Available online at www.sciencedirect.com
SCIENCE DIRECT
Meat Science 74 (2006) 66–75
MEAT SCIENCE
www.elsevier.com/locate/meatsci

Role of quantitative risk assessment and food safety objectives in managing *Listeria monocytogenes* on ready-to-eat meats

Isabel Walls *

International Life Sciences Institute, Research Foundation/Risk Science Institute, 1, Thomas Circle, NW, Washington, DC 20005, United States

Received 13 March 2006; received in revised form 28 April 2006; accepted 29 April 2006

Abstract

Listeria monocytogenes may be found on ready-to-eat (RTE) meats, posing a public health risk. To minimize the public health impact, an appropriate level of protection (ALOP) can be established for a population with respect to *L. monocytogenes*, and ideally should be based on a scientific assessment of the risk, as well as societal and economic factors. Food safety systems can be based on meeting the ALOP. Food safety objectives (FSO) provide a link between the ALOP and performance objectives that are established to control a foodborne hazard. An FSO can be used as a risk management tool for *L. monocytogenes* in RTE meats, as the FSO establishes the stringency of the measures being used to control the hazard, by specifying the frequency and/or cell number of the pathogen in the food that should not be exceeded at the time of consumption. Typically, this requires setting performance objectives or performance criteria at an earlier point in the food chain, to ensure that the product will meet the FSO. Establishing an FSO requires an assessment of the risk of the hazard to the population of interest. Risk management strategies such as use of HACCP systems and Good Manufacturing Practices can then be used to ensure that the FSO is met.
Published by Elsevier Ltd.

Keywords: Microbial risk assessment; Food safety objectives; *Listeria monocytogenes*

1. Introduction

Many different pathogens are found on meat and meat products, including *Escherichia coli* O157:H7, *Salmonella*, *Listeria monocytogenes* and *Staphylococcus aureus* (CDC, 1989; CDC, 1997; Gill, Jones, Bryant, & Brereton, 2000; Gill, McGinnis, & Bryant, 2001; McEvoy, Doherty, Sheridan, Blair, & McDowell, 2003; O'Brien et al., 2005; Pearce, Sheridan, & Bolton, 2006; Sheridan, Duffy, McDowell, & Blair, 1994; Sofos, Smith, Kochevar, & Reagan, 1999). Pathogens may be found on the hides of live animals and may contaminate the meat if cross contamination occurs during the slaughter, or they may colonize the intestines of animals, which could contaminate muscle meat at slaughter. Pathogens can be reduced by knife trimming or vacuuming any visible contamination off the carcass, through acid rinses or hot water or steam pasteurization of the carcass (Bacon et al., 2000; Gill, Bryant, & Bedard, 1999; Gill, Bryant, & McGinnis, 2000; Logue, Sheridan, & Harrington, 2005). Thorough cooking or processing of meat products should destroy vegetative pathogens, but contamination of cooked meat products can occur from the environment, after processing and before packaging in the processing plant. Further contamination or microbial growth can occur through improper handling during storage, at retail or in the home. Organisms can grow to large numbers if time and temperature controls are not in place. Therefore, food safety management strategies must be in place to protect public health. Food safety systems have typically been based on how well an industry is capable of performing, i.e., the concept of as low as reasonably achievable (ALARA) rather than a stated degree of stringency. Where feasible, an alternative approach is to set

* Present address: USDA Food Safety and Inspection Service, Office of Food Defense and Emergency Response, 1400 Independence Avenue, SW., Room 414, Aerospace Bldg, Washington, DC, 20024, USA.
E-mail address: Isabel.walls@fsis.usda.gov.

0309-1740/$ - see front matter. Published by Elsevier Ltd.
doi:10.1016/j.meatsci.2006.04.029

public health goals, or targets, for reducing the level of disease associated with specific pathogens. Setting public health goals requires consideration of societal and economic considerations as well as scientific information to balance the need to ensure protection of public health with the practical realities of food production. Goals should be developed with consideration to the prevailing levels of illness associated with a pathogen/food combination, and the need to reduce this level of illness such that continuous improvement in public health occurs, while acknowledging that zero risk cannot be attained. This manuscript describes the use of risk analysis for managing microbial pathogens in meat, including a case study on *Listeria monocytogenes* in ready-to-eat meats.

2. Appropriate level of protection (ALOP)

Public health goals are established to ensure continuous improvement in the health of the population. For microbial hazards, public health goals can be stated as a reduction in the current level of illnesses, for example, in the USA, a goal was set to reduce the incidence of disease caused by four key pathogens (*Salmonella* species, *Campylobacter* species, *Escherichia coli* O157:H7, and *Listeria monocytogenes*) by 50% by 2010 (HHS, 2000). For foodborne listeriosis, this represents a reduction from 0.50 to 0.25 cases per 100,000 population per year. The current level of listeriosis as measured by the US Centers for Disease Control and Prevention (CDC) is 0.3 cases per 100,000 population and is therefore approaching the public health goal (CDC, 2006). A public health goal is a statement of a country's appropriate level of protection (ALOP). The ALOP concept was introduced in the World Trade Organization Agreement on the Application of Sanitary and Phytosanitary Measures (the SPS Agreement), which promotes the use of risk assessment, based on objective and accurate scientific data, when setting food safety standards. The ALOP is defined as the level of protection deemed appropriate by the member-country establishing a sanitary or phytosanitary measure to protect human, animal or plant life or health within a territory. The ALOP is viewed as the degree of risk that a society is willing to tolerate, or accept, and measures what is achievable before "costs" to society become too great. Costs may be human, economic, ethical, medical, legal, etc. Risk managers are responsible for setting the ALOP. The ALOP will be influenced by the severity of the hazard, the degree of outrage associated with a hazard (e.g., if children are affected) and the perception of risk, which is a function of the ability of a consumer to control the hazard. The ALOP may include safety margins deemed appropriate for minimizing illnesses and to account for uncertainty. The safety margins employed should be proportional to the uncertainty as measured by the underlying risk assessment. As uncertainty is reduced through acquiring more information, the ALOP can be adjusted.

