Quantitative Risk Assessment for *Listeria monocytogenes* in Selected Categories of Deli Meats: Impact of Lactate and Diacetate on Listeriosis Cases and Deaths

ABANI K. PRADHAN,1* RENATA IVANEN,1 YRJÖ T. GRÖHN,1 IFIGENIA GEORNARAS,2 JOHN N. SOFOS,2 AND MARTIN WIEDMANN1

1Department of Population Medicine and Diagnostic Sciences, College of Veterinary Medicine, and 2Department of Food Science, College of Agriculture and Life Sciences, Cornell University, Ithaca, New York 14853; and 2Center for Meat Safety and Quality, Department of Animal Sciences, Colorado State University, Fort Collins, Colorado 80523, USA

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**ABSTRACT**

Foodborne disease associated with consumption of ready-to-eat foods contaminated with *Listeria monocytogenes* represents a considerable public health concern. In a risk assessment published in 2003, the U.S. Food and Drug Administration and the U.S. Food Safety and Inspection Service estimated that about 90% of human listeriosis cases in the United States are caused by consumption of contaminated deli meats. In this risk assessment, all deli meats were grouped into one of 23 categories of ready-to-eat foods, and only the postretail growth of *L. monocytogenes* was considered. To provide an improved risk assessment for *L. monocytogenes* in deli meats, we developed a revised risk assessment that (i) models risk for three subcategories of deli meats (i.e., ham, turkey, and roast beef) and (ii) models *L. monocytogenes* contamination and growth from production to consumption while considering subcategory-specific growth kinetics parameters (i.e., lag phase and exponential growth rate). This model also was used to assess how reformulation of the chosen deli meat subcategories with *L. monocytogenes* growth inhibitors (i.e., lactate and diacetate) would impact the number of human listeriosis cases. Use of product-specific growth parameters demonstrated how certain deli meat categories differ in the relative risk of causing listeriosis; products that support more rapid growth and have reduced lag phases (e.g., turkey) represent a higher risk. Although reformulation of deli meats with growth inhibitors was estimated to reduce by about 2.5- to 7.8-fold the number of human listeriosis cases linked to a given deli meat subcategory and thus would reduce the overall risk of human listeriosis, even with reformulation deli meats would still cause a considerable number of human listeriosis cases. A combination of strategies is thus needed to provide continued reduction of these cases. Risk assessment models such as that described here will be critical for evaluation of different control approaches and to help define the combinations of control strategies that will have the greatest impact on public health.

*L. monocytogenes* is a human pathogen that causes a rare but severe foodborne disease and represents a major public health concern for both regulatory agencies and the food industry. Pregnant women, the elderly, and persons with weakened immune systems are at increased risk for listeriosis (45, 49). Ready-to-eat (RTE) foods appear to represent the main source of sporadic foodborne listeriosis infections and have been implicated as the source of a number of listeriosis outbreaks (1, 16, 25, 29). The prevalence of *L. monocytogenes* in contaminated RTE foods differs considerably depending on type of RTE food, country of origin, and year (45). 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) estimated that about 90% of human listeriosis cases in the United States are caused by consumption of contaminated RTE deli meats (45). Human listeriosis outbreaks linked to consumption of RTE deli meats also have been reported. For example, a multistate listeriosis outbreak in 2002 was linked to sliceable turkey deli meat. This outbreak comprised 46 illness, 7 deaths, and 3 stillbirths or miscarriages in eight states in the northeastern United States (11). Consumption of turkey deli meat also has been linked to another multistate outbreak of listeriosis in 2000 involving 10 states in the United States and 29 illnesses with 4 deaths and 3 stillbirths or miscarriages (10).

