/lung/other/lungs_hd.pdf). The number of pathogens a person might be exposed to is calculated by multiplying this breathing volume by the concentration of pathogens in the simulated volume of air around the livestock yard.

The number of ill per million per day is calculated on the basis of a dose response equation described below (module R).

## *P. Public health risk: air contamination from processing & treatment (facility air)*

Exposure to pathogens in the air surrounding the processing and treatment site is calculated in the same way as for the livestock yard. The number of pathogens used to calculate the concentration is derived in module J.

## *Q. Public health risk: air contamination from spray irrigation (spray drift)*

The highest risk might be from irrigation aerosols as the pathogen is in an aqueous environment and is likely to survive transport for a greater distance/time.

According to MLA (1998), irrigation is practiced at many abattoir sites around Australia. Application rates are seasonal and the monthly variation needs to be taken into account (2-15 ML/ha/yr). The model assumes that all wastewater generated per day is irrigated that day, and not stored to allow for optimal irrigation rates.

Exposure to pathogens in the air surrounding the spray irrigation site is calculated in the same way as for the livestock yard. The number of pathogens used to calculate the concentration is derived from the water stream pathogen load after processing and treatment (module J), taking into account the estimated proportion of irrigation water dispersed as an aerosol (module K).

In addition, the model assumes that spray drift irrigation is sourced from untreated wastewater (no further treatment after the effluent treatment stage).

# Dose response model

## *R. Weibull-Gamma dose-response equation*

The outputs of the simulations described above are numbers of pathogens that could be ingested or inhaled by a person exposed to a contaminated source of water or air. Module R converts these levels of exposure to a probability of illness resulting from such exposure. This is expressed as the number of people per million exposed that could be expected to become ill from that level of exposure per day. There is no estimate of the number of people that could actually be exposed, for example, how many people are actually exposed to irrigated spray drift per day. Hence the magnitude of the risk needs to be interpreted/identified.

Pathogen specific data is commonly reported in terms of Weibull-Gamma or Beta Poisson parameters, where the probability of infection depends on the dose, *D*, and the dose-response parameters $\varepsilon$ and $\beta$. The Beta-Poisson equation is a simple form of the Weibull-Gamma equation where the chi ($\chi$) parameter is equal to 1. To keep the model flexible the more complex equation has been built in, so that parameters for either type can be used. The dose response, or probability of illness, is quantified in the generic model using the Weibull-Gamma dose-response equation, which has the form:

$$\text{Probability of infection} = 1 - [(1 + (\text{dose}^{\chi}/\beta))]^{-\varepsilon}$$

To start the quantitative risk assessment, values for the three Weibull-Gamma parameters $\chi$, $\beta$ and $\varepsilon$ are required. Alternative dose response equations can be introduced into the model, depending on the pathogen of interest.

Given the large variation in dose-response reported for most pathogens, the model allows the user to simulate the uncertainty by applying a probability distribution to one of the parameters. In the generic model the $\beta$ parameter is set to UNIFORM (x, y). By simulating the minimum and maximum pathogen loads the user can determine the range of doses that a person might be exposed to. A dose response curve can be plotted for this range in Excel and the $\beta$ parameter manually changed to determine the minimum and maximum value which will describe the curve in that dose range.

The model assumes that one microorganism can cause infection at a certain probability level set by the three pathogenicity parameters used in the generic equation. Dose-response models have been criticised at the low levels of contamination indicated here, since data have been derived at much higher levels of contamination and extrapolated across the dose spectrum. With this caveat in mind, risk assessment results can at least be used to establish risk rankings even if the absolute number of expected infections is associated with large uncertainties.

## *S. Summary of model outputs*

Module S displays a summary of the model outputs based on each simulation being set to its most likely value. As a visual aid, three dynamic graphs show how the model is operating during a simulation. These display the pathogen intake per person, the dose response and the number of ill per million that is calculated.

# APPENDIX D. QUANTITATIVE RISK ASSESSMENT OF *CAMPYLOBACTER JEJUNI* EMISSIONS FROM ABATTOIRS

*Campylobacter jejuni* (*C. jejuni*) is now recognised as an important enteric pathogen. Before 1972, when methods were developed for its isolation from faeces, it was believed to be primarily an animal pathogen causing abortion and enteritis in sheep and cattle. *C. jejuni* is most often isolated from chickens, but can be found in the intestinal tract of a wide variety of wild or domesticated animals such as healthy cattle, birds and even flies.

Surveys in several countries have shown that *C. jejuni* is the leading cause of bacterial diarrheal illness. Although Campylobacteriosis is self-limiting and complications are uncommon, reactive arthritis may onset post-infection. Most outbreaks of Campylobacteriosis are due to foodborne illness associated with undercooked or raw poultry, however, *C. jejuni* can survive in water and contaminated (untreated) surface water has been implicated in a number of cases (Wallace, 1997). The infective dose of *C. jejuni* is considered to be small. Human feeding studies suggest that a dose of around 800 organisms may cause illness in some individuals (Black *et al*. 1988).

## Methods

The generic quantitative risk analysis model described in Appendix C was customised for *C. jejuni* by altering the following parameters (based on available information in the scientific literature):

- Pathogen prevalence in cattle and sheep
- Concentration of pathogen in faeces
- Dose-response relationship

The model was used to calculate the number of people ill / million exposed / days of exposure to airborne and waterborne *C. jejuni* sourced from a cattle and sheep abattoir. The volume of the groundwater supply was set to zero in the model, and hence the potential risk from exposure to contaminated groundwater was not simulated. Sensitivity analyses for a cattle abattoir scenario were performed for each output in order to determine which input parameters have the greatest influence on the estimated number of people ill.

### *Prevalence*

Input data on the prevalence of *C. jejuni* in cattle and sheep have been estimated by combining results from studies in Australia and the United Kingdom (Attachment 1). The constructed distributions for *C. jejuni* prevalence differ between cattle and sheep, with a much higher 'most likely' prevalence and broader range for cattle (Figure 1).

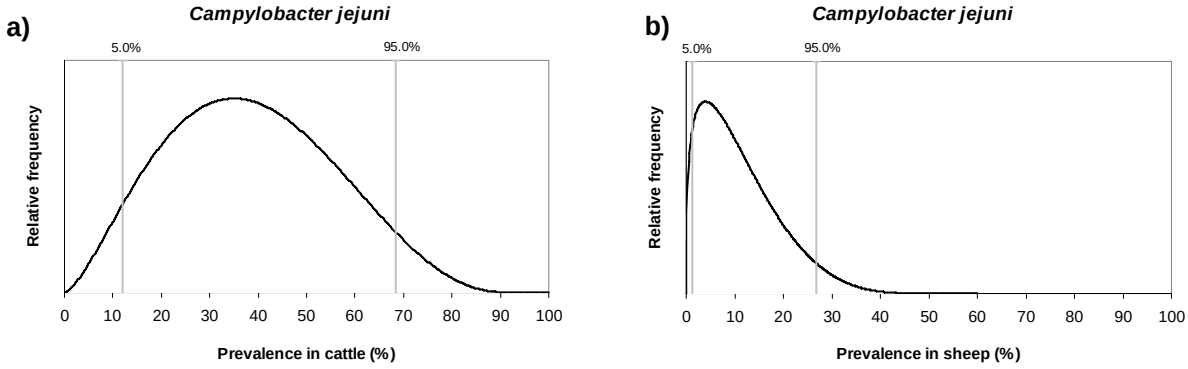



**Figure 1.** Distributions of the prevalence of *C. jejuni* in **a)** cattle and **b)** sheep used as inputs into the model.

## *Concentration*

The model assumes that *C. jejuni* is present in the gastro-intestinal system of infected cattle and sheep. The concentration of the organism in the faeces is assumed to be representative of the concentration in the entire gastro-intestinal tract. Input data on the concentration of *C. jejuni* in faeces have been estimated by combining results from studies in the United Kingdom (Attachment 1). The distributions for *C. jejuni* concentration differ between cattle and sheep, with a broader range but a lower 'most likely' concentration for cattle (Figure 2).

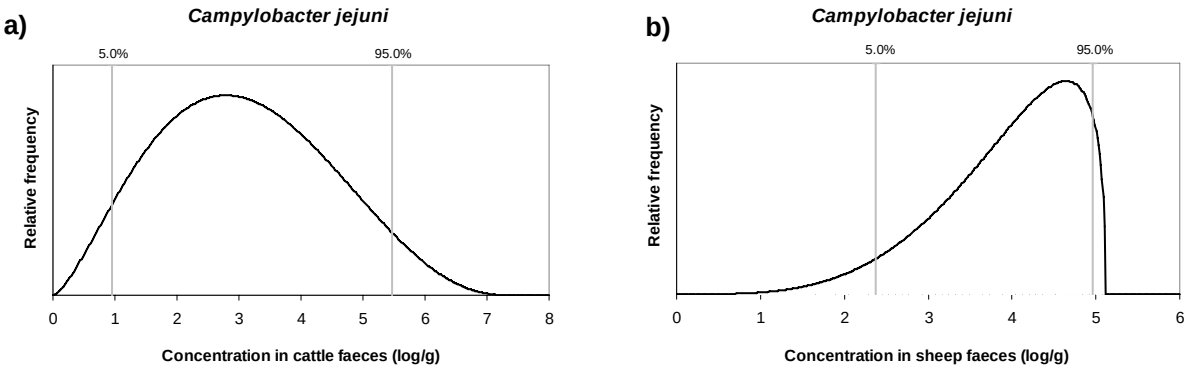



**Figure 2.** Distributions of the concentration of *C. jejuni* in **a)** cattle and **b)** sheep used as inputs into the model.

## *Dose response*

The dose response curve was derived from human studies for ingestion of *C. jejuni*. Available literature describes the human dose response curves as Weibull-Gamma and Beta Poisson distributions, where the probability of infection depends on the dose, *D*, and the dose-response parameters $\varepsilon$ and $\beta$. The Beta Poisson distribution is equivalent to the Weibull-Gamma distribution where the chi ($\chi$) parameter is equal to 1. The Weibull-Gamma dose-response equation has the form:

$$\text{Probability of infection} = 1 - [(1 + (\text{dose}^{\chi}/\beta))]^{-\varepsilon}$$

By varying the beta ($\beta$) parameter of the Weibull-Gamma distribution, we captured the full range of reported dose responses in the literature, within the range of pathogen ingestion/inhalation calculated by the model. The slope of the resulting dose response curve varies with $\beta$, which is simulated by a uniform distribution (Attachment 1). Depending on the $\beta$ value simulated, the dose response curve lies between the upper and lower curves shown in Figure 3. The dose range simulated by the model was low, with the $95^{th}$ percentile of approximately 5 microorganisms potentially inhaled in spray drift from irrigation water.

The model assumes that humans can be infected by *C. jejuni* through ingestion or inhalation of the pathogen and that the dose response is the same for both modes of infection. Dose-response studies are generally performed at high doses. Hence extrapolation from high doses studied in the literature to low doses simulated in this model may result in inaccurate estimates of the dose response.

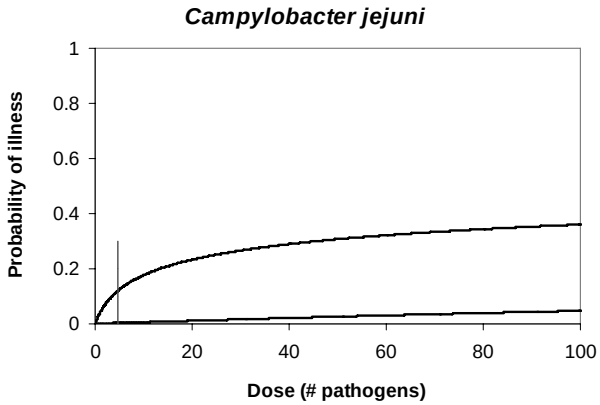



**Figure 3.** Dose response relationship for *Campylobacter jejuni*. The vertical line represents the 95$^{th}$ percentile pathogen dose for spray drift, simulated by the model for a cattle abattoir scenario. The upper and lower dose response curves represent the range of dose responses used in the model created by varying the $\beta$ parameter.

## Results

The model was run separately for a hypothetical cattle (1000/day) and a hypothetical sheep (3000/day) abattoir. Stabilisation of the model outputs took 4400 iterations for the cattle scenario and 3300 iterations for the sheep scenario.

Simulated input values for the three parameters unique to the *C. jejuni* model are shown in Table 1. The simulated outputs are given in Table 2 and illustrated graphically in Figures 4 and 5.

**Table 1.** Simulated inputs for the *Campylobacter jejuni* model from Figures 1, 2 and 3

| | **Prevalence of pathogen (%)** | | **Concentration of pathogen in faeces (log/g)** | | **Dose response parameter** | |
|---|---|---|---|---|---|---|
| | Cattle | Sheep | Cattle | Sheep | Cattle | Sheep |
| Minimum | 0.93 | 0.01 | 0.17 | 0.69 | 2.51 | 2.48 |
| 5$^{th}$ percentile | 12.31 | 1.27 | 0.92 | 2.39 | 12.45 | 12.17 |
| 50$^{th}$ percentile | 37.56 | 9.51 | 3.06 | 4.06 | 101.41 | 100.92 |
| 95$^{th}$ percentile | 68.52 | 26.75 | 5.44 | 4.94 | 189.58 | 190.76 |
| Maximum | 88.86 | 42.49 | 7.12 | 5.11 | 199.92 | 199.94 |

**Table 2.** Summary of simulated outputs: number of people ill / million exposed / days of exposure to airborne and waterborne *Campylobacter jejuni* from **a)** a cattle abattoir (1000 cattle) and **b)** a sheep abattoir (3000 sheep). See Figures 4 and 5 for graphical representation of results.

**a)**

**CATTLE**

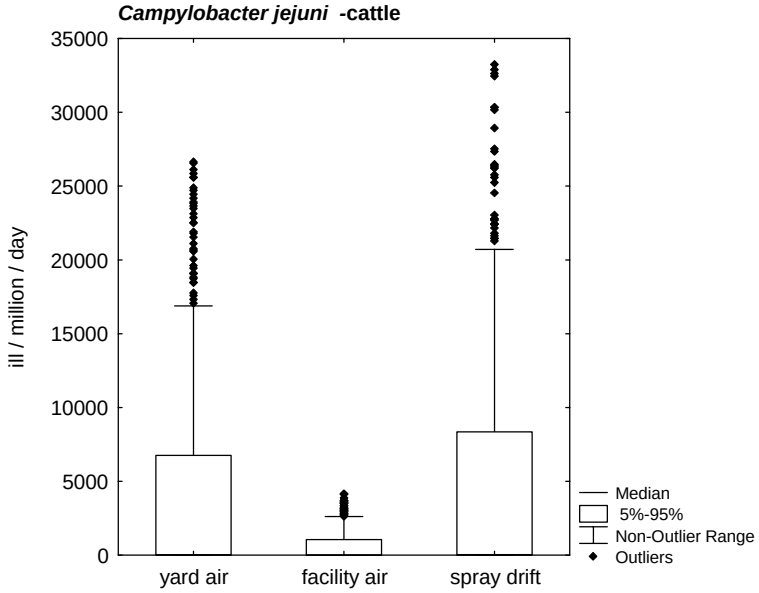
| Exposure route | Yard air | Facility air | Spray drift | Drinking water | Recreational water |
|---|---|---|---|---|---|
| Minimum | <1 | <1 | <1 | <1 | <1 |
| 5th percentile | <1 | <1 | <1 | <1 | <1 |
| 50th percentile | 21 | 3 | 27 | 0.0004 | 0.0034 |
| 95th percentile | 6736 | 1050 | 8345 | 0.1 | 1.1 |
| Maximum | 267385 | 117405 | 282196 | 31 | 190 |

**b)**

**SHEEP**

| Exposure route | Yard air | Facility air | Spray drift | Drinking water | Recreational water |
|---|---|---|---|---|---|
| Minimum | <1 | <1 | <1 | <1 | <1 |
| 5th percentile | <1 | <1 | <1 | <1 | <1 |
| 50th percentile | 14 | 2 | 19 | 0.0003 | 0.0023 |
| 95th percentile | 424 | 63 | 513 | 0.01 | 0.06 |
| Maximum | 13854 | 1556 | 17518 | 0.28 | 1.35 |

**a)**




**b)**

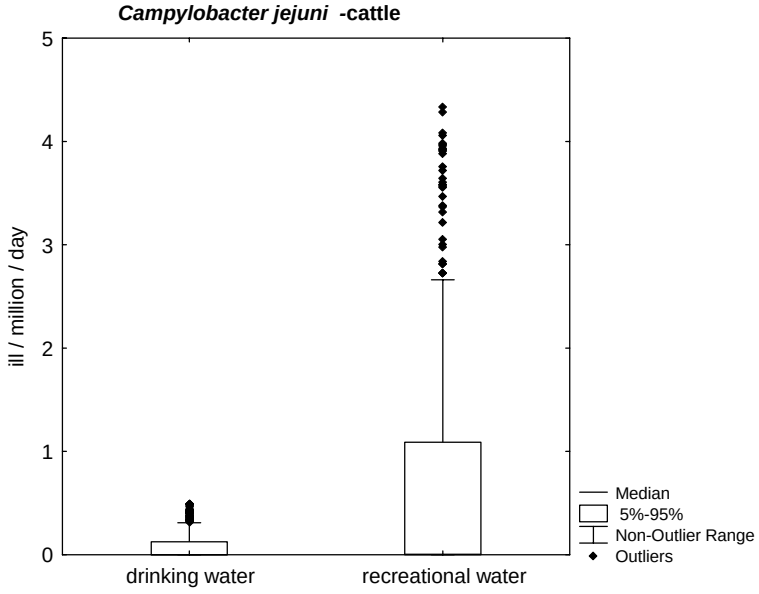
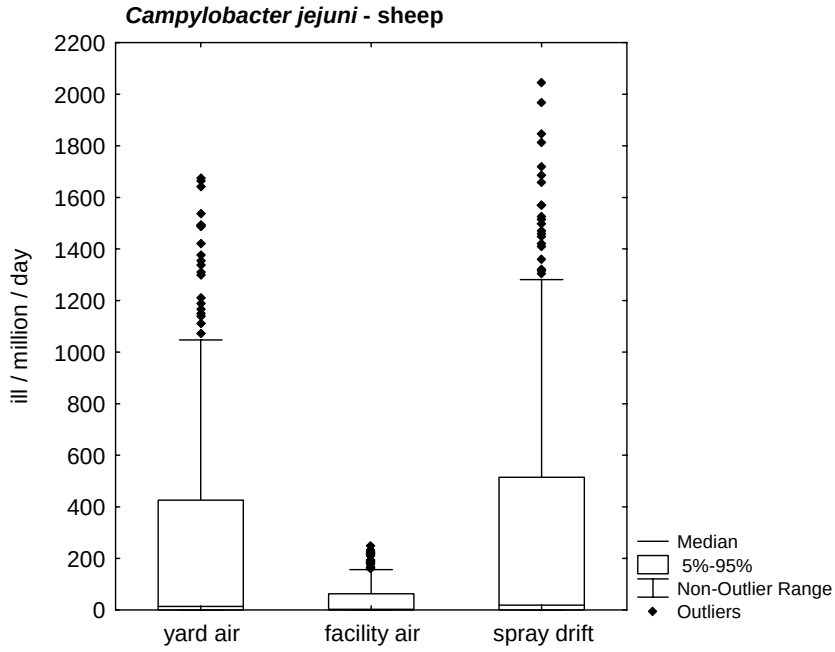



**Figure 4.** Number of people ill / million exposed / days of exposure to **a)** airborne, and **b)** waterborne *Campylobacter jejuni* from a cattle abattoir (1000 cattle). Refer to Table 2 for numerical values.

**a)**




**b)**

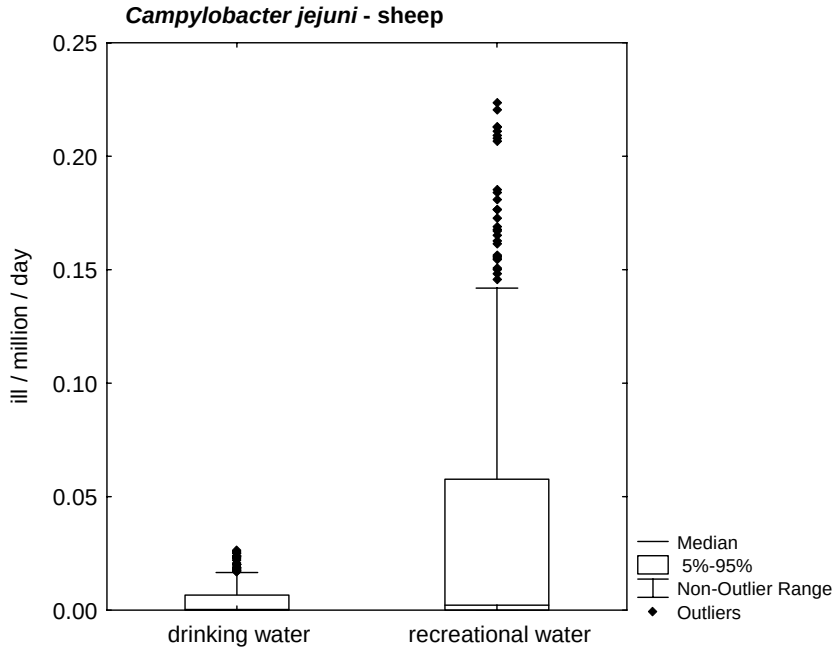



**Figure 5.** Number of people ill / million exposed / days of exposure to **a)** airborne, and **b)** waterborne *Campylobacter jejuni* from a sheep abattoir (3000 sheep). Refer to Table 2 for numerical values.

# Discussion

## *Relative risk of exposure pathways*

The results from the cattle (1000 cattle) and sheep (3000 sheep) abattoir scenarios (Table 2; Fig. 4, 5) suggest that the risk of human illness from *C. jejuni* is greatest for airborne exposure and least for waterborne exposure to the pathogen. For both cattle and sheep pathogen sources, the relative ranking of the 5 modelled exposure pathways is:

- Spray drift irrigation — *higher risk*
- Yard air
- Facility air
- Recreational water
- Drinking water — *lower risk*

For the three pathways of airborne exposure, contaminated spray drift would be expected to have the highest risk as the wastewater stream (after processing and treatment) has a higher pathogen load than the contaminated air released from the livestock yard or the air stream after processing and treatment in the abattoir (see total pathogen output in Appendix C). For waterborne exposure, contaminated drinking water would be expected to be lower risk than recreational water based on the assumption in the model that drinking water, unlike recreational water, undergoes a treatment step before possible ingestion.

## *Scale of the risk*

According to the model, if one million people per day were exposed to *C. jejuni* via contaminated spray drift from a cattle abattoir, a median of 27 people would be infected with enough pathogens to cause illness. This compares with 19 people infected for *C. jejuni* sourced from sheep. The median risk of human illness from exposure to contaminated air from the livestock yard is slightly lower at 21 people/day for a cattle abattoir and 14 people for a sheep abattoir. We note that there are no documented cases of human illness from exposure to *C. jejuni* via the inhalation route. For both waterborne pathways, the median estimates of risk are less than 1 person/day for the cattle and sheep abattoir scenarios. However, the maximum estimate indicates that for a cattle abattoir, approximately 190 people/day could be infected by *C. jejuni* from recreational water and 31 people from drinking water.

The median risk of exposure from a 1000 cattle/day abattoir is only slightly higher than from a 3000 sheep/day abattoir for all airborne and waterborne pathways, despite the far greater 'most likely' prevalence of *C. jejuni* in the faeces of cattle compared to sheep. This is consistent with the sensitivity analyses (Attachment 2), which indicate that the model is not very sensitive to pathogen prevalence. In addition, the median concentration of *C. jejuni* in faeces is higher in sheep than cattle, which probably shifts the relative risks such that there is only a small difference between the cattle and sheep abattoir results.

The estimated risk of illness from airborne and waterborne exposure to *C. jejuni* has a wide distribution (Fig. 4, 5). This is due to uncertainties in the input parameters such as pathogen prevalence, concentration in faeces and dose response. Sensitivity analyses for the cattle abattoir scenario (Attachment 2) indicate that each output in the model is highly sensitive to the concentration of the pathogen in cattle faeces. This means that the higher the concentration of *C. jejuni* in cattle faeces, the greater is the risk of illness from airborne or waterborne exposure.

The dose response curve and prevalence of the pathogen are the next most sensitive parameters. In the case of airborne exposure routes, the wind speed is also an important parameter in influencing the final outputs. This parameter has a negative correlation to the risk of illness because the volume of air potentially carrying pathogens is a function of the wind speed. The larger the volume, the more dilute is the effective concentration of the pathogen in the breathing volume of air. This means there is a lower risk of exposure to the pathogen.

## *Conclusion*

Given the sensitivity of the outputs to the model parameters, especially to the concentration of *C. jejuni* in faeces, the illness estimates generated by the model will be subject to a large amount of uncertainty

because the inputs themselves are uncertain. Although we have attempted to deal with this explicitly through a stochastic simulation approach, there is no guarantee that we have captured the full range of uncertainties. Further information on, say, dose response might reduce uncertainty or increase uncertainty by revealing a greater range of responses than previously documented. The results generated by the model, especially the absolute values, should therefore be interpreted with caution. Greater confidence can be placed in relative rankings, but even these could change as a result of different characteristics of individual abattoirs. The results of this 'generic abattoir' model should be used to identify where more information is needed and guide more specific investigations for individual abattoirs. We also note that the available input data for pathogen prevalence and concentration in animal faeces is not based solely on Australian studies.

In order to refine the estimates of risk for human exposure to *Campylobacter jejuni*, the model would require:

- Expert opinion on whether *C. jejuni* can be transmitted and survive in an airborne state
- Expert opinion on whether *C. jejuni* can cause human illness via inhalation exposure, and if so, information on the dose response curve
- Australian data on pathogen prevalence in sheep and the concentration in faeces of cattle and sheep
- A better understanding of the dose response at low doses.