3. Food safety objectives

Food safety standards should be set to meet the ALOP. One difficulty when implementing the ALOP concept is that the ALOP may not be described in terms that can be used by the food industry or government regulatory agencies to set a target for food safety systems, for example, the ALOP may be described as a reduction in illnesses, whereas industry or government need a target based on the number of organisms in a food. The International Commission on Microbiological Specifications for Foods (ICMSF, 2002) has proposed the establishment of Food Safety Objectives (FSO) to provide a link between the ALOP and target points in the supply chain. The intention of FSO is that they be established by government regulatory agencies and serve as a means of communicating public health goals to industry and other stakeholders in a form that can provide a measurable target (Walls & Buchanan, 2005). The FSO is defined as "the maximum frequency and/or concentration of a microbial hazard in a food considered tolerable for consumer protection at the time of consumption" (CAC, 2004). Setting the FSO at the time of consumption requires consideration of the likelihood and impact of contamination at all points further back in the food chain. Targets earlier in the food chain are termed performance objectives (PO) and performance criteria (PC). A PO is the maximum level (frequency and/or concentration) of a hazard in a food at a specified point in the food chain that should not be exceeded in order to achieve an FSO (CAC, 2004). A PC is the outcome of one or more steps in the food safety management system that must be met in order achieve an FSO. For example, the outcome could be a particular minimum reduction in the prevalence or numbers of the hazard in a specific unit of food required, or a maximum increase in the hazard level tolerable. Processors or legislative authorities may need to set POs or PCs at lower levels than the FSO to ensure that the FSO is met.

When establishing PO, consideration must be given to the initial levels of the organism and to any changes that may occur during production, distribution, storage, preparation, and use of a product. The PO can be expressed conceptually by the following equation introduced by the ICMSF (2002):

$$H_0 - \sum R + \sum I \leqslant \text{FSO}$$

where H_0 is the initial level of the hazard, $\sum R$ is the total (cumulative) reduction of the hazard, $\sum I$ is the total (cumulative) increase of the hazard and FSO is the food safety objective. FSO, H_0, R and I are expressed in $\log_{10}$ units.

4. Establishing a food safety objective – scientific considerations

Setting an FSO can involve the following actions (Walls & Buchanan, 2005):

(1) Identification of a public health concern and the need for management actions.
(2) Evaluation of the level of risk (e.g., by conducting a risk assessment).
(3) Articulation of the public heath goal.
(4) Determination of the maximum level of exposure that would achieve the public health goal (including consideration of the need to build in an extra margin of safety to account for variability in food safety management performance and uncertainty in our knowledge on the level of risk) – this is the FSO.
(5) Evaluation of the feasibility of complying with the FSO.
(6) Implementation of the FSO by the industry.

4.1. Risk analysis

Risk analysis can be used to identify public health concerns and evaluate risk (Buchanan, Dennis, & Miliotis, 2004; CAC, 2004; Stringer, 2004; Walls, 2004). Risk analysis comprises risk management, risk assessment and risk communication. Risk assessment provides a framework to collect and analyze the best available scientific information on a hazard. Risk managers consider the scientific information developed during the risk assessment process in light of other important non-scientific information (for instance costs, cultural and environmental factors, etc.), identify a range of appropriate options to manage that risk, and select the best option from the various possibilities to help make risk management decisions, i.e., determine the most appropriate ways to prevent or minimize harm from the hazard. The final decision on the risk management option should be based on all the available scientific, technical, economic and other relevant information.

Quantitative microbial risk assessment provides a systematic means for assessing the severity of microbial hazards and the likelihood that they will occur (Buchanan, Smith, & Long, 2000; CAC, 1999; ILSI RSI, 1996, 2000; Lammerding & Fazil, 2000.) Hazards have the potential to cause harm if an individual is exposed to the hazard. When assessing risks, the nature of the hazard, the likelihood that an individual or population will be exposed to the hazard, and the likelihood that exposure will result in an adverse health effect are considered. Quantitative microbial risk assessment includes: (i) hazard identification/risk profile; (ii) hazard characterization; (iii) exposure assessment; and (iv) risk characterization.

4.2. Developing a risk profile

Development of a risk profile is a planning step in which the goals, breadth and focus of the risk assessment are identified, as well as the regulatory and policy context of the assessment. The risk profile should include a brief description of the situation, product or commodity involved. The specific risk management questions to be addressed by the risk assessment should be determined at this point. Examples of risk management questions are:

(1) What is the risk associated with different levels of pathogens for normal and sensitive subpopulations (e.g., for *L. monocytogenes* in ready-to-eat meats, estimate the risk associated with 0 cfu/25 g, <1 cfu/g, <10 cfu/g, and <100 cfu/g at the point of consumption).
(2) What is the change in risk likely to occur from specific interventions (e.g., for *Salmonella* Enteritidis in raw chicken, estimate the change in risk if you reduce the prevalence of *Salmonella* Enteritidis positive flocks).
(3) What is the change in risk if I add preservatives to prevent the growth of *L. monocytogenes* in ready-to-eat meat?

Data needed to complete the risk assessment should be identified in the risk profile and the assumptions inherent in the analysis should be stated. A key element is that dialogue is established between risk assessors, risk managers and other stakeholders during this phase to ensure that the appropriate scientific concerns are addressed and that the risk assessment provides the information necessary for making sound risk management decisions. The risk profile should allow risk managers to determine whether a full risk assessment is needed, or whether the questions to be answered can be addressed with the existing knowledge. For example, implementation of known good manufacturing practices may be sufficient to resolve the issue. If there are illnesses or evidence of product contamination, immediate action may be needed, in which case a decision should be made based on existing knowledge, and a full risk assessment undertaken at a later stage.

4.3. Hazard identification

Hazard identification is the identification of biological, chemical and physical agents capable of causing adverse health effects and which may be present in a particular food or group of foods (CAC, 1999). In microbial risk assessment, usually the microorganism is known to be a hazard, based on epidemiological data on illnesses associated with the organism. Information may be available from the published literature or government databases (CDC, 2006; FDA, 2006).

4.4. Hazard characterization

Hazard characterization is the qualitative and/or quantitative evaluation of the nature of the adverse health effects associated with biological, chemical and physical agents, which may be present in food (CAC, 1999). Characterization of human health effects includes identifying the host population, and a dose–response assessment, if data are available. Information on the host could include: age,

health/immune status (including pregnancy), gender, ethnicity, location (urban, rural), profession and associated environment, education level and income level.

