Comparison of Public Health Impact of \textit{Listeria monocytogenes} Product-to-Product and Environment-to-Product Contamination of Deli Meats at Retail

ABANI K. PRADHAN,\textsuperscript{1,2*} RENATA IVANEK,\textsuperscript{3} YRJÖ T. GRÖHN,\textsuperscript{1} ROBERT BUKOWSKI,\textsuperscript{4} AND MARTIN WIEDMANN\textsuperscript{4}

\textsuperscript{1}Department of Population Medicine and Diagnostic Sciences, College of Veterinary Medicine, \textsuperscript{2}Computational Biology Service Unit, and \textsuperscript{3}Department of Food Science, College of Agriculture and Life Sciences, Cornell University, Ithaca, New York 14853; \textsuperscript{4}Department of Nutrition and Food Science & Center for Food Safety and Security Systems (CFS\textsuperscript{3}), University of Maryland, College Park, Maryland 20742; and \textsuperscript{5}Department of Veterinary Integrative Biosciences, College of Veterinary Medicine and Biomedical Sciences, Texas A&M University, College Station, Texas 77843, USA

MS 10-351: Received 23 August 2010/Accepted 11 June 2011

ABSTRACT

This study compared the relative public health impact in deli meats at retail contaminated with \textit{Listeria monocytogenes} by either (i) other products or (ii) the retail environment. Modeling was performed using the risk of listeriosis-associated deaths as a public health outcome of interest and using two deli meat products (i.e., ham and turkey, both formulated without growth inhibitors) as model systems. Based on reported data, deli meats coming to retail were assumed to be contaminated at a frequency of 0.4\%. Three contamination scenarios were investigated: (i) a baseline scenario, in which no additional cross-contamination occurred at retail, (ii) a scenario in which an additional 2.3\% of products were cross-contaminated at retail due to transfer of \textit{L. monocytogenes} cells from already contaminated ready-to-eat deli meats, and (iii) a scenario in which an additional 2.3\% of products were contaminated as a result of cross-contamination from a contaminated retail environment. By using a previously reported \textit{L. monocytogenes} risk assessment model that uses product-specific growth kinetic parameters, cross-contamination of deli ham and turkey was estimated to increase the relative risk of listeriosis-associated deaths by 5.9- and 6.1-fold, respectively, for contamination from other products and by 4.9- and 5.8-fold, respectively, for contamination from the retail environment. Sensitivity and scenario analyses indicated that the frequency of cross-contamination at retail from any source (other food products or environment) was the most important factor affecting the relative risk of listeriosis-associated deaths. Overall, our data indicate that retail-level cross-contamination of ready-to-eat deli meats with \textit{L. monocytogenes} has the potential to considerably increase the risk of human listeriosis cases and deaths, and thus precise estimates of cross-contamination frequency are critical for accurate risk assessments.

\textit{Listeria monocytogenes} is a foodborne bacterial pathogen that can cause listeriosis, a rare yet severe human disease, particularly in susceptible individuals such as the elderly, pregnant women, and immunocompromised persons \citep{11, 19, 41}. Contaminated ready-to-eat (RTE) foods are the major source of human listeriosis cases \citep{10, 38, 41}. In particular, contaminated RTE deli meats have been estimated to be responsible for approximately 90\% of listeriosis cases in the United States, based on a risk assessment conducted by the U.S. Food and Drug Administration (FDA) and the U.S. Department of Agriculture Food Safety and Inspection Service (FSIS) \citep{38}. Contamination of sliced RTE deli meats by \textit{L. monocytogenes} has resulted in major listeriosis outbreaks in the United States \citep{3–5}.

Contamination of RTE foods by \textit{L. monocytogenes} can occur at various points in the processing and distribution chain \citep{9–11, 19, 28}. Postprocessing contamination of RTE food products with \textit{L. monocytogenes} has been identified as the main source of \textit{L. monocytogenes} in RTE products \citep{1, 27, 36, 39}. Survey data specifically indicated that RTE deli meats handled at retail stores have a considerably higher prevalence of \textit{L. monocytogenes} contamination as compared with products prepackaged by the manufacturer and not handled in stores \citep{9, 37}. These data suggest that cross-contamination at retail level might contribute to the presence of \textit{L. monocytogenes} in RTE deli meats at the time of consumption \citep{32}. Indeed, recently conducted risk assessments for \textit{L. monocytogenes} in deli meats indicated that a vast majority of listeriosis cases and deaths associated with deli meats are likely due to contamination of products at retail \citep{8, 25, 37}. Specifically, it has been estimated that 83\% of human listeriosis cases and deaths attributable to deli meats are due to retail-sliced products \citep{8, 37}. Similarly, another risk assessment that used product-specific growth kinetic parameters indicated that, for deli ham and turkey, 63 to 84\% of human listeriosis deaths can be attributed to contamination at retail \citep{25}. Cross-contamination generally refers to the transfer of bacteria from a contaminated source (food product, equipment, food handlers,
etc.) to a noncontaminated product (23). While several reported studies (12, 33) modeled cross-contamination to better understand the cross-contamination pathways at food processing plants, similar studies at retail level are currently unavailable. For example, Ivanek et al. (12) developed a plant-specific (smoked fish processing plant) mathematical model of *L. monocytogenes* cross-contamination that described the transmission of *L. monocytogenes* contamination among food, food contact surfaces, employer’s gloves, and the environment.