# Attachment 1:

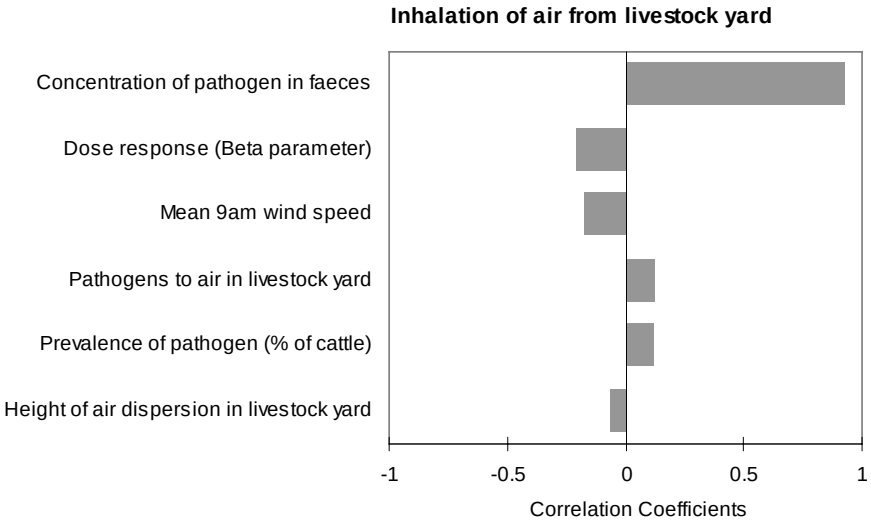
**Table A1.** Input data used to simulate *Campylobacter jejuni* load and dose response for pathogens entering and leaving an abattoir

| ***Campylobacter jejuni*** | **Distribution** | | | | **Source of data** | **Notes on data** |
|---|---|---|---|---|---|---|
| | | min | most likely | max | | |
| Prevalence of pathogen carrier cattle (%) | PERT | 0 | 35 | 92 | Unpublished Appendix to Vanselow & Hornitzky (2001) | Minimum, mean and maximum of 4 feedlot and 4 pasture properties in NSW and SE QLD, 25 cattle at each (faecal samples). |
| Faeces contamination in carrier cattle (pathogens log/g) | PERT | 0 | 2.79 | 7.38 | Stanley *et al.* (1998a) | Data for UK beef cattle (small intestine) |
| Prevalence of pathogen carrier sheep (%) | PERT | 0 | 4 | 51 | 1. Vanselow & Hornitzky (2001); 2. Jones *et al.* (1999); 3. Stanley *et al.* (1998b) | Most likely value set as the mean from 3 mutton sheep and 2 prime lamb properties in NSW, 25 sheep faecal samples at each (1). Data for UK sheep (2,3) to set the range (faecal samples). |
| Faeces contamination in carrier sheep (pathogens log/g) | PERT | 0 | 4.64 | 5.11 | Jones *et al.* 1999 | We used data for UK sheep (faecal samples) to set the range. The most likely was derived from average MPN in sheep faeces. |
| Weibull-Gamma dose response | | $\varepsilon$ | $\beta$ | $\chi$ | van Gerwen *et al.* (2000), Holcomb *et al.* (1999), Teunis *et al.* (1996), Medma *et al.* (1996), Rose & Gerba (1991), Black *et al.* (1988) | We altered the Beta parameter of the Weibull-Gamma dose response model to capture the full range of 4 reported human dose response curves within the range of pathogen loads calculated by the model. |
| | *fixed parameters* | 0.12 | | | | |
| | UNIFORM | min<br>max | 2.46<br>200 | 1 | | |

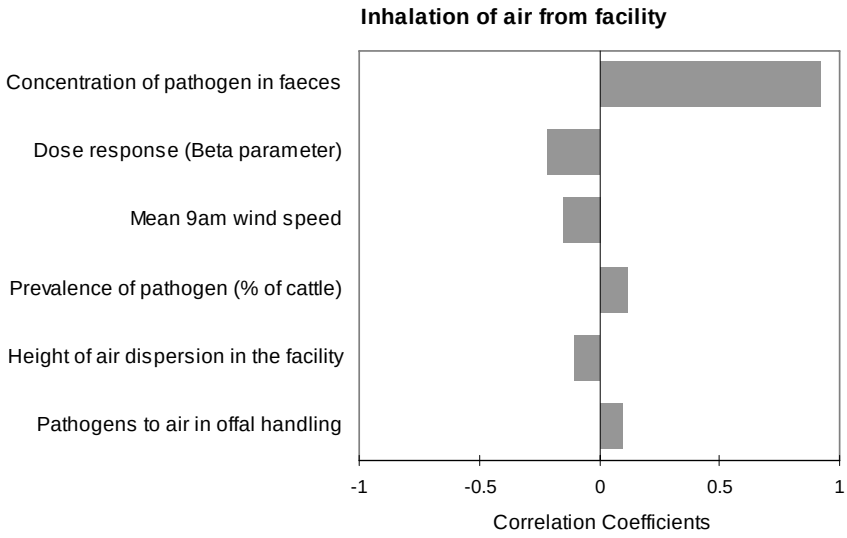
# Attachment 2:

**Figure A1.** Sensitivity analyses for number ill / million exposed / days exposed to airborne and waterborne *Campylobacter jejuni* based on a cattle abattoir scenario. **a)** inhalation of air from livestock yard; **b)** inhalation of air from the facility (abattoir); **c)** inhalation of spray drift; **d)** ingestion of drinking water; **e)** ingestion of recreational water. Sensitivities are only shown for parameters where the correlation value is greater than or equal to +/- 0.05.

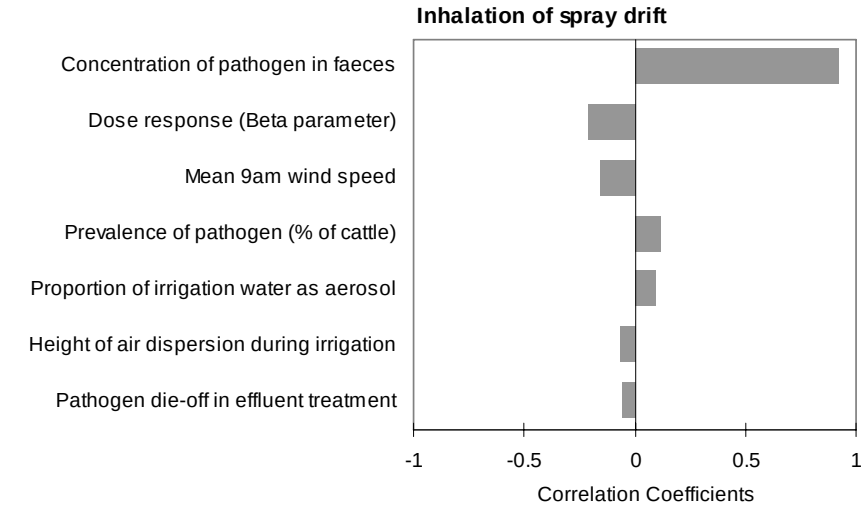
**a)**



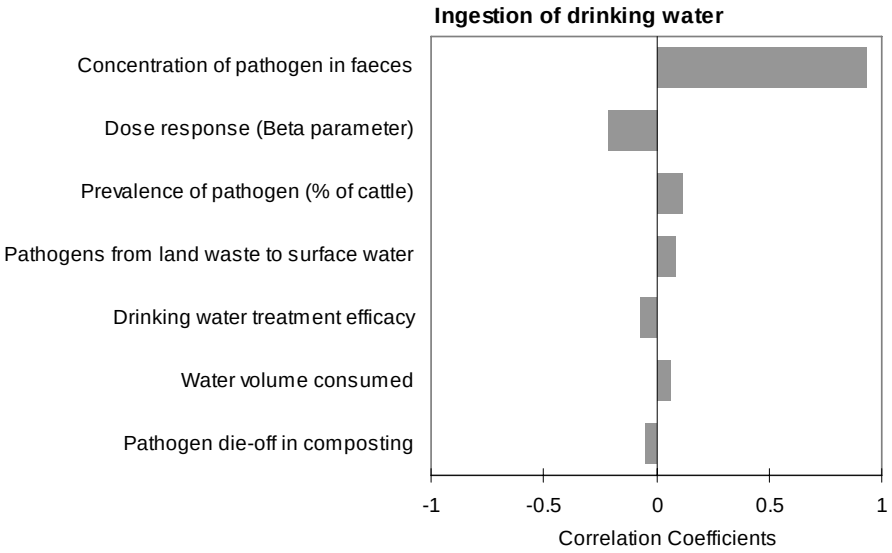

**b)**



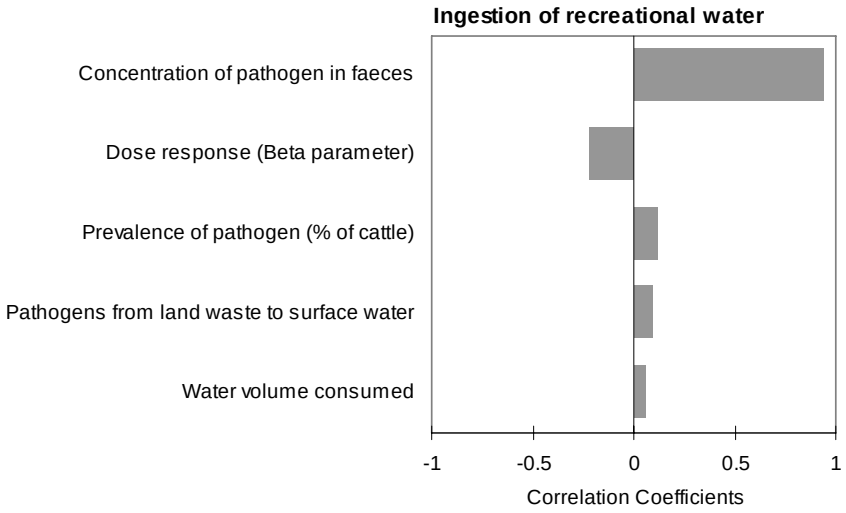
**c)**


Inhalation of spray drift
Concentration of pathogen in faeces
Dose response (Beta parameter)
Mean 9am wind speed
Prevalence of pathogen (% of cattle)
Proportion of irrigation water as aerosol
Height of air dispersion during irrigation
Pathogen die-off in effluent treatment
-1
-0.5
0
0.5
1
Correlation Coefficients

**d)**


Ingestion of drinking water
Concentration of pathogen in faeces
Dose response (Beta parameter)
Prevalence of pathogen (% of cattle)
Pathogens from land waste to surface water
Drinking water treatment efficacy
Water volume consumed
Pathogen die-off in composting
-1
-0.5
0
0.5
1
Correlation Coefficients

**e)**


Ingestion of recreational water
Concentration of pathogen in faeces
Dose response (Beta parameter)
Prevalence of pathogen (% of cattle)
Pathogens from land waste to surface water
Water volume consumed
-1
-0.5
0
0.5
1
Correlation Coefficients

# APPENDIX E. QUANTITATIVE RISK ASSESSMENT OF *COXIELLA BURNETTI* EMISSIONS FROM ABATTOIRS

Infection with the rickettsia *Coxiella burnetii* (*C. burnetii*) occurs in a wide range of wild and domestic animals without the animal itself displaying apparent signs of infection. In humans, the pathogen is responsible for the disease Q fever. The disease has flu-like symptoms that generally last 7-10 days, however approximately 20% of cases result in chronic disease (Cole *et al.* 1999). The organism is found worldwide and is common in Australia, but incidence of the disease is low except in certain occupational groups. In Australia, Q fever is considered primarily an occupational disease of people working with live and slaughtered cattle, sheep, goats and kangaroos in the livestock and meat industries (MRC, 1997). The risk of infection is substantially increased if humans are exposed to these animals during parturition (Welsh *et al*. 1958).

Humans can become infected with *C. burnetii* by inhalation of the organism in fine mist, small droplets or dust contaminated by the placenta, birth fluids, faeces or urine of infected animals. Indirect human exposure can occur if small droplets carrying the bacteria are dispersed in the air and disseminated at a distance from the source before settling and drying to form a highly infectious dust. Moving animals in the yards, pens or holding paddocks may also agitate contaminated surfaces and raise infective dusts. Infected dust on the ground, attached to buildings, machinery, stock transport vehicles, straw, wool, hides or work clothing may be blown (possibly for a kilometre or more) in dry and windy weather or transported on the above-mentioned materials and later released into the air, hence exposing individuals outside of the recognised risk environments to infection (MRC, 1997).

Contaminated aerosols, whether infected dust or droplets, are considered extremely infectious; a single organism of *C. burnetii* can cause human infection. Although the organism is unable to grow or replicate outside host cells, the spore-like form of the organism is extremely resistant to heat, pressure, desiccation and many standard antiseptic compounds. This allows *C. burnetii* to persist in the environment for long periods (weeks or months) under harsh conditions. As the organism can endure harsh conditions for many months in a dried state, the dust is a constant and often hidden source of infection.

## Methods

The generic quantitative risk analysis model described in Appendix C was customised for *Coxiella burnetii* by altering the following parameters (based on available information in the scientific literature):

- Pathogen prevalence in sheep and cattle
- Concentration of pathogen in faeces
- Concentration of pathogen in placenta
- Proportion of cattle and sheep shedding pathogen in placenta
- Other sources volume (placenta) in cattle and sheep
- Dose-response relationship

The model was used to calculate the number of people ill / million exposed / days of exposure to airborne *C. burnetii* sourced from a sheep and cattle abattoir. Human illness by ingestion of contaminated water was considered improbable (pers com. Anon.) and hence excluded from the model. However, inhalation of water contaminated with *C. burnetii* was incorporated into the model through exposure to spray-irrigated wastewater. The volume of the groundwater supply was set to zero in the model, and hence the potential risk from exposure to contaminated groundwater was not simulated. Sensitivity analyses for a sheep abattoir scenario (sheep data are more complete than for cattle) were performed for each output in order to determine which input parameters have the greatest influence on the estimated number of people ill.

## *Prevalence & Concentration*

The model assumes that *Coxiella burnetii* is present in the gastro-intestinal system and placenta of infected cattle and sheep. The concentration of the organism in the faeces of an infected animal is assumed to be representative of the concentration in the entire gastro-intestinal tract. Literature on *C. burnetii* prevalence and levels in animals is very old, dating back the 1950's, and is not comprehensive. Input data estimates on the prevalence of the pathogen and concentrations in the two carrier sources have been developed by combining results published in the literature with expert opinion (Attachment 1).

Serologic prevalence of *C. burnetii* was estimated by averaging published data from California and Australia. To convert the serologic prevalence (based on the presence of *C. burnetii* antibodies) to the number of animals actually excreting the organism, the simulated value for serologic prevalence was multiplied by 0.02 (i.e. 2% excretion rate; Stoker *et al*, 1955). Concentrations of the organism in faeces and placenta (reported in hamster or guinea pig infective doses) were only available for naturally infected sheep. In the absence of specific information on cattle, the same values have been used for cattle.

The model assumes that a flock of sheep or herd of cattle entering the abattoir is comprised of 50% females. The serologic prevalence and concentration of *C. burnetii* in faeces are assumed to be the same in both male and female animals, as is the probability of the organism being excreted/isolated from the faeces. Only a proportion of the female animals for slaughter would be expected to be pregnant and thus potentially harbour the pathogen in the placenta. This percentage of pregnant females is represented as a new distribution in the model (Appendix C, module A), which is further multiplied by 0.5 to give the proportion of cattle/sheep excreting *C. burnetii* in the placenta. In the case of sheep, this number is multiplied by an additional 0.5 on the assumption that 50% of slaughtered sheep are lambs and therefore could not be pregnant.

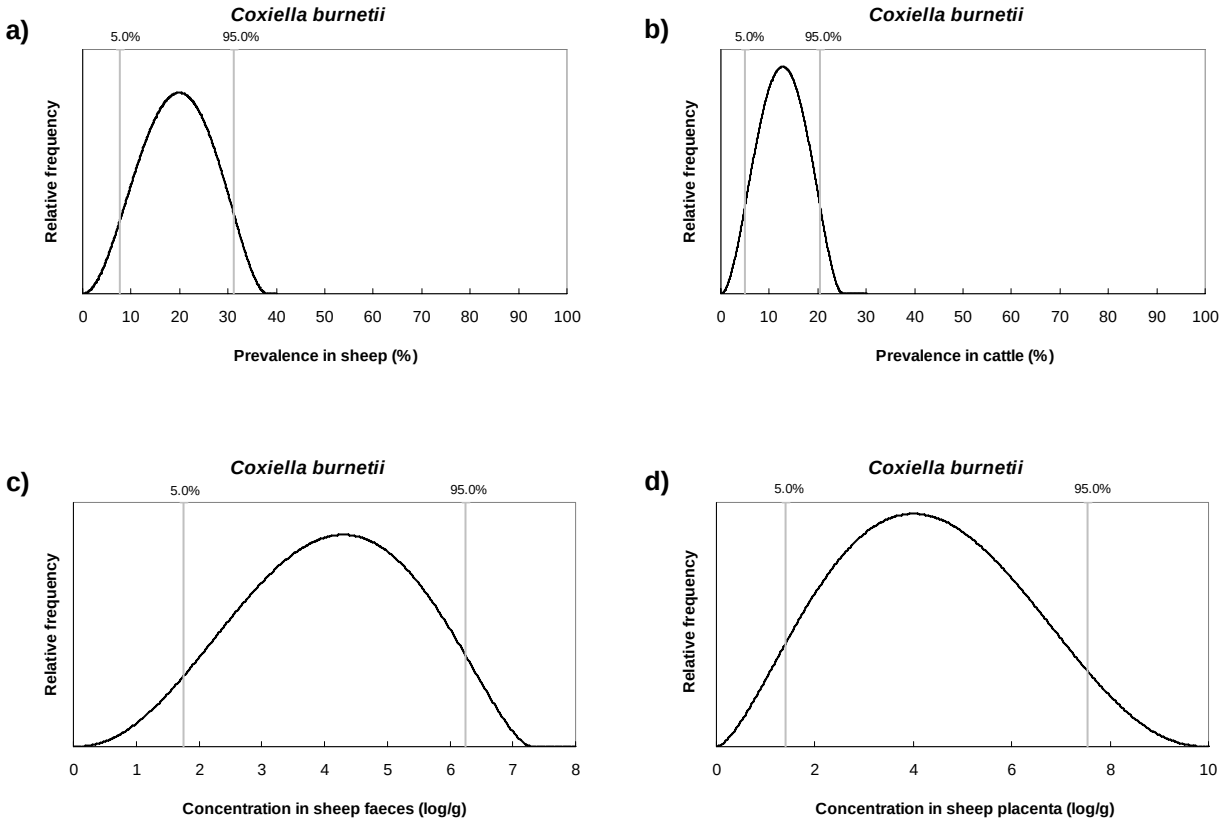



**Figure 1.** Distributions of the prevalence of *Coxiella burnetii* in **a)** sheep and **b)** cattle and the concentration of *C. burnetii* in **c)** sheep faeces and **d)** sheep placenta used as inputs into the model. In the absence of cattle-specific data, concentrations of *C. burnetii* in cattle faeces and placenta are assumed to be the same as those in sheep.

### *Dose response*

Published literature on human dose response to *C. burnetii* is very limited. Available information was obtained from studies on humans exposed by aerosol to graded doses of *C. burnetii* suspended in whole egg slurry (Tigertt and Benenson, 1956). Exposure units are given in guinea pig units, where one exposure unit is defined as the quantity of aerosolised *C. burnetii* producing infection in 50% of exposed guinea pigs (i.e. $ID_{50}$). The dose-to-incubation relationship in guinea pigs infected by the respiratory route is considered similar to that of humans (Williams, 1991) and is thus a reasonable surrogate. Based on clinical disease in humans the results from low dose exposure were used to construct a linear dose response curve, within the range of pathogen inhalation calculated by the model. The dose-response equation has the form:

Probability of infection = dose * slope parameter

where the slope of the curve is simulated by a uniform distribution (Attachment 1). Depending on the simulated slope variable, the dose response curve lies between the upper and lower curves shown in Figure 2. The dose range simulated by the model was low, with the 95$^{th}$ percentile of less than one microorganism potentially inhaled in spray drift from irrigation water.

The model assumes that humans can be infected only by inhalation of the pathogen through a dust or droplet medium. Dose-response studies are generally performed at high doses. Hence extrapolation from high doses studied in the literature to low doses simulated in this model may result in an inaccurate estimate of the dose response.

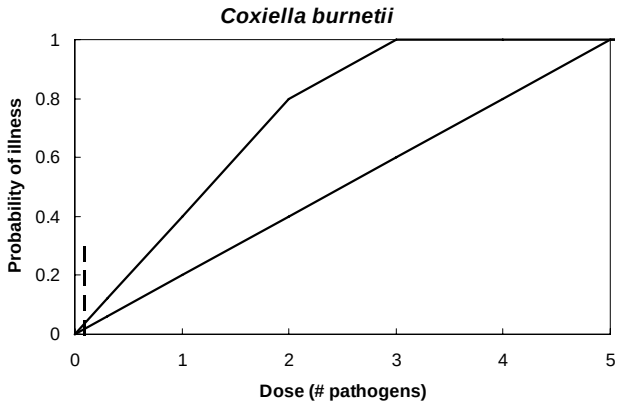



**Figure 2.** Dose response relationship for *Coxiella burnetii*. The vertical line represents the 95$^{th}$ percentile pathogen dose for spray drift, simulated by the model for a sheep abattoir scenario. The upper and lower dose response curves represent the range of dose responses used in the model created by varying the slope parameter.

## Results

The model was run separately for a hypothetical sheep (3000 sheep) and a hypothetical cattle (1000 cattle) abattoir. Stabilisation of the model outputs took 3800 iterations for the sheep scenario and 3500 iterations for the cattle scenario.

Simulated input values for four of the parameters unique to the *C. burnetii* model are shown in Table 1. The simulated outputs are given in Table 2 and illustrated graphically in Figure 3.

**Table 1.** Simulated inputs for the *Coxiella burnetii* model from Figures 1, 2

| | **Prevalence of pathogen (%)** | | **Concentration of pathogen in faeces (log/g)** | | **Concentration of pathogen in placenta (log/g)** | | **Dose response β parameter** | |
|---|---|---|---|---|---|---|---|---|
| | Cattle | Sheep | Cattle | Sheep | Cattle | Sheep | Cattle | Sheep |
| Minimum | 1.08 | 2.17 | 0.20 | 0.45 | 0.19 | 0.10 | 0.20 | 0.20 |
| 5$^{th}$ percentile | 4.71 | 7.89 | 1.78 | 1.69 | 1.37 | 1.32 | 0.21 | 0.21 |
| 50$^{th}$ percentile | 12.62 | 19.81 | 4.12 | 4.11 | 4.31 | 4.24 | 0.30 | 0.30 |
| 95$^{th}$ percentile | 20.80 | 31.58 | 6.27 | 6.22 | 7.50 | 7.53 | 0.39 | 0.39 |
| Maximum | 24.94 | 37.17 | 7.19 | 7.27 | 9.27 | 9.77 | 0.40 | 0.40 |

**Table 2.** Summary of simulated outputs: number of people ill / million exposed / days of exposure to airborne *Coxiella burnetii* from **a)** a sheep abattoir (3000 sheep) and **b)** a cattle abattoir (1000 cattle). See Figure 3 for graphical representation of results

**a)**

**SHEEP**

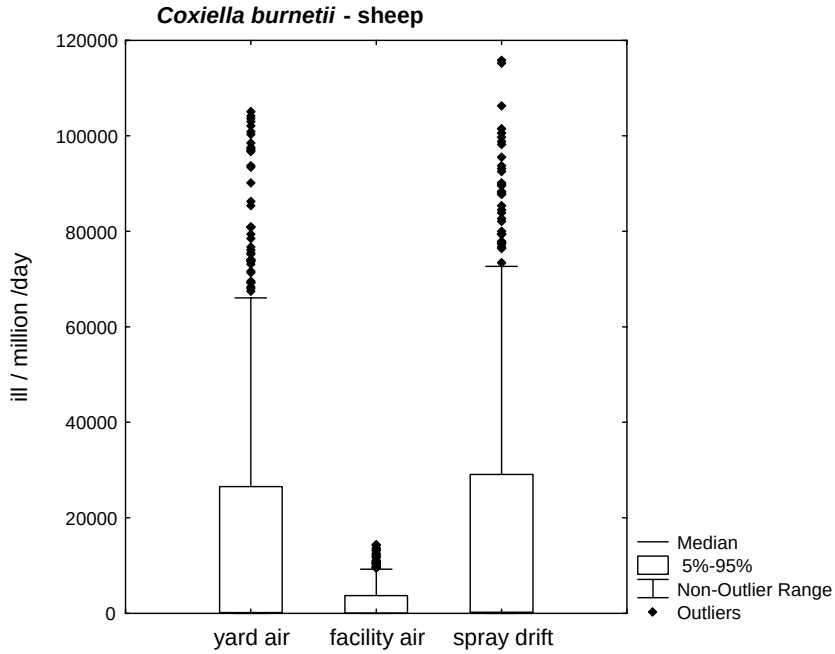
| **Exposure route** | **Yard air** | **Facility air** | **Spray drift** |
|---|---|---|---|
| Minimum | <1 | <1 | <1 |
| 5$^{th}$ percentile | 1 | <1 | 1 |
| 50$^{th}$ percentile | 174 | 29 | 239 |
| 95$^{th}$ percentile | 26481 | 3687 | 28996 |
| Maximum | 546146 | 143882 | 1000000 |

**b)**

**CATTLE**

| **Exposure route** | **Yard air** | **Facility air** | **Spray drift** |
|---|---|---|---|
| Minimum | <1 | <1 | <1 |
| 5$^{th}$ percentile | 3 | <1 | 3 |
| 50$^{th}$ percentile | 468 | 70 | 594 |
| 95$^{th}$ percentile | 54471 | 7164 | 72651 |
| Maximum | 1000000 | 395953 | 1000000 |

**a)**




**b)**

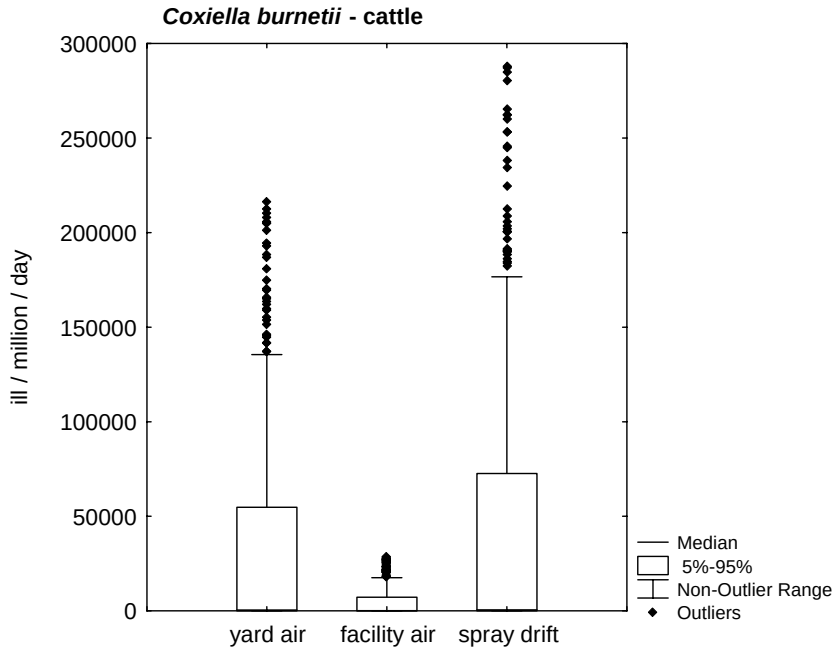



**Figure 3.** Number of people ill / million exposed / days of exposure to airborne *Coxiella burnetii* from **a)** a sheep abattoir (3000 sheep) and **b)** a cattle abattoir (1000 cattle). Refer to Table 2 for numerical values.

# Discussion

## *Relative risk of exposure pathways*

The results from the sheep (3000 sheep) and cattle (1000 cattle) abattoir scenarios (Table 2; Figure 3) suggest that the risk of human illness from *C. burnetii* is greatest for inhalation exposure to the pathogen in spray drift irrigation. For both sheep and cattle pathogen sources, the relative ranking of the 3 modelled exposure pathways is:

- Spray drift irrigation — *higher risk*
- Yard air
- Facility air — *lower risk*

For the three pathways of airborne exposure, contaminated spray drift would be expected to have the highest risk as the wastewater stream (after processing and treatment) has a higher pathogen load than the contaminated air released from the livestock yard or the air stream after processing and treatment in the abattoir (see total pathogen output in Appendix C).

## *Scale of the risk*

According to the model, if one million people/day were exposed to *C. burnetii* via contaminated spray drift from a sheep abattoir, a median of 239 people/day would be infected with enough pathogens to cause illness. This compares with a median of 594 people infected for *C. burnetii* sourced from cattle. The estimated median risk of human illness from exposure to contaminated air from the livestock yard is slightly lower at 174 people/day for a sheep abattoir and 468 people for a cattle abattoir.

Although the 'most likely' median prevalence of *C. burnetii* is higher in sheep than cattle (Table 1), the median number of ill is more than double for the cattle abattoir scenario for all exposure pathways modelled. This is consistent with the sensitivity analyses (Attachment 2), which indicate that the model is not very sensitive to pathogen prevalence. However, the difference between the cattle and sheep abattoir results is probably related to the volume of the gastro-intestinal system and the placenta, and hence the amount of contaminated faecal/placental material, which are much higher in cattle than sheep. In addition, the model assumes that the proportion of cattle shedding the organism in the placenta (50%) is higher for a generic cattle population entering the abattoir than for a sheep population (25%).

The estimated risk of illness from airborne exposure to *C. burnetii* has a wide distribution (Fig. 3). This is due to uncertainties in the input parameters such as pathogen prevalence, concentration in faeces and placenta and dose response. Sensitivity analyses from the sheep abattoir scenario (Attachment 2) indicate that each output in the model is highly sensitive to the concentration of the pathogen in sheep faeces. This means that the higher the concentration of *C. burnetii* in sheep faeces, the greater the risk of illness from airborne exposure. The wind speed and prevalence of the pathogen are the next most important parameters in influencing the final outputs. The wind speed has a negative correlation to the risk of illness because the volume of air potentially carrying pathogens is a function of the wind speed. The larger the volume, the more dilute is the effective concentration of the pathogen in the breathing volume of air. This means there is a lower risk of exposure to the pathogen.

