Quantitative risk assessment of *Listeria monocytogenes* in ready-to-eat meats in Australia

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ARTICLE INFO

ABSTRACT

Listeria monocytogenes is a food-borne pathogen that can contaminate processed meats and has caused outbreaks in several nations in which processed meats were the vehicle. Due to its ecology, the control of this organism in ready-to-eat meats is difficult. As a first step in improving risk management for this product: pathogen pair in Australia, a stochastic simulation model to predict the numbers of *L. monocytogenes* likely to be consumed in those products under a wide range of scenarios was developed. The predictions are based on data describing initial contamination levels of both lactic acid bacteria and *L. monocytogenes*, product formulation, times and temperatures of distribution and storage prior to consumption, and consumption patterns. The model was used to estimate the probable numbers of cases of listeriosis due to processed meats in Australia per year. The model predicted that processed meats could be responsible for up to ~40% of cases of listeriosis in Australia, a level considered credible by comparison with available epidemiological data. The reliability of the model, as well as data gaps and further research needs, is discussed.

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1. Introduction

In Australia, the production and processing of meat is regulated by the Australian Standard for the hygienic production and transportation of meat and meat products for human consumption (ANZFRMS, 2007). The Standard emphasises risk assessment and risk management and requires processors to implement HACCP-based food safety strategies, requiring the identification of potential hazards associated with all stages of the production of meat and meat products undertaken by the processor that may reasonably be expected to occur.

Barnes et al. (1989) noted that processed meats were potentially a vehicle of human listeriosis. Subsequent outbreaks of listeriosis have proven that potential, often with fatal consequences. In December 1998 an outbreak involving processed meats occurred in the US and caused 21 deaths among 101 victims (Anon., 1999). In the latter half of 2002 another outbreak related to turkey- and chicken-based processed meats was identified in USA and caused eight deaths, three miscarriages and more than 40 illnesses (Anonymous, 2002a). An outbreak in France involving pork rillets caused over 60 deaths (Ryser, 1999) and smaller French outbreaks also resulting in deaths from other processed meats have been documented (Dorozynski 2000; de Valk et al., 2001). In New Zealand listeriosis cases from corned beef (Anonymous, 2000a) have

been reported. Partly in response to outbreaks the United States Food and Drug Administration worked with the United States Food Safety Inspection Service to assess the risk of listeriosis from a range of ready-to-eat foods, including "deli-meats". The report of that assessment (CFSAN/FSIS, 2003) considered that, of 23 categories of 'ready-to-eat' foods, deli meats represented the greatest contribution to listeriosis in USA.

Although small listeriosis outbreaks implicating processed poultry meats have been recorded in Australia (Watson et al., 1990; Hall et al., 1996), until 2006 there were no documented cases of listeriosis related to Australian processed red meat products. Cognizant of outbreaks in other nations, the Australian meat industry has adopted a pro-active approach to management of the risk of listeriosis from processed meats and in 2002–03 undertook a risk profile of food safety hazards at all stages of the food chain from primary production to consumption (Pointon et al., 2005). The profile used both a qualitative and a semi-quantitative risk assessment matrix and concluded that *Listeria monocytogenes* in ready-to-eat (RTE) meat products represented a moderate risk (Sumner et al., 2005a,b). To better estimate the risk, and to explore potential risk management options, a quantitative risk assessment was undertaken of this hazard:product pairing, the purpose of which was to assess the public health risk to Australian consumers from *L. monocytogenes* in Australian-made processed meat products.

More specifically the assessment aimed to:

1. characterise the nature and size of the microbial food safety risk due to *L. monocytogenes* in processed red meat products;

2. identify where critical data and/or knowledge (to characterise that risk) were lacking;

3. characterise factors that contribute most significantly to the risk; and

4. assess the effectiveness of potential management strategies to reduce the food safety risk due to *L. monocytogenes*.

While increasing attention is being given to the potential role of *L. monocytogenes* in otherwise undiagnosed gastro-intestinal illness (Hof, 2001), that form of listeriosis was considered to be outside of the scope of the risk assessment.

There were no constraints on the time available to undertake the risk assessment. The only limitation related to the availability of data, discussed later.

The results of the risk assessment that address the first two aims are presented in this paper. Results of studies to address the other aims are presented separately in Ross et al. (in press).

2. Methods

The risk assessment was undertaken and documented in accordance with Codex guidelines for food safety risk assessment (CAC, 1999). A stochastic model was developed using @Risk stochastic simulation software (@Risk 4.2 for Excel, Palisade, Ithaca, USA). The @Risk software implements Monte Carlo simulation as an add-in to Microsoft Excel. The model and software were used to assess the effect of putative risk management options. The model is described in detail in the complete risk assessment document (~250 pp, available on request from the corresponding author) but is described briefly below. The model and summary supporting documentation are available for download at FoodRisk.org (http://foodrisk.org/exclusives/models/AU_listeria.cfm).

The mathematical model describes the post-production contamination, storage and distribution of Australian smallgoods, their effect on levels of *L. monocytogenes* and lactic acid bacteria in the product, and the public health consequences of those contamination levels and frequencies for Australian consumers.

The model uses Monte Carlo simulation modelling using Latin Hypercube sampling to predict the numbers of *L. monocytogenes* likely to be consumed in those products under a wide range of scenarios based on data describing initial contamination levels, product formulation, times and temperatures of distribution and storage prior to consumption and consumption patterns. Inputs to the model can be changed to investigate the likely effectiveness of different strategies intended to reduce the risk. In essence, the model allows the user to conduct experiments that would not be feasible or ethical to conduct with the real food system.

Effectively, from:

- data for contamination levels and frequencies on Australian processed meats at the time of their production
- times and temperatures of handling and storage between production and consumption, and
- knowledge of the microbial ecology of the product

the model estimates the range of concentrations of *L. monocytogenes* on servings of processed meats at the time of consumption and, from that estimate and the size of the servings, estimates the range of doses that would be ingested by consumers. Due to differences in product formulation and end use, the model separately assesses risk from three groups of processed meats products. These are:

- luncheon meats (i.e. those products that might be served sliced or shaved as part of a salad or included in a sandwich) including emulsion products, hams, whole cooked muscle meats, non-fermented sausages not intended for reheating, etc.
- cooked sausages intended for reheating before consumption, including viennas, cocktail sausages, frankfurters, etc, and
- pâtés.