In addition to a number of studies that have shown the presence and persistence of *L. monocytogenes* in a variety of food processing plants (2, 13, 14, 18, 30, 36), initial studies also indicate that *L. monocytogenes* is commonly found in retail environments and that cross-contamination of RTE meat products with *L. monocytogenes* originating from the retail environment is likely (31, 32). Continued presence of *L. monocytogenes* in a particular environmental site at retail (a “niche”) that is in proximity to exposed RTE product (or that can otherwise easily contaminate the product) is of particular concern; such sites may allow for continued cross-contamination of products from environmental sources, thus potentially adding considerable numbers of additional *L. monocytogenes* cells to products. RTE food products that arrive free of contamination at retail facilities may become contaminated at retail due to either (i) cross-contamination from other *L. monocytogenes*-contaminated RTE products (redistribution of *L. monocytogenes*) or (ii) cross-contamination from the retail environment during further product handling (addition of *L. monocytogenes* to products).

The mechanisms and specific pathways of cross-contamination at retail involving several factors (e.g., type of source, occurrence frequency, worker habits, transfer distributions, etc.) are not currently well understood. However, a risk analysis study by Perez-Rodriguez et al. (22) attempted to model a specific pathway in which different handling procedures (wearing or not wearing gloves and washing or not washing hands) were examined as potential sources of cross-contamination at the retail level.

In contrast, our study was not a comprehensive assessment of the actual cross-contamination risk at retail; it did not model specific pathways of cross-contamination at retail level. Although it is self-evident that contamination of RTE foods with *L. monocytogenes* (and subsequent growth of *L. monocytogenes* in the product during refrigerated storage) increases the public health risk, quantitative estimates of the relative public health impact of different types of contamination events (product-to-product and environment-to-product) at retail level are currently unavailable. For example, cross-contamination from environmental sources could be hypothesized to have a particularly high public health impact as it leads to addition of new *L. monocytogenes* cells that were not previously present in foods, allowing for transmission of *L. monocytogenes*. On the other hand, *L. monocytogenes* cells introduced from environmental sources are likely to experience a lag phase upon introduction into the food, whereas cross-contamination between foods (particularly between similar types of foods) may allow for immediate growth without a lag phase (37, 38, 41). We were interested in evaluating the effects on public health of product-to-product and environment-to-product contamination of *L. monocytogenes* in deli meats at retail level in a generic way. The objectives of the present study were thus (i) to compare the relative risk of human listeriosis deaths due to contamination of deli meats by *L. monocytogenes* at retail originating from either contaminated products or the retail environment and (ii) to perform sensitivity and scenario analyses to characterize the importance of different parameters affecting the number of human listeriosis deaths due to these (product-to-product and environment-to-product) contamination scenarios at retail.

### MATERIALS AND METHODS

**Food product types.** Similar to the previous risk assessments published by our group (25, 26), we evaluated two types of deli meats, i.e., ham and turkey formulated without growth inhibitors (GIs), for the overall category “RTE deli meats.” These products were chosen because they are consumed at a higher frequency than other products and there are sufficient available experimental data on *L. monocytogenes* growth in these products to estimate the product-specific growth parameters lag time (LT) and exponential growth rate (EGR). As the estimated consumption level for ham in the United States was the highest among all deli meats (26), more detailed and in-depth analyses (i.e., sensitivity and scenario analyses) were performed only for this product category.