Although the food industry and regulatory agencies around the world have endorsed and implemented various strategies to control *L. monocytogenes* in RTE foods (27, 38, 43, 50), control of this foodborne pathogen continues to represent a considerable challenge. Because *L. monocytogenes* can grow under refrigeration temperatures in RTE foods with appropriate physicochemical properties, prevention of postpasteurization contamination and reformulation of foods to inhibit *L. monocytogenes* growth have been identified as critical strategies likely to help reduce the public health risk associated with *L. monocytogenes* (27). Although various food additives and antimicrobials (e.g., nisin, organic acids, and acidic calcium sulfate) can reduce growth of *L. monocytogenes* in RTE foods (2, 16), lactate and diacetate salts appear to be particularly effective at in-

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*Author for correspondence. Tel: 607-255-1140; Fax: 607-257-8485; E-mail: akp49@cornell.edu.*
hibiting *L. monocytogenes* growth in RTE meat and poultry products stored at refrigeration temperatures, specifically when these compounds are used in combination (25, 29, 30, 32, 43). These two growth inhibitors (GIs) have been classified as generally recognized as safe by the FDA and thus can be used as a direct human food ingredient (12). Specifically, lactate and diacetate at levels not exceeding 4.8 and 0.25%, respectively, by weight of total formulation are permitted in various meat and poultry products for inhibition of microbial growth (13). Currently, manufacturers increasingly incorporate lactate and diacetate into many formulations of RTE foods (17, 25, 43). The use of GIs such as lactate and diacetate in reformulated products can extend lag phase and slow down exponential growth; these effects differ in foods, depending on various physicochemical product characteristics (e.g., pH) (4, 9, 17, 25). Although many researchers have evaluated the ability of lactate and diacetate to inhibit *L. monocytogenes* growth (4, 7, 25, 29, 30, 33, 41), no studies have been published on the effect on the number of human listeriosis cases of reformulation of RTE deli meats with these GIs.

Quantitative estimates on the relative risk of illness or death due to consumption of RTE foods contaminated with *L. monocytogenes* in the United States have been reported in a FDA-FSIS risk assessment (FFRA), which was published in 2003 (45). In the FFRA, the relative risk per annum and per serving of acquiring listeriosis was specifically calculated for 23 different RTE food categories (e.g., deli meats, pasteurized fluid milk, and smoked seafood). Although the FFRA estimate indicated that foods in the category “deli meats” were responsible for the vast majority of human listeriosis cases (i.e., approximately 90% of cases), this food category includes a diverse set of foods, such as bologna, corned, cooked, and roast beef, ham, beef sirloin, sliced chicken, chicken fillets, and sliced turkey. In the FFRA, a generic kinetic parameter, exponential growth rate (EGR), was used for the category deli meats (and for all other food categories). Although this approach was justified at the time because of the limited availability of growth kinetics data for different deli meats and the increased complexity of models with additional food categories (45), the category deli meats clearly includes foods that differ considerably in various characteristics (e.g., pH) that may affect *L. monocytogenes* survival and growth (9, 16–18).

The majority of contamination data for RTE foods originate from food samples collected at retail establishments. Therefore, the original FFRA modeled *L. monocytogenes* growth only between retail and consumption. For a few data collected at the manufacturing plant (production), contamination was adjusted to account for growth between production and retail. This modeling strategy implicitly assumed that bacterial cells at the retail level already have adapted to the food environment, i.e., they are beyond the lag phase and would therefore grow exponentially during the retail-consumption period. Because use of GIs in RTE deli meats will extend the *L. monocytogenes* lag phase, possibly into the retail-to-consumption storage period, the original FFRA model thus could not be readily used to evaluate the effects of reformulation with GIs on the number of human cases of *L. monocytogenes* infection in the United States. GIs also differ considerably in their effect on *L. monocytogenes* growth, depending on the type of deli meat (16, 25). The objective of this study was thus to revise the original FFRA model and use the new risk assessment model to estimate (i) the per annum risk of listeriosis associated with selected deli meats and (ii) the effect of deli meat reformulation with GIs on the number of human listeriosis cases and deaths.