Fermented meats were also considered. The risk assessment of fermented meat products, using the same level of data and stochastic simulation modelling techniques described below, indicated that the risk was negligible. A similar conclusion was reported in FAO/WHO (2004) which estimated a risk per serving from fermented meats of 2.1×10^{-12}. That estimate represents less than 1/50,000th of the risk from all meals consumed by an individual, based on current listeriosis rates of ~0.3/100 000 population per year. Accordingly, fermented products are not further considered in this report.

For each product category, variables in the model include:

- product formulation (pH, water activity, nitrite levels, lactic acid concentration),
- initial contamination levels of spoilage/lactic acid bacteria and *L. monocytogenes*,
- times and temperatures of storage at various stages in the production to consumption pathway etc.
- serving size and frequency
- consumer susceptibility and strain variability (implemented *via* a 'dose–response' model).

For the 'base-line' estimates reported here ten simulations of the model of 100 000 iterations each, representing the range of products and conditions in Australia, were executed for each category of product. For each product category the average and standard deviation of the average risk estimate from each of the ten simulations was calculated and is the primary risk estimate discussed here. The simulation averaging process follows the approach of FAO/WHO (2004) which was implemented because it was observed that, due to the extremely low probabilities associated with acquiring listeriosis from any single meal, "the estimates from risk characterization are very sensitive to extreme values from input distributions (the right-hand tails of the distributions)", and that when such values are sampled they greatly increase the risk estimate. In the modelling described here, co-efficients of variation for the average of the averages of the risk per serving from the three product types were ~0.15.

Using the exponential dose–response model developed by FAO/WHO (2004) the average probability of illness *per* serving was calculated from the results of the exposure assessment model and is the main measure of public health risk used in the risk characterisation.

An overview of the conceptual model for the exposure assessment is presented in Fig. 1 in the form of an influence diagram.

An innovative feature of the model is the joint modelling of *L. monocytogenes* and spoilage bacteria (presumed to be lactic acid bacteria). If the product is predicted by the modelling to be spoiled before consumption, the product is predicted to be discarded and, thus, does not contribute to the risk of illness. The model also predicts that high levels of lactic acid bacteria can be present for several weeks before product spoilage becomes evident and that such high levels will inhibit growth of *L. monocytogenes* according to the principle of the Jameson Effect (Stephens et al., 1987; Ross et al., 2000). The consequences of these aspects of the model are considered in detail in Ross et al. (in preparation).

3. Hazard identification

The risk assessment considered only food safety risks emanating from systemic listeriosis, the manifestations of which are serious and, in 20–30% of cases, fatal. Full details of the hazard presented to consumers by *L. monocytogenes* are presented in FAO/WHO (2004).

In Australia, on average, there have been approximately 65 (range 50–74) notified cases of listeriosis *per* year for the decade 1997 to 2007 (CDN, 2008). Outbreaks of listeriosis in Australia involving processed meats include an outbreak in a hospital maternity ward in which chicken liver pâté was implicated epidemiologically in six foetal/neo-natal deaths (Watson et al., 1990). In 1996 a cluster of listerioses was confirmed in an Adelaide hospital involving five patients, one of whom
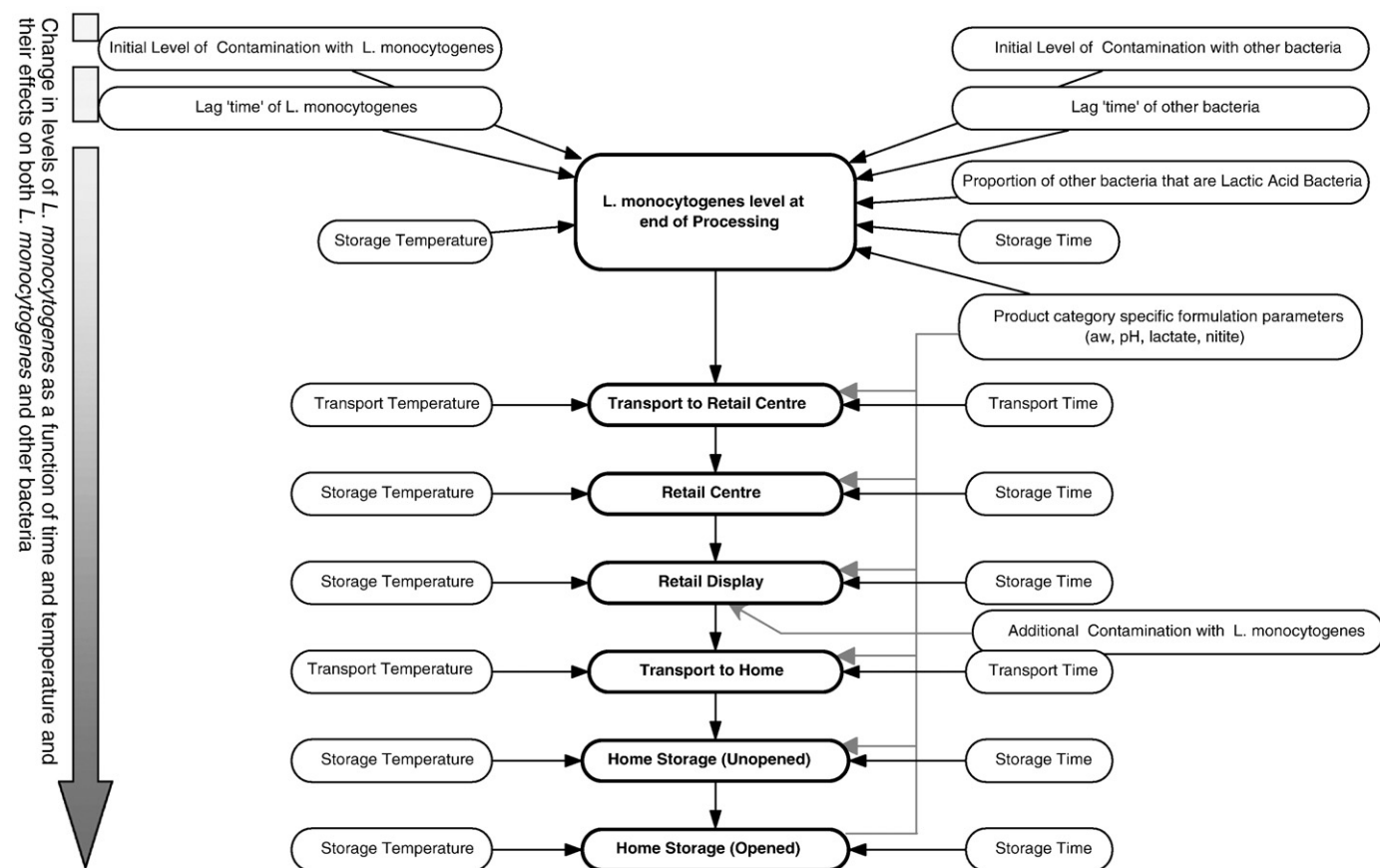
Fig. 1. An influence diagram showing the overall structure of the exposure assessment past of the model. The figure shows stages between processor and consumer that are discretely modelled and shows model inputs. For each of the stages shown the numbers of *L. monocytogenes* and lactic acid bacteria are calculated, based on the levels at the end of the previous stage and the additional model inputs. Arrows from one input or calculation denote that the values influence the calculation of the value to which the arrow points. Estimated frequencies and concentrations of *L. monocytogenes* at the time of consumption are then combined with the FAO/WHO (2004) dose–response model to estimate consumer risk.