A dose–response analysis is undertaken to characterize the relationship between dose, infectivity and the likelihood and severity of the spectrum of adverse health effects associated with the hazard in an exposed population. A major assumption in dose-response modeling for microbial risk assessment is that a single microbial cell has a very small but finite probability of causing illness. Dose–response relationships may be determined by human volunteer feeding trials, but for many microorganisms, such trials may not be ethical as the organisms may cause life-threatening diseases. Animal models have been used to develop dose–response models, but their utility is limited due to uncertainties in the correlation with the human response to the pathogen. One approach to estimating the dose–response relationship for a specific subpopulation is to collect data during an outbreak of foodborne disease, including quantitative data on contamination levels of the implicated food, food consumption levels and attack rate data (Kasuga et al., 2004). However, these data also have limitations; typically quantitative data on contamination levels of the implicated food are not available; when they are, the estimated dose–response may be based on a single data point, which depending on when the food is sampled, may be an over-estimate of the actual amount consumed. A further confounding factor is that there is no consideration of the impact of stomach acid or the human immune system on reducing the number of organisms in the food before organisms reach the target site to cause infection.

4.5. Exposure assessment

Exposure assessment has been defined as the qualitative and/or quantitative evaluation of the likely intake of biological, chemical and physical agents via food as well as exposures to other sources if relevant (CAC, 1999). When characterizing exposure to microbial pathogens, data are needed on the frequency of contamination (prevalence) and the numbers of microorganisms (concentration) in a specific food. The amount of food consumed is also needed.

Ideally the contamination levels should be determined at the time of consumption, but typically foods are sampled at earlier points in the food chain. The amount of pathogen growth or inactivation prior to consumption can be estimated for the given scenario from mathematical models, e.g., Combase (http://wyndmoor.arserrc.gov/combase/), or developed for a specific pathogen/food combination (Buchanan, Golden, & Phillips, 1997; Walls & Scott, 1997a; Walls & Scott, 1997b; Whiting, 1995.) Uncertainties in the data should be estimated and quantified where possible.

Consumption data are necessary to complete the exposure scenario. Data needs include the estimated number of servings per year and the average size of a typical serving. Consumption data may be available from national nutrition surveys, e.g., in the USA nationally representative data are collected on the amount of food consumed by an individual (based on consumers' 24-h recall of dietary intakes), the number of servings of a food consumed per year, and demographic characteristics of the consumer e.g. continuing survey of food intakes by individuals (CSFII) (Murphy, 2003; US Department of Agriculture, 2000).

4.6. Risk characterization

Risk characterization is the qualitative and/or quantitative estimation, including attendant uncertainties, of the probability of occurrence and severity of known or potential adverse health effects in a given population based on hazard identification, hazard characterization and exposure assessment (CAC, 1999). In risk characterization, the results from the hazard characterization and exposure assessment are combined to give an estimate of the risk. The magnitude and severity of the risk should be quantified and the attendant variability and uncertainties should be documented, along with the assumptions made when doing the risk assessment. The results should allow the questions identified in the risk profile to be answered.

5. Case example: *Listeria monocytogenes* in ready-to-eat meats

In the USA a risk assessment was undertaken to provide a risk ranking of various ready-to-eat foods, to determine which foods provided a greater risk to human health than others (HHS/USDA, 2003). Ready to eat food categories used in the assessment were selected based on their potential for *L. monocytogenes* contamination, history of causing listeriosis, and availability of food contamination and consumption data. Food categories included meats (frankfurters, dry/semi-dry fermented sausages, delicatessen meats and pate); seafood, dairy, fruits and vegetables and delicatessen salads.

5.1. Hazard identification

Listeria monocytogenes is a foodborne pathogen that can cause listeriosis. Listeriosis is a very severe disease with an estimated case fatality rate of 20–40%, considerably higher than most other foodborne pathogens (Mead et al., 1999). The current level of listeriosis in the USA is estimated to be around three cases per 1,000,000 population per year, based on the US FoodNet surveillance data (CDC, 2006). Clearly, *L. monocytogenes* poses a public health concern and risk management actions are required to reduce the levels of listeriosis that currently exist.

5.2. Hazard characterization

In adults, listeriosis occurs in an invasive or a noninvasive form. After initial flu-like symptoms (fever, fatigue,

malaise, nausea, cramps, vomiting, and diarrhea), invasive listeriosis in adults is characterized by the onset of septicemia and meningitis. In a pregnant woman, invasive listeriosis can lead to spontaneous abortion (CDC, 1998; Linnan et al., 1988). Invasive listeriosis typically occurs in susceptible individuals who have one or more underlying conditions that depress immune function, which predispose them to this disease. Susceptible individuals include patients with cancer or undergoing treatment with steroids or cytotoxic drugs; pregnant women or neonates; renal transplant recipients; patients with acquired immunodeficiency syndrome (AIDS); and the elderly (Gellin & Broome, 1989; Jensen, Frederiksen, & Gerner-Smidt, 1994; Goulet & Marchetti, 1996; Slutsker & Schuchat, 1999). A noninvasive form of listeriosis resulting in febrile gastroenteritis has been documented in several outbreaks (Dalton et al., 1997; Salamina et al., 1996). The frequency of febrile gastroenteritis as a result of *L. monocytogenes* infection is undetermined, as are host characteristics associated with this syndrome.

Dose–response relationships cannot be determined for *L. monocytogenes* using human volunteer feeding trials, because such trials are not ethical as listeriosis is a life-threatening disease and may not be meaningful if conducted in healthy adults, because healthy adults are not the at-risk population and rarely contract listeriosis. Mice have been used to develop dose–response models for *L. monocytogenes*, but mice are more susceptible to listeriosis than humans, so using mice will overestimate the risk (Golnazarian, Donnelly, Pintauro, & Howard, 1989). In addition, there is considerable variation among strains of *L. monocytogenes* in their ability to cause disease, and this should be considered when developing dose–response curves. Despite these uncertainties, dose–response relationships have been estimated for *L. monocytogenes* based on studies in animal models and human illness data for both the normal healthy population and for many at risk populations (Buchanan, Damart, Whiting, & Van Schothorst, 1997; Farber, Ross, & Harwig, 1996; Lindqvist & Westoo, 2000; HHS/USDA, 2003).