**MATERIALS AND METHODS**

**Deli meat types.** Although the deli meats category in the FFRA includes several types of products, we considered only three major types of deli meats: ham, turkey, and roast beef. These three products were selected because (i) they represent some of the most commonly consumed deli meats in the United States and (ii) sufficient data were available for these products to allow estimation of product-specific (PS) *L. monocytogenes* growth kinetic parameters in products formulated with and without GIs (i.e., lactate and diacetate). The three deli meat types selected also represent a range of intrinsic factors (e.g., pH) that affect *L. monocytogenes* growth kinetics, including products with relatively high pH values (e.g., turkey meats with pH 6.2 to 6.4 (9, 18)) allowing for more rapid growth and products with lower pH values (e.g., roast beef with pH 5.4 to 6.0 (9)), thus reducing growth.

To assess the different levels of risk attributed to three types of deli meats, estimates are needed on the consumption pattern of these products in the United States. Although data from two large nationwide U.S. food consumption surveys (Continuing Survey of Food Intakes by Individuals, 1994 to 1996; and the Third National Health and Nutrition Examination Survey, 1988 to 1994) were used to estimate consumption of different food categories in the FFRA (45), these food consumption surveys did not provide appropriate consumption data for the three deli meats considered in our study. Therefore, we elicited an expert opinion on the relative production volumes of RTE ham, turkey, and roast beef deli meats as compared to total RTE deli meats produced in the United States.

**Growth parameters and distributions.** The original FFRA model that estimated *L. monocytogenes* growth between retail (R) and consumption (C) included the initial *L. monocytogenes* contamination (at R), EGR, food storage temperature, storage time, and the maximum growth level (i.e., maximal population density as defined by Pérez-Rodríguez et al. (36)), which is dependent on storage temperature, to calculate *L. monocytogenes* contamination at the time of consumption. In our revised risk assessment for the considered food products (ham, turkey, and roast beef), each formulated with and without GIs, we (i) estimated growth parameters (lag phase and EGR), (ii) used the estimated growth parameters to model separately *L. monocytogenes* growth during production-to-retail and retail-to-consumption phases, and (iii) modeled separately the associated number of human listeriosis cases and deaths. In our risk assessment, a temperature-dependent restriction on the maximal population density was applied and modeled in the same manner as in the original FFRA model (45) for deli meats formulated with and without GIs, assuming that the maximal population density depends only on storage temperature (36).

Data on the distributions of *L. monocytogenes* lag time (*Lt*) and EGR for the three deli meat categories considered (ham, turkey, and roast beef) for products both with or without the addition of GIs (i.e., lactate and diacetate) were taken from published lit-
eration (5–9, 14–18, 20, 23–26, 30, 31), the FFRA (45), and unpublished data (34) as appropriate for our study (detailed data available at: http://www.foodsci.cornell.edu/cals/foodsci/research/labs/wiedmann/links/pradhane-tal-dei-meat-risk.cfm). In addition to numerically reported \( L_t \) and EGR values, data were extracted from growth curves available in the literature and in the Combined Database for Predictive Microbiology (ComBase: http://www.combase.cc/about.html). Growth data extracted as described above were fitted with DMFit from ComBase whenever necessary (http://ifrswwwdev.ifrn.bbsrc.ac.uk/CombasePMP/DMFi.html). In DMFit, three models were initially used for curve fitting: (i) Baranyi and Robert’s complete model (3), (ii) Baranyi and Robert’s no-asymptote model (3), and (iii) the biphasic no-asymptote model. Values for EGR and \( L_t \) representing the best fit (based on coefficient of determination \([R^2]\) and standard error of fit) were used as kinetic growth parameters in our model. Several studies (14, 15, 21, 30, 46) have previously indicated that curing ingredients such as sodium nitrite may inhibit \( L. \) monocytogenes growth. Although a few studies (34) evaluating cured and uncured products concurrently have produced different \( L. \) monocytogenes growth rates for cured and uncured products, the number of data points for each category (cured and uncured) was too small to allow separate analysis.