died. All of those patients were immunosuppressed, due to chronic disease. Diced chicken prepared offsite was used in sandwiches and *L. monocytogenes* was isolated from the chicken and from the hospital kitchen in which the sandwiches were prepared. Of sporadic listeriosis cases reported earlier in 1996 at least two were linked with a medical centre (Hall et al., 1996).

Prior to 2005 there were no confirmed outbreaks of listeriosis related to Australian RTE meats, although ongoing surveillance (Tan et al., 1995; A. Tan, Microbiology Diagnostic Unit, University of Melbourne, *pers. comm.*, 2003) has provided circumstantial evidence of the involvement of RTE meats in sporadic listeriosis cases. In late-2005 RTE meats were implicated in a listeriosis outbreak among five patients in South Australian hospitals of whom three died (SADH, 2006; Givney, 2006).

4. Exposure assessment

4.1. Prevalence

Data describing the frequency of contamination with *L. mono-cytogenes* of Australian processed meats at the point of production were obtained from two sources: i) the Health Department of Western Australia (S. Goodchild, *pers. comm.*) and ii) the Australian Meat Industry Council ('AMIC'; C. Blaney, *pers. comm.*), from a poll of their membership. Collectively, over 4000 data were obtained for the period 1997–2003. AMIC data was available for 2003 only. Both data sets were derived from random surveys, i.e. not linked to food-borne disease

investigations. Average contamination rates over the period 1997–2003 for three processed meat categories are shown in Table 1.

The prevalence of contamination is consistent with that in other developed nations but appears to be lower than that reported for Australian smallgoods at retail prior to 2000 (e.g. Arnold and Coble, 1995; Grau and Vanderlinde, 1992; Varabioff, 1992). Despite the apparent reduction in contamination rates there is no evidence of a corresponding decline in Australian incidence of listeriosis over the last decade.

4.2. Concentration

The Western Australian data described above involved further testing of *L. monocytogenes*-positive samples, providing 177 estimates of *L. monocytogenes* concentration in samples in which *L. monocytogenes*

Table 1
Rates of contamination with *L. monocytogenes* of Australian RTE meats at the point of production.

	Number of data	Contamination rate (%)	Standard deviation of annual prevalence (1997–2003)
Processed (deli) meats	3351	4.77	2.62
Pâtés	568	1.20	1.93
Cooked sausages, frankfurters	1118	2.77	1.71

Estimates were derived from data supplied by the Australian Meat Industry Council and Health Department of Western Australia (see text for details).

was detected by enrichment culture. Those data were used to estimate the range and distribution of contamination levels. Reported contamination levels ranged from <3 MPN g^{-1} to >1100 MPN g^{-1}, and at certain discrete values in that range, governed by the sensitivity limits of the multiple tube dilution method used. As far as the limited data allow, contamination levels were not found to differ between the three product categories. A summary of those data, and comparison with a compilation of analogous data from other nations, is shown in Table 2.

4.3. Estimating contamination levels at consumption

There are very few data which describe the level of contamination of foods with *L. monocytogenes* at the point of consumption. To overcome this limitation, predictive microbiology was used to estimate the level at consumption based on:

- known contamination frequencies and levels at production or retail
- product formulation (e.g. salt/water activity, pH, other additives)
- times and temperatures between production and consumption
- the ecology of *L. monocytogenes* in foods, including lag times and the effects of lactic acid bacteria, in vacuum-packed or modified-atmosphere-packed product.

To include potential effects of strain variability, two models for *L. monocytogenes* growth rate (Ross, 1999; Devlieghere et al., 2001) were used but expanded with additional 'gamma concept' (Zwietering et al., 1996) type terms to incorporate the effects of additional environmental parameters, e.g. nitrite, relevant to processed meats. Similarly modified versions of the models of Devlieghere et al. (2000) for *Lactobacillus sake* and Wijtzes et al. (2001) for *Lactobacillus curvatus* were used to predict lactic acid bacteria growth rates.