**Risk assessment modeling and model parameters.** All simulations reported here were performed using risk assessment models, which were based on the original FDA-FSIS risk assessment (FFRA (38)) and have previously been reported in detail (25, 26). *L. monocytogenes* growth is modeled using LT and EGR, growth kinetic parameters specific to ham or turkey. To estimate *L. monocytogenes* growth in contaminated products between retail and consumption, we used the previously described retail-to-consumption growth and exposure module with a one-dimensional probabilistic framework, similar to our previous study (25). To characterize the variability in model parameters, a one-dimensional framework was used that represents variations associated with the ability of individual servings of food to cause listeriosis. Sources of variation modeled include the parameters LT and EGR, the storage time and temperature in home refrigerators, and serving size (amount consumed per serving). The same distributions that were used in our previous studies (25, 26) were used here for EGR and LT (both at the reference temperature of 5°C); these distributions were based on data from the published literature, the FFRA, and unpublished data as previously detailed (26). Briefly, growth parameters in different foods from different studies, which used different storage temperatures, were transformed to a standard temperature (reference temperature of 5°C). Compiled data were fitted with BestFit software (Palisade Corp., Ithaca, NY) to characterize the distributions for LT and EGR at the reference temperature; these were subsequently used in the risk assessment models to predict *L. monocytogenes* growth in different deli meats (detailed data available at: [http://foodscience.cornell.edu/cals/foodsci/research/labs/wiedmann/links/pradhan-et-al-deli-meat-risk-cfm](http://foodscience.cornell.edu/cals/foodsci/research/labs/wiedmann/links/pradhan-et-al-deli-meat-risk-cfm)). In our study, storage temperature in home refrigerators was characterized by means of a discrete distribution based on data from a more recent survey conducted by Ecolab in 2007 (7), which is different from the 2005 consumer survey.
described in the report by Pouillot et al. (24). The cumulative distribution of storage time for retail-sliced products used in the 2009 FSIS draft risk assessment (37), which was based on data from the 2005 consumer survey (8, 37), was used to characterize home refrigerator storage time. As in the original FFRA, the serving size was modeled through an empirical cumulative distribution (median, 56 g per serving; 99th percentile, 196 g; extreme value on the right side, 648 g per serving) (38).

Additional parameters that affect the estimation of number of human listeriosis deaths due to contamination originating from other contaminated products or environment at retail, including (i) initial contamination prevalence (ICP), (ii) cross-contamination frequency (CCF), (iii) initial product contamination level (ICL), and (iv) transfer percentage (TP), were characterized based on data from published literature and reports. Although several studies regarding bacterial transfer are available, most of these studies represented a specific contamination pathway involving a contaminated source (e.g., kitchen surfaces, cutting boards, hands with or without gloves, and knife) and a food product (vegetable, meat, cheese, etc.), contaminated by different pathogenic bacteria (including surrogate microorganisms) (6, 15–17, 21, 23), and these studies are not suitable for estimating distributions for cross-contamination parameters relevant to our study. Data regarding the transfer of L. monocytogenes from other contaminated products and contaminated environment at retail level are currently unavailable to precisely characterize the probability distributions for parameters describing cross-contamination events. We thus chose the deterministic (fixed) values for these four parameters to model the average behavior of these parameters, an approach that also facilitated the tracking of L. monocytogenes transfer without any technical complexity. Nevertheless, to capture the variability, we used several different values of these parameters in subsequent sensitivity and scenario analyses.

The baseline value for ICP (representing the frequency of L. monocytogenes–contaminated deli meat products entering the retail environment) was set at 0.4%, and the baseline value for CCF was set at 2.3% (indicating that an additional 2.3% of deli meat products were contaminated due to cross-contamination at retail). These values were based on a survey conducted from 2000 to 2001 (9) that reported a 0.4% prevalence of L. monocytogenes in RTE deli meats (luncheon meats) that were packaged at manufacture and not handled in stores, whereas 2.7% of deli meats that were handled in stores were positive for L. monocytogenes (suggesting that 2.3% of products are contaminated at retail, as previously detailed (25)). The baseline value for ICL in L. monocytogenes–contaminated products was set at 10 CFU/g to represent the average contamination level of all initially contaminated products. This value was based on (i) L. monocytogenes contamination data from a 2005 to 2006 National Alliance for Food Safety and Security (NAFSS) study, which reported L. monocytogenes levels in contaminated RTE deli meat products ranging from 0.3 to 110 MPN/g (as reported in the 2009 FSIS draft comparative risk assessment for L. monocytogenes in RTE meat and poultry deli meats (8, 37)) and (ii) the survey by Gombas et al. (9), which reported that the majority of L. monocytogenes–positive samples were contaminated at levels of <10 CFU/g (to be conservative, treated as contaminated at 10 CFU/g), with a positive sample for deli meat product contaminated up to >10^4 to 10^5 CFU/g.