In the original FFRA model, the square root model for EGR was used to predict growth of \( L. \) monocytogenes in different foods. This model was chosen to characterize the growth kinetic parameters as a function of temperature because of its simplicity and its general acceptance as supported by documented use in microbiology literature (39). According to this model, EGR and \( L_t \) can be expressed as

\[
\sqrt{\text{EGR}} = a(T - T_0) \quad (1)
\]

\[
\frac{1}{L_t} = a(T - T_0) \quad (2)
\]

where EGR is the exponential growth rate (log CFU per day), \( T \) is the growth temperature (degrees C), \( T_0 \) is the extrapolated minimum notational growth temperature (degrees C), \( a \) is the slope parameter for \( L. \) monocytogenes in the specific food, and \( L_t \) is the lag time (days). For the FFRA the value for \( T_0 \) was estimated as \(-1.18^\circ C\) (based on four different sources) (45); the same \( T_0 \) value was used in our risk assessment.

Because different storage temperatures are used in different studies of \( L. \) monocytogenes growth parameters in different foods, EGR and \( L_t \) from these different studies must be transformed to a standard temperature. We used the approach reported in the FFRA for this transformation. Specifically, an equivalent EGR at \( 5^\circ C \) was calculated as presented in equation 3, which is a ratio and rearrangement of equation 1. Similarly, \( L_t \) at different temperatures was converted to equivalent values at \( 5^\circ C \) using equation 4 (which was obtained by rearranging equation 2):

\[
\frac{\text{EGR}_{5}}{\text{EGR}_{t}} = \left( \frac{a(T_{5} + 1.18)}{a(T_{t} + 1.18)} \right)^2 = \left( \frac{6.18}{(T_{5} + 1.18)} \right)^2 \quad (3)
\]

\[
\frac{L_{5}}{L_{t}} = \left( \frac{a(T_{5} + 1.18)}{a(T_{t} + 1.18)} \right)^2 = \left( \frac{6.18}{(T_{5} + 1.18)} \right)^2 \quad (4)
\]

where EGR\(_5\) and \( L_5 \) are kinetic parameters at \( 5^\circ C \), EGR\(_t\) and \( L_t \) are kinetic parameters at the study temperature \( T_t \) (degrees C), and \( T_5 \) is the reference temperature of \( 5^\circ C \). The kinetic parameters (after conversion to values at the reference temperature of \( 5^\circ C \)) were compiled for the three types of deli meats for products formulated with or without GIs. Compiled data for a given deli meat type (either with or without GIs) were fitted with BestFit software (Palisade Corp., Ithaca, NY) to characterize the distributions for EGR and \( L_t \) at the reference temperature (see Table 1 for the parameter values for the fitted distributions for EGR and \( L_t \)). These fitted distributions were subsequently used in the risk assessment model to predict growth of \( L. \) monocytogenes from production (\( P \)) to \( C \) (i.e., at consumption) for different deli meats. The same distributions for EGR and \( L_t \) at the reference temperature of \( 5^\circ C \) (Table 1) were used during both phases, production to retail (\( P \) to \( R \)) and retail to consumption (\( R \) to \( C \)).

**Description of the original FFRA model.** Because the FFRA (45) was used as the basis of our revised model reported here, the original FFRA is briefly described here. In the FFRA, three age-based population groups were considered: (i) the perinatal group, which includes fetuses and neonates from 16 weeks after fertilization to 30 days postpartum, (ii) the elderly group, which includes individuals older than 60 years of age, and (iii) the intermediate age group, which includes both healthy individuals and certain susceptible groups in the remaining population.