In each iteration of the model, alternate product formulation and temperature and time assumptions are invoked. For each iteration, the fastest growth rate predicted by either of the lactic acid bacteria models, based on the selected formulation parameters, is used. Similarly, the fastest growth rate predicted by either of the *L. monocytogenes* models was used for calculation of *L. monocytogenes* growth in that iteration of the model. The assumption adopted in this study was a "middle-ground" to avoid overly conservative and overly non-conservative growth rates and conditions. The consequences of this assumption, compared to other possible assumptions, are explored in Ross et al. (in preparation).

4.3.1. Data sources

4.3.1.1. Initial lactic acid bacteria levels. The distribution of initial levels of lactic acid bacteria was estimated by combining data from published papers with unpublished data from Australian smallgood manufacturers. From this, a distribution of total aerobic counts at the points of manufacture was generated, and found to be consistent with

the observation of Borch et al. (1996), i.e. that the initial microbial count on cooked meat products is typically in the range 10^2–10^3 CFU g^{-1}. Data in Mol et al. (1971), Simard et al. (1983); Lee et al. (1984), Borch et al. (1996) and Samelis et al. (1998) were combined to estimate the proportion (described by a uniform distribution from 4 to 47%) of the initial total aerobic counts that are lactic acid bacteria.

4.3.1.2. Physico-chemical properties of processed meats. Product pH and water activity information was obtained by analysis of products purchased at retail. Surface pH and water activity of all products was measured from duplicate samples taken from each package.

Data (~300) concerning lactic acid concentration was obtained from industry and published (Gill, 1982; Devlieghere et al., 2000; Wallace et al., 2003) data. Consistent with the rest of this study, the dataset was divided into luncheon meats, pâtés and liverwursts, and cooked sausages.

Measured nitrite levels, post-cooking, were provided by one manufacturer for 'deli' meats and sausages but not pâtés, and supplemented with a small amount of published data (Devlieghere et al., 2000; Wallace et al., 2003). The degradation of nitrite over time was modelled by comparing these measured levels at manufacture with measured values at retail (Grau and Vanderlinde, 1992).

4.3.1.3. Temperature during storage, retail display, transport etc. Temperatures during transport, display and storage (manufacturer, wholesaler, consumer) were estimated from a number of sources including Microtech (1998), Alliance (1998), Audits International (1999) and supplemented with expert opinion, e.g. expert advice from representatives of national retail chains suggested that temperatures in distribution centres were always in the range 2–4 °C.

4.3.1.4. Processed meats shelf life. We were unable to find literature or reports specifying appropriate shelf lives for processed meat products. To resolve this, two surveys of RTE meats on retail display were undertaken to derive shelf lives used by Australian smallgoods manufacturers and also to determine the distribution of nominal shelf life remaining on processed meats at their time of purchase. These were obtained from the nominated use-by date on the packaging/labelling compared to the date of the survey, i.e., many labels also included date of manufacture information from which the nominal shelf life of the product, specified by the manufacturer, could be determined. The surveys included over 3000 units of over 300 different products. Consistent with the rest of this report, the data were divided into the three product groups identified above.

The analysis above was complemented by expert opinion to estimate the time that processed meats spend in different stages of the "factory-to-fork" pathway. Time of transport from point of purchase to consumers' homes was derived from Microtech (1998). The duration of storage in consumers' homes was estimated from experience and the known shelf life of the product but was considered in two distinct stages. Common experience indicates that after opening, vacuum-packed or modified-atmosphere-packed RTE meats, begin to spoil more rapidly due to the development of films and slimes but we were unable to find published data to specify this. From informal solicitation of meat processors, food microbiologists and consumers it was concluded that smallgoods (with the exception of fermented meats) are most likely consumed within five days of opening, in the case of vacuum-packed products, or within 5 days of purchase as sliced, loose, product purchased from butchers or delicatessen counters in stores. It was also assumed that, despite that deterioration is usually evident within 5 days, products may occasionally be held for 10 or 15 days before use.

4.4. Consumption of RTE meats in Australia

Several studies provide estimates of frequency of consumption and serving size in Australia for various categories of RTE meats according to

Table 2
Levels of *L. monocytogenes* on contaminated processed meats at production and retail.

Contamination level (CFU g^{-1})	Percentage of samples in contamination levels range					
	~<3	≤10	≤100	≤1000	≤10,000	≥10,000
CFSAN/FSIS (2003)[a] — retail	72.2	7.5	8.6	7.1	2.3	2.2
Cumulative total		79.7	88.34	95.44	97.8	100.0
Gombas et al. (2003) — retail (USA)	75.6	12.2	2.4	8.6	1.2	
Cumulative total		87.8	90.2	98.8	100.0	
Health Dept., WA — production	71.8	16.9	7.3	1.7	2.3[b]	
Cumulative total		88.7	96.0	97.7	100.0	

[a] Report based on a compilation of all contamination levels related to processed meats reported from all sources and for all nations for which data were available. Statistics shown are based on data for frankfurters, "deli meats", and pâtés and meat pastes.
[b] Samples in this range were ≥1100 MPN g^{-1}, with no estimate of an upper limit available.

consumer age, geographic region and gender (CDH/NHF, 1986; DCHS, 1988, Baghurst et al., 1987; ABS, 1999a,b). The data indicate that, on any day, between 20 and 50% of Australians consume RTE meats. The amount consumed varies according to the type of product, with pâtés typically having smaller serving sizes and cooked sausages having larger serving sizes. The overall range of serving sizes was reported to be ~20 to ~120 g. Differences in consumption frequency and serving size according to age and gender are also evident. For example, there is lower consumption of all product types (except pâté) by females than males in all age categories. While differences in consumption according to age and gender can be discerned from the data, they are not explicitly used in this risk assessment due to the absence of data for strong differences in consumption among subpopulations with greatly increased susceptibility to listeriosis.

Table 3 summarises serving size information for an "average" Australian consumer for the three categories considered in this risk assessment. Note that the values presented relate to the serving sizes of those who consume these products, not to daily intakes averaged across the total population.