The baseline value for TP was set at an approximately average value of 0.1 (10%) based on a recent study (29) that reported an efficiency of transfer between 0.05 and 0.23 for transfer of L. monocytogenes cells from stainless steel and high-density polyethylene surfaces to deli meats (sliced chicken and pork bologna). In a study to demonstrate the mathematical frameworks for modeling Listeria cross-contamination in processing plants, Schaffner (33) set the average transfer rate at 30%; we followed a similar approach for modeling TP at an average value of 0.1 (10%). Based on TP of 10% and ICL of 10 CFU/g, the initial L. monocytogenes contamination level in cross-contaminated products is 1 CFU/g. As use of a single TP may represent an oversimplification (e.g., as one would expect that a higher number of L. monocytogenes cells would transfer during the first versus the second versus the third transfer event), we performed initial simulations to evaluate cross-contamination from contaminated products under two scenarios, including (i) a TP of 10% (so contamination level was 1 CFU/g for an ICL of 10 CFU/g) for 2.3% of products cross-contaminated at retail and (ii) TPs of 30, 10, 5, 3, and 2% (with corresponding contamination levels of 3, 1, 0.5, 0.3, and 0.2 CFU/g for an ICL of 10 CFU/g), so that equal proportions of products (0.46%) would be cross-contaminated with each of these five TPs. In the initial simulations, five different TPs (30, 10, 5, 3, 2%) were each tested at a CCF value of 0.46% (so that the overall CCF is 0.46% × 5 = 2.3%) versus one TP (10%) at a CCF of 2.3% (baseline). However, different TPs were further evaluated in sensitivity and scenario analyses, keeping other parameters at their baseline (including CCF at 2.3%). Both scenarios yielded similar results for the estimated number of listeriosis deaths per year, and the simpler scenario of TP at one level (10%) was retained for baseline simulations. Because the level of L. monocytogenes in cross-contaminated products was dependent on the ICL and TP, these parameters were included in the model (for example, if 10 cells are present in a contaminated source and the average transfer percentage is 10%, then 1 cell is transferred to the cross-contaminated products). Although all results reported here are based on these assumptions, which are based on literature data, the models used here are available (25, 26) and can be rerun using different combinations of parameters. The estimated numbers of deaths due to product-to-product and environment-to-product contamination scenarios at retail reported in this study are meant for comparative purposes and do not represent accurate estimates of listeriosis deaths. For each product, the exposure simulations generated a distribution of predicted doses (set of dose bins, i.e., dose levels broken out into half-log intervals) (25, 26, 38), which were used as inputs for the dose-response model. Similar to our previous studies (25, 26), the framework and models of the original FFRA model were used for the dose-response model, which was used to estimate the annual number of listeriosis-associated deaths. Briefly, in the FFRA, the dose-response simulation was carried out in two steps. Initially, a two-dimensional (second order) Monte Carlo simulation (100,000 variability and 300 uncertainty iterations) was used to integrate the variability and uncertainty of the strain-virulence and host susceptibility factors that provided dose-adjustment factors. Subsequently, a one-dimensional (4,000 uncertainty iterations) dose-response simulation was run by incorporating results from the exposure assessment to estimate the number of listeriosis-associated deaths (38). Because most of the total deaths due to listeriosis occur in the elderly population group (11, 38), we only assessed the risk of listeriosis for this population group. We reported only the estimated numbers of deaths here; however, based on epidemiological data (38), the corresponding number of listeriosis cases (i.e., those requiring hospitalization) can be easily calculated using a multiplier of 3.7 for the elderly population group.

Simulations and sensitivity and scenario analyses. For L. monocytogenes growth between retail and consumption and for the exposure assessment, a one-dimensional Monte Carlo simulation
(100,000 variability iterations) was performed in which sources of variation (growth kinetic parameters, storage time and temperature in home refrigerators, and serving size) were modeled using their distributions, whereas parameters (ICP, ICL, CCF, TP) characterizing cross-contamination events were modeled as deterministic (fixed) values. Subsequently, for each scenario simulated, a one-dimensional (4,000 uncertainty iterations) dose-response simulation was performed by incorporating the results from the exposure assessment. During the dose-response simulation, an appropriate proportion of the total number of servings was used for deli ham and turkey, as described previously (26).