In comparison to the sheep scenario, sensitivity analyses for the cattle scenario (not shown) indicate that after the concentration of *C. burnetii* in cattle faeces and wind speed, the concentration of the organism in the placenta is the next most significant parameter in relation to the number of ill. The concentration in placenta probably has a greater influence in the cattle versus sheep scenario because of the potentially greater proportion of pregnant female cattle than female sheep, as discussed above. Importantly, the model indicates that in both the sheep and cattle scenarios, the slope of the dose response curve is not one of the most important parameters. This suggests that in refining the model, constraining uncertainties in the other parameters is more important.

## *Conclusion*

Given the sensitivity of the outputs to the model parameters, especially to the concentration of *C. burnetii* in faeces, the illness estimates generated by the model will be subject to a large amount of uncertainty because the inputs themselves are uncertain. Although we have attempted to deal with this explicitly through a stochastic simulation approach, there is no guarantee that we have captured the full range of uncertainties. Further information on, say, dose response might reduce uncertainty or increase uncertainty by revealing a greater range of responses than previously documented. The results generated by the model, especially the absolute values, should therefore be interpreted with caution. Greater confidence can be placed in relative rankings, but even these could change as a result of different characteristics of individual abattoirs. The results of this 'generic abattoir' model should be used to identify where more information is needed and guide more specific investigations for individual abattoirs. We note that there is a more complete dataset available for sheep than cattle and therefore in the absence of further data, the *Coxiella burnetti* model is perhaps more applicable to sheep abattoir situations.

In order to refine the estimates of risk for human exposure to *Coxiella burnetii*, the model would require:

- Australian data on pathogen prevalence, concentration in faeces and placenta in cattle
- Data on the human dose response curve for inhalation and a better understanding of the dose response at low doses.

# Attachment 1:

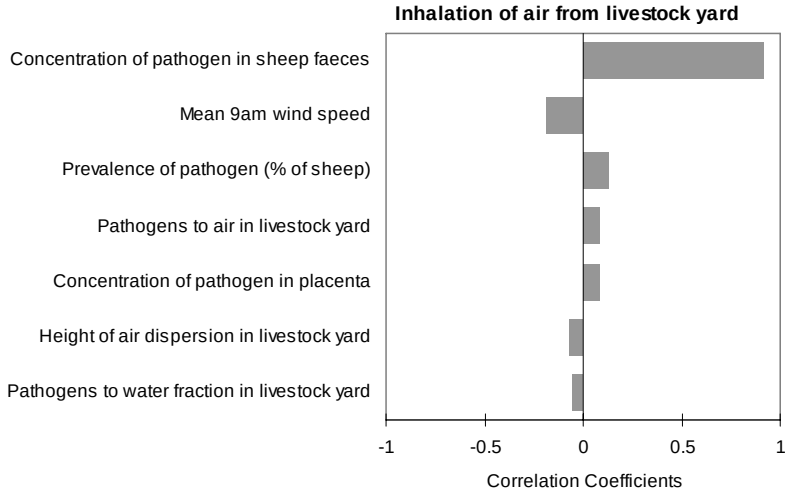
**Table A1.** Input data used to simulate *C. burnetii* load and dose response for pathogens entering and leaving an abattoir

| ***Coxiella burnetti*** | **Distribution** | | | | **Source of data** | **Notes on data** |
|---|---|---|---|---|---|---|
| | | min | most likely | max | | |
| Prevalence of pathogen carrier cattle (%) | PERT | 0 | 11.78 | 25.1 | Williams (1991), Lang (1990), Stoker *et al*. (1955) | Average of data from Australia and California to estimate mean and maximum values. Minimum estimated as zero. |
| Faeces contamination in carrier cattle (pathogens log/g) | PERT | 0 | 4.3 | 7.3 | Winn *et al.* (1953) | Assumed same as for sheep |
| Concentration in placenta of carrier cattle (pathogens log/g) | PERT | 0 | 4.0 | 10.0 | Abinanti *et al*. (1955), Anon pers com (2002) | Assumed same as for sheep |
| Proportion of cattle shedding pathogen in placenta | PERT | 0 | 1 | 4 | Estimate | Simulated value multiplied by 0.5 |
| Other sources (placenta) volume in cattle (kg/animal) | PERT | 4 | 6 | 8 | Kleeman *et al*. (2001) | Estimate based on placenta volume in sheep |
| Prevalence of pathogen carrier sheep (%) | PERT | 0 | 19.9 | 38.0 | Williams (1991), Lang (1990), Stoker *et al*. (1955) | Average of data from Australia and California to estimate mean and maximum values. Minimum estimated as zero. |
| Faeces contamination in carrier sheep (pathogens log/g) | PERT | 0 | 4.3 | 7.3 | Winn *et al*. (1953) | Minimum estimated as zero |
| Concentration in placenta of carrier cattle (pathogens log/g) | PERT | 0 | 4.0 | 10.0 | Abinanti *et al*. (1955), Anon pers com (2002) | Minimum estimated as zero |
| Proportion of sheep shedding pathogen in placenta | PERT | 0 | 1 | 4 | Estimate | Simulated value multiplied by 0.25 |
| Other sources (placenta) volume in sheep (kg/animal) | PERT | 4 | 6 | 8 | Kleeman *et al*. (2001) | Based on sheep studies |
| Linear dose response | UNIFORM | 0.2 | - | 0.4 | Tigertt & Benenson (1956) | We used the results from the low dose studies to construct a linear dose response curve. The most likely value is simulated by the model. |

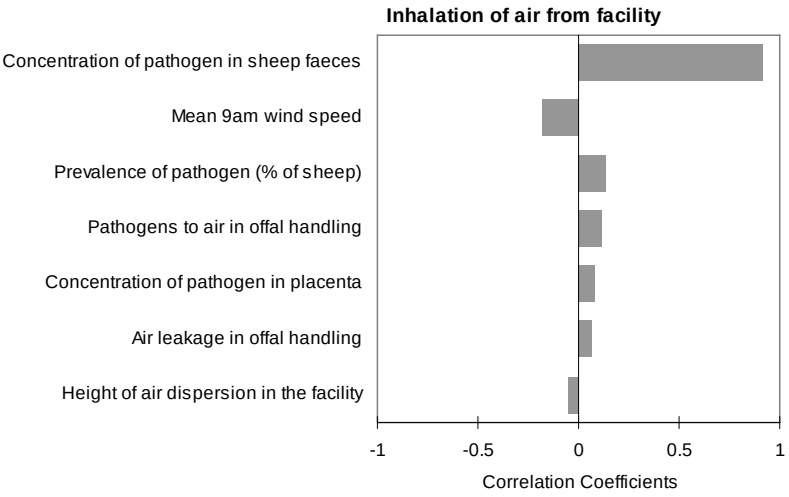
# Attachment 2:

**Figure A1.** Sensitivity analyses for number ill / million exposed / days exposed to airborne *Coxiella burnetii* based on a sheep abattoir scenario. **a)** inhalation of air from livestock yard; **b)** inhalation of air from the facility (abattoir); **c)** inhalation of spray drift. Sensitivities are only shown for parameters where the correlation value is greater than or equal to +/- 0.05.

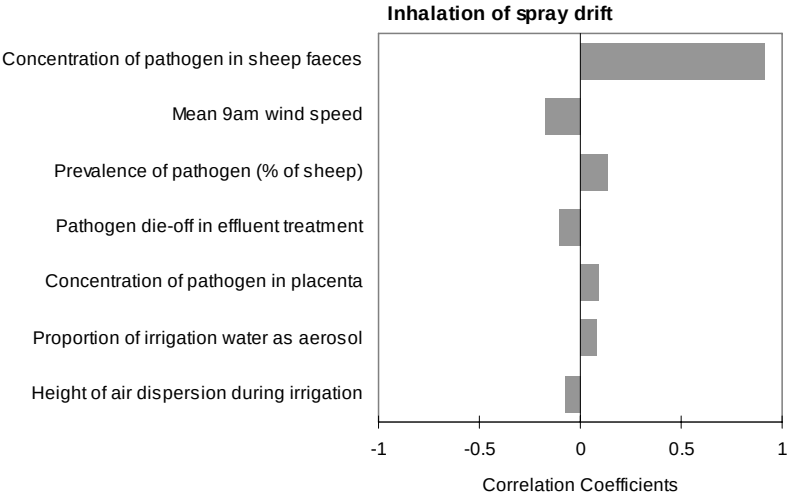
**a)**




**b)**




**c)**



# APPENDIX F. QUANTITATIVE RISK ASSESSMENT OF *ESCHERICHIA COLI* EMISSIONS FROM ABATTOIRS

*Escherichia coli* (*E. coli*) is the most common bacterium isolated from human faeces. There are many serotypes of *E. coli*, some of which are host adapted and do not typically cause disease, and others that cause a range of clinical diseases in both humans and livestock (Cole *et al*. 1999). Emerging strains belong to the enterohaemorrhagic *E. coli* (EHEC) group, of which the O157:H7 serotype is a typical example. While some strains of *E. coli* are asymptomatic, others cause gastroenteritis; infection with some strains of the EHEC group is associated with haemorrhagic colitis, haemolytic-uremic syndrome and even death in humans. The intestinal tract of ruminants, in particular cattle and sheep, is a major reservoir of *E. coli* O157:H7, although the microorganism is not associated with illness in these animals.

EHEC can spread through meat, other foods contaminated with animal faeces, and through environmental contamination. In addition to food-vehicles, outbreaks have also been associated with contaminated drinking water as well as recreational waters. EHEC strains of *E. coli* have a small infective dose (Desmarchelier and Grau, 1997).

## Methods

The generic quantitative risk analysis model described in Appendix C was customised for *E. coli* by altering the following parameters (based on available information in the scientific literature):

- Pathogen prevalence in sheep and cattle
- Concentration of pathogen in faeces
- Dose-response relationship

The model was used to calculate the number of people ill / million exposed / days of exposure to airborne and waterborne *E. coli* sourced from a cattle and sheep abattoir. The volume of the groundwater supply was set to zero in the model, and hence the potential risk from exposure to contaminated groundwater was not simulated. Sensitivity analyses for a cattle abattoir scenario (cattle data are more complete than for sheep) were performed for each output in order to determine which input parameters have the greatest influence on the estimated number of people ill.

### *Prevalence*

Input data on the prevalence of *E. coli* in cattle and sheep have been estimated by combining results from Australian studies (Attachment 1). The constructed distributions for *E. coli* prevalence are similar for cattle and sheep, although the 'most likely' prevalence is slightly higher for cattle (Figure 1).

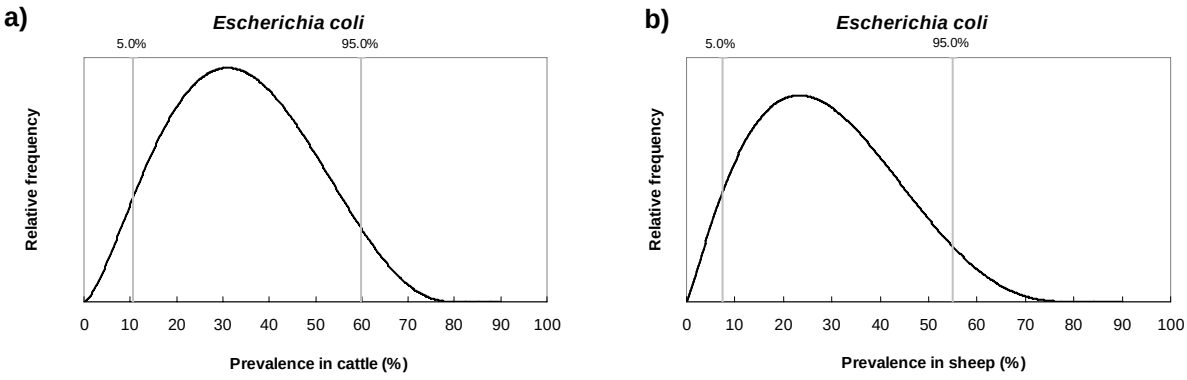



**Figure 1.** Distributions of the prevalence of *E. coli* in **a)** cattle and **b)** sheep used as inputs into the model.

## *Concentration*

The model assumes that *E. coli* is present in the gastro-intestinal system of infected cattle and sheep. The concentration of the organism in the faeces is assumed to be representative of the concentration in the entire gastro-intestinal tract. Input data on the concentration of *E. coli* in faeces have been estimated by combining results from studies in Australia and the United Kingdom (Attachment 1). The distributions for *E. coli* concentration in cattle and sheep are shown in Figure 2.

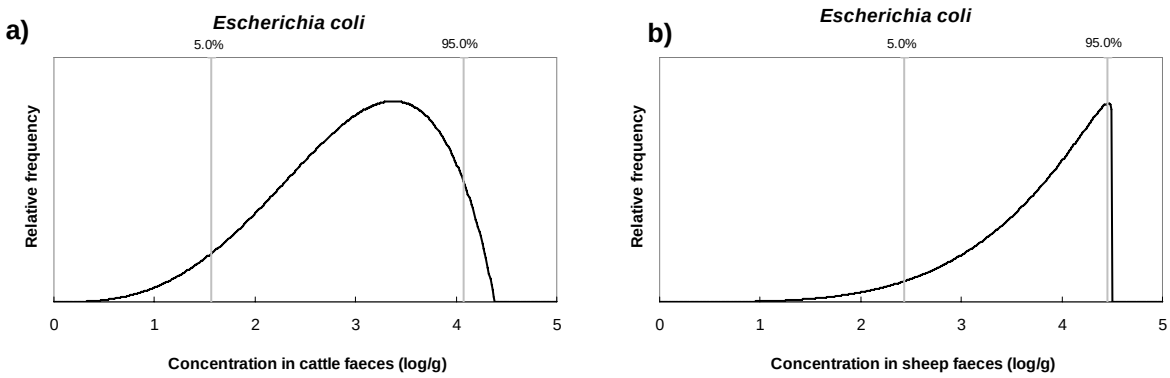



**Figure 2.** Distributions of the concentration of *E. coli* in **a)** cattle and **b)** sheep used as inputs into the model.

## *Dose response*

The dose response curve was derived from human and animal studies. The human studies were based on ingestion of strains of *E. coli* other than O157:H7, while the animal studies were based on rabbits inoculated with *E. coli* O157:H7. It has been suggested that *Shigella* can be used as a surrogate for *E. coli* 0157:H7 since the mechanisms of infection are quite similar (e.g. Marks *et al*. 1998). However, according to Haas *et al* (2000) the potency of *E. coli* in rabbits (which is considered similar to the potency in humans) is closer to the potency of other pathogenic *E.coli* than to *Shigella*. For this study we have thus chosen to use the data from animal studies to construct a dose-response curve rather than using *Shigella* as a surrogate for *E. coli* O157:H7.

Available literature describes the human dose response curve for *E. coli* as a Beta Poisson distribution, where the probability of infection depends on the dose, *D*, and the dose-response parameters $\varepsilon$ and $\beta$. The Beta Poisson distribution is equivalent to the Weibull-Gamma distribution where the chi ($\chi$) parameter is equal to 1. The Weibull-Gamma dose-response equation has the form:

$$\text{Probability of infection} = 1 - [(1 + (\text{dose}^{\chi}/\beta))]^{-\varepsilon}$$

By varying the beta ($\beta$) parameter of a Weibull-Gamma distribution, we captured the full range of reported dose responses in the literature, within the range of pathogen ingestion/inhalation calculated by the model. The slope of the resulting dose response curve varies with $\beta$, which is simulated by a uniform distribution (Attachment 1). Depending on the $\beta$ value simulated, the dose response curve lies between the upper and lower curves shown in Figure 3. The dose range simulated by the model was low, with the 95$^{th}$ percentile of less than one microorganism potentially inhaled in spray drift from irrigation water.

The model assumes that humans can be infected by *E. coli* through ingestion or inhalation of the pathogen and that the dose response is the same for both modes of infection. Dose-response studies are generally performed at high doses. Hence extrapolation from high doses studied in the literature to low doses simulated in this model may result in inaccurate estimates of the dose response.

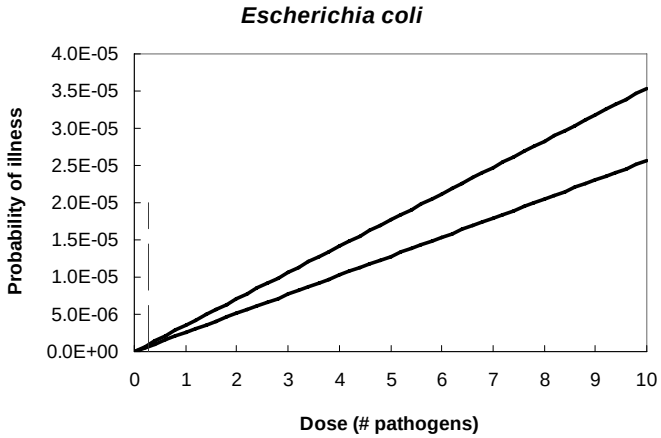



**Figure 3.** Dose response relationship for *E. coli*. The vertical line represents the 95$^{th}$ percentile pathogen dose for spray drift, simulated by the model for a cattle abattoir scenario. The upper and lower dose response curves represent the range of dose responses used in the model created by varying the β parameter.

# Results

The model was run separately for a hypothetical cattle (1000 cattle) and a hypothetical sheep (3000 sheep) abattoir. Stabilisation of the model outputs took 2300 iterations for the cattle scenario and 2700 iterations for the sheep scenario.

Simulated input values for the three parameters unique to the *E. coli* model are shown in Table 1. The simulated outputs are given in Table 2 and illustrated graphically in Figures 4 and 5.

**Table 1.** Simulated inputs for the *E. coli* model from Figures 1, 2 and 3

| | **Prevalence of pathogen (%)** | | **Concentration of pathogen in faeces (log/g)** | | **Dose response β parameter** | |
|---|---|---|---|---|---|---|
| | Cattle | Sheep | Cattle | Sheep | Cattle | Sheep |
| Minimum | 0.99 | 0.71 | 0.43 | 0.48 | 49508.7 | 49507.2 |
| 5$^{th}$ percentile | 11.63 | 7.36 | 1.58 | 2.44 | 50407.0 | 50450.4 |
| 50$^{th}$ percentile | 34.09 | 27.78 | 3.04 | 3.89 | 58623.9 | 58933.6 |
| 95$^{th}$ percentile | 60.70 | 55.01 | 4.12 | 4.45 | 67422.0 | 67343.5 |
| Maximum | 76.19 | 73.52 | 4.37 | 4.50 | 68297.2 | 68298.2 |

**Table 2.** Summary of simulated outputs: number of people ill / million exposed / days of exposure to airborne and waterborne *E. coli* from **a)** a cattle abattoir (1000 cattle) and **b)** a sheep abattoir (3000 sheep). See Figures 4 and 5 for graphical representation of results

**a)**

**CATTLE**

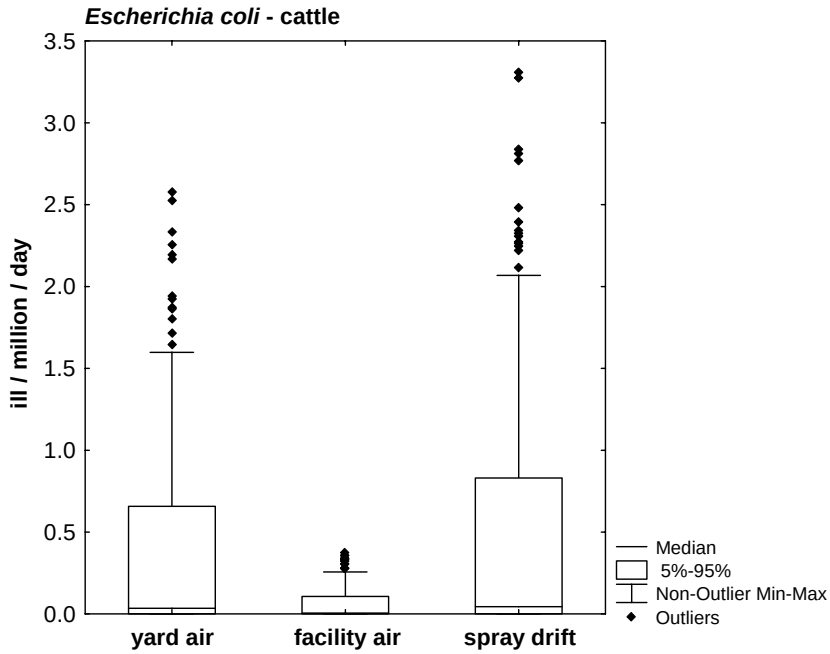
| Exposure route | Yard air | Facility air | Spray drift | Drinking water | Recreational water |
|---|---|---|---|---|---|
| Minimum | <1 | <1 | <1 | <1 | <1 |
| 5$^{th}$ percentile | <1 | <1 | <1 | <1 | <1 |
| 50$^{th}$ percentile | 0.03 | 0.01 | 0.04 | 6.50E-07 | 5.87E-06 |
| 95$^{th}$ percentile | 0.65 | 0.11 | 0.83 | 1.13E-05 | 8.85E-05 |
| Maximum | 6.08 | 1.62 | 8.21 | 5.44E-05 | 3.98E-04 |

**b)**

**SHEEP**

| Exposure route | Yard air | Facility air | Spray drift | Drinking water | Recreational water |
|---|---|---|---|---|---|
| Minimum | <1 | <1 | <1 | <1 | <1 |
| 5$^{th}$ percentile | <1 | <1 | <1 | <1 | <1 |
| 50$^{th}$ percentile | 0.05 | 0.01 | 0.07 | 9.55E-07 | 8.75E-06 |
| 95$^{th}$ percentile | 0.48 | 0.08 | 0.68 | 7.84E-06 | 6.51E-05 |
| Maximum | 4.95 | 0.56 | 16.14 | 2.52E-05 | 1.94E-04 |

**a)**




**b)**

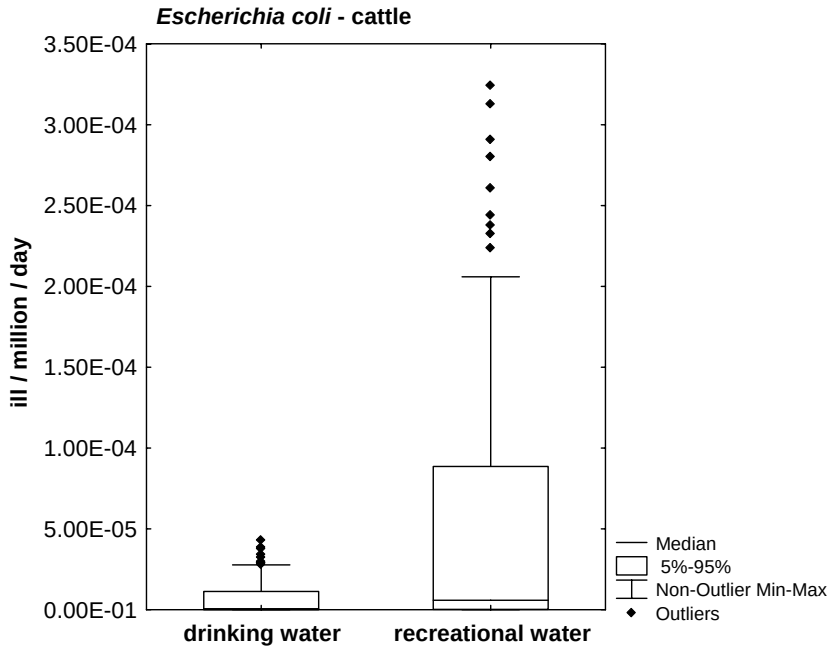
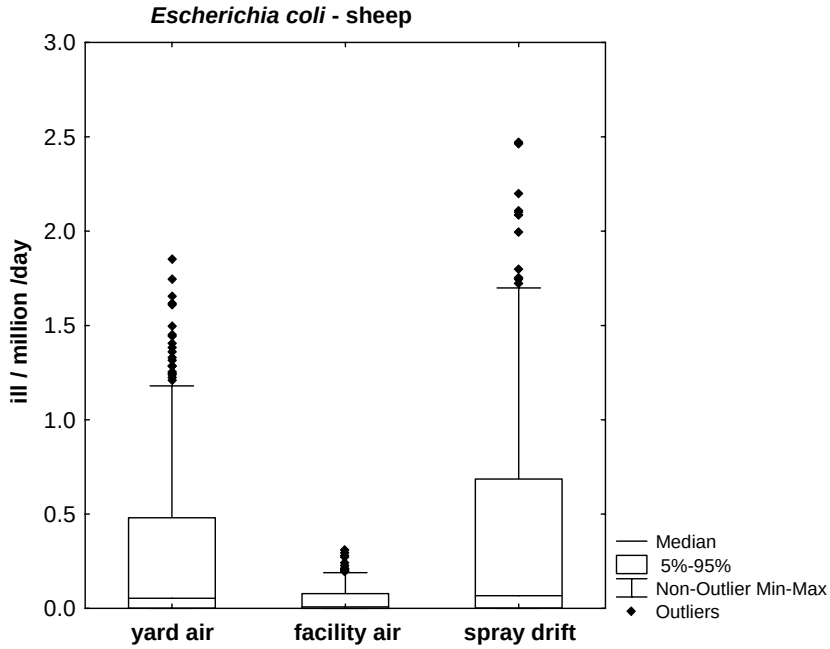



**Figure 4.** Number of people ill / million exposed / days of exposure to **a)** airborne, and **b)** waterborne *Escherichia coli* from a cattle abattoir (1000 cattle). Refer to Table 2 for numerical values.

**a)**




**b)**

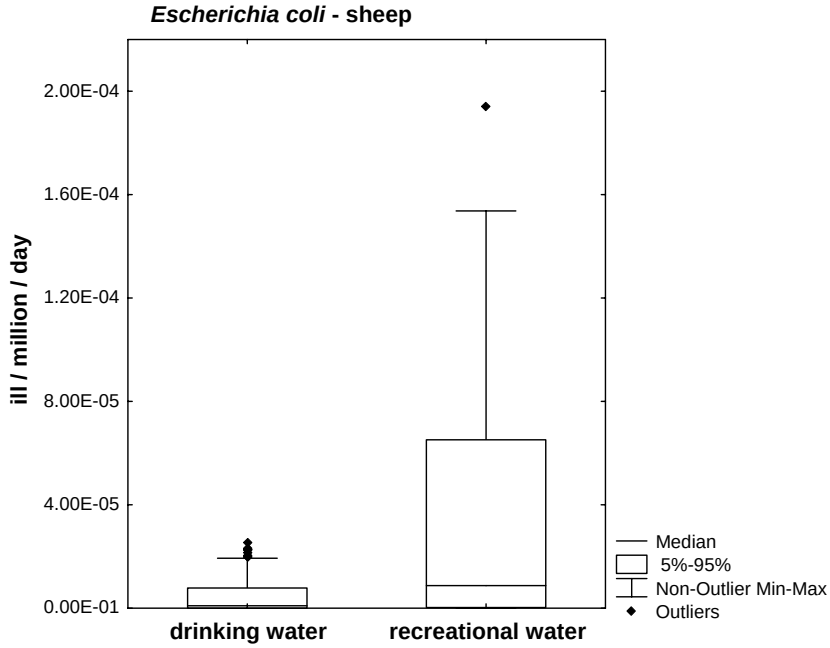



**Figure 5.** Number of people ill / million exposed / days of exposure to **a)** airborne, and **b)** waterborne *Escherichia coli* from a sheep abattoir (3000 sheep). Refer to Table 2 for numerical values.