In the HHS/USDA (2003) risk assessment, dose–response was estimated for three different populations: perinatal (16 weeks after conception to 30 days after birth); elderly (60 or more years of age); and intermediate-age (general population less than 60 years old, includes healthy people and people more susceptible to listeriosis). The drawback to this approach is that individuals who are immunocompromised but under 60 years of age will be included with the healthy population, confounding the

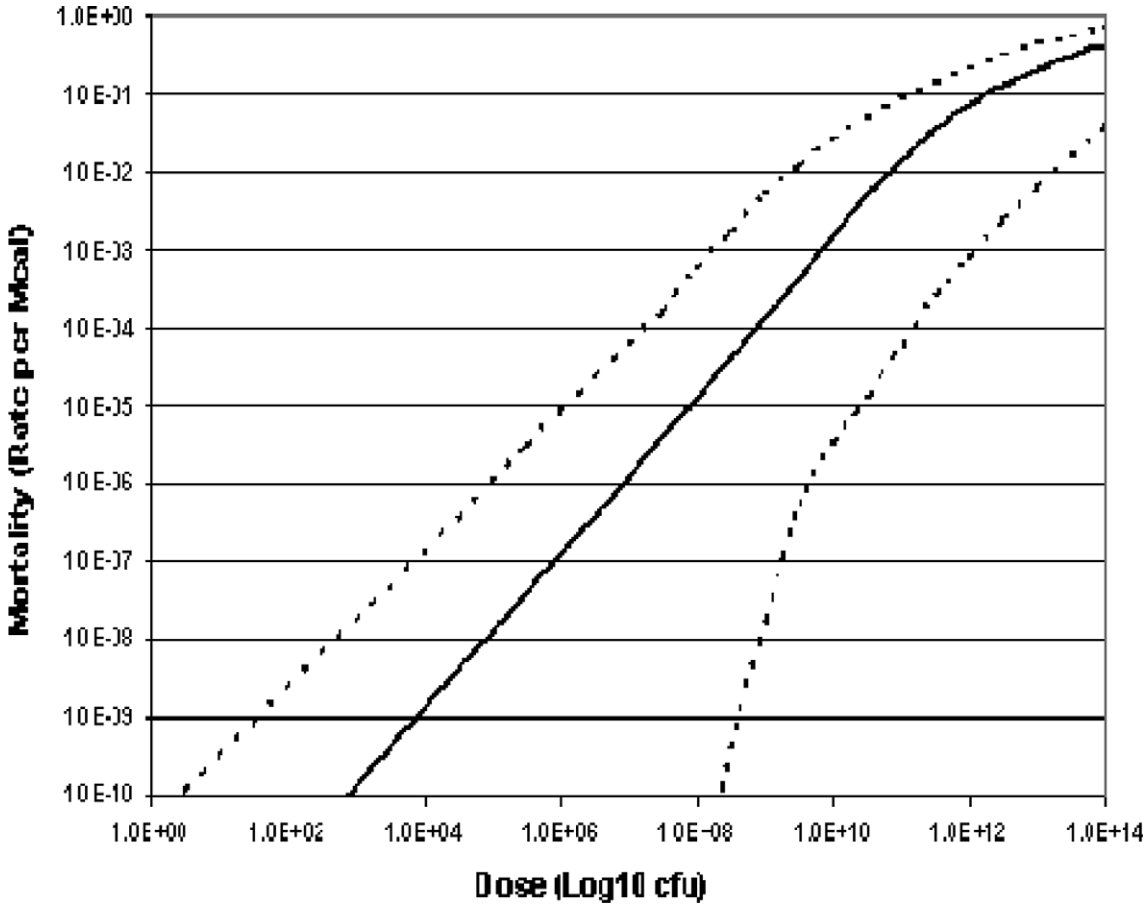

Fig. 1. Dose–response curve for *L. monocytogenes* for neonatal populations (newborns infected from contaminated foods consumed by their mothers before birth), showing the probability of mortality for a given dose (HHS/USDA, 2003).

dose–response assessment. However, insufficient data were available to separate these two groups in a meaningful way.

Dose–response was estimated by combining exposure assessment data with epidemiological data to derive a dose–response model for each population (Fig. 1). The shape of the dose–response curve was based on mouse lethality data for *L. monocytogenes* but the position of the dose–response curve was fixed by "anchoring" the curve to US annual disease statistics (CDC, 2006; Mead et al., 1999). Mice are much more susceptible to listeriosis than humans, so anchoring the curve to human disease statistics prevents the dose–response curve from over-estimating human fatalities to listeriosis. This provides the most accurate estimate of dose–response given the difficulties associated with developing dose–response curves.

5.3. Exposure assessment

A probabilistic assessment was made of the number of *L. monocytogenes* consumed based on the prevalence of contamination of foods, the extent of contamination, amount of growth before consumption, amount of food consumed at a serving and the frequency that a food was consumed. Table 1 shows the prevalence and contamination levels of *L. monocytogenes* in various ready-to-eat foods. Table 2 shows the frequency of consumption. Table 3 provides a summary of the exposure assessment data that was used for the risk characterization.

5.4. Risk characterization

In the risk characterization, the HHS/USDA risk assessment considered the risk per serving to an individual consumer and the risk per annum to various populations. Individual food category data and the dose–response model were used to predict the number of cases per serving and per year for each food category. This allowed foods to be ranked. An uncertainty analysis was performed and results were compared with existing knowledge to ensure that they conformed to within expected boundaries.

Table 2
Frequency of consumption (HHS/USDA, 2003)

Food categories	Servings/year-US (millions)	Amount/serving (g)
Smoked seafood	200	57
Soft unripened cheese	4410	29
Pasteurized milk	87,000	244
High fat dairy products	21,000	13
Fermented meats	1800	46
Deli meats	21,000	56
Deli-type salads	13,000	96
Pâté	120	57

Table 3
Summary of exposure to *L. monocytogenes* (HHS/USDA, 2003)

Listeria monocytogenes levels in food (per serving)	Number of servings (per year in the US)	Number of servings (per person per year)
<1.0	3.3×10^{11}	1300
1.0–1000	4.9×10^{9}	19
>1000 < 1,000,000	6.2×10^{8}	2.4
>I,000,000 < 1,000,000,000	1.3×10^{8}	0.5
>1,000,000,000	7.3×10^{7}	0.3

Fig. 2 is a risk characterization curve showing the results of a risk ranking for *Listeria monocytogenes* in various foods, based on the predicted cases of listeriosis associated with the foods, on a per serving basis. In the USA, deli meats, frankfurters (not reheated) and pâté and meat spreads pose a much greater risk (about 1 case of listeriosis per 10^7 servings is predicted) than hard cheeses, cultured milk products and processed cheeses, where the predicted level of illness is approximately 1 case of listeriosis per 10^{14} servings. The main reason for this is that the former group of foods supports the growth of *L. monocytogenes* to high numbers while the latter group does not.