Simulations were performed to estimate the relative risk of listeriosis-associated deaths in the elderly population group due to *L. monocytogenes* contamination of ham and turkey, under three contamination scenarios: scenario 1 (baseline), in which no additional cross-contamination occurred at retail, thus yielding a contamination frequency of 0.4%; scenario 2, in which an additional 2.3% of products were contaminated through cross-contamination, at retail, due to redistribution of *L. monocytogenes* cells from already contaminated RTE deli meat products; and scenario 3, in which an additional 2.3% of products were contaminated as a result of cross-contamination from contaminated retail environment. In scenario 1, 99.6% of products were noncontaminated, and 0.4% of initially contaminated products were contaminated at 10 CFU/g. Because of redistribution of *L. monocytogenes* cells from already contaminated products to noncontaminated products, mass balance (same total CFU per gram) between scenario 1 and scenario 2 was satisfied. For example, in scenario 1, 996 of 1,000 products are noncontaminated and 4 products are contaminated at 10 CFU/g, resulting in a total CFU per gram of 4 × 10 = 40; whereas in scenario 2, 973 products are noncontaminated, 4 initially contaminated products are now contaminated at 4.25 CFU/g (after losing bacteria to contaminate 23 noncontaminated products and assuming all initially contaminated products on average lose an equal number of bacteria), and 23 noncontaminated products are now contaminated at 1 CFU/g (TP 10% × ICL 10 CFU/g), resulting in a total CFU per gram of (4 × 4.25) + (23 × 1) = 40. Thus, in scenario 2, 97.3% of products were noncontaminated, 0.4% of initially contaminated products were contaminated at 4.25 CFU/g, and 2.3% of cross-contaminated products were contaminated at 1 CFU/g. In scenario 3, 97.3% of products were noncontaminated, 0.4% of initially contaminated products remained contaminated at 10 CFU/g, and 2.3% of cross-contaminated products were contaminated at 1 CFU/g from the retail environment. Because of the unavailability of appropriate data for cross-contamination from the retail environment, and to assure fair comparison to cross-contamination from contaminated foods, we assumed the environmental contamination level to be the same as the product contamination level. Thus, similar to scenario 2, with a TP of 10% and ICL of 10 CFU/g, in scenario 3, the cross-contaminated products were contaminated at 1 CFU/g; i.e., the contamination level of cross-contaminated products was assumed to be the same for product-to-product and environment-to-product contamination.

For scenarios 1 and 2, *L. monocytogenes* would not be expected to go through a lag phase, because *L. monocytogenes* should have already been adapted to the food matrix and should have completed the lag phase (37, 38, 41) (in scenario 2, this would depend on the food that is the source of cross-contamination: if the source food and the contaminated food are different in physicochemical and other parameters, *L. monocytogenes* may still experience a lag phase). For scenario 3, *L. monocytogenes* is likely to experience a lag phase, as the retail environment that represents the contamination source is likely to have considerably different physicochemical parameters than the food that is contaminated. While simulations for all scenarios were initially performed both with and without lag phase, sensitivity and scenario analyses were based on simulations in which no lag phase was used in the growth model for scenario 2, and lag phase was used in the growth model for scenario 3. For modeling without lag phase, the growth time used for *L. monocytogenes* in contaminated products was the storage time between retail and consumption (i.e., home refrigerator storage time), whereas for modeling with lag phase, *L. monocytogenes* growth time used was the storage time between retail and consumption minus the lag phase at the sampled storage temperature between retail and consumption.

To identify important parameters affecting listeriosis-associated deaths due to cross-contamination at retail, we performed sensitivity analyses by changing the values of different parameters from baseline to other values. Input values for different parameters were selected so that cross-contamination due to redistribution of *L. monocytogenes* cells from contaminated food products would occur by satisfying mass balance between scenario 1 and scenario 2, i.e., total CFU per gram were the same for scenario 1 and scenario 2. Sensitivity analysis was carried out by changing the ICP from a baseline value of 0.4% to values of 0.25, 0.3, 0.5, or 0.6%; the ICL from a baseline value of 10 CFU/g to values of 6, 8, 12, or 15 CFU/g; the CCF from a baseline value of 2.3% to values of 1, 1.7, 2.9, or 3.6%; and the TP from a baseline value of 10% to 5, 7, 12, or 15%.

Because CCF was observed to be the most important parameter in sensitivity analyses, we further performed several “what-if” scenario analyses to evaluate how extreme the additional parameters (other than CCF) would have to be to result in risk estimates similar to those observed for CCF in sensitivity analysis. Specifically, the impact of ICL on listeriosis deaths due to cross-contamination at retail was evaluated by increasing this parameter to 100, 1,000, 4,000, 5,000, and 10,000 CFU/g (from a baseline of 10 CFU/g). Because of the mass balance constraints between scenarios 1 and 2, further what-if scenario analyses were performed for scenario 3 only. For example, scenarios were established for TP by increasing this parameter from a baseline of 10% to 25, 50, and 90%. The impacts of CCF and ICP were tested by increasing these parameter values from a baseline of 2.3% (for CCF) up to 15% and from a baseline of 0.4% (for ICP) up to 5%. What-if scenario analysis was also performed by increasing the contamination level of the cross-contaminated products from 1 CFU/g to 10,000 CFU/g (for contamination originated from the environment).