# Discussion

## *Relative risk of exposure pathways*

The results from the cattle (1000 cattle) and sheep (3000 sheep) abattoir scenarios (Table 2; Figures 4, 5) suggest that the risk of human illness from *Escherichia coli* is greatest for airborne exposure and least for waterborne exposure to the pathogen. For both cattle and sheep pathogen sources, the relative ranking of the 5 modelled exposure pathways is:

- Spray drift irrigation
- Yard air
- Facility air
- Recreational water
- Drinking water

*higher risk*

*lower risk*

For the three pathways of airborne exposure, contaminated spray drift would be expected to have the highest risk as the wastewater stream (after processing and treatment) has a higher pathogen load than the contaminated air released from the livestock yard or the air stream after processing and treatment in the abattoir (see total pathogen output in Appendix C). For waterborne exposure, contaminated drinking water would be expected to be lower risk than recreational water based on the assumption in the model that drinking water, unlike recreational water, undergoes a treatment step before possible ingestion.

## *Scale of the risk*

According to the model, if one million people/day were exposed to *E. coli* via any of the 3 airborne routes from a cattle or sheep abattoir, a median of less than 1 person/day would be infected. However, the maximum estimates of risk suggest that if one million people/day were exposed to *E. coli* via contaminated spray drift from a cattle abattoir, approximately 8 people/day could be infected with enough pathogens to cause illness. This compares with a maximum estimate of 16 people infected by *E. coli* sourced from sheep. We note that there are no documented cases of human illness from exposure to *E. coli* via the inhalation route. The maximum estimates of risk (number of people ill/ million exposed/ days of exposure) for both waterborne pathways are less than 1 person/day for the cattle and sheep abattoir scenarios.

The median risk of exposure from a sheep abattoir is slightly higher than from a cattle abattoir for all airborne and waterborne pathways. This is most likely due to the slightly higher median concentration of *E. coli* in the faeces of sheep compared to cattle. Although the median prevalence of *E. coli* and the gastro-intestinal volume in cattle are higher than in sheep, sensitivity analyses (Attachment 2) indicate that the concentration of the pathogen in faeces is the more important parameter in influencing the number of ill and hence the relative risks.

The estimated risk of illness from airborne and waterborne exposure to *E. coli* has a wide distribution (Fig. 4, 5). This is due to uncertainties in the input parameters such as pathogen prevalence, concentration in faeces and dose response. As noted above, sensitivity analyses (Attachment 2) for the cattle scenario indicate that each output in the model is highly sensitive to the concentration of the pathogen in cattle faeces. This means that the higher the concentration of *E. coli* in cattle faeces, the greater the risk of illness from airborne or waterborne exposure. The wind speed (in the case of airborne pathways) and prevalence of the pathogen are the next most important parameters in influencing the final outputs. The wind speed has a negative correlation to the risk of illness because the volume of air potentially carrying pathogens is a function of the wind speed. The larger the volume, the more dilute is the effective concentration of the pathogen in the breathing volume of air. This means there is a lower risk of exposure to the pathogen. Importantly, the model indicates that the slope of the dose response curve is not one of the most important parameters. This suggests that in refining the model, constraining uncertainties in the other parameters is more important.

## *Conclusion*

Given the sensitivity of the outputs to the model parameters, especially to the concentration of *E. coli* in faeces, the illness estimates generated by the model will be subject to a large amount of uncertainty because the inputs themselves are uncertain. Although we have attempted to deal with this explicitly through a stochastic simulation approach, there is no guarantee that we have captured the full range of uncertainties. Further information on, say, dose response might reduce uncertainty or increase uncertainty by revealing a greater range of responses than previously documented. The results generated by the model, especially the absolute values, should therefore be interpreted with caution. Greater confidence can be placed in relative rankings, but even these could change as a result of different characteristics of individual abattoirs. The results of this 'generic abattoir' model should be used to identify where more information is needed and guide more specific investigations for individual abattoirs. We note that based on Australian studies there is a more complete input dataset available for cattle than sheep, and therefore in the absence of further data, the *E. coli* model is perhaps more applicable to cattle abattoir situations in Australia.

In order to refine the estimates of risk for human exposure to *Escherichia coli*, the model would require:

- Expert opinion on whether *E. coli* can be transmitted and survive in an airborne state
- Expert opinion on whether *E. coli* can cause human illness via inhalation exposure, and if so, information on the dose response curve
- Australian data on the concentration of the pathogen in the faeces of sheep
- A better understanding of the human dose response at low doses.

# Attachment 1:

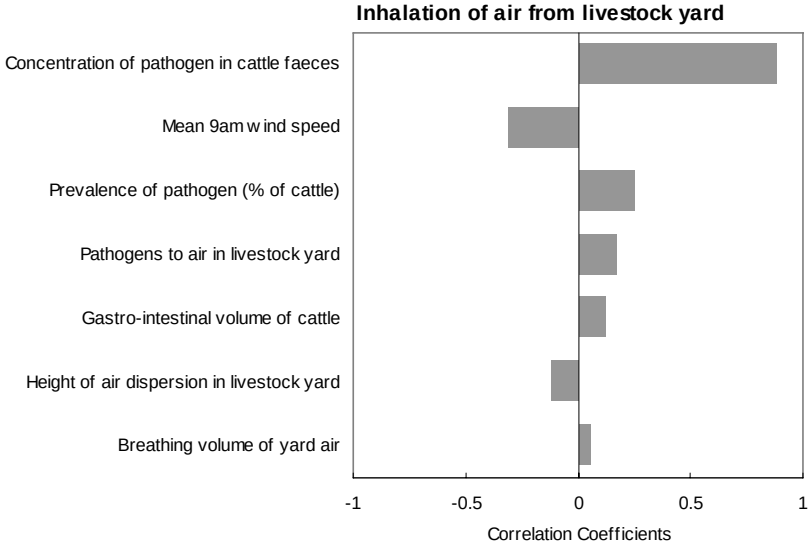
**Table A1.** Input data used to simulate *E. coli* load and dose response for pathogens entering and leaving an abattoir

| ***Escherichia coli*** | **Distribution** | | | | **Source of data** | **Notes on data** |
|---|---|---|---|---|---|---|
| | | min | most likely | max | | |
| Prevalence of pathogen carrier cattle (%) | PERT | 0 | 31 | 80 | FSA (2002) | Minimum, mean and maximum of 5 samples from each of 28 herds (14 grass-fed and 14 grain-fed) in cattle entering an abattoir in SE Queensland (toxigenic 0157). |
| Faeces contamination in carrier cattle (pathogens log/g) | PERT | 0.00 | 3.37 | 4.38 | FSA (2002) | Average of 12 grainfed and 10 grassfed cattle faeces samples with positive results for toxigenic *E. coli* 0157 from 28 herds from QLD and NSW. |
| Prevalence of pathogen carrier sheep (%) | PERT | 0 | 23.3 | 80 | Vanselow & Hornitzky (2001) (appendix A4 and A5) | Minimum, mean and maximum of results from 25 animals sampled from each of 94 flocks of prime lambs and sheep in NSW, VIC and TAS. (note: serotype 0157 was uncommon, most serotypes that were found are not associated with human disease). |
| Faeces contamination in carrier sheep (pathogens log/g) | PERT | 0.00 | 4.46 | 4.50 | Strachan *et al*. (2001) | Mean of results for UK ewes and lambs, plus 10% to estimate maximum, zero estimated minimum. |
| Weibull-Gamma dose response | | $\varepsilon$ | $\beta$ | $\chi$ | Haas *et al*. (1999); Haas *et al*. (2000) | We altered the Beta parameter of the Weibull-Gamma dose response model to capture the range of 2 reported dose response curves for *E. coli* in rabbits and humans. The study in rabbits was for *E. coli* 0157:H7 whereas the human study was for other strains. |
| | *fixed parameters* | 0.175 | | 1.00 | | |
| | UNIFORM | min | 49500 | | | |
| | | max | 68300 | | | |

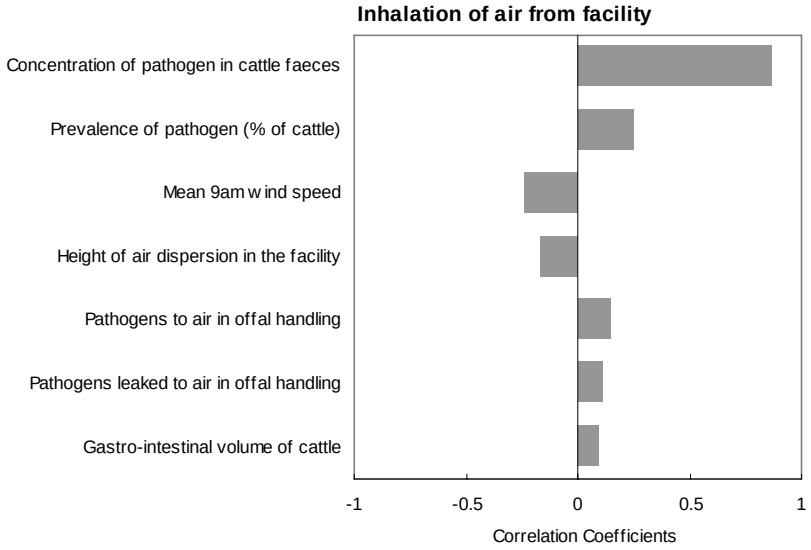
# Attachment 2:

**Figure A1.** Sensitivity analyses for number ill / million exposed / days exposed to airborne *Escherichia coli* based on a cattle abattoir scenario. **a)** inhalation of air from livestock yard; **b)** inhalation of air from the facility (abattoir); **c)** inhalation of spray drift; **d)** ingestion of drinking water; **e)** ingestion of recreational water. Sensitivities are only shown for parameters where the correlation value is greater than or equal to +/- 0.05.

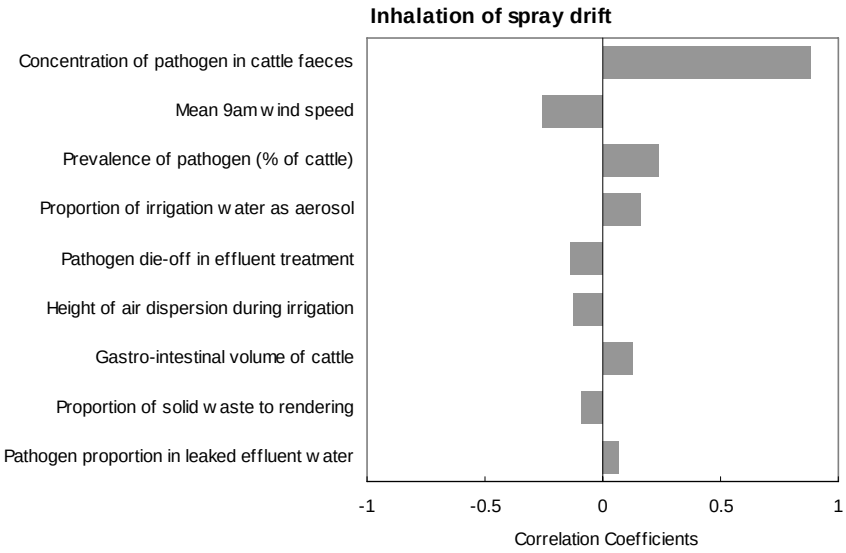
**a)**



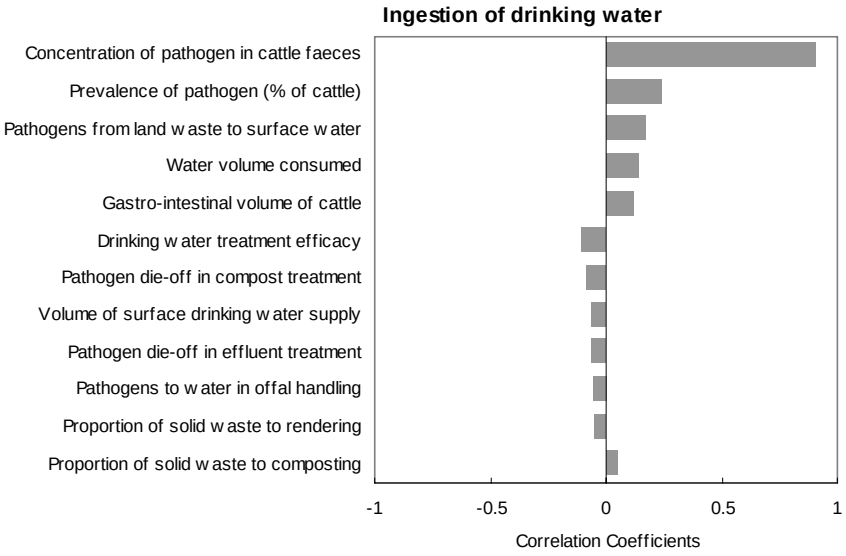

**b)**



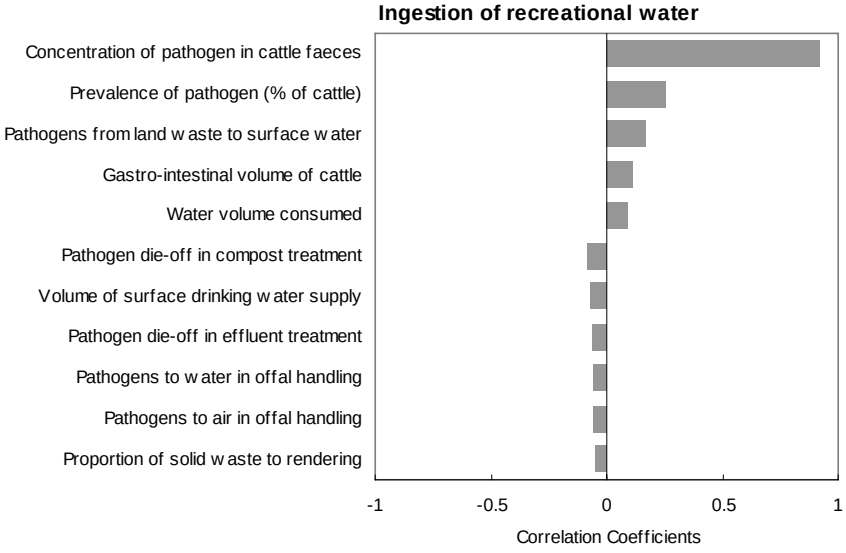
**c)**


Inhalation of spray drift
Concentration of pathogen in cattle faeces
Mean 9am wind speed
Prevalence of pathogen (% of cattle)
Proportion of irrigation water as aerosol
Pathogen die-off in effluent treatment
Height of air dispersion during irrigation
Gastro-intestinal volume of cattle
Proportion of solid waste to rendering
Pathogen proportion in leaked effluent water
-1
-0.5
0
0.5
1
Correlation Coefficients

**d)**


Ingestion of drinking water
Concentration of pathogen in cattle faeces
Prevalence of pathogen (% of cattle)
Pathogens from land waste to surface water
Water volume consumed
Gastro-intestinal volume of cattle
Drinking water treatment efficacy
Pathogen die-off in compost treatment
Volume of surface drinking water supply
Pathogen die-off in effluent treatment
Pathogens to water in offal handling
Proportion of solid waste to rendering
Proportion of solid waste to composting
-1
-0.5
0
0.5
1
Correlation Coefficients

**e)**


Ingestion of recreational water
Concentration of pathogen in cattle faeces
Prevalence of pathogen (% of cattle)
Pathogens from land waste to surface water
Gastro-intestinal volume of cattle
Water volume consumed
Pathogen die-off in compost treatment
Volume of surface drinking water supply
Pathogen die-off in effluent treatment
Pathogens to water in offal handling
Pathogens to air in offal handling
Proportion of solid waste to rendering
-1
-0.5
0
0.5
1
Correlation Coefficients

# APPENDIX G. QUANTITATIVE RISK ASSESSMENT OF *SALMONELLA* SPP. EMISSIONS FROM ABATTOIRS

Salmonellosis is recognised as one of the most important public and animal health disease problems, causing worldwide morbidity and mortality of humans and animals. Some serovars are associated with gastroenteritis in humans, while other types such as *S. typhi*, which leads to Typhoid fever (a rare disease in developing countries), is a more serious form of salmonellosis. Salmonellae are found in the intestinal tract of a wide variety of animals whether domesticated, pet or wild, mammalian or avian, warm blooded or cold-blooded. Salmonellosis is a communicable disease readily transmissible from animals to humans, either directly or through contaminated products of plant or animal origin. Animals colonised by salmonellae often shed the organism without signs of illness. Salmonellae shed in faeces can contaminate soil, pasture, streams and lakes; organisms in soil can survive for months.

Over 2000 serotypes of salmonellae have been identified on a global basis. Although all salmonellae are considered potentially pathogenic, some serovars are host specific for humans, while other serovars occurring frequently in animals and animal products are rarely responsible for human disease. The infectivity of salmonellae in humans varies with the strain, the food vehicle and the age and health status of the person. Infectious doses of as few as 1-10 cells have been found to cause outbreaks of salmonellosis (Jay *et al*. 1997).

## Methods

The generic quantitative risk analysis model described in Appendix C was customised for *Salmonella* spp. by altering the following parameters (based on available information in the scientific literature):

- Pathogen prevalence in sheep and cattle
- Concentration of pathogen in faeces
- Dose-response relationship

The model was used to calculate the number of people ill / million exposed / days of exposure to airborne and waterborne salmonellae sourced from a cattle and sheep abattoir. The volume of the groundwater supply was set to zero in the model, and hence the potential risk from exposure to contaminated groundwater was not simulated. Sensitivity analyses for a cattle abattoir scenario (cattle data are more complete than for sheep) were performed for each output in order to determine which input parameters have the greatest influence on the estimated number of people ill.

### *Prevalence*

Input data on the prevalence of *Salmonella* spp. in cattle and sheep have been estimated by combining results from studies undertaken in various states of Australia (Attachment 1). The constructed distributions for salmonellae prevalence differ between cattle and sheep, with a much higher 'most likely' prevalence and broader range for cattle (Figure 1).

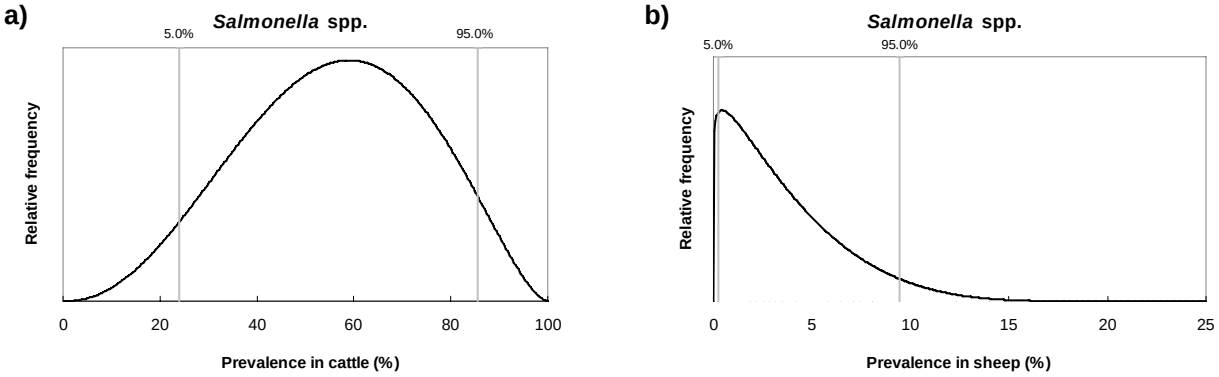



**Figure 1.** Distributions of the prevalence of *Salmonella* spp. in **a)** cattle and **b)** sheep used as inputs into the model.

## *Concentration*

The model assumes that *Salmonella* spp. are present in the gastro-intestinal system of infected cattle and sheep. The concentration of the organism in the faeces is assumed to be representative of the concentration in the entire gastro-intestinal tract. Input data on the concentration of salmonellae in cattle faeces have been estimated by combining results from studies in Queensland and NSW (Attachment 1). In the absence of specific information for sheep, the same concentration values have been used as for cattle. The distribution for salmonellae concentration in cattle is shown in Figure 2.

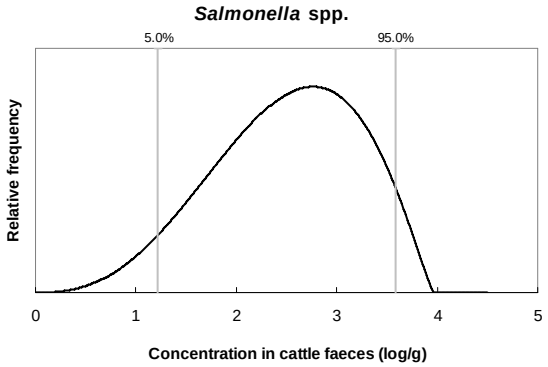



**Figure 2.** Distribution of the concentration of *Salmonella* spp. in cattle used as an input into the model. In the absence of sheep-specific data, the concentration of *Salmonellae* spp. in sheep is assumed to be the same as in cattle.

## *Dose response*

The dose response curve was derived from human studies for ingestion of different strains of *Salmonella*. Available literature describes the human dose response curves as Weibull-Gamma and Beta Poisson distributions, where the probability of infection depends on the dose, *D*, and the dose-response parameters $\varepsilon$ and $\beta$. The Beta Poisson distribution is equivalent to the Weibull-Gamma distribution where the chi ($\chi$) parameter is equal to 1. The Weibull-Gamma dose-response equation has the form:

$$\text{Probability of infection} = 1 - [(1 + (\text{dose}^{\chi}/\beta))]^{-\varepsilon}$$

By varying the beta ($\beta$) parameter of the Weibull-Gamma distribution, we captured the full range of 8 reported dose response models in the literature, within the range of pathogen ingestion/inhalation calculated by the model. The slope of the resulting dose response curve varies with $\beta$, which is simulated by a uniform distribution (Table A1). Depending on the $\beta$ value simulated, the dose response curve lies between the upper and lower curves shown in Figure 3. The dose range simulated by the model was low, with the 95$^{th}$ percentile of less than one microorganism potentially inhaled in spray drift from irrigation water.

The model assumes that humans can be infected by *Salmonella* spp. through ingestion as well as inhalation of the pathogen and that the dose response is the same for both modes of infection. Dose-response studies are generally performed at high doses. Hence extrapolation from high doses studied in the literature to low doses simulated in this model may result in an inaccurate estimate of the dose response.

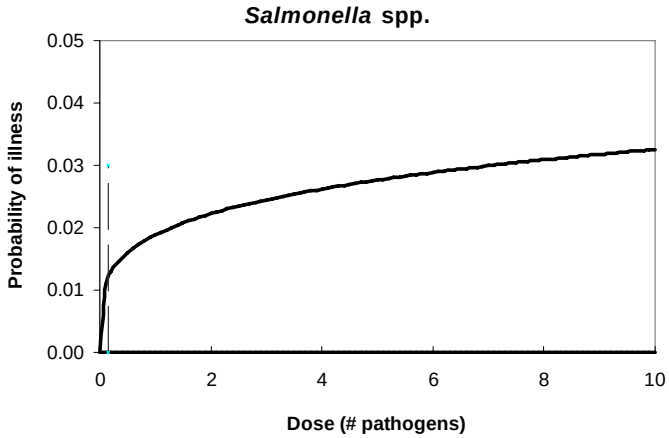



**Figure 3.** Dose response relationship for *Salmonella* spp. The vertical line represents the 95$^{th}$ percentile pathogen dose for spray drift, simulated by the model for a cattle abattoir scenario. The upper and lower dose response curves (the lower curve sits on the x-axis) represent the range of dose responses used in the model created by varying the dose response parameter $\beta$.

## Results

The model was run separately for a hypothetical cattle (1000/day) and a hypothetical sheep (3000/day) abattoir. Stabilisation of the model outputs took 4200 iterations for the cattle scenario and 5700 iterations for the sheep scenario.

Simulated input values for the three parameters unique to the *Salmonella* spp. model are shown in Table 1. The simulated outputs are given in Table 2 and illustrated graphically in Figures 4 and 5.

**Table 1.** Simulated inputs for the *Salmonella* spp. model from Figures 1, 2 and 3

| | **Prevalence of pathogen (%)** | | **Concentration of pathogen in faeces (log/g)** | | **Dose response parameter** | |
|---|---|---|---|---|---|---|
| | Cattle | Sheep | Cattle | Sheep | Cattle | Sheep |
| Minimum | 6.78 | 0.0012 | 0.33 | 0.30 | 1.38E+12 | 3.25E+11 |
| 5$^{th}$ percentile | 24.58 | 0.26 | 1.20 | 1.24 | 2.56E+14 | 2.57E+14 |
| 50$^{th}$ percentile | 56.58 | 2.88 | 2.57 | 2.57 | 2.49E+15 | 2.50E+15 |
| 95$^{th}$ percentile | 85.62 | 9.40 | 3.59 | 3.59 | 4.76E+15 | 4.75E+15 |
| Maximum | 97.87 | 16.17 | 3.95 | 3.96 | 5.00E+15 | 5.00E+15 |

**Table 2.** Summary of simulated outputs: number of people ill / million exposed / days of exposure to airborne and waterborne *Salmonella* spp. from **a)** a cattle abattoir (1000 cattle) and **b)** a sheep abattoir (3000 sheep). See Figures 4 and 5 for graphical representation of results

**a)**

**CATTLE**

| Exposure route | Yard air | Facility air | Spray drift | Drinking water | Recreational water |
|---|---|---|---|---|---|
| Minimum | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 |
| 5th percentile | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 |
| 50th percentile | 0.079 | 0.000 | 0.079 | 0.000 | 0.000 |
| 95th percentile | 0.40 | 0.32 | 0.47 | 0.000 | 0.08 |
| Maximum | 91 | 52 | 64 | 7 | 11 |

**b)**

**SHEEP**

| Exposure route | Yard air | Facility air | Spray drift | Drinking water | Recreational water |
|---|---|---|---|---|---|
| Minimum | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 |
| 5th percentile | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 |
| 50th percentile | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 |
| 95th percentile | 0.16 | 0.08 | 0.16 | 0.000 | 0.000 |
| Maximum | 118 | 80 | 148 | 9 | 15 |

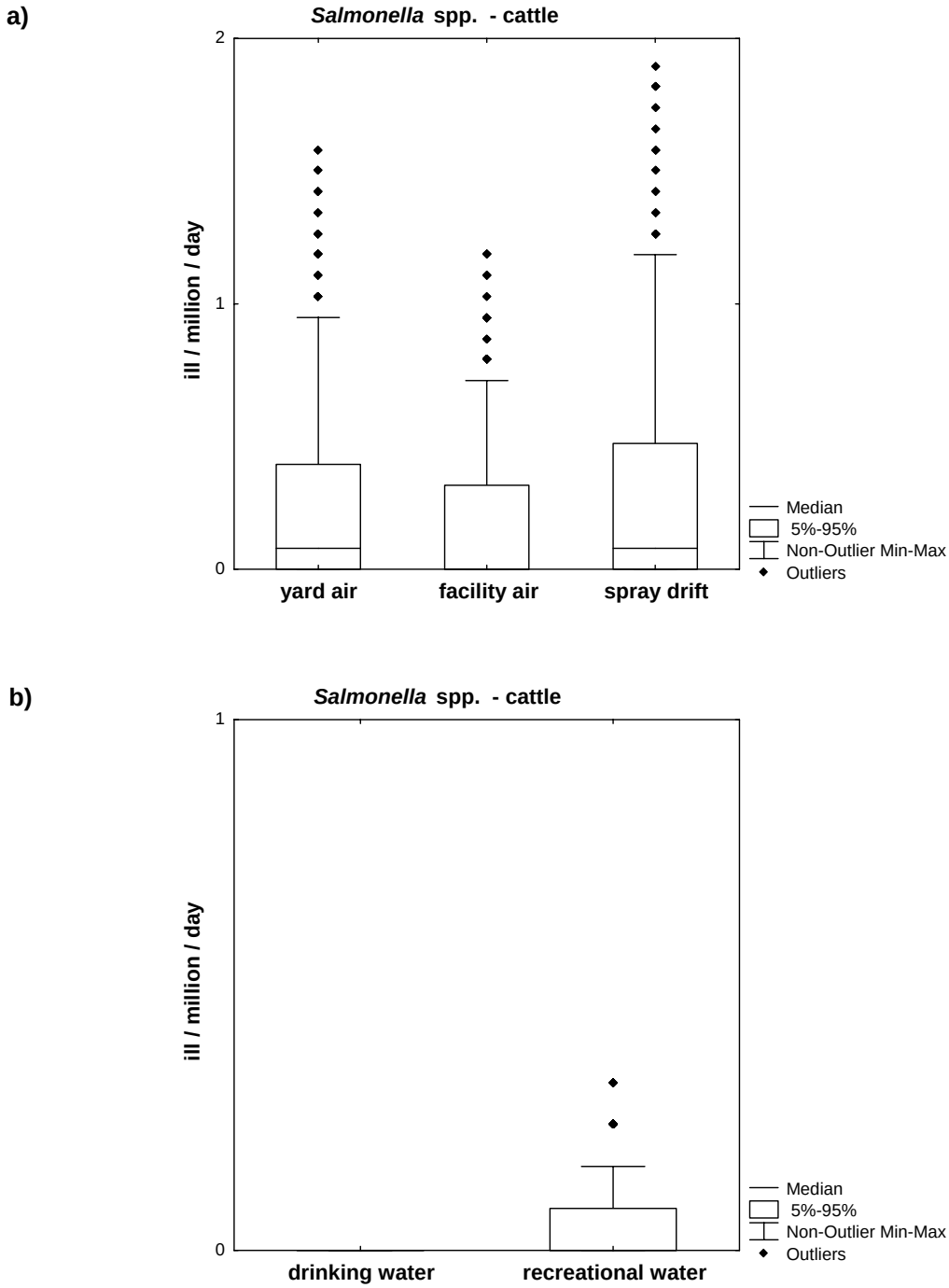
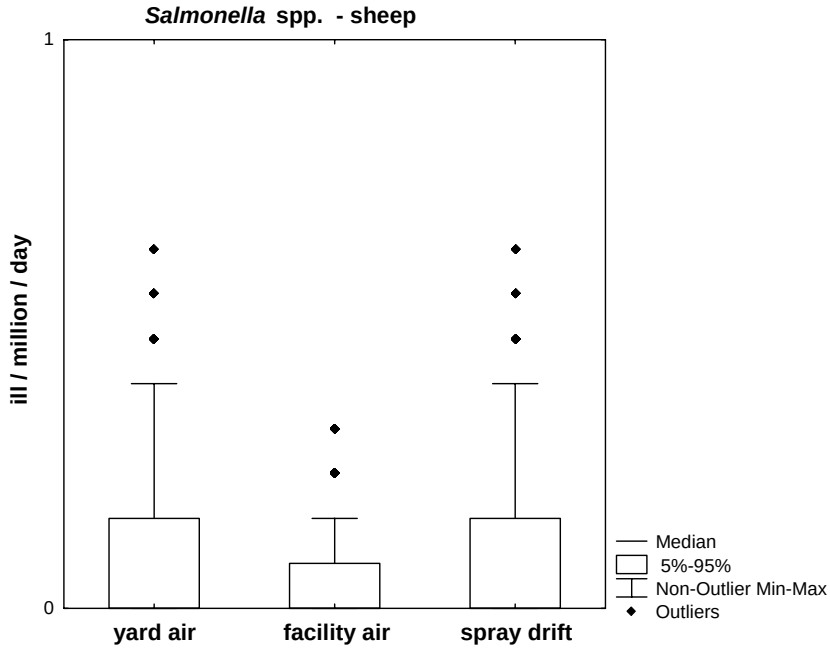



**Figure 4.** Number of people ill / million exposed / days of exposure to **a)** airborne, and **b)** waterborne *Salmonella* spp. from a cattle abattoir (1000 cattle). Refer to Table 2 for numerical values.