Food categories were grouped as follows:

5.4.1. Risk designation – very high

This group included delicatessen meats and frankfurters – not reheated. Risk assessors predicted a high relative risk per serving and per annum, due to relatively high rates of

Table 1
Prevalence and contamination levels of *L. monocytogenes* in various ready-to-eat foods in 2000–2001 (ILSI, 2005)

Product categories	No. positive (no. tested)	Number of positive samples in each concentration range (cfu/g)							
		0.04[a] –0.1	>0.1–1	>1–10	>10–10^2	>10^2–10^3	>10^3–10^4	>10^4–10^5	>10^5–10^6
Deli salads	202 (8549)	162	28	9	2	0	1	0	0
Deli meats	82 (9199)	42	20	10	2	7	1	0	0
Fresh soft cheeses	5 (2931)	2	0	0	3	0	0	0	0
Bagged salads	22 (2966)	17	1	1	2	1	0	0	0
Blue-veined cheeses	23 (1623)	18	3	1	1	0	0	0	0
Mold-ripened cheeses	14 (1347)	12	0	2	0	0	0	0	0
Seafood salads	115 (2446)	82	19	10	2	2	0	0	0
Smoked seafood	114 (2644)	67	11	19	8	6	1	0	2
Total	577 (31,705)	402	82	52	20	16	3	0	2

[a] Positive in 25 g of a sample.

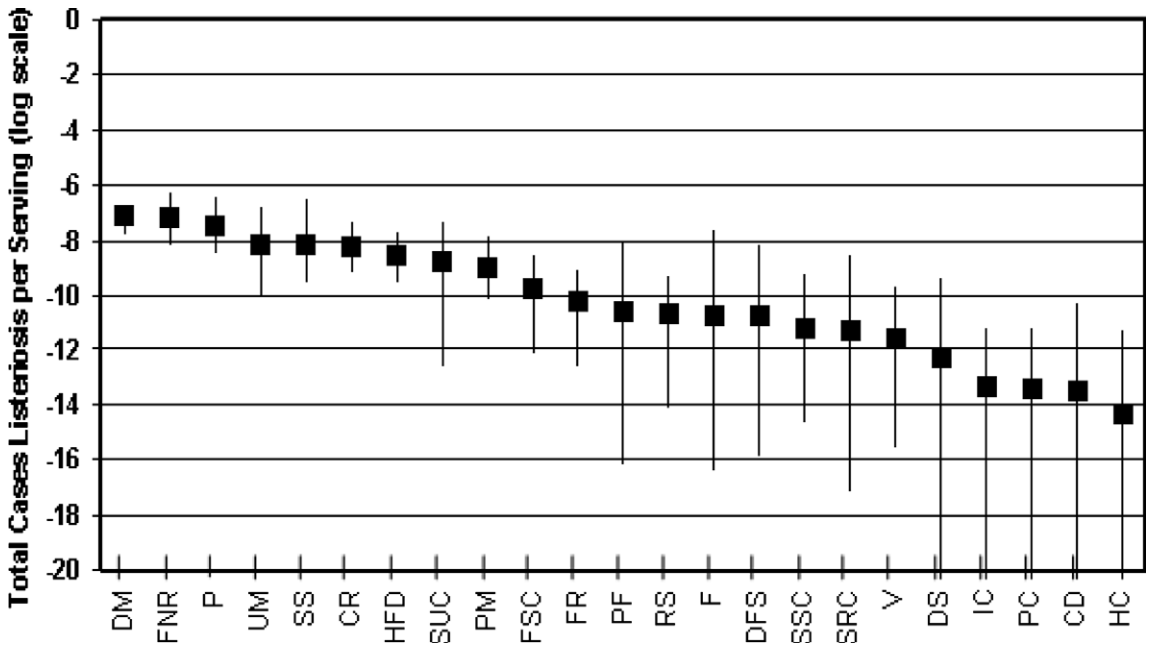

Fig. 2. Predicted cases of listeriosis (log scale) associated with food categories for the total united states population on a per serving Basis (HHS/USDA, 2003). [The box indicates the median predicted number of cases of listeriosis (log scale) and the bar indicates the lower and upper bounds (i.e., the 5th and 95th percentiles). The *y*-axis values are presented on a log scale. For example a log of −6 is equivalent to 1 case of listeriosis in a million servings.] DM, deli meats; FNR, frankfurters (not reheated); P, pâté and meat spreads; UM, unpasteurized fluid milk; SS, smoked seafood; CR, cooked ready-to-eat crustaceans; HFD, high fat and other dairy products; SUC, soft unripened cheese; PM, pasteurized fluid milk; FSC, fresh soft cheese; FR, frankfurters (reheated); PF, preserved fish; RS, raw seafood; F, fruits; DFS, dry/semi-dry fermented sausages; SSC, semi-soft cheese; SRC, soft ripened cheese; V, vegetables; DS, Deli-type salads; IC, ice cream and frozen dairy products; PC, processed cheese; CD, cultured milk products; HC, hard cheese.

contamination with *L. monocytogenes*, and the potential for rapid growth of *L. monocytogenes* to high numbers. Products were stored for extended periods, and had relatively high consumption rates. Immediate risk management action is needed for these types of products, which could include development of new control strategies and/or consumer education programs.

5.4.2. Risk designation – high

This group included pâté and meat spreads, smoked seafood, and unpasteurized fluid milk. Risk assessors predicted a high relative risk per serving or per annum. Products support the growth of *L. monocytogenes* to high numbers, and are contaminated at a moderate to high rate, but have low consumption rates. New control measures and/or continued avoidance of these products is warranted by those at high risk of listeriosis.

5.4.3. Risk designation – moderate

This group includes dry/semi-dry fermented sausages and frankfurters – reheated. These products include a bactericidal step or inhibitors, which reduces their risk as growth of *L. monocytogenes* to high numbers is prevented or retarded. Presence of *L. monocytogenes* is primarily associated with post-processing recontamination.

