Health risk assessment of *Listeria monocytogenes* in Canada

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Abstract

In this review, the major steps used in the formulation of a health risk assessment for *Listeria monocytogenes* in foods are discussed. Data is given on the numbers of human listeriosis cases reported in Canada along with the current Canadian regulatory policy on *L. monocytogenes*. Four major steps in the health risk assessment of this organism in foods, namely, hazard identification, hazard characterization, exposure assessment and risk characterization, were examined. For hazard characterization, since it is known that no direct human dose response data is available for *L. monocytogenes*, a flexible dose response model called the Weibull-Gamma model was evaluated. For the exposure assessment, pâté and soft cheese, both high-risk foods in terms of listeriosis infection, were used as prototypes in some of the models that were used. Using disappearance data for cheese and 100 g as a typical serving, the data suggested an average of 102 servings per capita, per year in Canada. As a rough approximation, for *L. monocytogenes*, reference ID\(_{10}\) and ID\(_{90}\) dose levels of response for both normal and high risk populations were given as 10\(^7\) and 10\(^9\) for normal individuals, and 10\(^5\) and 10\(^7\) for high-risk people. The corresponding dose response models were graphically displayed. These models exhibited a higher degree of susceptibility and less host/pathogen heterogeneity for the higher risk group. The range of doses between the ID\(_{10}\) and ID\(_{90}\) reference values corresponded roughly to levels associated with cases of listeriosis. In the risk characterization stage, dose response data was combined with some predictive
growth modeling data of *L. monocytogenes* on pâté, assuming an initial exposure of a single cell for food stored at 4°C and 8°C. Storage of pâté at 4°C for more than 35 days resulted in a rapidly increasing risk for the high risk population, while storage at 8°C produced a similar risk after about 13 days. In addition, an equation, used to calculate the average probability of acquiring human listeriosis in Canada from soft and semi-soft cheese consumption, was formulated. Computations derived from this equation indicated a substantial level of consistency between reported data and assumptions of the risk assessment model. An important part of risk characterization or possibly risk management is characterizing the economic and social consequences of estimated risks. The total annual estimated cost of listeriosis illnesses and deaths in Canada was estimated to be between 11.1 and 12.6 million dollars.

**Keywords:** *Listeria monocytogenes*; Incidence; Cost; Health risk assessment; Dose response models; Regulatory policy

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1. Introduction to risk assessment in the Health Protection Branch

The Canadian regulatory policy on the control of foodborne pathogens is based on current knowledge and stresses a requirement for health risk analysis. However, health risk analysis for foodborne microorganisms in the Health Protection Branch (HPB) is still in its infancy. To date, there is no general agreement on the risk-related terms and definitions that should be used in such risk analysis, but there is a trend to use those of the Codex Alimentarius. Presumably, Codex Alimentarius risk assessment methodologies will be adopted by the HPB once developed and accepted internationally. Quantitative microbial risk assessments are increasingly being applied within Health Canada for policy direction, resource allocation, and determining priorities in research and surveillance projects. In this context, quantitative risk assessment refers to the formal synthesis of data derived from both experimental and observational studies with other qualitative information for the purposes of risk assessment. It has been realized, however, that research should be directed toward better quantification of overall risks, thereby providing the data required for such a task.

In this paper, the major steps used in the formulation of a health risk assessment for *Listeria monocytogenes* will be discussed. In addition, a health risk assessment of *L. monocytogenes* in pâté and cheese will be used as examples.

2. Human listeriosis in Canada

The history of human listeriosis in Canada dates back to the early 1950s and one of the earliest descriptions of the organism was made by a renowned Canadian scientist, Dr. E.G.D. Murray (Murray et al., 1926). Since then, only around 15 cases a year were reported and the foodborne aspects of the disease were not recognized until 1982, when the first well documented outbreak of foodborne listeriosis in the world occurred in the Maritime provinces of Canada. The
outbreak, traced to contaminated packaged coleslaw mix, was thought to have occurred via raw cabbage which was fertilized during the growing period with sheep manure, originating from farms known to have had cases of ovine listeriosis (Schlech et al., 1983). Rather ironically, in Canada, no further outbreaks of listeriosis have occurred since this outbreak, although numerous other foodborne listeriosis outbreaks have occurred worldwide (McLauchlin, 1993).