**a)**




**b)**

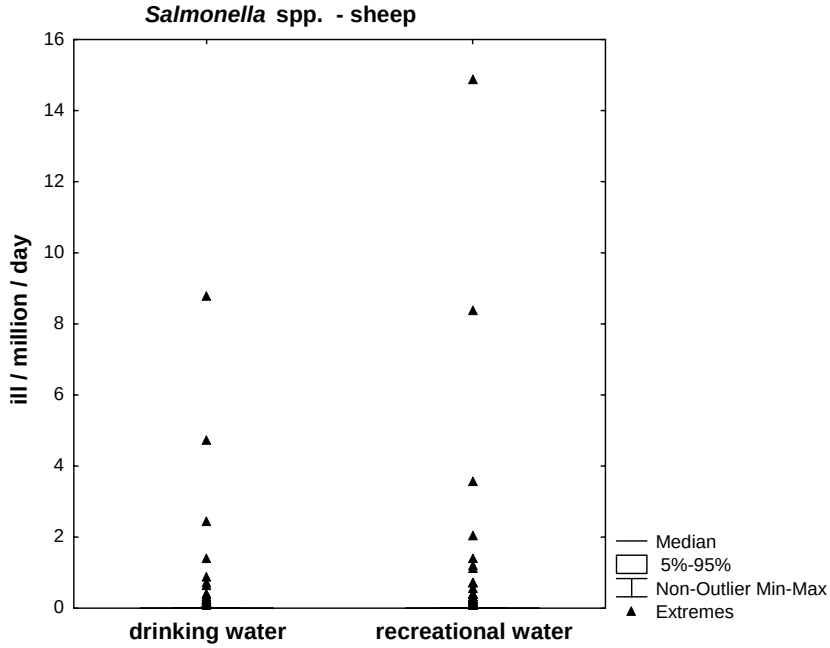



**Figure 5.** Number of people ill / million exposed / days of exposure to **a)** airborne, and **b)** waterborne *Salmonella* spp. from a sheep abattoir (3000 sheep). Refer to Table 2 for numerical values. Note that the 5$^{th}$, 50$^{th}$ and 95$^{th}$ percentiles for exposure to contaminated drinking water and recreational water are zero and thus the box plot shows the range of extreme values.

# Discussion

## *Relative risk of exposure pathways*

The results from the cattle (1000 cattle) and sheep (3000 sheep) abattoir scenarios (Table 2; Fig. 4, 5) suggest that the risk of human illness from *Salmonella* spp. is greatest for airborne exposure to the pathogen in spray drift irrigation and yard air. For both cattle and sheep pathogen sources, the relative ranking of the 5 modelled exposure pathways is:

- Spray drift irrigation & yard air — *higher risk*
- Facility air
- Recreational & drinking water — *lower risk*

Based on the median result, the model indicates similar risks for spray drift and yard air and recreational and drinking water. For the three pathways of airborne exposure, contaminated spray drift would be expected to have the highest risk as the wastewater stream (after processing and treatment) has a higher pathogen load than the contaminated air released from the livestock yard or the air stream after processing and treatment in the abattoir (see total pathogen output in Appendix C). For waterborne exposure, contaminated drinking water would be expected to be lower risk than recreational water based on the assumption in the model that drinking water, unlike recreational water, undergoes a treatment step before possible ingestion.

## *Scale of the risk*

According to the model, if one million people per day were exposed to *Salmonella* spp. via contaminated spray drift or air from the livestock yard from a cattle or sheep abattoir, a median of less than 1 person/day would be infected. However, the maximum estimates of risk suggest that if one million people/day were exposed to *Salmonella* spp. via contaminated spray drift or air from the yard of a cattle abattoir, over 60 people/day could be infected with enough pathogens to cause illness. We note that there are no documented cases of human illness from exposure to *Salmonella* spp*.* via the inhalation route. For both waterborne pathways, the median estimates of risk are less than 1 person/day for the cattle and sheep abattoir scenarios. However, the maximum estimate indicates that for a cattle abattoir, approximately 11 people/day could be infected by *Salmonella* spp. from recreational water and 7 people from drinking water.

The median and 95$^{th}$ percentile risks of exposure from a 1000 cattle/day abattoir are only slightly higher than from a 3000 sheep/day abattoir for all airborne and waterborne pathways, despite the far greater 'most likely' prevalence of *Salmonella* spp*.* in the faeces of cattle compared to sheep. This is consistent with the sensitivity analyses (Attachment 2), which indicate that the model is not very sensitive to pathogen prevalence.

The estimated risk of illness from airborne and waterborne exposure to *Salmonella* spp. has a wide distribution (Fig. 4, 5). This is due to uncertainties in the input parameters such as pathogen prevalence, concentration in faeces and dose response. Sensitivity analyses for the cattle abattoir scenario indicate that each output in the model is highly sensitive to the dose response parameter $\beta$. This is not unexpected given the wide range of possible values for the $\beta$ parameter in the model. This analysis suggests that in refining the model it would be important to constrain the uncertainty in the $\beta$ parameter. The concentration of the pathogen in cattle faeces is the next most important parameter. This means that the higher the concentration of *Salmonella* spp. in cattle faeces, the greater the risk of illness from airborne or waterborne exposure. In the case of airborne pathways, the wind speed is the next most important parameter in influencing the final outputs. The wind speed has a negative correlation to the risk of illness because the volume of air potentially carrying pathogens is a function of the wind speed. The larger the volume, the more dilute is the effective concentration of the pathogen in the breathing volume of air. This means there is a lower risk of exposure to the pathogen.

## *Conclusion*

Given the sensitivity of the outputs to the model parameters, especially to the dose response parameter and the concentration of *Salmonella* spp. in faeces, the illness estimates generated by the model will be subject to a large amount of uncertainty because the inputs themselves are uncertain. Although we have attempted to deal with this explicitly through a stochastic simulation approach, there is no guarantee that we have captured the full range of uncertainties. Further information on, say, dose response might reduce uncertainty or increase uncertainty by revealing a greater range of responses than previously documented. The results generated by the model, especially the absolute values, should therefore be interpreted with caution. Greater confidence can be placed in relative rankings, but even these could change as a result of different characteristics of individual abattoirs. The results of this 'generic abattoir' model should be used to identify where more information is needed and guide more specific investigations for individual abattoirs. We note that there is a more complete dataset available for cattle than sheep and therefore in the absence of further data, the *Salmonella* spp. model is perhaps more applicable to cattle abattoir situations.

In order to refine the estimates of risk for human exposure to *Salmonella* spp., the model would require:

- Expert opinion on whether *Salmonella* spp. can be transmitted and survive in an airborne state
- Expert opinion on whether *Salmonella* spp. can cause human illness via inhalation exposure, and if so, information on the dose response curve
- Australian data on pathogen concentration in the faeces of sheep
- A better understanding of the dose response at low doses.

# Attachment 1:

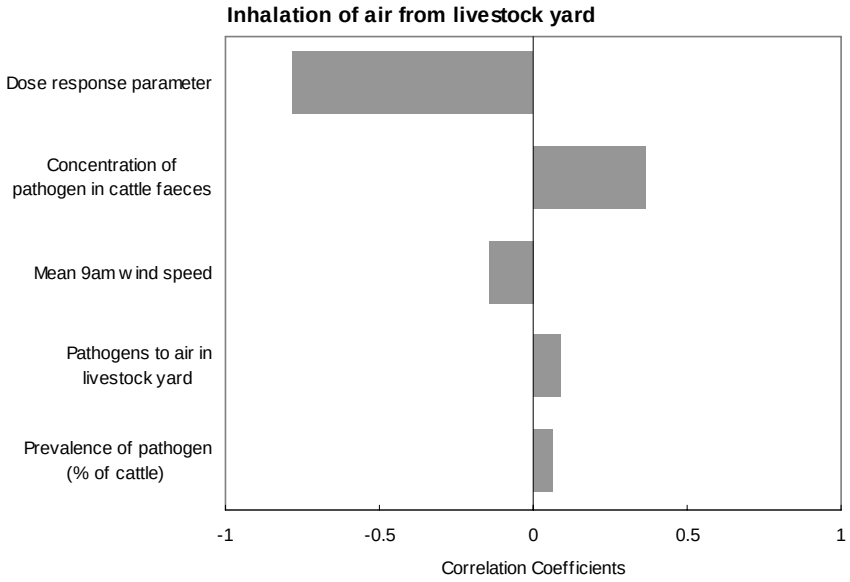
**Table A1.** Input data used to simulate *Salmonella* spp. load and dose response for pathogens entering and leaving an abattoir

| ***Salmonellae*** | **Distribution** | | | | **Source of data** | **Notes on data** |
|---|---|---|---|---|---|---|
| | | min | most likely | max | | |
| Prevalence of pathogen carrier cattle (%) | PERT | 0 | 59 | 100 | FSA (2002) | Minimum, mean and maximum of 5 samples from each of 28 herds (14 grass-fed and 14 grain-fed) in cattle entering an abattoir in SE Queensland. |
| Faeces contamination in carrier cattle (pathogens log/g) | PERT | 0 | 2.76 | 3.97 | FSA (2002) | Average of 12 grainfed and 12 grassfed cattle faeces samples from 28 herds from Qld and NSW. |
| Prevalence of pathogen carrier sheep (%) | PERT | 0 | 0.44 | 20 | Vanselow & Hornitzky (2001) (appendix A4 and A5) | Minimum, mean and maximum of results from 25 animals sampled from each of 94 flocks of prime lambs and sheep in NSW, VIC and TAS. |
| Faeces contamination in carrier sheep (pathogens log/g) | PERT | 0 | 2.76 | 3.97 | No data available | Assumed same as for cattle. |
| Weibull-Gamma dose response | | $\varepsilon$ | $\beta$ | $\chi$ | Messner *et al*. (2001) | We altered the Beta parameter of the Weibull-Gamma dose response model to capture the full range of 8 reported human dose response curves within the range of pathogen loads calculated by the model. |
| *fixed parameters* | | 3.56E+08 | | 0.24 | | |
| UNIFORM | | min | 1.87E+10 | | | |
| | | max | 5.00E+15 | | | |

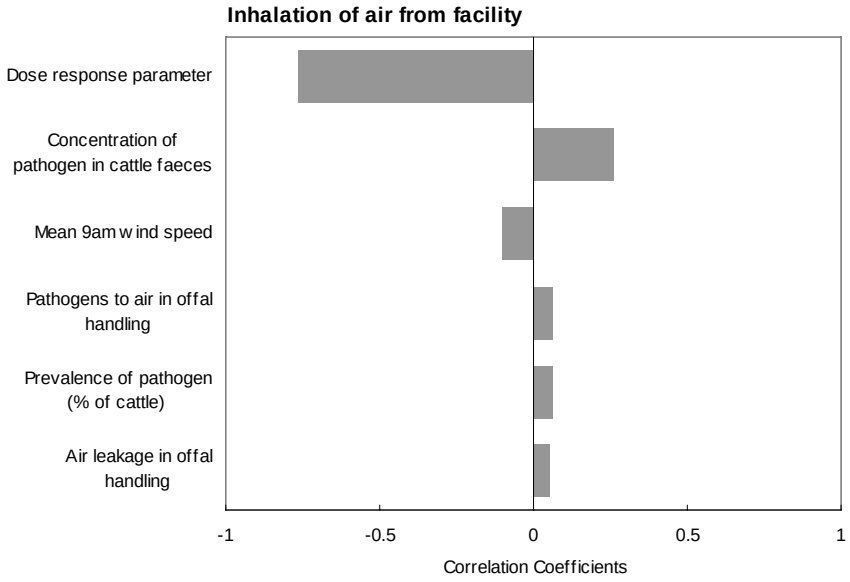
# Attachment 2:

**Figure A1.** Sensitivity analyses for number ill / million exposed / days exposed to airborne and waterborne *Salmonella* spp. based on a cattle abattoir scenario. **a)** inhalation of air from livestock yard; **b)** inhalation of air from the facility (abattoir); **c)** inhalation of spray drift; **d)** ingestion of drinking water; **e)** ingestion of recreational water. Sensitivities are only shown for parameters where the correlation value is greater than or equal to +/- 0.05.

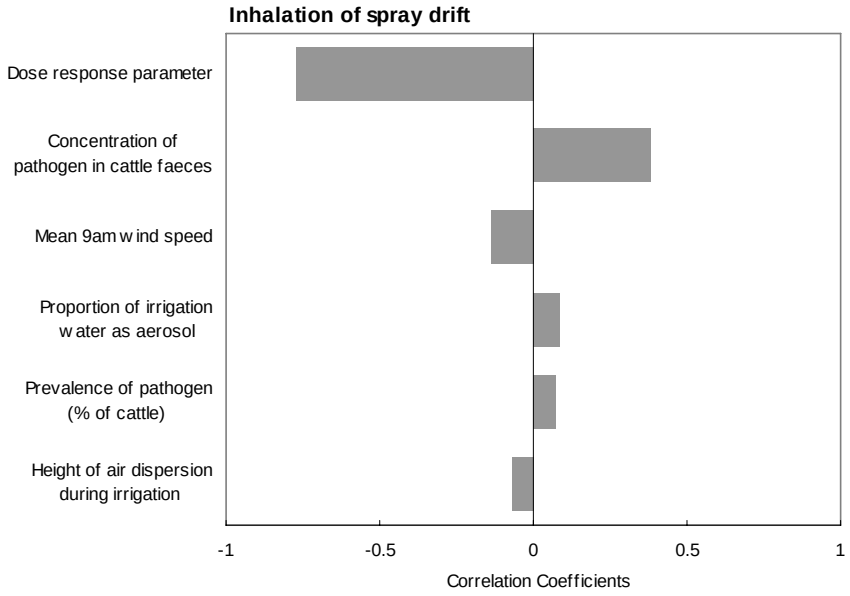
**a)**



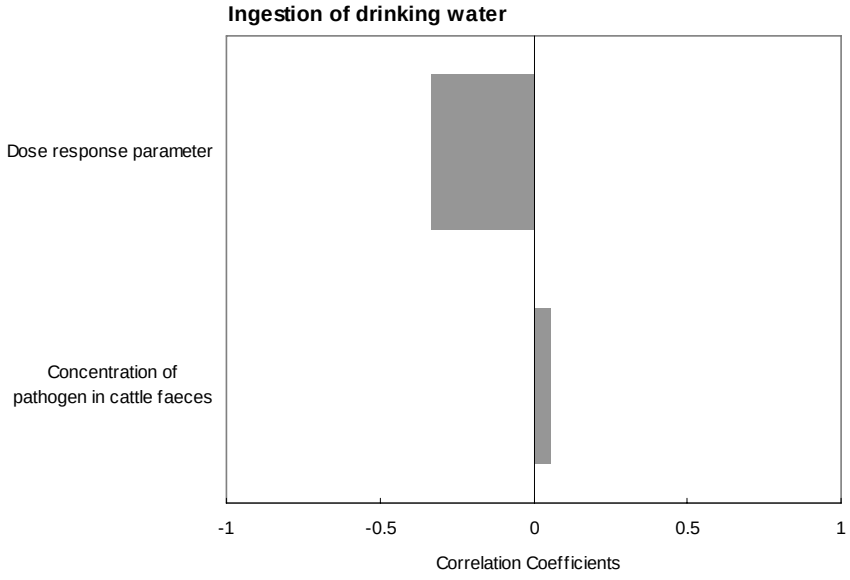

**b)**



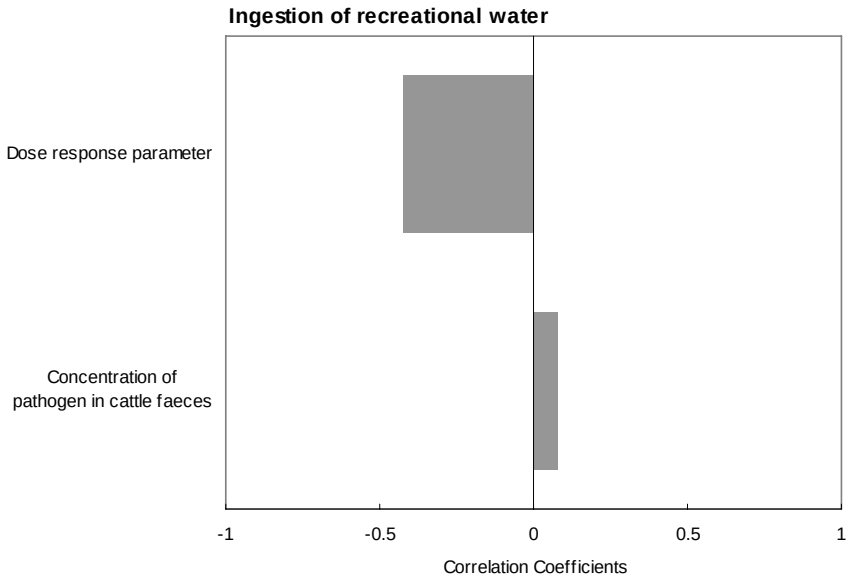
**c)**


Inhalation of spray drift
Dose response parameter
Concentration of pathogen in cattle faeces
Mean 9am w ind speed
Proportion of irrigation w ater as aerosol
Prevalence of pathogen (% of cattle)
Height of air dispersion during irrigation
-1
-0.5
0
0.5
1
Correlation Coefficients

**d)**


Ingestion of drinking water
Dose response parameter
Concentration of pathogen in cattle faeces
-1
-0.5
0
0.5
1
Correlation Coefficients

**e)**


Ingestion of recreational water
Dose response parameter
Concentration of pathogen in cattle faeces
-1
-0.5
0
0.5
1
Correlation Coefficients

# APPENDIX H. QUANTITATIVE RISK ASSESSMENT OF *CRYPTOSPORIDIUM PARVUM* EMISSIONS FROM ABATTOIRS

*Cryptosporidium* spp. are parasites of a wide range of vertebrate species, including reptiles, birds and mammals including humans. Young livestock animals such as calves generally show clinical disease (Cole *et al*. 1999). Although a number of species have been recorded from humans, *Cryptosporidium parvum* (*C. parvum*) is commonly reported during outbreaks. Cryptosporidiosis is considered world wide in distribution, with human infection mostly originating from contaminated water. The pathogen is known to cause gastrointestinal disease, which is self-limiting in most healthy individuals. The most sensitive populations to enteric microorganisms include the young, elderly, malnourished and the immunocompromised for whom the disease may be life-threatening (Gibson *et al*. 1998; Teunis *et al*. 2002).

*C. parvum* is thought to be readily passed from animals to humans through the faecal-oral route. There have been reports of at least two different genotypes of *C. parvum*, one of which is exclusively isolated from humans, and one of which can be isolated from both humans and cattle. It was previously assumed that *Cryptosporidium* infections in humans were zoonotic. This assumption has now been questioned and the contribution made by the human and bovine forms needs further clarification. *C. parvum* oocysts are resistant to most disinfectants and are stable in the environment (Cole *et al*. 1999). *Cryptosporidium* spores can remain viable for about 18 months in a cool or wet environment.

Compared to previously published risk assessments, the model presented here is simplified in that it does not take into account uncertainty in the viability of the cysts or oocysts that cause infection. The actual number of *C. parvum* oocysts required to cause illness is reported to be highly variable between isolates, ranging from an $ID_{50}$ (median infectious dose) of 2066-1042 oocysts for one isolate to 12.1-9 oocysts for another. This indicates substantial variation in the infectivity of *Cryptosporidium* for humans (DuPont *et al* 1995; Messner *et al* 2001; Teunis *et al* 2002).

## Methods

The generic quantitative risk analysis model described in Appendix C was customised for *C. parvum* by altering the following parameters (based on available information in the scientific literature):

- Pathogen prevalence in sheep and cattle
- Concentration of pathogen in faeces
- Drinking water treatment efficacy
- Dose-response relationship

The model was used to calculate the number of people ill / million exposed / days of exposure to airborne and waterborne *C. parvum* sourced from a cattle and sheep abattoir. The volume of the groundwater supply was set to zero in the model, and hence the potential risk from exposure to contaminated groundwater was not simulated. Sensitivity analyses for a cattle abattoir scenario (cattle data are more complete than for sheep) were performed for each output in order to determine which input parameters have the greatest influence on the estimated number of people ill.

### *Prevalence*

Input data on the prevalence of *C. parvum* in cattle have been estimated by combining results from studies in California and Scotland (Attachment 1). In the absence of specific information for sheep, the same prevalence values have been used as for cattle (Figure 1).

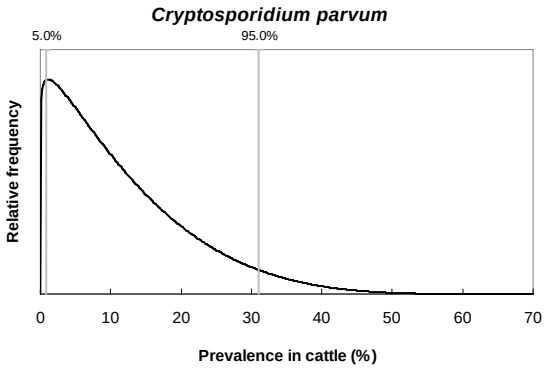



**Figure 1.** Distribution of the prevalence of *C. parvum* in cattle used as an input into the model. In the absence of sheep-specific data, the prevalence of *C. parvum* in sheep is assumed to be the same as in cattle.

## *Concentration*

The model assumes that *C. parvum* is present in the gastro-intestinal system of infected cattle and sheep. The concentration of the organism in the faeces is assumed to be representative of the concentration in the entire gastro-intestinal tract. Input data on the concentration of *C. parvum* in faeces have been estimated by combining results from studies in the United Kingdom (Attachment 1). The distributions for *C. parvum* concentration in cattle and sheep are shown in Figure 2.

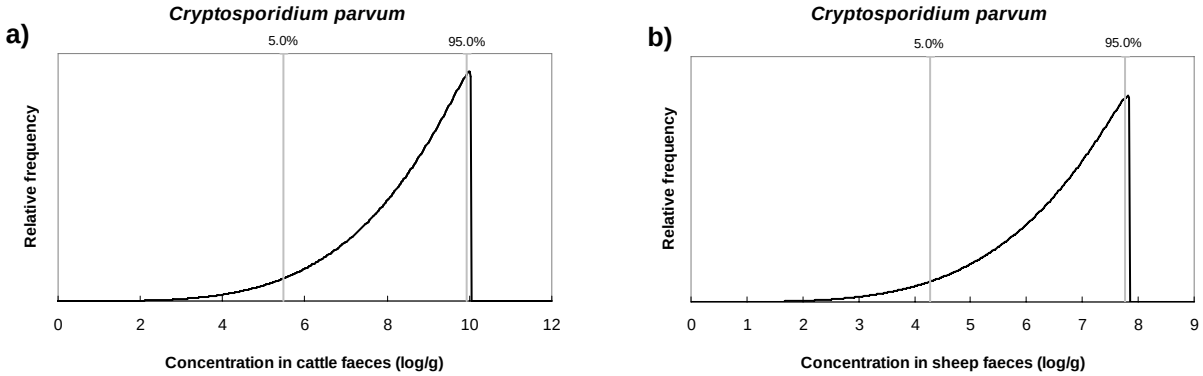



**Figure 2.** Distributions of the concentration of *C. parvum* in **a)** cattle and **b)** sheep used as inputs into the model.

## *Dose response*

The dose response curve was derived from three human studies for ingestion of known doses of *C. parvum* oocysts from calves. The studies involved different isolates of *C. parvum*: the IOWA, TAMU and UCP isolates. In these studies, infection was defined as the presence of oocysts in stool and/or diarrheal illness characteristic of cryptosporidiosis. Available literature describes the human dose response for *C. parvum* by an exponential model, where the probability of infection depends on the dose, *D*, and the unknown dose-response parameter *k* (Messner *et al.* 2001).

The dose-response equation has the form:

$$\text{Probability of infection} = P(D,K) = 1 - e^{-D/k}$$

A different *k* parameter is reported in the literature for the three strains of *C. parvum*. By varying the *k* parameter of the exponential distribution, we captured the full range of reported dose responses in the literature, within the range of pathogen ingestion/inhalation calculated by the model. The shape of the resulting dose response curve varies with *k*, which is simulated by a uniform distribution (Attachment 1).

Depending on the *k* value simulated, the dose response curve lies between the upper and lower curves shown in Figure 3. The dose range simulated by the model was high, with the 50$^{th}$ percentile of approximately 1900 microorganisms potentially inhaled in spray drift from irrigation water.

The model assumes that humans can be infected by *C. parvum* through ingestion as well as inhalation of the pathogen and that the dose response is the same for both modes of infection. Dose-response studies are generally performed at high doses. Hence extrapolation from high doses studied in the literature to low doses simulated in this model may result in inaccurate estimates of the dose response.