5.4.4. Risk designation – low

This group includes preserved fish and raw seafood. These products may have natural barriers to *L. monocytogenes* growth, e.g., competitive flora, or low water activity. They have low to moderate contamination and low consumption rates.

5.4.5. Risk designation – very low

This group includes cultured milk products, hard cheese, and ice cream. Risk assessers predicted a low per serving and per annum risk. These products do not support the growth of *L. monocytogenes* to high numbers, due to low water activity or being frozen. They are not likely to be a significant source of foodborne listeriosis.

Risk assessors found that the major factors that affect risk of listeriosis included the susceptibility of an individual, the amount and frequency of consumption of contaminated foods, the frequency and levels of contamination of the foods, the ability of the food to support growth of *L. monocytogenes* to high numbers and the opportunity for growth – which is a function of refrigerated storage temperature and refrigerated storage time.

6. Risk management options

The risk assessment results estimate that more than 99% of illnesses are associated with foods containing greater than 10,000 cfu *L. monocytogenes* per serving (Buchanan, 2003; Table 4). Therefore, strategies that reduce the likelihood of contamination of foods with *L. monocytogenes* or reduce the likelihood of growth of *L. monocytogenes* to high numbers will be effective at reducing listeriosis

Table 4
Estimated relationships between *Listeria monocytogenes* (Lm) dose per serving at the time of consumption and incidence of foodborne listeriosis in the US, based on the HHS/USDA (2003) risk assessment (Buchanan, 2003)

Level of Lm in food at consumption (cfu/serving)	% Servings annually at that level	% Cases of listeriosis attributable to that level
0.04	96.37	0.02
0.1	1.90	<0.01
1	0.91	0.01
10	0.43	0.03
100	0.21	0.13
1000	0.10	0.60
10,000	0.05	2.85
100,000	0.02	13.47
1,000,000 or greater	0.01	82.89

(ILSI, 2005). This can be achieved by implementing good hygiene practices (GHP), good management practices (GMP) and hazard analysis critical control point (HACCP) systems (Gallagher, Ebel, & Kause, 2003). A broad range of food control measures are available, including: (a) reformulating foods to include antimicrobials to prevent/retard growth of *L. monocytogenes* to high numbers; (b) post packaging listericidal treatments, (c) reduction of shelf life, or (d) use of competitive flora to minimize growth of *L. monocytogenes*.

An FSO can be used as a risk management tool for *L. monocytogenes* in ready-to-eat foods, because the FSO establishes the stringency of the measures that will be used to control *L. monocytogenes* by articulating the frequency or cell number of *L. monocytogenes* in the food that should not be exceeded at the time of consumption. The public health impact of setting an FSO for *L. monocytogenes* in ready-to-eat foods depends on how effectively industry can meet the FSO. The current standard for *L. monocytogenes* in the USA for regulatory purposes is no *L. monocytogenes* cells detected in the sample size tested. For a sample size of 25 g, this standard equates to <0.04 cfu/g. If this were achieved for all foods, the predicted number of cases of listeriosis would be <1 per year, based on risk assessment estimates (HHS/USDA, 2003). As there are an estimated 2500 cases of listeriosis in the USA per year (Mead et al., 1999) this standard is clearly not being achieved for all foods.

Science-based education messages targeted to susceptible populations are a critical component to the risk management of listeriosis (ILSI, 2005). One challenge lies in adequately defining susceptible populations. At the extreme, there may be individuals (e.g., transplant patients immediately after surgery) who are so susceptible to *L. monocytogenes* that the only protective FSO would be the total exclusion of foodborne exposure to the pathogen until the patients once again have a reasonable level of immune function (Lyytikäinen et al., 2000). In these populations, strict avoidance of foods that pose an increased risk of listeriosis may be necessary, and the only practical safety strategy may be the consumption of only commercially-sterile foods. Individuals at increased risk, such as pregnant women and the elderly, will need guidance on healthy eating, including avoid high-risk foods or cooking them before consumption. However, most of the population is at very low risk for listeriosis. Comprehensive food safety education should be provided to the general population, including good food handling practices, starting in preschool (ILSI, 2005).

FSOs will generally have to be implemented via the establishment of POs and PCs because an FSO is at the time of consumption so that it can be related directly to the public health goal. In the case of a ready-to-eat product that does not support growth of *L. monocytogenes*, the PC or PO at the point of manufacture may be the same as the FSO (e.g., the frequency and level of *L. monocytogenes* in a hard cheese). Alternatively, the PO or PC for a product may be substantially different at specific steps in the food chain in order to achieve the stated FSO. For example, if a ready-to eat product supports the growth of *L. monocytogenes* during normal refrigerated storage (e.g., cooked turkey) the PO at the point of manufacture will likely be more stringent than the FSO to account for the potential growth of the microorganism during distribution and home-use. Conversely, if a product is reliably and consistently cooked just prior to consumption (e.g., reheated frankfurters), a PO set at the time of manufacture could be less stringent than the FSO. However, care must be taken in such instance to ensure that *L. monocytogenes* infections are not caused by the product cross-contaminating other foods before they are reheated. By defining food safety goals in terms of FSO and their corresponding PO and PC, the focus is on defining what needs to be accomplished and allows the manufacturers to decide what strategy will be effective for their products and technological capabilities. This flexibility is one of the advantages of the FSO concept.

When establishing the FSO, an evaluation should be made to determine whether the FSO is achievable, i.e., whether food safety management systems can be implemented that will ensure that the FSO is met. For certain products it may be that current technologies in the industry do not allow the FSO to be met. In such instance, the government regulatory agency and the industry have effectively three choices, revise the FSO, identify a surrogate product (e.g., consumption of pasteurized milk cheeses instead of raw milk cheeses), or remove the product from commerce.

Setting a food safety objective, and designing strategies for meeting the objective, are effective means for reducing foodborne listeriosis, provided that the FSO is met. If the FSO is being met, the public health goal upon which it is based should be met. A reduction in the level of illness in the population related to the particular hazard should become apparent through disease surveillance. Verification through the acquisition of disease and food surveillance data is needed to estimate the burden of disease and relate it to the level of compliance to the FSO. Implementing effective food safety control measures, which ensure that

the FSO is being met consistently, is a key factor for reducing foodborne listeriosis.