**RESULTS**

Effects of contamination source on estimated number of listeriosis-associated deaths from consumption of ham and turkey. Simulations for all scenarios were initially performed assuming either immediate exponential growth after cross-contamination (i.e., no lag phase) or an initial lag phase followed by exponential growth (see Table 1 for all results). When assuming an initial lag phase for ham, the estimated median number of deaths per annum attributable to this product was 1.7 for products without any additional contamination at retail (scenario 1), 10.1 for products with additional contamination from contaminated products (scenario 2), and 10.2 for products with additional contamination from contaminated retail environment (scenario 3) (Table 1). Under this scenario, cross-contamination
at a frequency of 2.3% thus was estimated to increase the number of listeriosis deaths by approximately 5.9-fold (regardless of contamination source). Overall, for both products tested (deli ham and turkey; assuming an initial lag phase in all cases), cross-contamination at a frequency of 2.3% was estimated to increase the number of listeriosis deaths by 5.9- to 6.2-fold (relative to an initial contamination frequency [before cross-contamination] of 0.4%); differences between cross-contamination sources (i.e., contaminated products or retail environment) were limited with contamination from retail environments yielding only marginally higher numbers of deaths.

For all products and scenarios tested, the estimated numbers of deaths obtained when assuming immediate exponential growth after cross-contamination (i.e., no lag phase) were numerically higher as compared with the number of deaths estimated when assuming an initial lag phase (Table 1). Furthermore, the fold increase in the relative risk of listeriosis deaths due to cross-contamination at retail (either from contaminated products or from retail environment) relative to the baseline scenario of 0.4% contamination of incoming products (without additional cross-contamination) was similar for simulations assuming immediate exponential growth after cross-contamination and for simulations assuming an initial lag phase (see Table 1).

As detailed above, we surmised that simulations for scenario 3 (i.e., contamination from retail environment) assuming a lag phase and simulations for scenarios 1 and 2 assuming no lag phase represent the simulations with the most relevant and realistic assumptions, and, thus, we chose the results from these simulations (highlighted in bold in Table 1) to compare, although others could be chosen. Overall, the absolute and relative fold increase in the estimated number of listeriosis deaths were numerically higher when contamination originated from contaminated products (assuming no lag phase) as compared with contamination originating from retail environment (assuming an initial lag phase). For deli ham and turkey, cross-contamination at a frequency of 2.3% was estimated to increase the number of listeriosis deaths by 5.9- and 6.1-fold, respectively, for cross-contamination from other products, and by 4.9- and 5.8-fold, respectively, for cross-contamination from the retail environment (relative to an initial contamination frequency [before cross-contamination] of 0.4%).

### Sensitivity analysis to identify the important parameters affecting listeriosis deaths associated with cross-contamination

Sensitivity analysis performed to model the impact of CCF, ICP, TP, and ICL showed that CCF had the greatest impact on the estimated number of listeriosis deaths (Fig. 1). For example, for cross-contamination originating from contaminated products, an increase of CCF from 2.3 to 2.9% (i.e., by 26.1%) and from 2.3 to 3.6% (i.e., by 56.5%) increased the number of listeriosis deaths by 21.4% (from 12.2 to 14.8) and by 46.4% (from 12.2 to 17.8), respectively. Similarly, when CCF was decreased from 2.3 to 1.7% (i.e., by 26.1%) and from 2.3 to 1% (i.e., by 56.5%), the number of listeriosis deaths decreased by 21.6% (from 12.2 to 9.5) and by 47% (from 12.2 to 6.5), respectively. Overall, ICP, ICL, and TP had negligible effects on the predicted number of deaths (Fig. 1). Of the two parameters in scenario 1, ICP and ICL, sensitivity analysis showed ICP to be the more influential. For example, for ham, this study estimated numbers of listeriosis deaths in the elderly population per year were 1.1 at lower-bound ICP (0.25%) and 2.6 at upper-bound ICP (0.6%) compared with 1.7 at lower-bound ICL (6 CFU/g) and 1.8 at upper-bound ICL (15 CFU/g); the greater the range in the estimated number of deaths between

---

**Table 1. Estimated number of listeriosis-associated deaths in the U.S. elderly population group per annum due to L. monocytogenes cross-contamination of deli ham and turkey that was formulated without growth inhibitors (GIs)**

<table>
<thead>
<tr>
<th>Deli meat</th>
<th>Contamination scenario</th>
<th>Growth models with LT</th>
<th>Growth models without LT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ham</td>
<td>Scenario 1</td>
<td>1.7 (0.3, 9.0)</td>
<td>2.1 (0.3, 10.9)</td>
</tr>
<tr>
<td></td>
<td>Scenario 2</td>
<td>10.1 (1.7, 53.1)</td>
<td>12.2 (2.0, 63.9)</td>
</tr>
<tr>
<td></td>
<td>Scenario 3</td>
<td><strong>10.2 (1.7, 53.4)</strong></td>
<td>12.3 (2.0, 64.4)</td>
</tr>
<tr>
<td>Turkey</td>
<td>Scenario 1</td>
<td>1.4 (0.2, 7.6)</td>
<td><strong>1.5 (0.3, 8.1)</strong></td>
</tr>
<tr>
<td></td>
<td>Scenario 2</td>
<td>8.8 (1.5, 46.3)</td>
<td><strong>9.4 (1.6, 49.4)</strong></td>
</tr>
<tr>
<td></td>
<td>Scenario 3</td>
<td><strong>8.9 (1.5, 46.7)</strong></td>
<td>9.5 (1.6, 49.8)</td>
</tr>
</tbody>
</table>

* L. monocytogenes originated from either contaminated products or the retail environment and was predicted with L. monocytogenes growth models with or without lag time (LT).