Canada's national Listeria surveillance program and data collection on cases of human listeriosis began in 1987. However, in reality, only four provinces (Ontario, Manitoba, Saskatchewan and Newfoundland) have reported cases. A summary of the cases occurring in Canada and the U.S.A. is presented in Table 1. The actual number of cases occurring with only mild upper gastrointestinal (GI) symptoms, is not known but mild diarrheal-type episodes can occur, as evidenced by several recent outbreaks outside Canada (Riedo et al., 1994; Proctor et al., 1995).

3. Canadian regulatory policy on *Listeria monocytogenes*

The Canadian regulatory policy on *Listeria* contaminated foods is based on the principles of HACCP and contains elements of a health risk assessment approach. Canada's updated policy, effective November 1, 1994, reflects current knowledge that the risk of contamination by *L. monocytogenes* can be reduced, but the organism cannot always be eradicated from finished product or the environment (Anonymous, 1994a). Intended to be applied at the manufacturing level, the policy directs inspection and compliance action to ready-to-eat (RTE) foods which are capable of supporting growth of the organism. More specifically, the highest priority is given to those RTE foods which have been causally linked to listeriosis and those with a greater than 10 day shelf life. The policy is based on a combination of inspection, environmental sampling and product testing, with foods being placed into three categories, based upon health risk (Table 2).

**Table 1**
Human listeriosis cases in Canada and the USA

<table>
<thead>
<tr>
<th>Year</th>
<th>Canada</th>
<th>Ontario</th>
<th>Canada - adjusted (Ontario)</th>
<th>USA (5 areas)</th>
<th>USA (projected)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1987</td>
<td>44</td>
<td>16</td>
<td>44 (1.7)&lt;sup&gt;d&lt;/sup&gt;</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1988</td>
<td>63</td>
<td>23</td>
<td>63 (2.4)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1989</td>
<td>63</td>
<td>26</td>
<td>71 (2.7)</td>
<td>151</td>
<td>1965 (7.9)</td>
</tr>
<tr>
<td>1990</td>
<td>49</td>
<td>29</td>
<td>79 (3.0)</td>
<td>147</td>
<td>1914 (7.7)</td>
</tr>
<tr>
<td>1991</td>
<td>49</td>
<td>40</td>
<td>109 (4.0)</td>
<td>117</td>
<td>1523 (6.1)</td>
</tr>
<tr>
<td>1992</td>
<td>32</td>
<td>21</td>
<td>57 (2.1)</td>
<td>88</td>
<td>1146 (4.6)</td>
</tr>
<tr>
<td>1993</td>
<td>56</td>
<td>48</td>
<td>128 (4.5)</td>
<td>84</td>
<td>1092 (4.4)</td>
</tr>
<tr>
<td>1994</td>
<td>46</td>
<td>34</td>
<td>91 (3.1)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup>80–90% of cases are thought to be food-related.
<sup>b</sup>Figures were projected for the whole country based on the data obtained from Ontario.
<sup>c</sup>Area of 19.1 million (see Tappero et al., 1995)
<sup>d</sup>Numbers in bracket signifies cases per million population.
<table>
<thead>
<tr>
<th>Category</th>
<th>Action level for LM</th>
<th>GMP status</th>
<th>Immediate action</th>
<th>Follow-up action</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. RTE foods causally linked to listeriosis (This list presently includes: soft cheese, liver pâté, coleslaw mix with shelf-life &gt; 10 days, jellied pork tongue)</td>
<td>&gt; 0 cfu/50 g</td>
<td>n/a</td>
<td>Class I recall to retail level</td>
<td>Consideration of public alert</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Appropriate follow-up at plant level</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. All other RTE foods supporting growth of LM with refrigerated shelf-life &gt; 10 days</td>
<td>&gt; 0 cfu/25 g</td>
<td>n/a</td>
<td>Class II recall to retail level</td>
<td>Consideration of public alert</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Appropriate follow-up at plant level</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. RTE foods supporting growth of LM with refrigerated shelf-life ≤ 10 days and all RTE foods not supporting growth</td>
<td>≤ 100 cfu/g&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Adequate GMP</td>
<td>Allow sale</td>
<td>Appropriate follow-up at plant level</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>≤ 100 cfu/g&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Inadequate or no GMP&lt;sup&gt;e&lt;/sup&gt;</td>
<td>Consideration of class II recall or stop sale</td>
<td>Appropriate follow-up at plant level</td>
</tr>
<tr>
<td></td>
<td>&gt; 100 cfu/g&lt;sup&gt;c&lt;/sup&gt;</td>
<td>n/a</td>
<td>Class II recall or stop sale</td>
<td>Appropriate follow-up at plant level</td>
</tr>
</tbody>
</table>