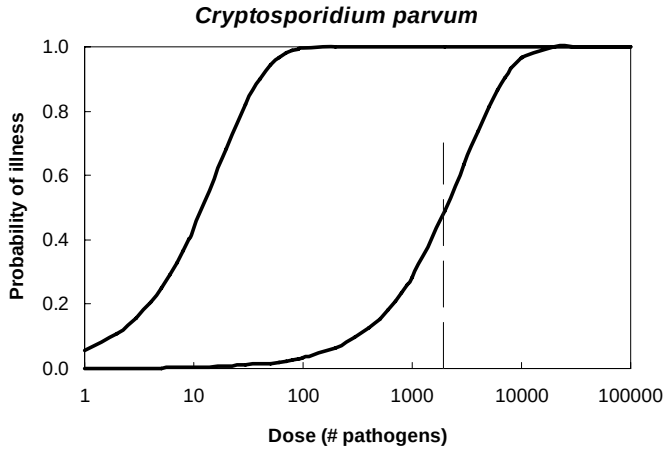



**Figure 3.** Dose response relationship for *Cryptosporidium parvum*. The vertical line represents the 50$^{th}$ percentile pathogen dose for spray drift, simulated by the model for a cattle abattoir scenario. The upper and lower dose response curves represent the range of dose responses used in the model created by varying the dose response parameter, *k*.

## Results

The model was run separately for a hypothetical cattle (1000 cattle) and a hypothetical sheep (3000 sheep) abattoir. Stabilisation of the model outputs took 2700 iterations for the cattle scenario and 3600 iterations for the sheep scenario.

Simulated input values for the three parameters unique to the *C. parvum* model are shown in Table 1. The simulated outputs are given in Table 2 and illustrated graphically in Figures 4 and 5.

**Table 1.** Simulated inputs for the *C. parvum* model from Figures 1, 2 and 3

| | **Prevalence of pathogen (%)** | | **Concentration of pathogen in faeces (log/g)** | | **Dose response parameter** | |
|---|---|---|---|---|---|---|
| | Cattle | Sheep | Cattle | Sheep | Cattle | Sheep |
| Minimum | 6.25E-04 | 2.01E-03 | 1.74 | 1.02 | 18.0 | 17.8 |
| 5$^{th}$ percentile | 0.87 | 0.80 | 5.53 | 4.24 | 167.5 | 175.5 |
| 50$^{th}$ percentile | 9.46 | 9.12 | 8.71 | 6.82 | 1526.6 | 1482.5 |
| 95$^{th}$ percentile | 31.08 | 31.14 | 9.93 | 7.77 | 2841.2 | 2830.5 |
| Maximum | 56.62 | 53.47 | 10.04 | 7.85 | 2979.0 | 2979.3 |

**Table 2.** Summary of simulated outputs: number of people ill / million exposed / days of exposure to airborne and waterborne *C. parvum* from **a)** a cattle abattoir (1000 cattle) and **b)** a sheep abattoir (3000 sheep). See Figures 4 and 5 for graphical representation of results

**a)**

**CATTLE**

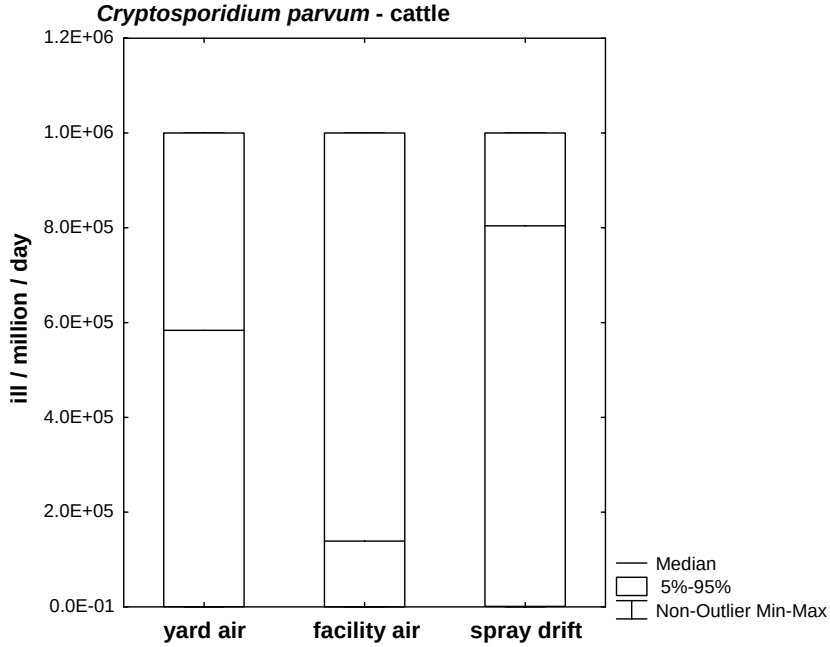
| Exposure route | Yard air | Facility air | Spray drift | Drinking water | Recreational water |
|---|---|---|---|---|---|
| Minimum | 6.20E-04 | 1.46E-04 | 9.61E-04 | 1.61E-07 | 1.97E-07 |
| 5th percentile | 574 | 95 | 1061 | 0.06 | 0.10 |
| 50th percentile | 583085 | 138627 | 803407 | 114 | 168 |
| 95th percentile | 1000000 | 999937 | 1000000 | 8005 | 10248 |
| Maximum | 1000000 | 1000000 | 1000000 | 579888 | 422028 |

**b)**

**SHEEP**

| Exposure route | Yard air | Facility air | Spray drift | Drinking water | Recreational water |
|---|---|---|---|---|---|
| Minimum | 3.82E-03 | 5.69E-04 | 0.014 | 1.48E-06 | 1.74E-06 |
| 5th percentile | 8 | 1 | 13 | 8.03E-04 | 1.20E-03 |
| 50th percentile | 3321 | 573 | 5892 | 0.42 | 0.63 |
| 95th percentile | 136408 | 22646 | 243053 | 15 | 22 |
| Maximum | 987401 | 861290 | 1000000 | 1143 | 1208 |

**a)**




**b)**

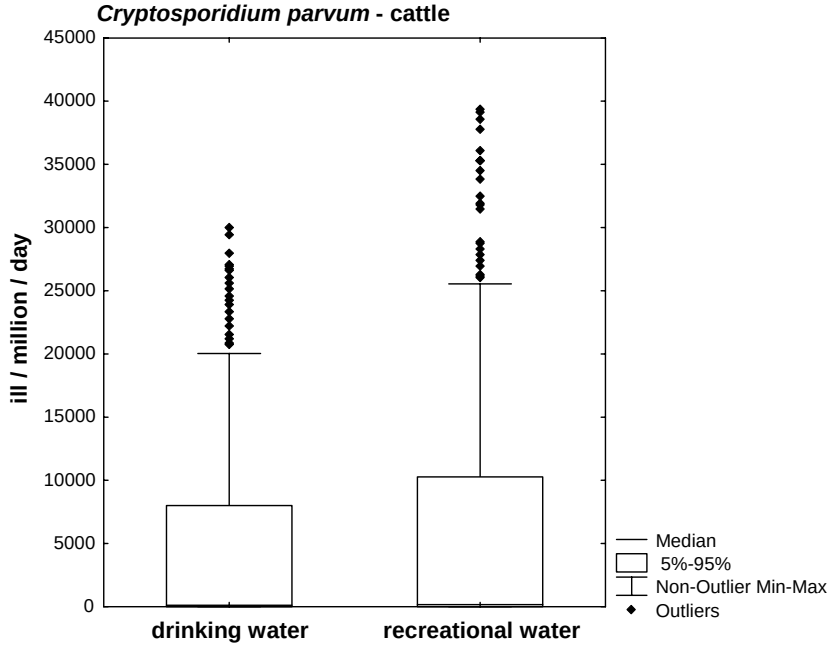
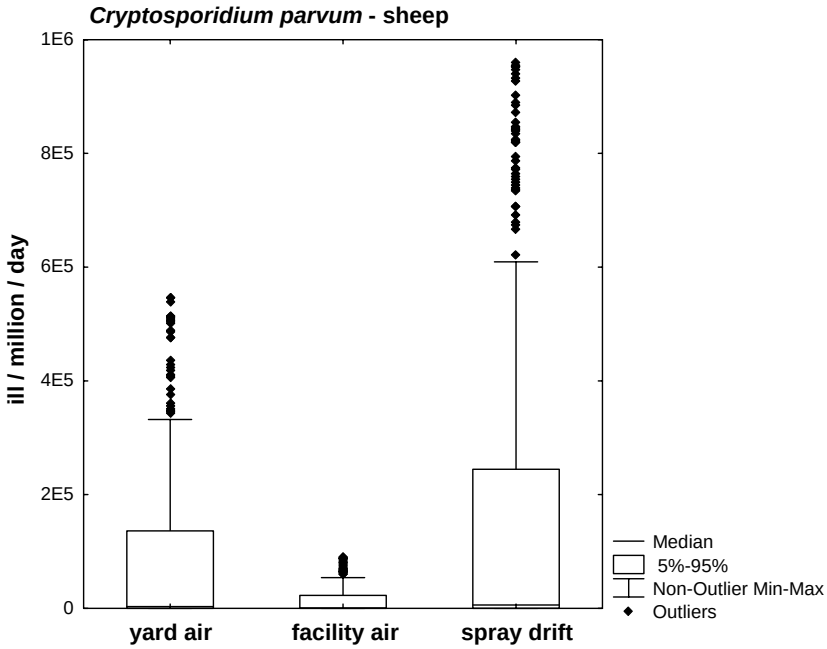



**Figure 4.** Number of people ill / million exposed / days of exposure to **a)** airborne, and **b)** waterborne *Cryptosporidium parvum* from a cattle abattoir (1000 cattle). Refer to Table 2 for numerical values.

**a)**




**b)**

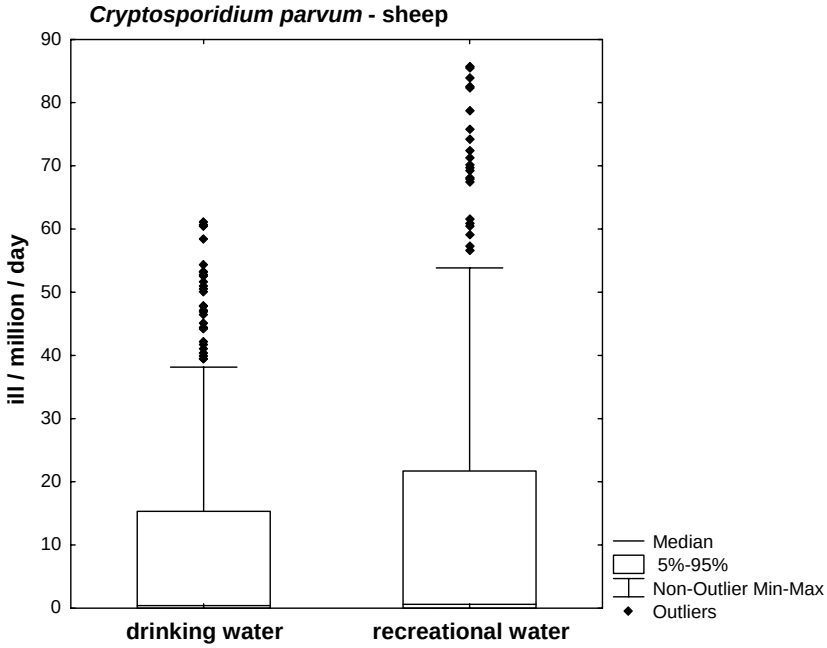



**Figure 5.** Number of people ill / million exposed / days of exposure to **a)** airborne, and **b)** waterborne *Cryptosporidium parvum from* a sheep abattoir (3000 sheep). Refer to Table 2 for numerical values.

# Discussion

## *Relative risk of exposure pathways*

The results from the cattle (1000 cattle) and sheep (3000 sheep) abattoir scenarios (Table 2; Fig. 4, 5) suggest that the risk of human illness from *Cryptosporidium parvum* is greatest for airborne exposure and least for waterborne exposure to the pathogen. For both cattle and sheep pathogen sources, the relative ranking of the 5 modelled exposure pathways is:

- Spray drift irrigation
- Yard air
- Facility air
- Recreational water
- Drinking water

*higher risk*

*lower risk*

For the three pathways of airborne exposure, contaminated spray drift would be expected to have the highest risk as the wastewater stream (after processing and treatment) has a higher pathogen load than the contaminated air released from the livestock yard or the air stream after processing and treatment in the abattoir (see total pathogen output in Appendix C). For waterborne exposure, contaminated drinking water would be expected to be lower risk than recreational water based on the assumption in the model that drinking water, unlike recreational water, undergoes a treatment step before possible ingestion.

## *Scale of the risk*

According to the model, if one million people/day were exposed to *C. parvum* via contaminated spray drift from a cattle abattoir, a median of 803400 people would be infected with enough pathogens to cause illness. This compares with 5890 people infected by *C. parvum* sourced from sheep. The median estimates of risk for waterborne exposure to *C. parvum* from a cattle abattoir are 168 people/day from contaminated recreational water and 114 people/day from drinking water. In comparison, for a sheep abattoir, the median number of people ill/ million exposed/ days of exposure, is less than 1 person/day for both waterborne pathways. For the airborne cattle and sheep abattoir scenarios, the estimated maximum number of people ill per million is 1 million. This is an artefact of the dose response model; as $D/k$ in the dose response equation become larger, $e^{-D/k}$ approaches zero (refer to the dose response equation in an earlier section). We note that there are no documented cases of human illness from exposure to *C. parvum* via the inhalation route. Therefore, although the estimated median and maximum number of people ill is large for airborne exposure, these estimates may be irrelevant if inhalation is not a plausible exposure route.

The median risk of exposure from a 1000 cattle/day abattoir is a lot higher than from a 3000 sheep/day abattoir for all airborne and waterborne pathways. This is most likely due to the higher median concentration of *C. parvum* in the faeces of cattle compared to sheep, consistent with sensitivity analyses (Attachment 2), which indicate that the model is most sensitive to the concentration of the pathogen in faeces. The prevalence of the pathogen is also slightly higher in cattle than sheep, and hence there is a greater chance of infected cattle entering the abattoir.

The estimated risk of illness from airborne and waterborne exposure to *C. parvum* has a wide distribution (Fig. 4, 5). This is due to uncertainties in the input parameters such as pathogen prevalence, concentration in faeces and dose response. As noted above, sensitivity analyses for the cattle abattoir scenario indicate that each output in the model is highly sensitive to the concentration of the pathogen in cattle faeces (Attachment 2). This means that the higher the concentration of *C. parvum* in cattle faeces, the greater the risk of illness from airborne or waterborne exposure. The prevalence of the pathogen, dose response parameter and the wind speed (in the case of airborne pathways) are the next most important parameters in influencing the final outputs. The wind speed has a negative correlation to the risk of illness because the volume of air potentially carrying pathogens is a function of the wind speed. The larger the volume, the more dilute is the effective concentration of the pathogen in the breathing volume of air. This means there is a lower risk of exposure to the pathogen.

Importantly, for the airborne pathways, the sensitivity analysis suggests that the wind speed is not one of the most important parameters in influencing the model outputs. This suggests that in refining the model it

might be important to constrain the uncertainty in the other parameters before attempting to refine the way in which air emissions are modelled.

## *Conclusion*

Given the sensitivity of the outputs to the model parameters, especially to the concentration of *C. parvum* in faeces, the illness estimates generated by the model will be subject to a large amount of uncertainty because the inputs themselves are uncertain. Although we have attempted to deal with this explicitly through a stochastic simulation approach, there is no guarantee that we have captured the full range of uncertainties. Further information on, say, dose response might reduce uncertainty or increase uncertainty by revealing a greater range of responses than previously documented. The results generated by the model, especially the absolute values, should therefore be interpreted with caution. Greater confidence can be placed in relative rankings, but even these could change as a result of different characteristics of individual abattoirs. The results of this 'generic abattoir' model should be used to identify where more information is needed and guide more specific investigations for individual abattoirs. We note that there is a more complete dataset available for cattle than sheep and therefore in the absence of further data, the *Cryptosporidium parvum* model is perhaps more applicable to cattle abattoir situations. We also note that the available input data for pathogen prevalence and concentration in animal faeces is not based on Australian studies.

In order to refine the estimates of risk for human exposure to *Cryptosporidium parvum*, the model would require:

- Expert opinion on whether *C. parvum* can be transmitted and survive in an airborne state
- Expert opinion on whether *C. parvum* can cause human illness via inhalation exposure, and if so, information on the dose response curve
- Australian data on pathogen prevalence and concentration in faeces for cattle and sheep
- A better understanding of the human dose response at low doses.

# Attachment 1:

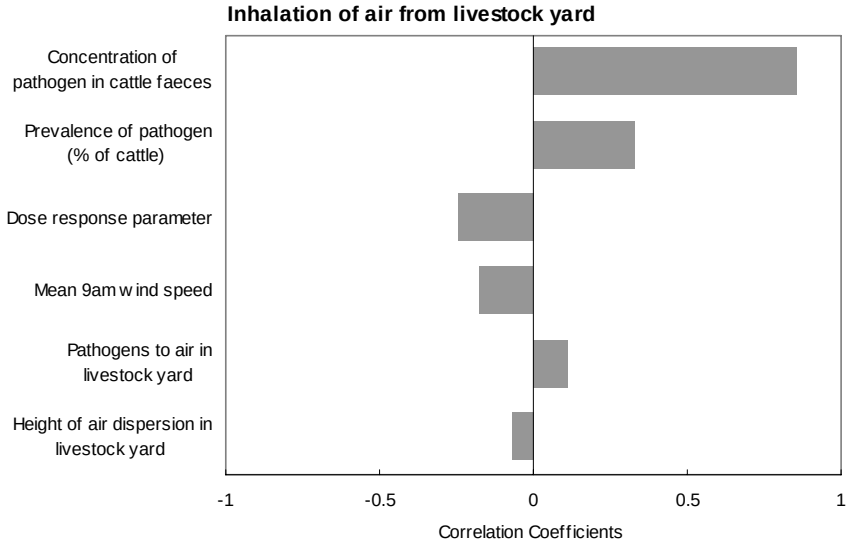
**Table A1.** Input data used to simulate *C. parvum* load and dose response for pathogens entering and leaving an abattoir

| ***Cryptosporidium parvum*** | **Distribution** | | | | **Source of data** | **Notes on data** |
|---|---|---|---|---|---|---|
| | | min | most likely | max | | |
| Prevalence of pathogen carrier cattle (%) | PERT | 0 | 1.1 | 66.4 | 1. Hoar *et al*. (2001); 2. Scott *et al*. (1995) | 1. Most likely, based on Californian cattle; 2. Maximum, based on cattle in Scotland. Minimum estimated as zero. |
| Faeces contamination in carrier cattle (pathogens log/g) | PERT | 0 | 10 | 10.04 | Anon 1998e (Nicholson report) | UK cattle result plus 10% to estimate maximum, zero estimated minimum. |
| Prevalence of pathogen carrier sheep (%) | PERT | 0 | 1.1 | 66.4 | No data available | Assumed same as for cattle. |
| Faeces contamination in carrier sheep (pathogens log/g) | PERT | 0 | 7.81 | 7.85 | Svoboda *et al*. (1997) | UK lambs result plus 10% to estimate maximum, zero estimated minimum. |
| Drinking water treatment efficacy | PERT | 0.9 | 0.928 | 0.99 | Rose *et al*. (1996) | |
| Exponential dose response | UNIFORM (k value) | 17.5 | simulated | 2980 | Messner *et al*. (2001) | The range of exponential human dose response curves based on studies of 3 isolates of *C. parvum* from calves. |

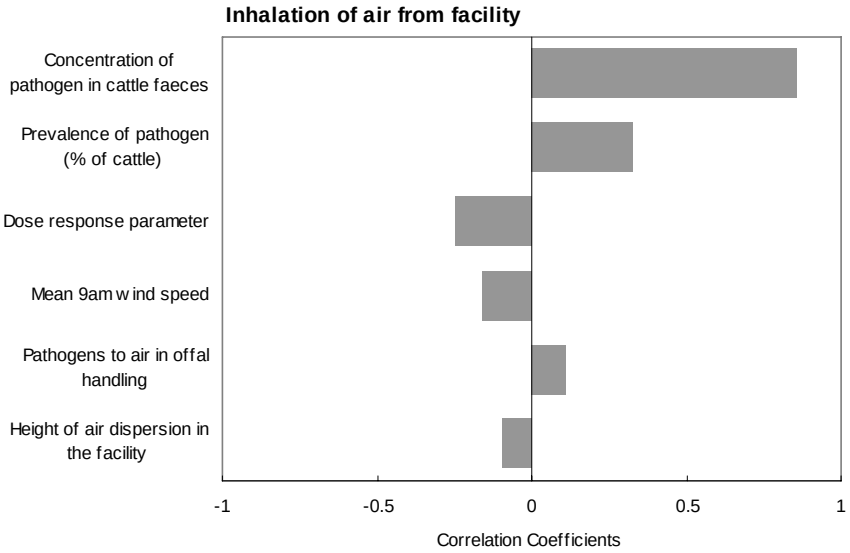
# Attachment 2:

**Figure A1.** Sensitivity analyses for number ill / million exposed / days exposed to airborne and waterborne *Cryptosporidium parvum* based on a cattle abattoir scenario. **a)** inhalation of air from livestock yard; **b)** inhalation of air from the facility (abattoir); **c)** inhalation of spray drift; **d)** ingestion of drinking water; **e)** ingestion of recreational water. Sensitivities are only shown for parameters where the correlation value is greater than or equal to +/- 0.05.

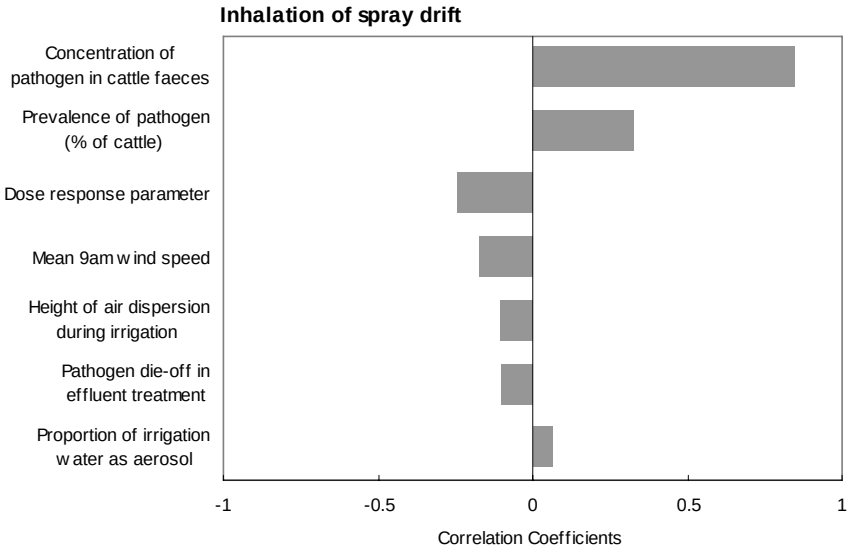
**a)**



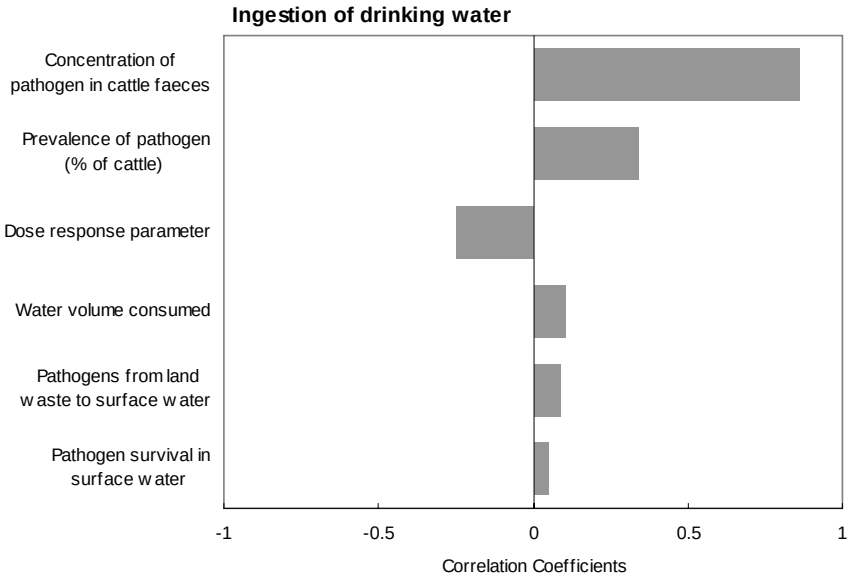

**b)**



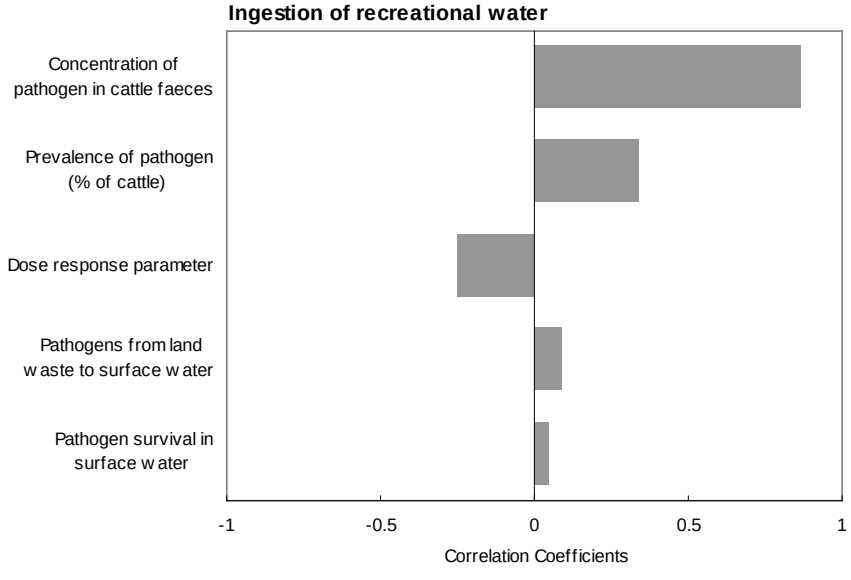
**c)**


Inhalation of spray drift
Concentration of pathogen in cattle faeces
Prevalence of pathogen (% of cattle)
Dose response parameter
Mean 9am wind speed
Height of air dispersion during irrigation
Pathogen die-off in effluent treatment
Proportion of irrigation water as aerosol
-1
-0.5
0
0.5
1
Correlation Coefficients

**d)**


Ingestion of drinking water
Concentration of pathogen in cattle faeces
Prevalence of pathogen (% of cattle)
Dose response parameter
Water volume consumed
Pathogens from land waste to surface water
Pathogen survival in surface water
-1
-0.5
0
0.5
1
Correlation Coefficients

**e)**


Ingestion of recreational water
Concentration of pathogen in cattle faeces
Prevalence of pathogen (% of cattle)
Dose response parameter
Pathogens from land waste to surface water
Pathogen survival in surface water
-1
-0.5
0
0.5
1
Correlation Coefficients

# APPENDIX I. QUANTITATIVE RISK ASSESSMENT OF *LISTERIA MONOCYTOGENES* EMISSIONS FROM ABATTOIRS

Sporadic cases of human listeriosis are reported in Australia caused by *Listeria monocytogenes* (*L. monocytogenes*). The highest risk groups include pregnant women and their foetuses, neonates, the elderly, and immunocompromised adults. However, there is increasing evidence to suggest that normal, healthy individuals can become infected by the organism in an outbreak. The symptoms of listeriosis range from mild febrile gastroenteritis and flu-like symptoms to the reproductive forms which may cause abortions, stillborns or premature births in pregnant women. The source of the organism can be domestic and wild animals, poultry, soil, fish, crustaceans, vegetables, water, sewage and mud. In Australia, the disease in animals occurs largely in ruminants and small marsupials.