* Based on reported data, three contamination scenarios at retail were investigated: scenario 1 (baseline), deli meats coming to retail were contaminated at a frequency of 0.4% and no additional cross-contamination occurred at retail; scenario 2, an additional 2.3% of products were contaminated through cross-contamination, at retail, due to redistribution of L. monocytogenes cells from other contaminated food products; and scenario 3, an additional 2.3% of products were contaminated as a result of cross-contamination from contaminated retail environment.

* Median and 5th and 95th percentiles values were obtained after L. monocytogenes growth and exposure assessment (100,000 variability iterations) and dose-response simulation (4,000 uncertainty iterations) (25, 26, 38).

* Results highlighted in bold represent the most relevant and realistic assumptions (i.e., simulation of scenario 3 assuming a lag phase and simulations of scenarios 1 and 2 assuming no lag phase) and provide relevant comparison among scenarios.
the parameter’s lower and upper bounds, the more influential is that parameter.

Scenario analysis for the extreme changes to different parameters in the estimation of listeriosis-associated deaths. From our sensitivity analysis, we found that CCF had the greatest impact on the predicted number of listeriosis-associated deaths. To determine whether this observation was related to the range of parameter values used for CCF, we further evaluated what-if scenarios to see how extreme changes to other parameters would have to be to result in changes to the risk estimates comparable to that observed for CCF in the sensitivity analysis. For ICL, an increase in the risk estimate comparable to that observed when CCF was increased from 2.3 to 3.6% was only achieved when ICL was increased from 10 to 4,000 CFU/g (Fig. 2A); with an ICL of 4,000 CFU/g, the estimated number of deaths increased from 12.2 to 18.2 and from 10.2 to 15.3 (relative to an ICL baseline of 10 CFU/g), for scenario 2 and scenario 3, respectively. For TP, on the other hand, even relatively extreme changes produced only a very limited impact on the estimated number of listeriosis deaths (Fig. 2B).

Although in sensitivity analysis we showed that an increase of CCF from 2.3 to 3.6% had the greatest impact on estimated number of listeriosis deaths, we further evaluated the effect of CCF on the number of listeriosis deaths when CCF was increased to higher values. When CCF was increased from 2.3 to 15%, the estimated number of deaths increased from 10.2 to 57.2 (Fig. 3A). An increase of ICP from 0.4 to 5% increased the estimated number of listeriosis deaths from 10.2 to 29.8 (Fig. 3B). Simulations were also performed by increasing the contamination level of the cross-contaminated products for contamination originating from the retail environment (i.e., scenario 3) to evaluate situations where large numbers of L. monocytogenes are present in the environment at a location where cross-contamination would occur. An increase in the cross-contamination level from 1 to 100 CFU/g increased the estimated number of deaths by 30.3%, and an increase of cross-contamination level to 10,000 CFU/g increased the estimated number of deaths by 74% (Fig. 4).

DISCUSSION

Contamination of deli meats at retail from other food products or from retail environment has a similar effect on increasing human listeriosis-associated deaths.
In this study, the estimates for the number of listeriosis deaths are consistent with previous reports that suggest that cross-contamination of cooked and RTE foods with *L. monocytogenes* is a serious public health concern (9, 34, 35, 40). We found that cross-contamination of deli meats with *L. monocytogenes* by contaminated products or by environment at retail has a considerable impact on the number of listeriosis-associated deaths (assuming a cross-contamination frequency of 2.3%).

Conceptually, cross-contamination at retail can represent two different scenarios. Specifically, cross-contamination of uncontaminated products or product batches that enter retail as contaminated; while this contamination could occur through direct product-to-product contact, the more likely scenario is that a food contact surface serves as a transfer vehicle for this type of contamination (with no or limited growth on this product surface). In this type of cross-contamination, the same number of *L. monocytogenes* cells are redistributed to a larger number of products. Several studies have indicated that *L. monocytogenes* can spread from contaminated products during slicing of deli meats through slicing equipment (34, 35, 39, 40), thus supporting this type of cross-contamination. Cross-contamination at retail can also occur from the retail environment; this scenario basically represents an addition to RTE food products of *L. monocytogenes* cells that were not previously located on food products. An increasing number of studies suggest that *L. monocytogenes* is commonly found at various sites in retail environments, including food contact surfaces (31, 32), suggesting likely contamination of deli meats with *L. monocytogenes* from environmental sources at retail.