5. Hazard characterisation

5.1. Australian populations susceptible to listeriosis

The reported incidence of listeriosis in Australia is 0.2–0.4 per 100 000 population *per annum* (CDN, 2008) which is consistent with, but slightly lower than, many other developed nations. An analysis of the proportion of Australians at increased risk of listeriosis due to a range of conditions yields an estimate of between 15% (including people over 65 years only) and 19% (including people above age 60). The composition of this group at increased risk is given in greater detail in Table 4. Lindqvist and Westöö (2000) derived an estimate of 20.1% for Sweden (including the population >65 years old) while Buchanan et al. (1997) and CAST (1994) estimated the susceptible USA population at 20% of the total population. Similarly, Hitchins (1996) estimated that 15% of the total population of USA was at increased risk of listeriosis due to known predisposing factors. These estimates are somewhat arbitrary because the increase in susceptibility with age is gradual and commences with adulthood (Kirk et al., 2002).

The likelihood of an individual being in more than one of the susceptibility classes increases with age, with the exception of HIV/AIDS and pregnancy, and possibly alcoholism. Hitchins (1996) considered it highly likely that the inclusion of both aged persons and those with cancer or diabetes would lead to double-counting and overestimation of the prevalence of susceptible people within a community.

The largest contribution to the 'at-risk' estimate is from people >60 years old. Even without underlying disease such as cancer or diabetes, elderly Australians are still more susceptible to infectious disease because the immune system diminishes with age. Taking the extreme assumption that other debilitating illnesses including cancer, diabetes, kidney and liver disease only affect those above 60, an estimate of the actual proportion of people in the YOPI (*Y*oung, *O*ld, *P*regnant, *I*mmunocompromised) group that discounts multiple predisposing

Table 3
Summary statistics for the ranges of values characterising distributions of RTE meat servings sizes consumed in Australia.

	Range of estimates of serving sizes (g)		
	Minimum	Range of averages	Maximum
Processed meats	15	28–58	84
Cooked sausages, frankfurters	42	63–108	140
Pâté and meat paste	7	40–56	140

Table 4
Estimate of the proportion of the Australian population at increased risk of food-borne illness.

Predisposing condition	Proportion of Australian population affected (%)
Age >65 years	10.16
Age >60 years	13.70
Age <30 days	0.11
Organ transplant recipients — kidney	0.03
Organ transplant recipients — other	0.01
HIV	0.10
AIDS	0.01
Cancers (non-melanoma)	1.42
Pregnancy	0.99
Kidney disease or dialysis	0.03
Cirrhosis, chronic liver disease	0.12
Diabetes (reported, Type 1 only)	1.97
Hepatitis (all forms)	0.03
Alcohol dependency	0.10
Total (including age >65 years)	15.2
Total (including age >60 years)	18.7

factors includes: neonates, those above 60, pregnant women and their foetuses, alcoholics, HIV and AIDS patients. From Table 4, this group is estimated to comprise ~15% of Australia's population. Thus, we estimate that the effect of double counting is likely to amount to no more 3–4% of the total population. In comparison with other sources of uncertainty in the model this level of uncertainty is relatively minor but is included in the risk estimate by allowing the size of the susceptible population to vary between 15 and 20% in each iteration of the simulation.

5.2. Dose–response model

To convert the predicted frequency and doses of *L. monocytogenes* ingested into an estimate of public health risk requires definition of the relationship between the dose ingested and the probability of illness resulting from that dose, i.e. a 'dose–response model'.

There are insufficient data from which to build a reliable dose–response model for *L. monocytogenes* either from experimental outbreak data, human volunteer feeding trials or animal experiment data. CFSAN/FSIS (2003) used an approach based on the use of a "dose–response scaling factor" to "correct" a mouse-derived model for the range of virulence to make it applicable to humans, but that model varies with every iteration of the risk assessment and is neither readily reproduced nor readily defined.

The CFSAN/FSIS (2003) study, and its pre-cursor in USDA/FDA/CDC (2001) can, however, be used to infer a global dose–response model without the need for adjustment factors using essentially the same approach as Buchanan et al. (1997). Effectively, the CFSAN/FSIS (2003) exposure assessment represents the most complete estimate of the exposure of a population to food-borne *L. monocytogenes*. Using the knowledge of the incidence of listeriosis in USA (Mead et al., 1999) together with the proportions of people of varying susceptibility in that population, a series of exponential dose–response models can be derived by combining the exposure data with the dose–response model and finding values of the exponential parameter, *r*, such that the model predicts the number of cases observed. This extends the Buchanan et al. (1997), Lindqvist and Westöö (2000) and Chen et al. (2003) derivations of *L. monocytogenes* dose–response relationships because it is based on 20 different ready-to-eat food commodities rather than one, and also considers the effect of growth of *L. monocytogenes* in the products between the time of "sampling" for the presence and concentration of *L. monocytogenes*, and the concentration at the time of consumption.

That approach was adopted, and further refined, by FAO/WHO (2004) using the exposure assessment from USDA/FDA/CDC (2001). The full derivation of that model, including assumptions and

Table 5
Predicted average[a] risk of listeriosis *per* serving of Australian processed meats.

	Processed meats	Pâtés	Cooked sausages
Average	1.00×10^{-8}	2.28×10^{-9}	7.06×10^{-9}
Standard deviation	1.37×10^{-9}	3.55×10^{-10}	1.11×10^{-9}

[a] Calculated as the average of the risk from all servings sampled in the stochastic simulation. The risk of listeriosis in every iteration of the model is calculated using an exponential dose–response model (FAO/WHO, 2004) and the sampled dose of *L. monocytogenes*, i.e., the product of contamination level (CFU g^{-1}) and serving size (g). Note that this value will be highly influenced by very high doses so that the average risk is much larger than the risk estimated from the average concentration measured on a log(CFU g^{-1}) scale (see text for more explanation).

consequent caveats concerning its use, is described in FAO/WHO (2004). The general form of the exponential dose–response model is:

$$P = 1 - e^{(-rD)} \tag{1}$$

where:

P	is the probability of severe illness
D	is the number of *L. monocytogenes* consumed, and
r	is the parameter that defines the dose–response relation for the population being considered.