Acknowledgement

This paper is based in part on a report by the ILSI RSI Working Group on *Listeria monocytogenes* in foods "Achieving Continuous Improvement In Reductions In Foodborne Listeriosis – A Risk Based Approach." Financial support for this project was provided by the American Frozen Food Institute, American Meat Institute, Cargill, Con Agra Foods, International Dairy Foods Association, ILSI North America Technical Committee on Food Microbiology, Joint Institute of Food Safety and Applied Nutrition (JIFSAN), Kraft Foods, Purac America Inc., Tyson Foods and the U.S. Department of Agriculture Food Safety and Inspection Service.

References

Bacon, R. T., Belk, K. E., Sofos, J. N., Clayton, R. P., Reagan, J. O., & Smith, G. C. (2000). Microbial populations on animal hides and beef carcasses at different stages of slaughter in plants employing multiple-sequential interventions for decontamination. *Journal of Food Protection, 63*(8), 1080–1086.

Buchanan, R. L. (2003). Comparison of the US and FAO/WHO *Listeria monocytogenes* risk assessments. *Presented at the 2003 CSL/JIFSAN joint symposium – food safety and nutrition: Risk analysis*. Adelphi, MD. Available from http://www.jifsan.umd. edu/presentations/csljifsan2003/robert_buchanan_2003.pdf.

Buchanan, R. L., Damart, W. G., Whiting, R. C., & Van Schothorst, M. (1997). Use of epidemiological and food survey data to estimate a purposefully conservative dose response relationship for *Listeria monocytogenes* levels and incidence of listeriosis. *Journal of Food Protection, 60*, 918–922.

Buchanan, R. L., Dennis, S., & Miliotis, M. (2004). Initiating and managing risk assessments within a risk analysis framework: FDA/CFSAN'S practical approach. *Journal of Food Protection, 67*(9), 2058–2062.

Buchanan, R. L., Golden, M. H., & Phillips, J. G. (1997). Expanded models for the non-thermal inactivation of *Listeria monocytogenes*. *Jouranl of Applied Microbiology, 82*(5), 567–577.

Buchanan, R. L., Smith, J. L., & Long, W. (2000). Microbial risk assessment: dose-response relations and risk characterization. *International Journal of Food Microbiology, 58*, 159–172.

Centers for Disease Control and Prevention (1989). Listeriosis associated with consumption of turkey franks. *MMWR, 38*, 267–268.

Centers for Disease Control and Prevention (1997). Outbreak of Staphylococcal food poisoning associated with precooked ham. *Morbidity and Mortality Weekly, 46*, 1189–1191.

Centers for Disease Control and Prevention (1998). Multistate outbreak of listeriosis – United States. *MMWR Morb. Mortal. Weekly Report, 47*, 1085–1086.

Centers for Disease Control and Prevention (CDC). (2006). Preliminary FoodNet Data on the Incidence of Infection with Pathogens Transmitted Commonly Through Food — 10 Sites, United States, 2005. MMWR Morb. Mortal. Wkly. Rep. April 14, 2006 /55(14); 392–395. <http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5514a2.htm?s_cid=mm5514a2_e#tab> Accessed 28.04.2006.

Codex Alimentarius Commission (CAC). (1999). Principles and guidelines for the conduct of a microbiological risk assessment. FAO, Rome. CAC/GL-30.

Codex Alimentarius Commission (CAC). (2004). Report of the thirty-sixth session of the Codex Committee on Food Hygiene ALINORM 04/27/13. Appendix III: Definitions to be included in the procedural manual. ftp://ftp.fao.org/docrep/fao/meeting/008/j2308e.pdf Accessed 28.04. 2006.

Dalton, C. B., Austin, C. C., Sobel, J., Hayes, P. S., Bibb, W. F., Graves, L. M., et al. (1997). An outbreak of gastroenteritis and fever due to *Listeria monocytogenes* in milk. *New England Journal of Medicine, 336*, 100–105.

Farber, J. M., Ross, W. H., & Harwig, J. (1996). Health risk assessment of *Listeria monocytogenes* in Canada. *International Journal of Food Microbiology, 30*, 145–156.

FDA Center for Food Safety and Applied Nutrition. (2006). Foodborne pathogenic microorganisms and natural toxins handbook (Bad Bug Book). <http://www.cfsan.fda.gov/~mow/intro.html> Accessed 28.04. 2006.

Gallagher, D. L., Ebel, E. D., & Kause, J. R. (2003). FSIS risk assessment for *Listeria monocytogenes* in Deli meats. <http://www.fsis.usda.gov/OPPDE/rdad/FRPubs/97-013F/ListeriaReport.pdf> Accessed 28.04.2006.

Gellin, B. G., & Broome, C. V. (1989). Listeriosis. *JAMA, 261*, 1313–1320.

Gill, C. O., Bryant, J., & Bedard, D. (1999). The effects of hot water pasteurizing treatments on the appearances and microbiological conditions of beef carcass sides. *Food Microbiology, 16*, 281–289.

Gill, C. O., Bryant, J., & McGinnis, C. (2000). Microbial effects of the carcass washing operations at three beef packing plants. *Fleishwirtsch International Issue, 3*, 46–48.

Gill, C. O., Jones, T., Bryant, J., & Brereton, D. A. (2000). The microbiological conditions of the carcasses of six species after dressing at a small abattoir. *Food Microbiology, 17*, 233–239.

Gill, C. O., McGinnis, J. C., & Bryant, J. (2001). Contamination of beef chucks with *Escherichia coli* during carcass breaking. *Jouranal of Food Protection, 64*, 1824–1827.

Golnazarian, C. A., Donnelly, C. W., Pintauro, S. J., & Howard, D. B. (1989). Comparison of infectious dose of *Listeria monocytogenes* F5817 as determined for normal versus compromised C57B1/6J mice. *Jouranal of Food Protection, 52*, 696–701.

Goulet, V., & Marchetti, P. (1996). Listeriosis in 225 non-pregnant patients in 1992: clinical aspects and outcome in relation to predisposing conditions. *Scandinavian Jouranl of Infectious Diseases, 28*, 367–374.

HHS. (2000). (US Department of Health and Human Services) *Healthy People 2010*. 2nd ed. With understanding and improving health and objectives for improving health. 2 vols. Washington, DC: US Government Printing Office.