<sup>a</sup>At present, this product is not commonly found in the Canadian marketplace.

<sup>b</sup>RTE food not supporting growth of LM includes the following:

(a) pH 5.0-5.5 and $a_w < 0.95$;
(b) pH < 5.0 regardless of $a_w$;
(c) $a_w \leq 0.92$ regardless of pH;
(d) frozen foods.

<sup>c</sup>Enumeration done by direct plating onto LPM and Oxford agar (Farber and Daley, 1995).

<sup>d</sup>n/a, not applicable.

<sup>e</sup>No information on GMP is considered as no GMP. Burden of proof remains with the legal agent.
4. Health risk assessment: introduction:

Health risk assessment as defined by Codex refers to the 'scientific evaluation of the probability of occurrence of known or potential adverse health effects resulting from human exposure to foodborne hazards' (Anonymous, 1995). The four major steps in the process include (i) hazard identification; (ii) hazard characterization, (iii) exposure assessment and (iv) risk characterization. The definition includes both quantitative and qualitative expressions of risk, along with their associated uncertainties.

In general, the quantification of risk for the assessment of any identified human health hazard requires characterizing two basic elements: (a) the exposure of the human population to the identified hazard, and (b) the corresponding tolerance of the population to a given level of exposure. In most cases, both elements are subject to inherent variability (Anonymous, 1994b). In addition, there is often considerable uncertainty, or lack of knowledge associated with both elements. This is no less true when attempting to quantify health risks from microbial hazards.

Individual adverse health effects related to microbial pathogens usually result from a single acute exposure, rather than long term chronic exposure to a microbial hazard. Characterizing these exposures requires determining the prevalence of the pathogen of interest in the food supply. However, prevalence will vary considerably between food lots, food types and across time. Low levels of pathogens inhomogeneously located in the food supply are difficult to detect and therefore estimate. Unlike many other hazards, bacterial pathogens may grow or be inactivated, causing additional fluctuations in the overall prevalence in the food supply (Anonymous, 1994b).

Consumption patterns vary between individuals. These differences may have strong demographic components such as sex, age, culture, and health status. Detailed information on consumption patterns is often not readily accessible or does not exist.

An important part of characterizing any hazard is the description of the susceptibility or tolerance of the exposed population. Individual tolerances will depend on major risk factors such as sex, age, and health status. In addition, it should be expected that tolerances will vary between individuals with identical risk profiles, and that an individual's tolerance to a particular pathogen will vary from day to day.

An individual’s susceptibility to a pathogen may also depend on the particular strain. Furthermore, each incident of exposure to a particular strain of a given pathogen represents a different subpopulation, or sample, from the overall population of microbes. This also contributes to the variability in observed dose response relationships. Finally, there is very little direct experimental evidence of human dose response for microbial pathogens (Anonymous, 1994b).

Despite the present shortcomings of health risk assessment, numerous benefits can result from the process. Some of the major ones would include the ability, (i) to estimate human risk; (ii) to be used as a framework for collecting and organizing data and for dividing up responsibility for analysis; (iii) to provide transparent and
uniform health safety information to risk managers; and (iv) to point out areas where insufficient information is available with which to make a reasonable decision regarding a particular risk (Anonymous, 1995).