In effect, r is the average probability (i.e. recognising variation in pathogen virulence and host susceptibility) with which a single *L. monocytogenes* cell would cause illness. Median r-values, generated by FAO/WHO (2004) for 'healthy' and 'susceptible' populations, were used in the risk characterisation (see Section 6), together with estimates of the proportion of Australians in two broad categories of susceptibility, namely 'healthy' and 'YOPI' to estimate the likelihood of listeriosis from ingestion of contaminated servings of processed meat. For the 'healthy' population the r-value used is 2.37×10^{-14}, while for the 'susceptible' population the r-value used is 1.06×10^{-12}. The difference in r-values for these populations reflects the differences in average susceptibility and implies that the YOPI group is, on average, ~40 times more susceptible than the 'healthy' population.

A significant vulnerability of the dose–response model used is that it relies on the validity of all assumptions and data used in the development of the USDA/FDA/CDC (2001) *L. monocytogenes* risk assessment. If any of the assumptions or data were altered in such a way as to alter the estimated level of exposure, the resulting dose–response model would, by inference, be changed, as would any estimates of risk based on it. The data of Gombas et al. (2003) and Levine et al. (2001), for example, suggest that more recent estimates of prevalence of processed meats contaminated with *L. monocytogenes* are lower by a factor of two or three than that apparently used in USDA/FDA/CDC (2001). A general change in the assumption of storage times or temperatures, particularly for higher risk products, could significantly affect this estimation process.

The FAO/WHO (2004) approach leads to estimates consistent (to within an order of magnitude) with other estimates of the r-value when differences in assumptions are accounted for (e.g., number of RTE foods contributing to the exposure, extent of growth between retail and consumption etc.) and, due to its broader data-base, is probably the most-preferred of the *L. monocytogenes* dose–response models currently available.

6. Risk characterisation

A mathematical model, an overview of which is presented in Fig. 1, was developed to incorporate all of the above factors that affect the risk of listeriosis to Australian consumers from consumption of RTE meats and to characterise that risk. The model describes product formulation factors and times and temperatures during the storage and distribution of Australian RTE meats after production. It incorporates the data and models described in the Exposure Assessment and Hazard Characterisation sections above and uses @Risk© simulation modelling software to predict, from initial contamination levels, the levels of *L. monocytogenes* likely to be consumed under a wide range of scenarios relevant to Australia. In stochastic models, input variables are described by a distribution of possible values rather than a single 'best' estimate. The advantage of this type of model is that it incorporates variability and uncertainty, although in the model described here their effects are not explicitly differentiated.

In the model 100 000 iterations were executed for each category of product and that process was repeated ten times. The average and standard deviation of the average risk estimate from each of the ten 100 000 iterations for each product category was calculated. To save processing time, in each iteration it was assumed that the product was contaminated with *L. monocytogenes* so that computing time would not be wasted modelling meals/servings that were not contaminated and presented no risk of listeriosis. The actual prevalence of contaminated servings (see Section 4) was described using Betapert distributions giving rise to average contamination prevalence estimates of 6.4%, 1.4% and 3.2% for luncheon meats, pâtés/liverwursts and sausages, respectively, and these prevalence estimates were incorporated into the *per* serving risk estimate in each iteration. Thus. given that ~5% of the servings of processed meats are contaminated with *L. monocytogenes*, the simulations represent the exposure from ~20 million servings of each of the three categories of RTE meats considered.

By combining the dose estimate and contamination prevalence estimates with the dose–response model the average probability of illness *per* serving is calculated and is the main measure of risk used in this risk characterisation. Further, by combining the risk per serving with the total annual number of servings in each category it is also possible to estimate the number of cases of listeriosis in the Australian population associated with consumption of those meals.

7. Results and discussion

The estimated distributions for the risk of listeriosis for three processed meat categories considered are summarised in Table 5 and shown in detail in Fig. 2. These estimates are based on the knowledge, data and assumptions summarised in Sections 4 and 5.

The distributions shown in Fig. 2 illustrate that, while the distribution of estimated *per* serving risk of listeriosis for processed meats and sausages is similar, the distribution for pâté is shifted slightly to the left, i.e. to lower levels. This implies a lower likelihood of illness from any serving and is most likely due to the lower prevalence of
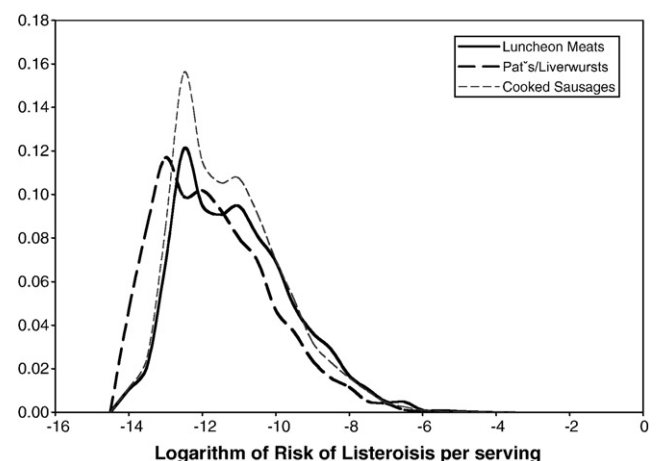
Fig. 2. Distributions of estimated logarithm of risk of listeriosis *per* serving of processed meats according to product type.
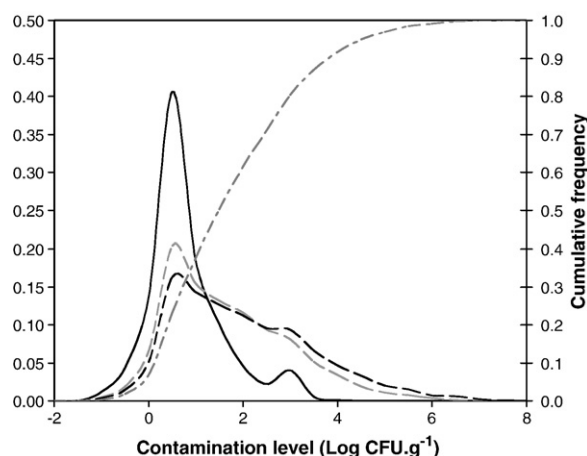
Fig. 3. Predicted levels of *L. monocytogenes* on contaminated Australian processed meats at the point of production (solid line); point of purchase (light broken line); and at consumption (heavy broken line). Also shown is the cumulative frequency of predicted contamination levels at the point of consumption for luncheon meats (dashed-dotted line). From the cumulative frequency plot the proportion of contaminated samples containing greater than 100 CFU g^{-1} at the time of consumption is estimated to be ~39%.