HHS/USDA. (2003). (US Department of Health and Human Services/US Department of Agriculture.) Quantitative assessment of the relative risk to public health from foodborne *Listeria monocytogenes* among selected categories of ready-to-eat foods. Available from http://www.foodsafety.gov/~dms/lmr2-toc.html.

ICMSF (International Commission on Microbiological Specifications for Foods) (2002). *Microorganisms in Foods 7 – microbiological testing in food safety management*. New York, NY: Klewer Academic, pp. 290–291.

International Life Sciences Institute Risk Science Institute (ILSI RSI) (1996). A conceptual framework to assess the risks of human disease following exposure to pathogens. *Risk Analysis, 16*, 841–848.

ILSI RSI (2000). *Revised framework for microbial risk assessment*. Washington, DC: ILSI Press.

ILSI Research Foundation/Risk Science Institute Expert Panel on *Listeria monocytogenes* in Foods. (2005). Achieving continuous improvement in reductions in foodborne listeriosis – a risk-based approach, *Journal of Food Protection*, *68*, 1932–1994.

Jensen, A., Frederiksen, W., & Gerner-Smidt, P. (1994). Risk for listeriosis in Denmark, 1989–1990. *Scandinavian Journal of Infectious Diseases, 26*, 171–178.

Kasuga, F., Hirota, M., Wada, M., Yunokawa, T., Toyofuku, H., Shibatsuji, M., et al. (2004). Archiving of food samples from restaurants and caterers – quantitative profiling of outbreaks of

foodborne salmonellosis in Japan. *Journal of Food Protection, 67*, 2024–2032.

Lammerding, A. M., & Fazil, A. (2000). Hazard identification and exposure assessment for microbial food safety risk assessment. *International Journal of Food Microbiology, 58*, 147–157.

Linnan, M. J., Mascola, L., Lou, X. D., Goulet, V., May, S., Salminen, C., et al. (1988). Epidemic listeriosis associated with Mexican-style cheese. *New England Journal of Medicine, 319*, 823–828.

Lindqvist, R., & Westoo, A. (2000). Quantitative risk assessment for *Listeria monocytogenes* in smoked or gravad salmon and rainbow trout in Sweden. *International Journal of Food Microbiology, 58*(3), 181–196.

Logue, C. M., Sheridan, J. J., & Harrington, D. (2005). Studies of steam decontamination of beef inoculated with *Escherichia coli* O157:H7 and its effect on subsequent storage. *Journal of Applied Microbiology, 98*(3), 741–751.

Lyytikäinen, O., Autio, T., Maijala, R., Runtu, P., Honkanen-Buzzalski, T., Mjettinen, M., et al. (2000). An outbreak of *Listeria monocytogenes* serotype 3a infections from butter. *Jouranl of Infectious Diseases, 181*, 1838–1841.

McEvoy, J. M., Doherty, A. M., Sheridan, J. J., Blair, I. S., & McDowell, D. A. (2003). The prevalence of *Salmonella* spp. in bovine faecal, rumen and carcass samples at a commercial abattoir. *Journal of Applied Microbiology, 94*, 693–700.

Mead, P. S., Slutsker, L., Dietz, V., McCaig, L. F., Bresee, J. S., Shapiro, C., et al. (1999). Food-related illness and death in the United States. *Emerging Infectious Diseases, 5*, 607–625. Available fromwww.cdc.gov/ncidod/eid/vol5no5/mead.htm. .

Murphy, S. P. (2003). Collection and analysis of intake data from the integrated survey. *Journal of Nutrition, 133*, 585S–589S.

O'Brien, S. B., Duffy, G., Carney, E., Sheridan, J. J., McDowell, D. A., & Blair, I. S. (2005). Prevalence and numbers of *Escherichia coli* O157 on bovine hides at a beef slaughter plant. *Journal of Food Protection, 68*(4), 660–665.

Pearce, R. A., Sheridan, J. J., & Bolton, D. J. (2006). Distribution of airborne microorganisms in commercial pork slaughter processes. *International Journal of Food Microbiology, 107*(2), 186–191.

Salamina, G., Donne, E. D., Niccolini, A., Poda, G., Cesaroni, D., Bucci, M., et al. (1996). A foodborne outbreak of gastroenteritis involving *Listeria monocytogenes*. *Epidemiological Infections, 117*, 429–436.

Sheridan, J. J., Duffy, G., McDowell, D. A., & Blair, I. S. (1994). The occurrence and initial numbers of *Listeria* in Irish meat and fish products and the recovery of injured cells from frozen products. *International Journal of Food Microbiology, 22*(2–3), 105–113.

Slutsker, L., & Schuchat, A. (1999). Listeriosis in humans. In E. T. Ryser & E. H. Marth (Eds.), *Listeria*, listeriosis and food safety (2nd ed.) (pp. 75–95). New York, NY: Marcel Dekker, Inc.

Sofos, J. N., Smith, G. C., Kochevar, S. L., & Reagan, J. O. (1999). Incidence of *Salmonella* on beef carcasses relating to the US. *Meat and Poultry Inspection Regulations Journal of Food Protection, 62*(5), 467–473.

Stringer, M. (2004). ILSI Europe Report on Food Safety Objectives – Role in Microbiological Food Safety Management. Available from http://europe.ilsi.org/publications/pubslist.cfm?publicationid=551.

US Department of Agriculture, Food Surveys Research Group. (2000). Survey Nutrient Database. CD-ROM (PB2000-500027) 2000 National Technical Information Service Springfield, VA.

Walls, I. (2004). Using risk-based approaches for managing microbial food safety. *Nutrition in Clinical Care, 7*(4), 156–160.

Walls, I., & Buchanan, R. L. (2005). Use of food safety objectives as a tool for reducing foodborne listeriosis. *Food Control, 16*, 795–799.

Walls, I., & Scott, V. N. (1997a). Validation of predictive mathematical models describing growth of *Listeria monocytogenes*. *Journal of Food Protection, 60*, 1142–1145.

Walls, I., & Scott, V. N. (1997b). Use of predictive microbiology in microbial food safety risk assessment. *International Journal of Food Microbiology, 36*, 2–3.

Whiting, R. C. (1995). Microbial modeling in foods. *Critical Reviews in Food Science and Nutrition, 35*(6), 464–494.