4.1. Hazard identification

*Listeria monocytogenes* is a facultative intracellular bacterial pathogen of both humans and animals. It causes listeriosis in humans, with a variety of symptoms including mild diarrhea, meningitis, and septicemia (Marth, 1988). Epidemiological evidence suggests that most exposure is foodborne (Ciesielski et al., 1988; Broome et al., 1990; Farber and Peterkin, 1991; McLauchlin, 1993). Although listeriosis occurs infrequently, at somewhere between 2 and 7 cases per million population, between 20 to 40% of the cases are fatal (McLauchlin, 1993; Rocourt, 1994). In addition, *L. monocytogenes* is found in many different foods (Farber and Peterkin, 1991). However, illness is associated with only a few virulent strains (Farber and Peterkin, 1991; McLauchlin, 1993; Rocourt, 1994). Major risk factors include immunosuppression, pregnancy and age (Gellin and Broome, 1989; Schuchat et al., 1991). This broad based prevalence in the food system, together with the high mortality rate of listeriosis suggests that *L. monocytogenes* represents an important, emerging hazard to human health.

4.2. Hazard characterization

Hazard characterization represents the qualitative and quantitative evaluation of the nature of the adverse effects. Although the potential effects of exposure to *L. monocytogenes* are wide ranging in severity, listeriosis is the common precursor. As a result, efforts at hazard characterization have concentrated on establishing a dose response model. Such a model provides the functional relationship between the probability that an individual will contract listeriosis and a specified dose, or level of exposure to a virulent strain of *L. monocytogenes*.

Work by Furumoto and Mickey (Furumoto and Mickey, 1967a,b) and Haas (Haas, 1983) derived the Beta-Poisson (BP) model for microbial dose response data. This model provides reasonable fits to several available dose response data sets (Haas, 1983; Haas et al., 1993). The BP model combines a one-hit model for individual dose response with host/organism heterogeneity modelled by the beta distribution. The model calculates the average probability of infection or illness, assuming that the level of exposure follows a Poisson law (hence the name Beta-Poisson).

There is no experimental dose response data on humans available for *Listeria*, i.e. the minimum infectious dose (MID) of *L. monocytogenes* for humans is unknown. For *L. monocytogenes*, as well as many other foodborne pathogens, the MID will depend on factors such as the virulence of the strain, the type and amount of food consumed, the levels of the organism in the food and the state of the host. Some animal data is available, mainly using the mouse model (Audurier et al., 1980; Golnazarian et al., 1989). However, extrapolation of the mouse data to the human situation is tenuous, at best.
Since no direct dose response data is available, the choice model must be flexible enough to accommodate all qualitative information and also be adaptable to both healthy and high risk groups. A flexible dose response model is provided by the Weibull model (Krewski and van Ryzin, 1980):

$$P(d) = 1 - e^{-ad^b}$$

Here, $P(d)$ denotes the probability of illness for an individual exposed to $d$ L. monocytogenes cells. The properties of this relationship are determined by the parameters $a$ and $b$. The parameter $a$ is related to the probability of illness given exposure to a single Listeria cell. Specifically, using $P$ to denote this probability, $a = -\ln (1 - P)$. Host/pathogen heterogeneity for a particular risk group can be described by specifying a probability distribution to either $a$ or $P$.

The parameter $b$ determines the shape of the individual dose response curve. For example, values of $b > 2$ give sigmoidal-shaped curves associated with extremely low probabilities of illness at low doses, followed by a rapid increase in probability at doses near the ID$_{50}$ level. In addition, a plot of $\log(P(d))$ versus $\log(d)$ has slope $b$ at low doses. Therefore, at low doses, $1/b$ is the number of log reductions in dose required to give a one log reduction in risk.

Host/pathogen heterogeneity can be described by specifying a Gamma distribution with parameters $\alpha$ and $\beta$ for the Weibull parameter $a$. As a result, the average probability of illness given a dose $d$, is

$$P_I(d) = 1 - \left[1 + (d^b)/\beta\right]^{-\alpha}$$

$P_I(d)$ will be referred to as the Weibull-Gamma (WG) model. Several well-known models are obtained as special cases of the WG model. For example, if $b = 1$, then the WG model reduces to the BP dose response relationship. Similarly, setting $\alpha = 1$, results in the log-logistic model which is a common alternative to the lognormal dose response model.