L. monocytogenes contamination of these products. The apparently abrupt lower limit to risk *per* serving evident in Fig. 2 is due in part to the selection of 'bin' sizes (0.5 log$_{10}$ intervals) for the frequency histograms upon which plots are based and also to the scenario of the smallest servings of products, consumed by a 'healthy' consumer, that are contaminated at the lowest level (1 CFU per 25 g) and for which no growth is predicted to occur under the product formulation and time/temperature parameters sampled in the simulation modelling. In fact, this estimate is unrealistically low because, in the model, the lowest concentration (1/25 g) can be combined with the smallest serving size (7 g for pâté) to yield an estimate based on less than one cell per serving. The minimum risk per serving based on consumption of a single cell by a non-susceptible consumer in the smallest servings of each product type is in the range $10^{-13.4}$ to $10^{-14.2}$. The 'bumps' in the predicted distributions arise because the contamination level data were derived from MPN data and were described as a series of contamination level *categories* in the model, and the data themselves. For example, the 7 MPN g^{-1} category has a low probability compared to the 4 and 9 MPN g^{-1} categories. Similarly, the >1100 MPN g^{-1} category, because it encompasses a wide range, is relatively large.

Also evident from Fig. 2 is that the most frequent estimate of risk, i.e. the risk of listeriosis most likely to be experienced upon consumption of a serving of processed meat, is 100 to 1000-fold lower than the average risk estimate (Table 5). This arises from long right hand tail of the distribution and the direct proportionality between dose ingested and probability of illness predicted from the dose–response model for all realistic levels of *L. monocytogenes* contamination. Those iterations of the model that predict high levels of *L. monocytogenes* have more influence in the calculation of the average risk. For example, the dose–response model infers that one instance (one iteration or scenario) of consumption of product contaminated with one million *L. monocytogenes* CFU g^{-1} contributes as much to the determination of the average risk as do one thousand simulated instances of consumption at a concentration of 1000 CFU g^{-1}.

The predicted levels of *L. monocytogenes* on contaminated serves of luncheon meats at the point of production, point of purchase and point of consumption are shown in Fig. 3 to illustrate how the estimated risk increases as a function of time since production. As expected, the distribution of contamination levels shifts to the right (i.e. higher contamination level) as the age of the product increases due to greater opportunity for *L. monocytogenes* growth. From the cumulative distributions it is possible to estimate the proportion of samples at

particular levels of contamination. A *L. monocytogenes* contamination level of 100 CFU g^{-1} has been suggested as a tolerable upper limit of contamination at the point of consumption in some European nations (European Commission, 1999; Nørrung et al., 1999). The simulation predicts that 30% of *contaminated* servings of luncheon meats, or ~1.5% of all servings would fail to satisfy this criterion at the time of retail purchase, while nearly 2% of all servings of luncheon meats are predicted to exceed the 'tolerable' level at the time of consumption.

The primary measure of risk used in this assessment is the risk of listeriosis per serving of processed meat. This is useful for comparing intrinsic risk from different product types under normal conditions of storage, distribution and sale. The concept of risk, however, also encompasses the severity of the consequences of exposure, whether severity of symptoms or numbers of people affected. Thus, to estimate public health risk one also has to consider the amount of each of the three types of processed meats that are consumed and the form in which they are eaten.

To do this, the average risk-per-serving estimates presented in Table 5 were combined with estimates of the number of servings of each product type per year in Australia. The latter was estimated by dividing the estimate of the total volume of each of the three product types that are consumed per year in Australia by the average serving size of those product types, predicted by the simulation model. The latter was estimated from the distributions used to model serving size for each product category (Table 3). In addition, it was assumed that 95% of pre-cooked sausages (e.g. frankfurters) are thoroughly cooked prior to consumption but that 5% are consumed without further cooking. Using these values, the predicted number of cases of listeriosis per year in Australia, due to those products, is presented in Table 6. Collectively, RTE meats, uncooked frankfurters and pâté are predicted to account for 44 cases. This equates to approximately one-third of Australia's listerioses cases, allowing for 50% under-reporting based on the estimates of Mead et al. (1999).

The annual risk estimates offer a different perspective to the per meal estimates because of the different consumption levels between the three categories. Thus, while processed meats and cooked sausages have a similar per serving risk (which could be considered as the intrinsic risk), the lower consumption and re-heating of sausages means that the risk to public health is considerably lower.

A quantitative risk assessment of listeriosis from selected ready-to-eat foods in USA (CFSAN/FSIS, 2003) estimated RTE meats, unheated frankfurters and pâté to be responsible for approximately 1600, 30 and 4, respectively of the approximately 2500 annual cases of listeriosis estimated by Mead et al. (1999), including unreported cases, in that country. This is a much higher attribution of the proportion of illness to the smallgoods industry in USA than is ascribed to the Australian industry in the present study. It must be remembered that risk estimates in both the USA and Australian risk assessments encompass a high degree of uncertainty. Nonetheless, the prediction that 44 cases of listeriosis in Australia may result from consumption of RTE meat products, in general, and from processed meats, in particular, appears credible when viewed in the context of recorded outbreaks and

Table 6
Number of cases of listeriosis per year in Australia due to consumption of various categories of RTE meat products predicted from the risk assessment model.