*L. monocytogenes* is recognised as a foodborne pathogen, it is not a zoonotic or soilborne disease as first thought (Sutherland and Porritt, 1997). Although humans have become infected after direct contact with diseased animals, and soil may often be the origin of the organism, a World Health Organisation (WHO) Informal Working Group considered *L. monocytogenes* as an environmental contaminant whose primary means of transmission to humans is through food contaminated during production and processing (Anon. 1988).

There are many possible routes for the transmission of *Listeria* to humans other than via food. There is evidence in the literature of animal to human, insect to human, human to human and plant/soil to human transmission. Cases of human listeriosis from inhaling airborne dust and dirt have also been reported. In meat processing environments, transport, chilled storage and packaging areas are considered the most important sites for isolation of *L. monocytogenes*. Reported epidemiological studies have shown that although the organism is widespread in foods and the environment, and humans are frequently exposed to it, listeriosis is comparatively rare. The minimum infective dose for foodborne listeriosis is not well known, although data suggests that greater than $10^3$ cfu/g may be needed to cause disease (Sutherland and Porritt, 1997).

## Methods

The generic quantitative risk analysis model described in Appendix C was customised for *L. monocytogenes* by altering the following parameters (based on available information in the scientific literature):

- Pathogen prevalence in sheep and cattle
- Concentration of pathogen in faeces
- Dose-response relationship

The model was used to calculate the number of people ill / million exposed / days of exposure to airborne and waterborne *L. monocytogenes* sourced from a cattle and sheep abattoir. The volume of the groundwater supply was set to zero in the model, and hence the potential risk from exposure to contaminated groundwater was not simulated. Sensitivity analyses for a cattle abattoir scenario were performed for each output in order to determine which input parameters have the greatest influence on the estimated number of people ill.

### *Prevalence*

Input data on the prevalence of *L. monocytogenes* in cattle and sheep have been estimated from studies undertaken in the UK or another temperature European country (Attachment 1). The constructed distributions for *L. monocytogenes* prevalence differ between cattle and sheep, with a much higher ‘most likely’ prevalence and broader range for cattle (Figure 1).

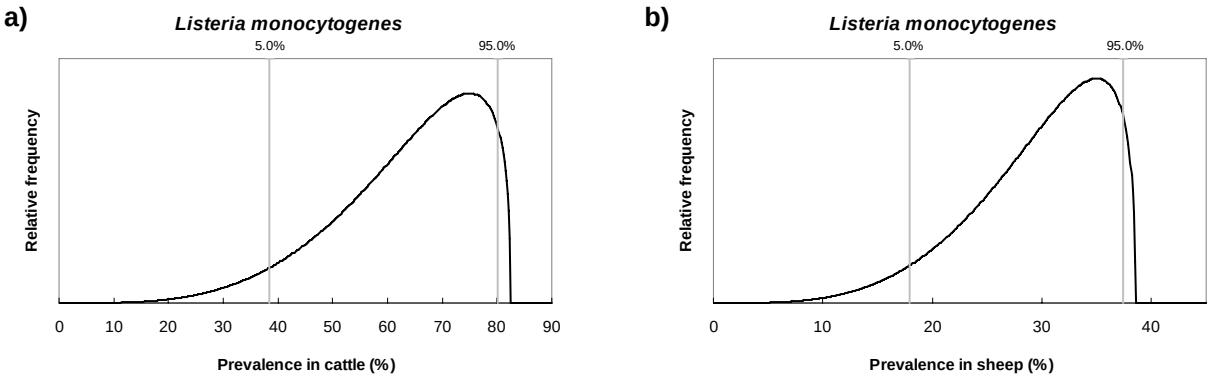



**Figure 1.** Distributions of the prevalence of *L. monocytogenes* in **a)** cattle and **b)** sheep used as inputs into the model.

## *Concentration*

The model assumes that *L. monocytogenes* are present in the gastro-intestinal system of infected cattle and sheep. The concentration of the organism in the faeces is assumed to be representative of the concentration in the entire gastro-intestinal tract. Input data on the concentration of *L. monocytogenes* in sheep faeces have been estimated from studies reported in the literature (Attachment 1). In the absence of specific information for cattle, the same values for concentration have been used as for sheep. The distribution for *L. monocytogenes* concentration in sheep is shown in Figure 2.

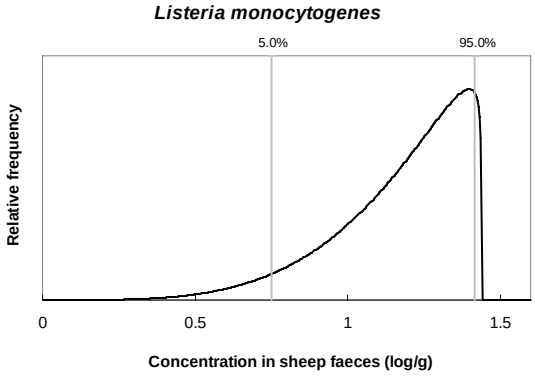



**Figure 2.** Distribution of the concentration of *L. monocytogenes* in sheep used as an input into the model. In the absence of cattle-specific data, the concentration of *L. monocytogenes* in cattle is assumed to be the same as in sheep.

## *Dose response*

No human dose-response data was available in the literature for *L. monocytogenes*. Instead, the dose response used in the model was derived by combining results from 2 animal dose-response studies, in which mice were orally dosed with *L. monocytogenes*. Available literature describes these dose response curves as Beta Poisson distributions, where the probability of infection depends on the dose, *D*, and the dose-response parameters $\varepsilon$ and $\beta$. The Beta Poisson distribution is equivalent to the Weibull-Gamma distribution where the chi ($\chi$) parameter is equal to 1.

The Weibull-Gamma dose-response equation has the form:

$$\text{Probability of infection} = 1 - [(1 + (\text{dose}^{\chi}/\beta))]^{-\varepsilon}$$

By varying the beta (β) parameter of the Weibull-Gamma distribution, we captured the results from the reported dose response models, within the range of pathogen ingestion/inhalation calculated by the model. The slope of the resulting dose response curve varies with β, which is simulated by a uniform distribution (Table A1). Depending on the β value simulated, the dose response curve lies between the upper and lower curves shown in Figure 3. The dose range simulated by the model was low, with the 95$^{th}$ percentile of less than one microorganism potentially inhaled in spray drift from irrigation water.

The model assumes that humans can be infected by *L. monocytogenes* through ingestion as well as inhalation of the pathogen and that the dose response is the same for both modes of infection. Dose-response studies are generally performed at high doses. Hence extrapolation from high doses studied in the literature to low doses simulated in this model may result in inaccurate estimates of the dose response.

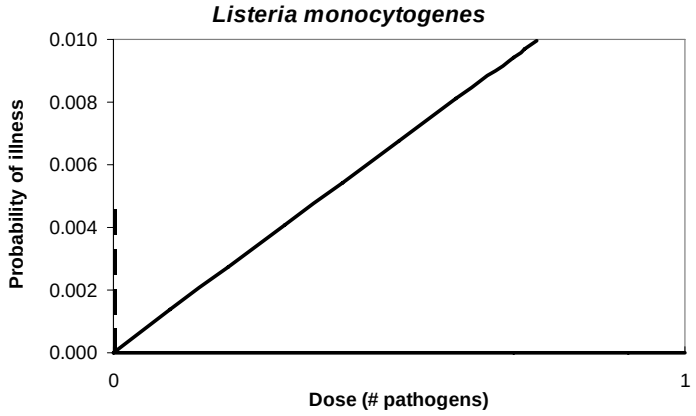



**Figure 3.** Dose response relationship for *L. monocytogenes*. The vertical line represents the 95$^{th}$ percentile pathogen dose for spray drift, simulated by the model for a cattle abattoir scenario. The upper and lower dose response curves (the lower curve sits on the x-axis) represent the range of dose responses used in the model created by varying the dose response parameter β.

## Results

The model was run separately for a hypothetical cattle (1000 cattle) and a hypothetical sheep (3000 sheep) abattoir. Stabilisation of the model outputs took 4400 iterations for the cattle scenario and 3700 iterations for the sheep scenario.

Simulated input values for the three parameters unique to the *L. monocytogenes* model are shown in Table 1. The simulated outputs are given in Table 2 and illustrated graphically in Figures 4 and 5.

**Table 1.** Simulated inputs for the *L. monocytogenes* model from Figures 1, 2 and 3

| | **Prevalence of pathogen (%)** | | **Concentration of pathogen in faeces (log/g)** | | **Dose response parameter** | |
|---|---|---|---|---|---|---|
| | Cattle | Sheep | Cattle | Sheep | Cattle | Sheep |
| Minimum | 11.93 | 4.25 | 0.27 | 0.28 | 31.0 | 23.0 |
| 5$^{th}$ percentile | 38.49 | 18.25 | 0.74 | 0.75 | 1903.6 | 1860.4 |
| 50$^{th}$ percentile | 66.13 | 30.91 | 1.22 | 1.22 | 18347.1 | 17934.2 |
| 95$^{th}$ percentile | 80.36 | 37.45 | 1.42 | 1.42 | 34260.9 | 34249.9 |
| Maximum | 82.48 | 38.47 | 1.44 | 1.44 | 35976.8 | 35995.1 |

**Table 2.** Summary of simulated outputs: number of people ill / million exposed / days of exposure to airborne and waterborne *L. monocytogenes* from **a)** a cattle abattoir (1000 cattle) and **b)** a sheep abattoir (3000 sheep). See Figures 4 and 5 for graphical representation of results

**a)**

**CATTLE**

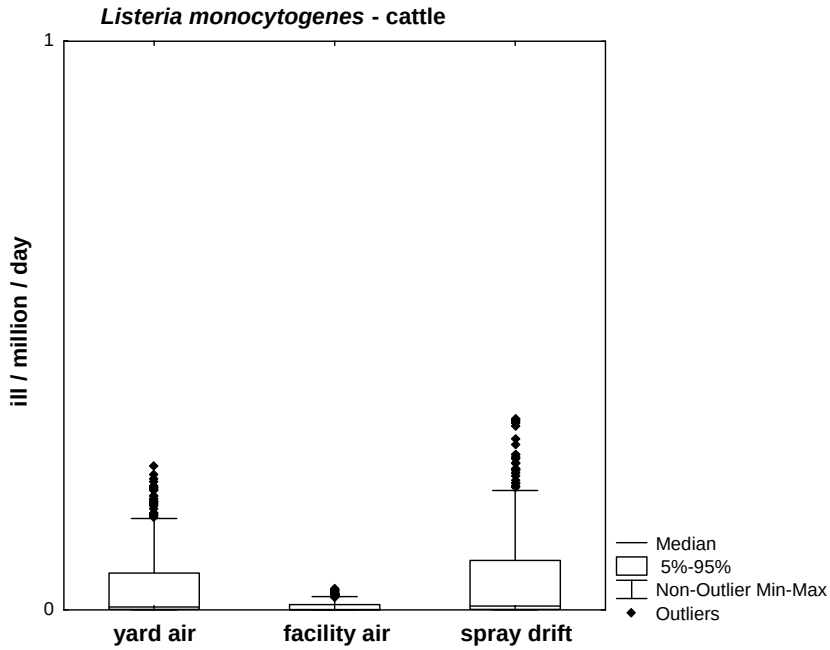
| Exposure route | Yard air | Facility air | Spray drift | Drinking water | Recreational water |
|---|---|---|---|---|---|
| Minimum | 0.00015 | 0.00001 | 0.00011 | 2.00E-09 | 2.42E-08 |
| 5$^{th}$ percentile | 0.0009 | 0.0001 | 0.0010 | 1.78E-08 | 1.64E-07 |
| 50$^{th}$ percentile | 0.005 | 0.001 | 0.007 | 9.77E-08 | 8.62E-07 |
| 95$^{th}$ percentile | 0.06 | 0.01 | 0.09 | 1.02E-06 | 8.46E-06 |
| Maximum | 16 | 2 | 12 | 0.0001 | 0.0003 |

**b)**

**SHEEP**

| Exposure route | Yard air | Facility air | Spray drift | Drinking water | Recreational water |
|---|---|---|---|---|---|
| Minimum | 1.30E-05 | 2.94E-06 | 1.62E-05 | 4.44E-10 | 4.00E-09 |
| 5$^{th}$ percentile | 1.24E-04 | 1.77E-05 | 1.50E-04 | 2.22E-09 | 2.31E-08 |
| 50$^{th}$ percentile | 0.0008 | 0.0001 | 0.0010 | 1.38E-08 | 1.22E-07 |
| 95$^{th}$ percentile | 0.009 | 0.002 | 0.013 | 1.53E-07 | 1.29E-06 |
| Maximum | 1.75 | 0.23 | 2.55 | 1.61E-05 | 0.0001 |

**a)**




**b)**

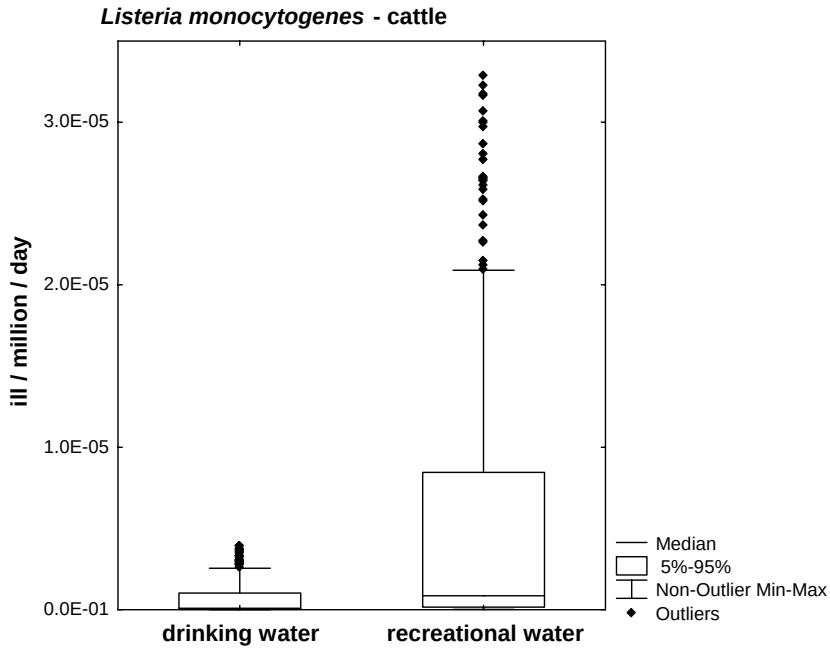
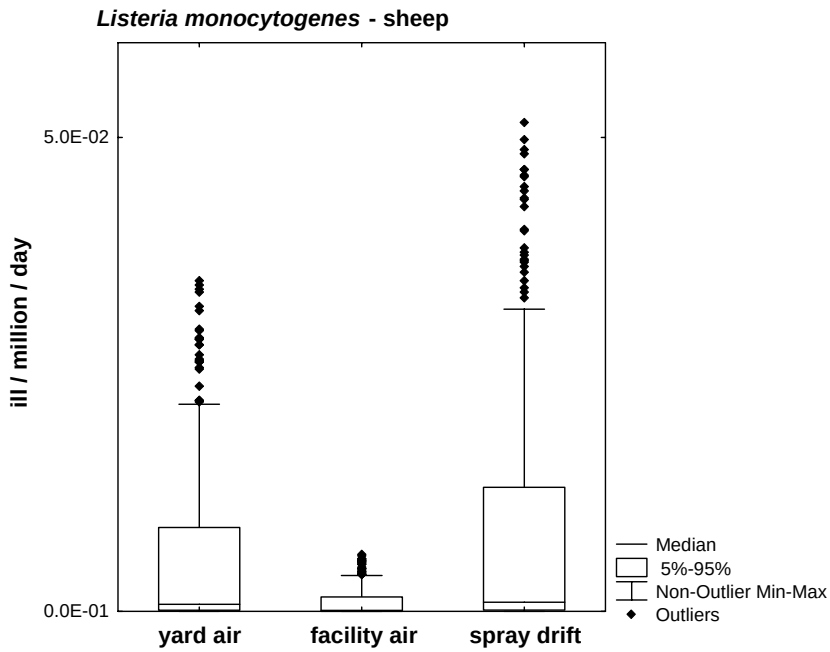



**Figure 4.** Number of people ill / million exposed / days of exposure to **a)** airborne, and **b)** waterborne *L. monocytogenes* from a cattle abattoir (1000 cattle). Refer to Table 2 for numerical values.

**a)**




**b)**

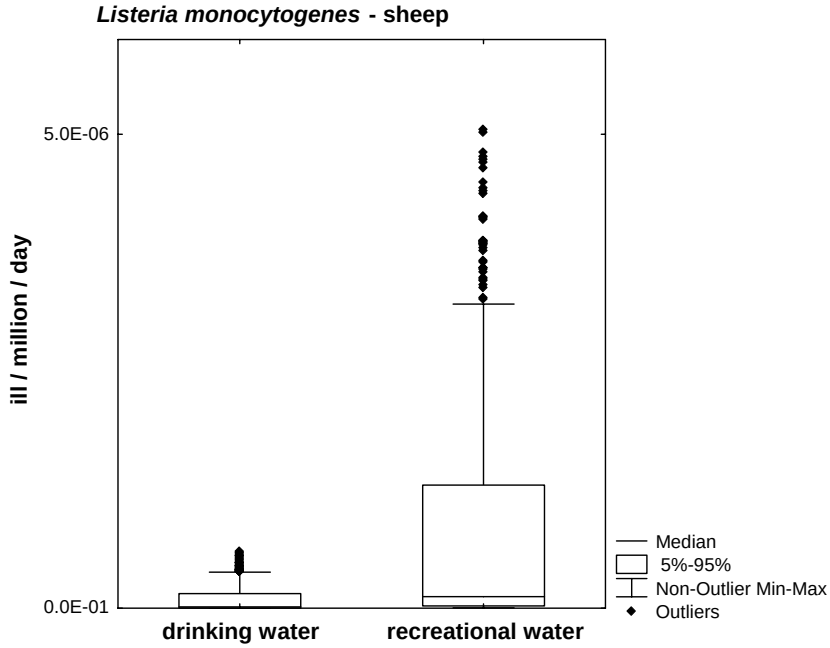



**Figure 5.** Number of people ill / million exposed / days of exposure to **a)** airborne, and **b)** waterborne *L. monocytogenes* from a sheep abattoir (3000 sheep). Refer to Table 2 for numerical values.

# Discussion

## *Relative risk of exposure pathways*

The results from the sheep (3000 sheep) and cattle (1000 cattle) abattoir scenarios (Table 2; Fig. 4, 5) suggest that the risk of human illness from *L. monocytogenes* is greatest for airborne exposure and least for waterborne exposure to the pathogen. For both cattle and sheep pathogen sources, the relative ranking of the 5 modelled exposure pathways is:

- Spray drift irrigation
- Yard air
- Facility air
- Recreational water
- Drinking water

*higher risk*

*lower risk*

Based on the median result, the model indicates similar risks for exposure to spray drift and yard air. For the three pathways of airborne exposure, contaminated spray drift would be expected to have the highest risk as the wastewater stream (after processing and treatment) has a higher pathogen load than the contaminated air released from the livestock yard or the air stream after processing and treatment in the abattoir (see total pathogen output in Appendix C). For waterborne exposure, contaminated drinking water would be expected to be lower risk than recreational water based on the assumption in the model that drinking water, unlike recreational water, undergoes a treatment step before possible ingestion.

## *Scale of the risk*

According to the model, if one million people/day were exposed to *L. monocytogenes* via any of the 3 airborne routes from a cattle or sheep abattoir, less than 1 person/day would be infected. However, the maximum estimates of risk suggest that if one million people/day were exposed to *L. monocytogenes* via contaminated yard air from a cattle abattoir, approximately 16 people/day could be infected with enough pathogens to cause illness. This compares with a maximum estimate of 2 people infected by *L. monocytogenes* sourced from sheep. We note that there is little documented evidence of human illness from exposure to *L. monocytogenes* via the inhalation route. For both waterborne pathways, the maximum estimate of risk (number of people ill / million exposed / days of exposure) is less than 1 person/day for the cattle and sheep abattoir scenarios.

The median risk of exposure from a 1000 cattle/day abattoir is only slightly higher than from a 3000 sheep/day abattoir for all airborne and waterborne pathways despite the far greater prevalence of *L. monocytogenes* in the faeces of cattle compared to sheep. This is consistent with the sensitivity analyses (Attachment 2), which indicate that the model is not very sensitive to pathogen prevalence.

The estimated risk of illness from airborne and waterborne exposure to *L. monocytogenes* has a wide distribution (Fig. 4, 5). This is due to uncertainties in the input parameters such as pathogen prevalence, concentration in faeces and dose response. Sensitivity analyses for the cattle abattoir scenario indicate that each output in the model is highly sensitive to the dose response parameter $\beta$. This is not unexpected given the wide range of possible values for the $\beta$ parameter in the model. This analysis suggests that in refining the model it would be important to constrain the uncertainty in the $\beta$ parameter. We note that the dose response curve was derived from animal studies rather than humans. The wind speed (in the case of airborne pathways) and the concentration of the pathogen in cattle faeces are the next most important parameters in influencing the final outputs. The wind speed has a negative correlation to the risk of illness because the volume of air potentially carrying pathogens is a function of the wind speed. The larger the volume, the more dilute is the effective concentration of the pathogen in the breathing volume of air. This means there is a lower risk of exposure to the pathogen. Conversely, the concentration in faeces has a positive correlation, which means that the higher the concentration of *L. monocytogenes* in cattle faeces, the greater the risk of illness from airborne or waterborne exposure.

## *Conclusion*

Given the sensitivity of the outputs to the model parameters, especially to the dose response parameter and the concentration of *L. monocytogenes* in faeces, the illness estimates generated by the model will be subject to a large amount of uncertainty because the inputs themselves are uncertain. Although we have attempted to deal with this explicitly through a stochastic simulation approach, there is no guarantee that we have captured the full range of uncertainties. Further information on, say, dose response might reduce uncertainty or increase uncertainty by revealing a greater range of responses than previously documented. The results generated by the model, especially the absolute values, should therefore be interpreted with caution. Greater confidence can be placed in relative rankings, but even these could change as a result of different characteristics of individual abattoirs. The results of this 'generic abattoir' model should be used to identify where more information is needed and guide more specific investigations for individual abattoirs. We note that there is a more complete dataset available for sheep than cattle and therefore in the absence of further data, the *L. monocytogenes* model is perhaps more applicable to sheep abattoir situations. We also note that the available input data for pathogen prevalence and concentration in animal faeces is not based on Australian studies and that the dose response information is based on animal studies rather than humans.

In order to refine the estimates of risk for human exposure to *Listeria monocytogenes*, the model would require:

- Expert opinion on whether *L. monocytogenes* can be transmitted and survive in an airborne state
- Expert opinion on whether *L. monocytogenes* can cause human illness via inhalation exposure, and if so, information on the dose response curve
- Data on the human dose response curve for ingestion and a better understanding of the dose response at low doses
- Australian data on pathogen prevalence and concentration in faeces for cattle and sheep.

# Attachment 1:

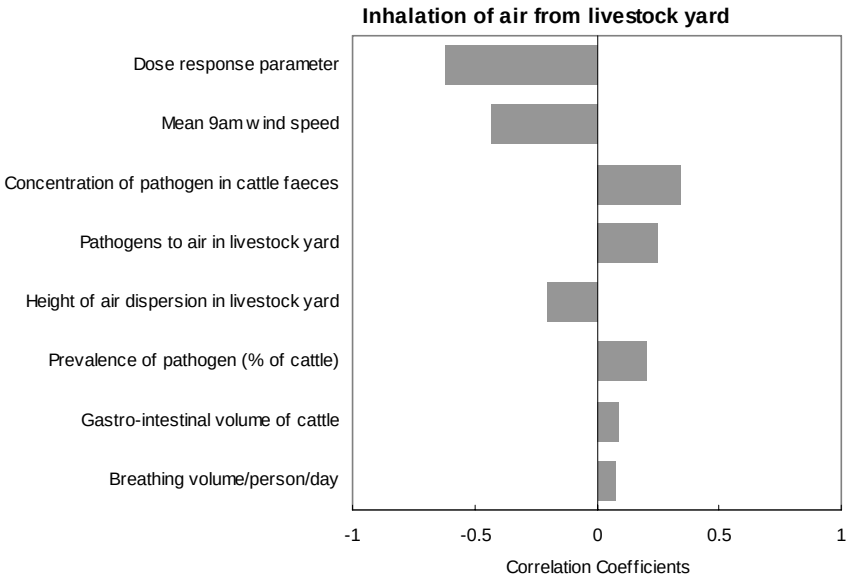
**Table A1.** Input data used to simulate *L. monocytogenes* load and dose response for pathogens entering and leaving an abattoir

| ***L. monocytogenes*** | **Distribution** | | | | **Source of data** | **Notes on data** |
|---|---|---|---|---|---|---|
| | | min | most likely | max | | |
| Prevalence of pathogen carrier cattle (%) | PERT | 0 | 75 | 82.5 | Nicholson *et al.* (1999) | 1. Most likely based on cattle in the UK or other temperature European country. 2. Minimum estimated as zero; maximum estimated as 10% greater than median. |
| Faeces contamination in carrier cattle (pathogens log/g) | PERT | 0 | 1.40 | 1.44 | Fenlon *et al.* (1996) in Nicholson *et al*. (1999) | Assumed same as for sheep |
| Prevalence of pathogen carrier sheep (%) | PERT | 0 | 35 | 38.5 | Nicholson *et al.* (1999) | 1. Most likely based on cattle in the UK or other temperature European country. 2. Minimum estimated as zero; maximum estimated as 10% greater than median. |
| Faeces contamination in carrier sheep (pathogens log/g) | PERT | 0 | 1.40 | 1.44 | Fenlon *et al.* (1996) in Nicholson *et al*. (1999) | 1. Most likely based on reported level in sheep. 2. Minimum estimated as zero; maximum estimated as 10% greater than median. |
| Weibull-Gamma dose response | | $\varepsilon$ | $\beta$ | $\chi$ | Audurier *et al*. (1980) and Donnelly *et al*. (1989) in Haas *et al*. (1998) | We altered the Beta parameter of the Weibull-Gamma dose response model to capture the full range of 2 reported animal dose response curves within the range of pathogen loads calculated by the model. |
| *fixed parameters* | | 0.25 | | 1 | | |
| UNIFORM | | min | 18.13 | | | |
| | | max | 36000 | | | |

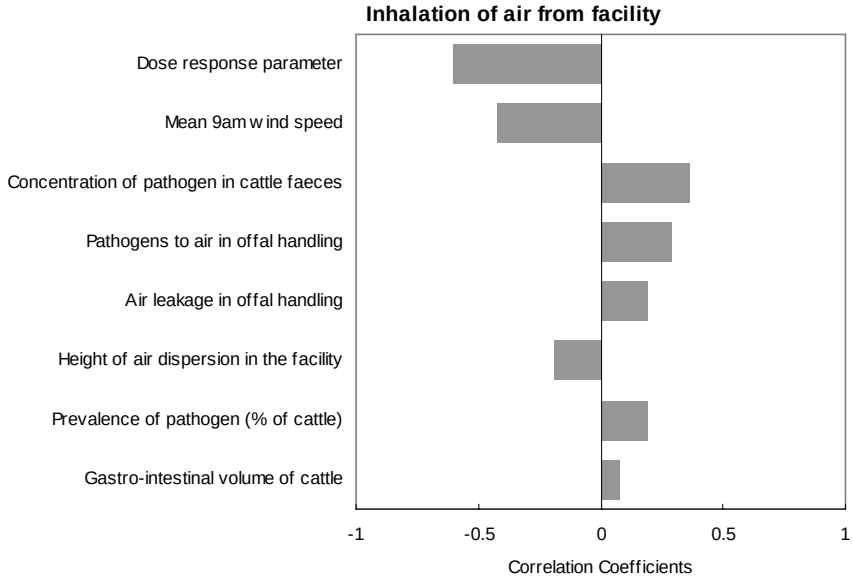
# Attachment 2:

**Figure A1.** Sensitivity analyses for number ill / million exposed / days exposed to airborne and waterborne *L. monocytogenes* based on a cattle abattoir scenario. **a)** inhalation of air from livestock yard; **b)** inhalation of air from the facility (abattoir); **c)** inhalation of spray drift; **d)** ingestion of drinking water; **e)** ingestion of recreational water. Sensitivities are only shown for parameters where the correlation value is greater than or equal to +/- 0.05.