The FFRA model comprises two major components: exposure assessment and dose-response. Simulation modeling for the risk assessment includes (i) a growth module to calculate growth between \( R \) and \( C \), which assumes that growth after \( R \) is exponential, and (ii) a dose-response module to estimate the relative risk of listeriosis and associated deaths from exposure. Models for both components were written and simulated in Visual Basic for Applications (VBA; Microsoft, Redmond, WA). The exposure assessment modeled \( L. \) monocytogenes levels and contamination frequency for different foods considering (i) frequency and extent of contamination at \( R \), (ii) EGR for \( L. \) monocytogenes in a given a food category, (iii) length of refrigerated storage, (iv) refrigeration temperature, (v) maximum \( L. \) monocytogenes growth (stationary phase) that a given food can support (i.e., maximal population density), and (vi) consumption patterns. The FFRA model in the exposure assessment included a two-dimensional probabilistic framework incorporating variability and uncertainty for the concentration of \( L. \) monocytogenes at \( R \), storage time, and EGR and a one-dimensional framework for other parameters. For example, in the original FFRA, the set of 300 lognormal distributions were used to characterize the variability and uncertainty of \( L. \) monocytogenes concentration at \( R \). Each lognormal distribution characterized the variability, and the set of 300 distributions represented the uncertainty about this parameter.

A two-dimensional Monte-Carlo simulation (100,000 variability and 300 uncertainty iterations) was used for the exposure assessment, and results were then carried forward to the dose-response simulations, where a separate simulation was constructed for each of the three subpopulation groups. The dose-response simulation was carried out in two steps. First, a two-dimensional 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 functions for each of the subpopulations to provide dose adjustment factors. Second, a one-dimensional (4,000 uncertainty iterations) dose-response simulation was run by selecting one of the 300 sets of dose bins (dose levels broken out into half-log intervals) from the exposure assessment. The number of listeriosis-associated deaths was modeled initially, and the total number of cases of listeriosis requiring hospitalization was then estimated by multiplying the predicted number of deaths by a multiplier that was based on epidemiological data (45).

**Description of the new risk assessment model.** Based on the original FFRA (45), we developed a new risk assessment model (our model is available at: http://www.foodsci.cornell.edu/cals/
### TABLE 1. Parameters of the fitted distributions for exponential growth rate and lag time at the reference temperature 5°C used in the risk assessment model