	Total volume (*t*)	Mean serving size (g)	Servings (per year)	Predicted number of cases per year (95% CI)
Processed meats	194,600	45.34	4.29 billion[a]	43 (54.5 to 31.2)
Pâtés/liverwursts	8400	53.01	0.16 billion	0.36 (0.48 to 0.25)
Cooked sausages[b]	60,400	87.50	0.69 billion	0.24 (0.32 to 0.17)
Total				44 (55 to 31)

[a] i.e. 1000 million.
[b] For the estimate of the risk of cooked sausages, 95% were assumed to be eaten after normal cooking (immersion in boiling water for several minutes) which is listericidal.

Table 7
Recorded outbreaks and sporadic cases of listeriosis in Australia from 1990–2007 for which the aetiology is known.

	Food implicated	Cases (deaths)	Setting	Reference
1990	Pâté	9 (6)	Maternity ward	Watson et al. (1990)
1991	Smoked mussels	3	Home	Misrachi et al. (1991)
1994	Sandwiches	2	Hospital	Anonymous (2002b)
1994	Minced peas (?)	1	Hospital	Anonymous (2002b)
1996	Chicken	2	Hospital	Hall et al. (1996)
1996	Diced, cooked chicken	5(1)	Hospital	Hall et al. (1996)
1996	Sandwiches, meat salad	5	Hospital	Anonymous (2002b)
1997	Cooked chicken	1	Maternity ward	Kirk et al. (2002)
1997–99	Fruit salad	9 (6)	Nursing home	Anonymous (2000b)
1998	Ham and potato bake	32	Catered function	Anonymous (2002b)
2005	Corned beef	5 (3)	Hospital	Givney (2006)

sporadic cases of listeriosis from all sources in Australia (Table 7). This listing, however, accounts for only 45 of the more than 1000 notified listerioses that occurred in Australia during the period 1990–2007, emphasising the sporadic nature of the disease in this country. A case–control study of sporadic cases of listeriosis could yield valuable epidemiological information that could also be used to evaluate the validity of the risk assessment model.

A striking feature of Table 7 is the association of the listeriosis outbreaks with hospital and aged care settings. In reviewing foodborne disease outbreaks in Australia between 1995 and 2000, Dalton et al. (2004) noted that outbreaks in aged-care and hospital settings were associated with 35% of all deaths but only with 5% of outbreaks and <3% of cases. The authors stated that preventing high-risk patients from receiving high-risk foods could prevent many of these deaths, noting that case-fatality rates were ten times higher in aged care and hospital settings. Increasing incidence of listeriosis has been reported in France, Germany and the United Kingdom, with a trend to a higher proportion of cases in older people (Hedberg, 2006; ACMSF, 2008).

The development of a stochastic risk assessment model, including collation and integration of exposure assessment and hazard characterisation information into the risk assessment, is a resource intensive activity. The value of these activities can be enhanced if they can be used to assess mitigation strategies. One outcome of the present risk assessment was that the Australian smallgoods industry requested the risk assessors to investigate a number of mitigation strategies. This activity was undertaken and is reported in a complementary report (Ross et al., in press).

It is encouraging that in this risk assessment, estimates of disease burden from listeriosis are realistic and credible. These estimates were achieved using real data and knowledge of microbial ecology, credible assumptions where specific data or knowledge were lacking, and without any arbitrary factors to 'calibrate' model outcomes to expected results. This lends support to the thesis that microbial food safety risk can be estimated by inductive reasoning using the risk assessment paradigm proposed by CAC (1999) and implemented using stochastic simulation modelling.

Nonetheless, there are assumptions in the model, and some variables in the model are not able to be well characterised from the available data. From experimentation with the model and modification of the assumptions on which it is based, sometimes in seemingly minor ways but still within realistic limits, we observed that relatively large changes (up to 10-fold) in the predicted number of cases of listeriosis could occur.

While there are many sources of uncertainty, two sources contribute most to uncertainty in the risk estimates — that relating to the potential growth of *L. monocytogenes* in processed meats and that relating to the probability of infection from ingestion of a given dose of *L. monocytogenes*. These knowledge gaps are considered below, and additional research needs are suggested.

The limitations of the dose–response model were described briefly in Section 5.2. Due to the difficulties in obtaining novel dose–response data, it is unlikely that this component of the model will be improved by research in the near future. For example, a recent study using 10 pregnant rhesus monkeys (Smith et al., 2003) took several years to establish and to conduct the trial and cost many millions of dollars. Epidemiological data are unreliable due to the often long incubation period between ingestion of contaminated food and the onset of symptoms.

There is evidence (Torvaldsen et al., 1999; Ogunmodede et al., 2005) that pregnant women may reduce their consumption of some foods to reduce their exposure to *L. monocytogenes*. Similarly, consumption of processed meats varies by age and gender, and it is known that susceptibility to listeriosis is correlated with age. The model used in this study did not attempt to differentiate the exposure of different demographic groups but more detailed consumption data may enable more accurate assessment of relative risk to different types of consumers and the development of better targeted risk minimization strategies.

Also, changes in estimated *L. monocytogenes* growth rate can have a large effect on the risk estimate. Thus models that encompass the full variability of responses of different strains of *L. monocytogenes* and their interaction with lactic acid bacteria in long shelf life RTE meats are needed to characterise the risk and to optimise risk management approaches.

Despite this, the model has utility because it can provide support for risk management decisions by giving estimates of the *change* in risk under different scenarios relative to that for the *status quo* i.e. even though there is uncertainty in the absolute predictions of the model, the structure and detail in the model offer greater confidence in predicted differences between scenarios and specific sets of conditions.

The model includes relatively novel aspects including the consideration of the influence of lactic acid bacteria on the potential for growth of *L. monocytogenes*, modelling of reduction of nitrite levels, and consideration of shelf life and spoilage of the product and its potential disposal prior to consumption. The influence of these factors in the model and assumptions surrounding them, and other assumptions discussed above, will be explored in detail in a subsequent publication (Ross et al., in preparation).

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