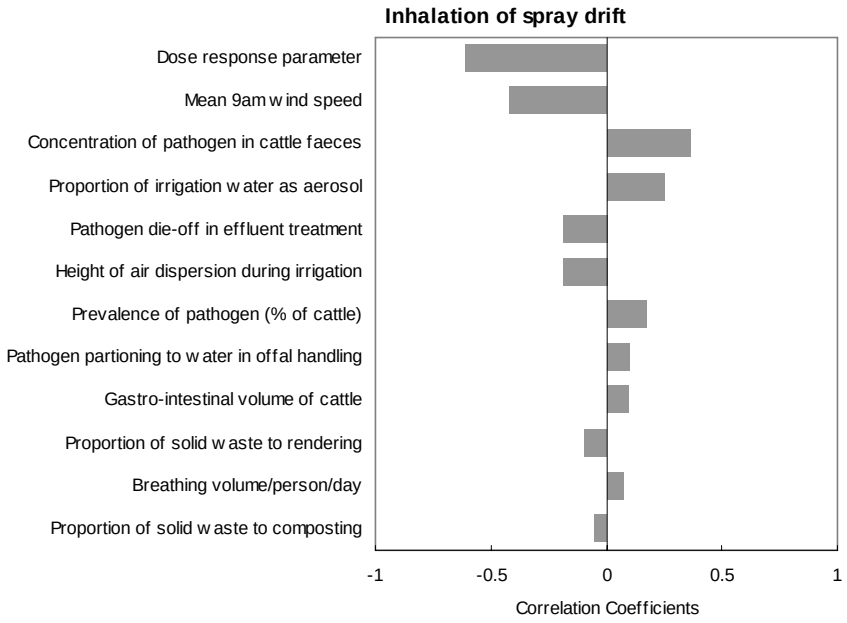
**a)**



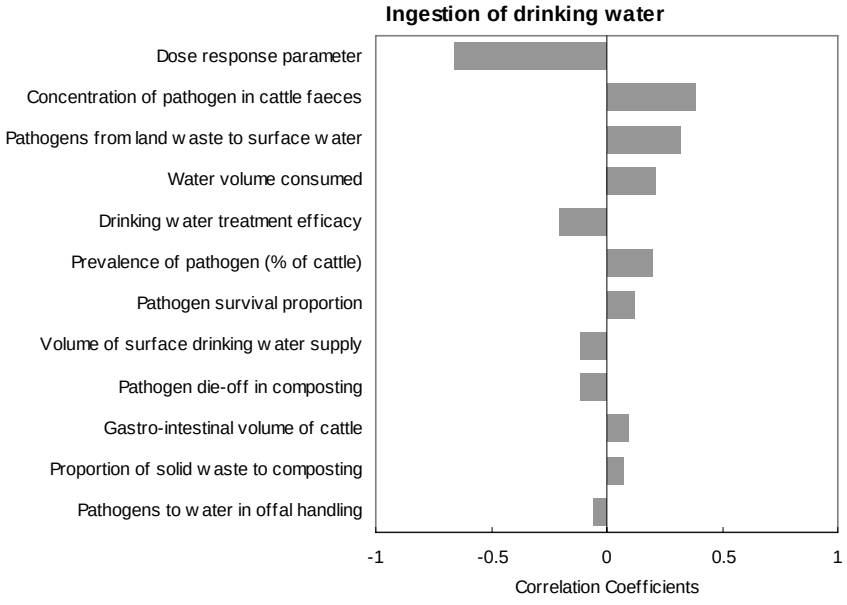

**b)**



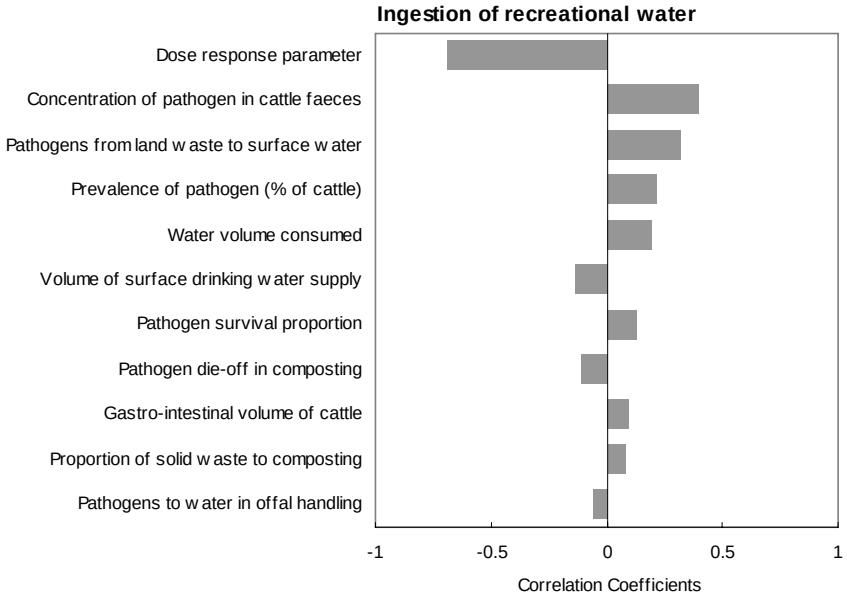
**c)**


Inhalation of spray drift
Dose response parameter
Mean 9am wind speed
Concentration of pathogen in cattle faeces
Proportion of irrigation water as aerosol
Pathogen die-off in effluent treatment
Height of air dispersion during irrigation
Prevalence of pathogen (% of cattle)
Pathogen partioning to water in offal handling
Gastro-intestinal volume of cattle
Proportion of solid waste to rendering
Breathing volume/person/day
Proportion of solid waste to composting
-1
-0.5
0
0.5
1
Correlation Coefficients

**d)**


Ingestion of drinking water
Dose response parameter
Concentration of pathogen in cattle faeces
Pathogens from land waste to surface water
Water volume consumed
Drinking water treatment efficacy
Prevalence of pathogen (% of cattle)
Pathogen survival proportion
Volume of surface drinking water supply
Pathogen die-off in composting
Gastro-intestinal volume of cattle
Proportion of solid waste to composting
Pathogens to water in offal handling
-1
-0.5
0
0.5
1
Correlation Coefficients

**e)**


Ingestion of recreational water
Dose response parameter
Concentration of pathogen in cattle faeces
Pathogens from land waste to surface water
Prevalence of pathogen (% of cattle)
Water volume consumed
Volume of surface drinking water supply
Pathogen survival proportion
Pathogen die-off in composting
Gastro-intestinal volume of cattle
Proportion of solid waste to composting
Pathogens to water in offal handling
-1
-0.5
0
0.5
1
Correlation Coefficients

# SPREADSHEET FOR THE *CAMPYLOBACTER JEJUNI* MODEL

## Quantitative risk assessment of microbial emissions from abattoirs

**A stochastic model based on Monte Carlo simulations in @RISK**

To run this model the Excel Add-In "@risk" must be installed (Palisade)
Information must be entered or confirmed in each white cell to run the model

User defined fixed input that may be based on a particular abattoir
Number derived directly from a Monte Carlo simulation based on user defined parameters derived from the literature
Value derived from other inputs and/or outputs, fixed or simulated
Key model outputs

| Date | *Campylobacter jejuni* |
|---|---|
| 28/10/2002 | |

| | Module and parameter | Fixed & derived values | Pathogens distributed to the environment (log/day) | | | Variable input values | | | Units | *Record source of input data in this column* |
|---|---|---|---|---|---|---|---|---|---|---|
| A | ***Pathogen input calculations*** | | Air | Water | Solids | Min | Most likely | Max | Units | |
| 1 | **Cattle per day** | **0** | | | | | | | animals/day | User input |
| | Expressed as tonnes of Hot Standard Carcase Weight | 0.00 | | | | | | | tHSCW | User input of 0.25 t/animal |
| 2 | **Prevalence of pathogen carriers** | **0.39** | | | | 0 | 35 | 92 | percentage | Vanselow & Hornitzky (2001) |
| 3 | **Concentration of pathogen in carrier faeces** | **3.09** | | | | 0 | 2.79 | 7.38 | log/g | Stanley *et al.* (1998a) |
| 4 | Gastro-intestinal volume kg/animal | 51.7 | | | | 40 | 50 | 70 | kg/animal | Ockerman & Hansen (2000) |
| 5 | Concentration of pathogen in other contamination sources in carrier | 0 | | | | 0 | 0 | 0 | log/g | No data |
| 6 | Other sources volume | 0 | | | | 0 | 0 | 0 | kg/animal | No data |
| 7 | **Sheep per day** | **3,000** | | | | | | | animals/day | User input |
| | Expressed as tonnes of Hot Standard Carcase Weight | 60.00 | | | | | | | tHSCW | User input of 0.02 t/animal |
| 8 | **Prevalence of pathogen carriers** | **0.11** | | | | 0 | 4 | 51 | percentage | Vanselow & Hornitzky (2001), Jones *et al.* (1999), Stanley *et al.* (1998b) |
| 9 | **Concentration of pathogen in carrier faeces** | **3.95** | | | | 0 | 4.64 | 5.11 | log/g | Jones *et al.* (1999) |
| 10 | Gastro-intestinal volume | 5.2 | | | | 4.0 | 5.0 | 7.0 | kg/animal | Ockerman & Hansen (2000) |
| 11 | Concentration of pathogen in other contamination sources in carrier | 0 | | | | 0 | 0 | 0 | log/g | No data |
| 12 | Other sources volume | 0 | | | | 0 | 0 | 0 | kg/animal | No data |
| | **Total pathogen load** | **10.19** | | | | | | | **log/day** | |

| B | ***Pathogen output: Livestock yard*** | | Air | Water | Solids | Min | Most likely | Max | Units | |
|---|---|---|---|---|---|---|---|---|---|---|
| 13 | Pathogen load released proportion | 0.25 | | | | 0.20 | 0.25 | 0.30 | proportion | Pers. com. Mike Johns (MLA, 2002) |
| | ***Solids*** | | | | | | | | | |
| | Pathogen partitioning - solids share | 0.95 | | | | | | | proportion | Fraction not partitioned to water/air |
| | Solids contamination pathogens | | | | 9.56 | | | | log/day | |
| | ***Water*** | | | | | | | | | |
| 14 | Pathogen partioning - water share | 0.038 | | | | 0.020 | 0.04 | 0.05 | proportion | Estimate |
| | Water contamination pathogens | | | 8.17 | | | | | log/day | |
| | ***Air*** | | | | | | | | | |
| 15 | Pathogens partitioning - air share | 0.010 | | | | 0.0025 | 0.01 | 0.02 | proportion | Estimate |
| 16 | Air leakage | 0.99 | | | | 0.95 | 0.99 | 1.00 | proportion | Estimate |
| | Air contamination pathogens | | 7.60 | 5.78 | | | | | log/day | |

| | PROCESSING | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| C | ***Pathogen output: Carcase processing*** | | Air | Water | Solids | Min | Most likely | Max | Units | |
| 17 | Pathogen load released proportion | 0.10 | | | | 0.05 | 0.10 | 0.15 | proportion | Pers. com. Mike Johns (MLA, 2002) |
| | ***Solids*** | | | | | | | | | |
| | Pathogen partitioning - solids share | 0.51 | | | | | | | proportion | Fraction not partitioned to water/air |
| | Solids contamination pathogens | | | | 8.89 | | | | log/day | |
| | ***Water*** | | | | | | | | | |
| 18 | Pathogen partioning - water share | 0.49 | | | | 0.44 | 0.48 | 0.58 | proportion | Estimate |
| | Water contamination pathogens | | | 8.88 | | | | | log/day | |
| | ***Air*** | | | | | | | | | |
| 19 | Pathogens partitioning - air share | 0.004 | | | | 0.0001 | 0.004 | 0.010 | proportion | Estimate |
| 20 | Air leakage | 0.02 | | | | 0.01 | 0.02 | 0.05 | proportion | Estimate |
| | Air contamination pathogens | | 5.19 | 6.81 | | | | | log/day | |
| D | ***Pathogen output: Hide processing*** | | Air | Water | Solids | Min | Most likely | Max | Units | |
| 21 | Pathogen load released proportion | 0.03 | | | | 0.01 | 0.03 | 0.05 | proportion | Pers. com. Mike Johns (MLA, 2002) |
| | ***Solids*** | | | | | | | | | |
| | Pathogen partitioning - solids share | 0.71 | | | | | | | proportion | Fraction not partitioned to water/air |
| | Solids contamination pathogens | | | | 8.52 | | | | log/day | |
| | ***Water*** | | | | | | | | | |
| 22 | Pathogen partioning - water share | 0.28 | | | | 0.18 | 0.28 | 0.37 | proportion | Estimate |
| | Water contamination pathogens | | | 8.11 | | | | | log/day | |
| | ***Air*** | | | | | | | | | |
| 23 | Pathogens partitioning - air share | 0.01 | | | | 0.00001 | 0.01 | 0.02 | proportion | Estimate |
| 24 | Air leakage | 0.06 | | | | 0.02 | 0.06 | 0.10 | proportion | Estimate |
| | Air contamination pathogens | | 5.44 | 6.64 | | | | | log/day | |
| E | ***Pathogen output: Offal handling*** | | Air | Water | Solids | Min | Most likely | Max | Units | |
| | Pathogen load released proportion | 0.62 | | | | | | | proportion | Fraction not partitioned to previous steps |
| | ***Solids*** | | | | | | | | | |
| | Pathogen partitioning - solids share | 0.66 | | | | | | | proportion | Fraction not partitioned to water |
| | Solids contamination pathogens | | | | 9.80 | | | | log/day | |
| | ***Water*** | | | | | | | | | |
| 25 | Pathogen partioning - water share | 0.33 | | | | 0.10 | 0.30 | 0.70 | proportion | Estimate |
| | Water contamination pathogens | | | 9.50 | | | | | log/day | |
| | ***Air*** | | | | | | | | | |
| 26 | Pathogens partitioning - air share | 0.01 | | | | 0.00001 | 0.01 | 0.02 | proportion | Estimate |
| 27 | Air leakage | 0.06 | | | | 0.02 | 0.06 | 0.10 | proportion | Estimate |
| | Air contamination pathogens | | 6.76 | 7.95 | | | | | log/day | |
| F | **Total pathogens per stream from processing** | | **6.79** | **9.63** | **10.04** | | | | **log/day** | |

| | TREATMENT | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| G | ***Pathogen output: Rendering*** | | Air | Water | Solids | Min | Most likely | Max | Units | |
| 28 | Proportion of solid waste to rendering | 0.48 | | | | 0.30 | 0.50 | 0.60 | proportion | Estimate |
| | Input pathogens (solids) log/day | 9.73 | | | | | | | log/day | |
| 29 | Pathogen die-off (solids treatment) | 0.98 | | | | 0.96 | 0.98 | 0.99 | proportion | Estimate |
| | Pathogen remaining after treatment log/day | 8.06 | | | | | | | log/day | |
| | Pathogen proportion in solids | 0.99 | | | | | | | proportion | Fraction not partitioned to water/air |
| 30 | Pathogen proportion in water leakage | 0.01 | | | | 0.005 | 0.008 | 0.012 | proportion | Estimate |
| 31 | Pathogen proportion in air leakage | 0.00005 | | | | 0.00001 | 0.00005 | 0.00008 | proportion | Estimate |
| | **Total pathogens per stream from rendering** | | **3.75** | **5.97** | **8.06** | | | | **log/day** | |
| H | ***Pathogen output: Composting - manure & paunch content*** | | Air | Water | Solids | Min | Most likely | Max | Units | |
| 32 | Proportion of solid waste to composting (manure & paunch content) | 0.30 | | | | 0.20 | 0.30 | 0.40 | proportion | Estimate |
| | Input pathogens (solids) log/day | 9.52 | | | | | | | | |
| 33 | Pathogen die-off (solids treatment) | 0.60 | | | | 0.40 | 0.60 | 0.80 | proportion | Estimate |
| | Pathogen remaining after treatment log/day | 9.12 | | | | | | | | |
| | Pathogen proportion in solids | 0.99 | | | | | | | proportion | Fraction not partitioned to water/air |
| 34 | Pathogen proportion in water leakage | 0.01 | | | | 0.005 | 0.008 | 0.012 | proportion | Estimate |
| 35 | Pathogen proportion in air leakage | 0.00005 | | | | 0.00001 | 0.00005 | 0.00008 | proportion | Estimate |
| | **Total pathogens per stream from manure treatment** | | **4.80** | **7.03** | **9.12** | | | | **log/day** | |
| I | ***Pathogen output: Effluent treatment*** | | Air | Water | Solids | Min | Most likely | Max | Units | |
| | Proportion of solids to effluent treatment (suspended solids) | 0.22 | | | | | | | proportion | Fraction not partitioned to rendering/composting |
| | Input pathogens log/day | 9.83 | | | | | | | log/day | |
| 36 | Pathogen die-off (solids treatment) | 0.95 | | | | 0.92 | 0.95 | 0.98 | proportion | Estimate |
| | Pathogen remaining after treatment log/day | 8.52 | | | | | | | log/day | |
| | Pathogen proportion in sludge | 0.50 | | | | | | | proportion | Fraction not partitioned to water/air |
| 37 | Pathogen proportion in water leakage | 0.50 | | | | 0.40 | 0.50 | 0.60 | proportion | Estimate |
| 38 | Pathogen proportion in air leakage | 0.00005 | | | | 0.00001 | 0.00005 | 0.00008 | proportion | Estimate |
| | **Total pathogens per stream from effluent treatment** | | **4.21** | **8.22** | **8.22** | | | | **log/day** | |
| J | ***Pathogen output: Total*** | | **Air** | **Water** | **Solids** | | | | **Units** | |
| | **Total pathogens per stream from processing & treatment** | | **6.79** | **8.22** | **9.20** | | | | **log/day** | Air from livestock yard is excluded in total |

| | WATER CONTAMINATION | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| K | ***Water*** | | | Min | Most likely | Max | Units | |
| 40 | Water volume from abattoir (without rendering) | 500000 | | 2000 | 8000 | 16000 | L/tHSCW/day | MLA (1998) |
| 41 | Water volume from rendering plant | 53 | | 0.75 | 0.875 | 1 | L/tHSCW/day | MLA (1998) |
| | Total water volume to irrigation (L/day) | 500053 | | | | | L/tHSCW/day | |
| | Proportion of irrigation water dispersed as aerosol | 0.31 | | 0.04 | 0.33 | 0.5 | proportion | http://www.agric.wa.gov.au/agency/Pubns/farmnote/1992/f04892.htm |
| | Volume of irrigation water dispersed as aerosol | 155016 | | | | | L/day | |
| | Proportion of irrigation reaching the ground | 0.69 | | | | | proportion | Fraction not dispersed in air |
| 42 | Proportion of irrigation leaked to ground & surface waters | 0.13 | | 0.00 | 0.10 | 0.40 | proportion | Estimate |
| | Volume of irrigation water leaked to surface water | 23002 | | | | | L/day | |
| | Volume of irrigation water leaked to groundwater | 23002 | | | | | L/day | |
| | | | | | | | | |
| L | ***Public health risk: surface drinking water*** | | | Min | Most likely | Max | Units | |
| | Pathogen number from irrigation run-off to surface water | 6.89 | | | | | log/day | |
| 43 | Pathogen proportion from landspread waste to surface water | 0.10 | | 0.01 | 0.10 | 0.20 | proportion | Estimate |
| | Pathogen number from landspread waste to surface water | 8.21 | | | | | log/day | |
| | Total pathogen number in run-off to surface water | 8.23 | | | | | log/day | |
| 44 | Pathogen survival proportion | 0.08 | | 0.05 | 0.08 | 0.12 | proportion | Estimate |
| | Pathogens surviving in leaked surface water | 7.14 | | | | | log/day | |
| 45 | Volume of surface drinking water supply | 732 | | 488 | 732 | 976 | GL | Wyangala Dam near Cowra at 40, 60 and 80% of full capacity |
| | Pathogen concentration in untreated drinking water supply | -4.72 | | | | | log/L | |
| 46 | Drinking water treatment efficacy (pathogen reduction) | 0.99 | | 0.980 | 0.990 | 0.995 | proportion | Estimate |
| | Pathogen concentration in treated drinking water | -6.69 | | | | | log/L | |
| 47 | Water volume consumed | 1.08 | | 0.50 | 1.00 | 2.00 | L/person/day | Estimate |
| | Pathogen intake/person/day surface drinking water | -6.65 | | | | | log/person/day | |
| | Probability of individual using water source becoming ill/day | 2.63E-10 | | | | | probability | |
| | **ill per million per day surface water ingestion** | **2.63E-04** | | | | | number | |
| | | | | | | | | |
| M | ***Public health risk: aquifer (groundwater) as drinking water*** | | | Min | Most likely | Max | Units | |
| | Pathogen number from irrigation run-off to groundwater | 6.89 | | | | | log/day | |
| 48 | Pathogen proportion from landspread waste to groundwater | 0.05 | | 0.01 | 0.05 | 0.10 | proportion | Estimate |
| | Pathogen number from landspread waste to groundwater | 7.91 | | | | | log/day | |
| | Total pathogen number in run-off to groundwater | 7.95 | | | | | log/day | |
| 49 | Pathogen survival proportion | 0.08 | | 0.05 | 0.08 | 0.12 | proportion | Estimate |
| | Pathogens surviving in leaked groundwater | 6.87 | | | | | log/day | |
| 50 | Volume of aquifer drinking water supply | 0 | | 0 | 0 | 0 | GL | No groundwater supply for this example |
| | Pathogen concentration in untreated drinking water supply | 0.00 | | | | | log/L | |
| 51 | Drinking water treatment efficacy (pathogen reduction) | 0.99 | | 0.980 | 0.990 | 0.995 | proportion | Estimate |
| | Pathogen concentration in treated drinking water | 0.00 | | | | | log/L | |
| 52 | Water volume consumed | 0.00 | | 0.00 | 0.00 | 0.00 | L/person/day | No groundwater used in this case |
| | Pathogen intake/person/day | 0.00 | | | | | log/person/day | |
| | Probability of individual using water source becoming ill/day | 0.00E+00 | | | | | probability | |
| | **ill per million per day groundwater ingestion** | **0.00E+00** | | | | | number | |
| | | | | | | | | |
| N | ***Public health risk: recreational swimming*** | | | Min | Most likely | Max | Units | |
| 53 | Water volume consumed | 0.10 | | 0.05 | 0.10 | 0.15 | L/person/day | 100 mL used by Haas *et al.* (2000), Haas (1983) |
| | Pathogen intake/person/day recreational water | -5.72 | | | | | log/person/day | Assumes pathogen concentration of untreated surface drinking water |
| | Probability of becoming ill/day | 2.24E-09 | | | | | probability | |
| | **ill per million per day recreational water ingestion** | **2.24E-03** | | | | | number | |

**AIR CONTAMINATION**

| O | *Public health risk: air contamination from livestock yard (dust)* | | Min | Most likely | Max | Units | |
|---|---|---|---|---|---|---|---|
| 54 | Mean 9am wind speed | 4.8 | 1.2 | 4.9 | 7.7 | km/hr | Wodonga windspeed 1973-1984 (BOM 2001) |
| | Radius of air dispersion | 57000 | | | | m | |
| 55 | Height of air dispersion | 4 | 1 | 4 | 6 | m | Estimate |
| | Volume of air potentially carrying pathogens | 3.91E+10 | | | | $m^3$ | |
| | Pathogen number in volume of infectious air | -3.00 | | | | $log/m^3$ | |
| 56 | Breathing volume/person/day | 10 | 8 | 10 | 12 | $m^3$ | http://www.nhlbi.nih.gov/health/public/lung/other/lungs_hd.pdf |
| | Inhaled pathogens/person/day air contamination | -2.00 | | | | log/person/day | |
| | Probability of becoming ill/day | 1.19E-05 | | | | probability | |
| | **ill per million per day yard air inhalation** | **1.19E+01** | | | | number | |

| P | *Public health risk: air contamination from processing and treatment (facility)* | | Min | Most likely | Max | Units | |
|---|---|---|---|---|---|---|---|
| 54 | Mean 9am wind speed | 4.8 | 1.2 | 4.9 | 7.7 | km/hr | Wodonga windspeed 1973-1984 (BOM 2001) |
| | Radius of air dispersion | 57000 | | | | m | |
| 55 | Height of air dispersion | 4 | 1 | 4 | 6 | m | Estimate |
| | Volume of air potentially carrying pathogens | 3.91E+10 | | | | $m^3$ | |
| | Pathogen number in volume of infectious air | -3.80 | | | | $log/m^3$ | |
| 56 | Breathing volume/person/day | 10 | 8 | 10 | 12 | $m^3$ | http://www.nhlbi.nih.gov/health/public/lung/other/lungs_hd.pdf |
| | Inhaled pathogens/person/day air contamination | -2.80 | | | | log/person/day | |
| | Probability of becoming ill/day | 1.89E-06 | | | | probability | |
| | **ill per million per day facility air inhalation** | **1.89E+00** | | | | number | |

| Q | *Public health risk: air contamination from spray irrigation (droplets)* | | Min | Most likely | Max | Units | |
|---|---|---|---|---|---|---|---|
| | Pathogen number from spray irrigation to aerosol | 7.72 | | | | log/day | |
| 54 | Mean 9am wind speed | 4.8 | 1.2 | 4.9 | 7.7 | km/hr | Wodonga windspeed 1973-1984 (BOM 2001) |
| | Radius of air dispersion | 57000 | | | | m | |
| 55 | Height of air dispersion | 4 | 1 | 4 | 6 | m | Estimate |
| | Volume of air potentially carrying pathogens | 3.91E+10 | | | | $m^3$ | |
| | Pathogen number in volume of infectious air | -2.88 | | | | $log/m^3$ | Assumes pathogen concentration in treated effluent |
| 56 | Breathing volume/person/day | 10 | 8 | 10 | 12 | $m^3$ | http://www.nhlbi.nih.gov/health/public/lung/other/lungs_hd.pdf |
| | Inhaled pathogens/person/day air contamination | -1.88 | | | | log/person/day | |
| | Probability of becoming ill/day | 1.57E-05 | | | | probability | |
| | **ill per million per day spray drift inhalation** | **1.57E+01** | | | | number | |

**DOSE RESPONSE MODEL**

| R | *Dose response* | ε | β | χ | Units | |
|---|---|---|---|---|---|---|
| 57 | Oral ingestion pathogenicity data - Weibull-Gamma dose response model(s) | 0.12 | 101.23 | 1 | parameter | van Gerwen et al. (2000), Holcomb et al. (1999), Teunis et al (1996), Medma et al (1996), Rose & Gerba (1991), Black et al (1988) |
| 58 | Variable parameter (minimum) | Min | 2.46 | | parameter | |
| 59 | Variable parameter (maximum) | Max | 200 | | parameter | |

| S | *Summary of model ouputs* | Estimate* | 5% | 50% | 95% |
|---|---|---|---|---|---|
| | **ill per million per day surface water ingestion** | **2.63E-04** | 2.63E-04 | 2.63E-04 | 2.63E-04 |
| | **ill per million per day groundwater ingestion** | **0.00E+00** | 0.00E+00 | 0.00E+00 | 0.00E+00 |
| | **ill per million per day recreational water ingestion** | **2.24E-03** | 2.24E-03 | 2.24E-03 | 2.24E-03 |
| | **ill per million per day yard air inhalation** | **1.19E+01** | 1.19E+01 | 1.19E+01 | 1.19E+01 |
| | **ill per million per day facility air inhalation** | **1.89E+00** | 1.89E+00 | 1.89E+00 | 1.89E+00 |
| | **ill per million per day spray drift inhalation** | **1.57E+01** | 1.57E+01 | 1.57E+01 | 1.57E+01 |

* based on all simulated inputs being set to their most likely value