<table>
<thead>
<tr>
<th>Notation</th>
<th>Definition</th>
<th>Distribution</th>
<th>Mean (5th, 95th percentiles)</th>
<th>n&lt;sup&gt;d&lt;/sup&gt;</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>EGR H without GIs</td>
<td>Exponential growth rate for ham RTE products without growth inhibitors</td>
<td>Logistic (0.1941, 0.0472)</td>
<td>0.1941 (0.055, 0.3332)</td>
<td>34</td>
<td>1, 2, 5, 7, 9, 16, 18, 20, 29, 32, 34, 45</td>
</tr>
<tr>
<td>Lt H without GIs</td>
<td>Lag time for ham RTE products without growth inhibitors</td>
<td>Triangular (0.405, 0.405, 16.941)</td>
<td>5.917 (0.82, 13.24)</td>
<td>7</td>
<td>7–9, 34</td>
</tr>
<tr>
<td>EGR H with GIs</td>
<td>Exponential growth rate for ham RTE products with growth inhibitors</td>
<td>Logistic (0.1065, 0.0282)</td>
<td>0.1065 (0.0235, 0.1895)</td>
<td>24</td>
<td>2, 7, 25, 29, 34</td>
</tr>
<tr>
<td>Lt H with GIs</td>
<td>Lag time for ham RTE products with growth inhibitors</td>
<td>Triangular (6.111, 6.111, 34.623)</td>
<td>15.615 (6.83, 28.25)</td>
<td>15</td>
<td>25, 29, 34</td>
</tr>
<tr>
<td>EGR T without GIs</td>
<td>Exponential growth rate for turkey RTE products without growth inhibitors</td>
<td>Logistic (0.2755, 0.0723)</td>
<td>0.2755 (0.0626, 0.4883)</td>
<td>34</td>
<td>9, 14, 15, 17, 26, 30, 34, 41, 45</td>
</tr>
<tr>
<td>Lt T without GIs</td>
<td>Lag time for turkey RTE products without growth inhibitors</td>
<td>Triangular (0.460, 0.460, 5.547)</td>
<td>2.156 (0.589, 4.410)</td>
<td>14</td>
<td>9, 15, 26, 34</td>
</tr>
<tr>
<td>EGR T with GIs</td>
<td>Exponential growth rate for turkey RTE products with growth inhibitors</td>
<td>Logistic (0.0975, 0.0253)</td>
<td>0.0975 (0.0231, 0.1720)</td>
<td>21</td>
<td>30, 34, 50</td>
</tr>
<tr>
<td>Lt T with GIs</td>
<td>Lag time for turkey RTE products with growth inhibitors</td>
<td>Triangular (2.387, 2.387, 23.874)</td>
<td>9.549 (2.93, 19.07)</td>
<td>7</td>
<td>30, 34, 50</td>
</tr>
<tr>
<td>EGR RB without GIs</td>
<td>Exponential growth rate for roast beef RTE products without growth inhibitors</td>
<td>Logistic (0.2722, 0.0646)</td>
<td>0.2722 (0.0819, 0.4625)</td>
<td>22</td>
<td>6, 14, 18, 23, 24, 33, 34, 41, 45</td>
</tr>
<tr>
<td>Lt RB without GIs</td>
<td>Lag time for roast beef RTE products without growth inhibitors</td>
<td>Triangular (1.125, 1.125, 13.036)</td>
<td>5.096 (1.43, 10.37)</td>
<td>11</td>
<td>6, 14, 18, 20, 23, 24</td>
</tr>
<tr>
<td>EGR RB with GIs</td>
<td>Exponential growth rate for roast beef RTE products with growth inhibitors</td>
<td>Logistic (0.1258, 0.0517)</td>
<td>0.1258 (−0.0265, 0.2780)</td>
<td>11</td>
<td>6, 33, 34</td>
</tr>
<tr>
<td>Lt RB with GIs</td>
<td>Lag time for roast beef RTE products with growth inhibitors</td>
<td>Triangular (2.685, 2.685, 22.814)</td>
<td>9.395 (3.19, 18.31)</td>
<td>5</td>
<td>6, 33, 34</td>
</tr>
</tbody>
</table>

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<sup>a</sup> Data at the reference temperature of 5°C for exponential growth rate (EGR<sub>5</sub>) and lag time (Lt<sub>5</sub>) were fitted with BestFit software (Palisade Corp., Ithaca, NY) to characterize the distributions used in the risk assessment models.

<sup>b</sup> Logistic distribution with two parameters (alpha and beta) and triangular distribution with three parameters (minimum value, most likely value, and maximum value) obtained from the best-fit process were used to define EGR<sub>5</sub> and Lt<sub>5</sub>, respectively. Minimum and most likely values for the triangular distributions were the same as those obtained by fitting the data with the BestFit software.

<sup>c</sup> Statistics of the fitted distributions (mean with 5th and 95th percentiles) are presented to show the uncertainty of the parameters.

<sup>d</sup> Total number of samples from different studies used in the best-fit process.
**FIGURE 1. Schematic of different scenarios for lag time \( (Lt) \) that may arise because of use of growth inhibitors \( (GIs) \) such as lactate and diacetate or because of natural \( Lt \) \( (without GIs) \) attributed to the food matrix, storage condition, and growth ability of the pathogen. Three scenarios may exist: (i) \( Lt \) is less than the storage time between production \( (P) \) and retail \( (R) \), (ii) \( Lt \) is carried forward to the storage period between \( R \) and consumption \( (C) \), and (iii) \( Lt \) is carried forward to the storage period beyond \( C \), i.e., no \( L. \) monocytogenes growth occurs before consumption of the deli meat.