The parameters of the model are chosen to reflect our present understanding of the properties of the dose response relationship. This can be accomplished by setting reference values which the dose response model should approximate. As a rough approximation, for L. monocytogenes, reference ID$_{10}$ and ID$_{90}$ dose levels (i.e. dose causing illness in 10 and 90% of the population, respectively) of response for both normal and high risk populations are given respectively as $10^7$ and $10^9$ for normal individuals, and $10^5$ and $10^7$ for high-risk people. The corresponding dose response models are displayed graphically in Fig. 1. These models exhibit a higher degree of susceptibility and less host/pathogen heterogeneity for the higher risk group. The range of doses between the ID$_{10}$ and ID$_{90}$ reference values corresponds roughly to levels associated with cases of listeriosis (Farber and Peterkin, 1991; McLauchlin, 1993).

4.3. Exposure characterization

Data from Agri-Food and Agriculture Canada demonstrates average incidence data for L. monocytogenes of 4.4 and 1.2% for meats and dairy products, respec-
Fig. 1. Dose response curve for *Listeria monocytogenes* infections derived using the Weibull-Gamma model. Solid line, normal population; broken line, high risk population.

tively (Agriculture and Agri-Food Canada, unpublished data). These are values similar to those reported from other countries (McLauchlin and Gilbert, 1990). However, these values represent the presence of all *L. monocytogenes* strains, regardless of their pathogenic potential, thereby providing an overestimate of prevalence. In terms of the levels of *L. monocytogenes* present in these foods, the organism can be present in both pâté and soft cheese (the two foods that we have chosen to focus on) at levels up to $10^6$ to $10^7$ organisms per g (Farber and Peterkin, 1991). It is difficult to make any definitive statements about the distribution of the organisms in these two foods, although it is known that for soft cheese, the organisms tend to be in higher concentrations in and/or near the rind because of the higher pH values found in these areas. For pâté, and probably other refrigerated ready-to-eat meats, post-processing contamination can occur either at the manufacturing or the retail level, and the organism is assumed to be more heavily concentrated on the top slice or surface of the meat product.

Accurate data on individual consumption patterns are not available for either cheese or liver pâté. Disappearance data for cheese, available from Statistics Canada indicates that per capita consumption of soft and semi-soft cheese has been consistent over the past five years at near 5.5 kg. Using 100 g as a typical serving, this data suggests an average of 55 servings per capita, per year. There is no published disappearance data for pâté.
It is well known that temperature abuse occurs quite often at the retail level and that temperatures of 8°C or higher would not be unexpected. Temperature abuse is likely to be partly responsible for the wide range of levels of \textit{L. monocytogenes} observed in foods at the retail level.

The effect of temperature abuse on the levels of \textit{L. monocytogenes} was calculated using our own growth kinetic model for pâté. The growth model shows that at 4°C levels can increase from 1 cell to $10^5$ cells in just under 40 days, while at 8°C the same levels are achieved in about 15 days (Farber et al., 1995).

4.4. Risk characterization

Characterizing the risk associated with \textit{L. monocytogenes} in foods involves a consideration of all the information gathered in the hazard identification, hazard characterization and exposure assessment steps. It can be helpful in determining the cause of the risk and in providing managers with background information to do risk management. This information can be combined to assess various outputs, i.e., the annual incidence of listeriosis, the impact of heat abuse on probability of illness, and the effectiveness of various exposure reduction strategies. Although the quantitative risk assessment approach is preferred over the qualitative approach, it is not

Fig. 2. Effect of 4°C and 8°C (temperature abuse) storage of pâté on the probability of \textit{Listeria monocytogenes} infections for both normal and high-risk populations calculated using the Weibull-Gamma model and the growth kinetics model of Farber et al. (1995). Growth is assumed to originate from a single cell. Solid lines, normal population; broken lines, high risk population; no star, 4°C; star, 8°C storage.
yet clear whether the former approach is possible and/or appropriate for characterizing the risks associated with foodborne bacterial pathogens.

The impact of growth on the probability of illness can be illustrated graphically for various scenarios. Fig. 2 combines the dose response model with the pâte growth model for L. monocytogenes, assuming an initial exposure of a single cell for food stored at 4 and 8°C, respectively. It can be observed that a single cell presents negligible risk even to the high risk population. However, storage at 4°C for more than 35 days results in rapidly increasing risk for the high risk population, while storage at 8°C produces a similar risk after about 13 days (Fig. 2).