While it is tempting to speculate that contamination from retail environments may have more severe public health consequences (the introduction of additional “new” *L. monocytogenes* onto product), our analyses indicate that the relative public health impacts of contamination from retail environments and from other products are very similar. These findings suggest that preventing contamination from contaminated products and from the retail environment is equally critical in reducing the number of human listeriosis deaths; however, our findings also indicate that determining the relative frequency of cross-contamination occurring from these two sources is critical for an accurate risk assessment.

**Frequency of cross-contaminated products at retail is the most important factor affecting the estimation of number of listeriosis-associated deaths.** Our sensitivity analyses indicated that CCF is the most important parameter affecting the public health impact of cross-contamination at retail. Contamination data collected by Gombas et al. (9) indicated that RTE deli meats handled at retail have a...
considerably higher \textit{L. monocytogenes} prevalence (i.e., 2.7\%) as compared with manufacturer-packaged samples, which showed a contamination prevalence of 0.4\%; our study used these data to estimate the baseline CCF at retail as 2.3\%.

It is important to note that considerable variability exists in cross-contamination parameters such as CCF and TP. In a model simulation, if data are not precise and robust to describe an event, different probability distributions (such as normal, triangular, etc.) would yield different model behavior and results. Because of the current unavailability of data to precisely characterize the probability distributions of these parameters appropriate to our study, instead of any arbitrary distribution, we preferred to use deterministic (fixed) values of these parameters. However, it would be more prudent to use precise probability distributions to characterize these parameters, when appropriate data are available in future. Although we used fixed values to characterize these parameters, the variability associated with these parameters was evaluated by using several different values of these parameters (representing distinct values sampled from a distribution) in our sensitivity and scenario analyses.

Our sensitivity analyses indicated that ICL of RTE deli meats entering retail as well as the TP had a very limited impact on the estimated number of listeriosis deaths due to retail-level cross-contamination events. Consistent with sensitivity analyses results, scenario analyses also indicated that, as the frequency of cross-contaminated products increased, the estimated number of listeriosis deaths increased considerably. These data indicate that a larger number of deli meat products contaminated with lower levels of \textit{L. monocytogenes} at retail represent a higher public health risk than a smaller number of products contaminated with higher levels of \textit{L. monocytogenes} at retail. When interpreted in conjunction with previous \textit{L. monocytogenes} risk assessments, which indicated that home refrigerator storage temperature has a major effect on the estimated number of human listeriosis cases (11, 25, 38), these data indicate that, under current typical time and temperature storage parameters, enough products, even if cross-contaminated at low levels at retail, allow for sufficient \textit{L. monocytogenes} growth to cause a considerable increase in the number of human listeriosis cases. Our previous study indicated that reducing storage temperature in home refrigerators to consistently below 7°C would greatly reduce the risk of human listeriosis deaths, and limiting home refrigerator temperatures to \(\leq 4\)°C was estimated to reduce the number of listeriosis deaths in the elderly population group that could be attributable to ham (formulated with or without GIs) to less than one death per year in the United States (25). Furthermore, lag phase was more important for the production-to-retail stage than the retail-to-consumption stage, which is likely due to the higher home refrigerator storage temperatures (which will lead to shorter bacterial lag phase) as compared to the production-to-retail storage temperatures (25).

In our what-if scenario analyses, we tested how extreme the value of various parameters would have to be to produce the same effect observed for CCF, the most important parameter in this study. This analysis is valuable because there is a potential for products occasionally to be contaminated with \textit{L. monocytogenes} at very high levels (such as 10,000 CFU/g) (9). Although the extreme values are possible and well documented, they are rare; thus, it is highly unlikely that the parameters tested in the scenario analyses are responsible for the estimated public health risks.

Overall, our findings support the need for implementation of appropriate control strategies (e.g., stringent and standardized cleaning and sanitation procedures, education and training of personnel in deli operations, sanitary equipment design (8, 20, 32)) to reduce the number of cross-contaminated products at retail. As some studies have suggested that retail environments could serve as niches where \textit{L. monocytogenes} survives over time (31, 32) with the potential for repeat cross-contamination, appropriate control strategies aimed at reducing environmental persistence of \textit{L. monocytogenes} at retail facilities is critical.

\section*{ACKNOWLEDGMENTS}

This project was supported through a grant from the National Integrated Food Safety Initiative (Special Emphasis Grant no. 2005-51110-03278) of the Cooperative State Research, Education, and Extension Service, U.S. Department of Agriculture. Any opinions, findings, conclusions, or recommendations expressed in this publication are those of the authors and do not necessarily reflect those of the U.S. Department of Agriculture.

\section*{REFERENCES}