An important measure of the level of risk to listeriosis through exposure from a specified food source is the annual incidence rate, i.e. actual annual number of human listeriosis cases. The incidence of listeriosis depends on the proportion of the population exposed to L. monocytogenes (prevalence), the level of exposure of each individual at risk, and their individual tolerances.

The incidence of listeriosis can be interpreted as the product of the annual incidence of exposure and the average probability of illness. The average probability of illness represents an average across the exposed population. It is the sum of two components — one due to the normal sub-population, the other due to the high risk subpopulation. It depends on the distribution of dose levels across the exposed population as well as food consumption patterns.

The incidence of exposure is a combination of the prevalence of virulent L. monocytogenes strains in the total annual servings of the specified food and the number of servings per capita.

In order to examine this equation in more detail, the following notation is introduced:

- \( p \) — incidence of L. monocytogenes in foods,
- \( c \) — consumption of typical servings (servings/capita/year),
- \( u_i \) — proportion of virulent L. monocytogenes strains,
- \( n \) — the proportion of the exposed population with a normal risk profile,
- \( P_{1,N} \) — the average probability of illness for individuals having a normal risk profile,
- \( P_{1,H} \) — the average probability of illness corresponding to a high risk profile,
- \( I \) — the reported annual incidence of listeriosis due to a specified food source,
- \( u_2 \) — rate of under reporting.

The incidence of listeriosis can be expressed symbolically by the equation:

\[ p c u_i [nP_{1,N} + (1 - n)P_{1,H}] = I u_2. \]

The use of this equation can be illustrated by considering the incidence of listeriosis from exposure to L. monocytogenes in soft and semi-soft cheeses. Using 100 g as a typical serving with \( c = 55 \), data from Farber and Peterkin (1991) suggests that a typical dose of L. monocytogenes from contaminated cheese would be \( 10^3 \text{–} 10^4 \) cells/serving. About 80% of the population has normal risk, so that \( n = 0.80 \). As a result, the average probability of illness is computed to be \( 2.5 \times 10^{-6} \) to \( 2.5 \times 10^{-4} \). Using \( p = 0.012 \) as the incidence of L. monocytogenes, with \( u_i \) in the range 0.01 to 0.1, the left hand side of the equation yields estimates of the incidence of listeriosis in the range \( 1.7 \times 10^{-8} \) to \( 1.7 \times 10^{-3} \).
Using the right hand side of the equation, Table 1 shows the reported annual incidence of human listeriosis in Canada ranges from 1.7 to 4.5 per million population. If one assumes that 10–20% of these cases are attributed to exposure to *L. monocytogenes* through cheese consumption, and the under reporting rate, \( u_2 \), is in the range 10 to 100, then the incidence of listeriosis is placed in the range \( 1.7 \times 10^6 \) to \( 9.0 \times 10^{-5} \). The fact that the range of incidence values is wider for computations for the left hand side is to be expected, since a larger number of uncertain values are used in that computation. The two computations indicate a substantial level of consistency between reported data and assumptions of the risk assessment model.

The previous computation, although simple, exhibits the pervasive nature of uncertainty in microbial risk assessment. For more complex calculations, a detailed analysis of the impact of uncertainty on predicted outcomes is important. Such an analysis requires identifying a joint probability distribution for all inputs to the risk model. A probability distribution for predicted outputs can then be generated (usually by simulation) and various measures of precision for these predicted values established.

An important addition to risk characterization (or possibly risk management) is characterizing the economic and social consequences of estimated risks. Taking the average number of cases from 1990 to 1995 as 93 (Ontario-adjusted figures, see Table 1), and using the figures from Roberts (1989) and Todd (1989), the total annual estimated cost of listeriosis illnesses and deaths in Canada would be either 12.6 or 11.1 million dollars, respectively. If one uses a conservative estimate of 10 actual cases per every case reported (E. Todd, personal communication), then the estimated annual cost of human listeriosis in Canada would range from 111 to 126 million dollars. It should also be noted that this excludes cases where only mild upper GI symptoms occur (Riedo et al., 1994; Proctor et al., 1995).

**References**