Food-chain stage

<table>
<thead>
<tr>
<th>Production ( (P) )</th>
<th>Retail ( (R) )</th>
<th>Consumption ( (C) )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Storage time between ( P ) and ( R ) ( (St_{PR}) )</td>
<td>Storage time between ( R ) and ( C ) ( (St_{RC}) )</td>
<td></td>
</tr>
</tbody>
</table>

Scenario 1 \( (Lt < St_{PR}) \)
- Lag time \( (Lt) \)
- Growth time \( (Gt) = St_{PR} + St_{RC} / Lt \)

Scenario 2 \( (Lt > St_{PR}) \)
- Lag time \( (Lt) \)
- Growth time \( (Gt) = St_{PR} + St_{RC} / Lt \)

Scenario 3 \( (Lt > St_{PR} + St_{RC}) \)
- Lag time \( (Lt) \)
- Growth time \( (Gt) = 0 \)

*foods/research/labs/wiedmann/links/pradhan-et-al-deli-meat-risk.cfm* running the models requires @Risk [Palisade Corp., Ithaca, NY], VBA, and FFRA DF [Dose Frequency] set up and dose-response files). The original FFRA model was provided to us by the FFRA risk analysis coordinator (Sherri Dennis, FDA, Washington, DC). Although our risk assessment used the unmodified dose-response model from the FFRA, we made considerable revisions and changes to the exposure model. To allow us to model \( L. \) monocytogenes growth from \( P \) to \( C \), we (i) developed a new module for \( L. \) monocytogenes growth from \( P \) to \( R \) and (ii) modified the \( R \) to \( C \) growth module from the original FFRA to allow consideration of \( Lt \) (e.g., for scenarios in which \( L. \) monocytogenes has a long lag phase that carries over into the \( R \)-to-\( C \) period).

Our new risk assessment can be described as a three-step model, which includes the following three modules: (i) \( P \) to \( R \) growth module, (ii) \( R \) to \( C \) growth and exposure module, and (iii) dose-response module. To include \( Lt \) in our \( P \) to \( R \) and \( R \) to \( C \) growth models, we defined three growth parameters: \( Lt \), EGR, and growth time \( (Gt) \). Conceptually, bacteria will be in either \( Lt \) or \( Gt \), where \( Gt \) is defined as the time during which bacteria grow at an EGR. Although \( Lt \) typically may be short (and \( L. \) monocytogenes hence may complete \( Lt \) during the \( P \) to \( R \) period), \( Lt \) also may be long and carry over into the \( R \) to \( C \) period (particularly for products formulated with GIs, which extend \( Lt \)). Consequently, three different scenarios of lag phase may arise during \( P \) to \( C \) period (Fig. 1). In the first scenario, \( Lt \) is shorter than the storage period between \( P \) and \( R \) \( (St_{PR}) \) and \( Gt \) would thus be the difference of \( St_{PR} \) and \( Lt \) plus the storage period between \( R \) and \( C \) \( (St_{RC}) \). In the second scenario, \( Lt \) exceeds \( St_{PR} \) and in this case \( Gt \) is the total storage period between \( P \) and \( C \) minus \( Lt \). In the third scenario, \( Lt \) exceeds the total storage period between \( P \) and \( C \) in which case \( Gt \) is zero, implying that no growth of \( L. \) monocytogenes will occur in the deli meat. More detailed descriptions of the three modules are provided below.

**P to R growth module.** This module was developed in the program @Risk to compute growth of \( L. \) monocytogenes between \( P \) and \( R \) with a focus on characterizing the variability in the model parameters (see Fig. 2 for a block diagram of this model). The \( L. \) monocytogenes contamination data used in the original FFRA model reflected contamination levels at \( R \), including data collected for foods at \( R \) and some data collected at the manufacturing plants (production) that were adjusted to reflect \( L. \) monocytogenes levels at \( R \). To estimate \( L. \) monocytogenes contamination levels at \( P \) by backcalculating from the contamination levels at \( R \), a scalar adjustment of 1.86 log units \((44, 45)\) was used on each distribution in the set of 300 distributions representing the contamination levels at \( R \) from the original FFRA model.

**R to C growth and exposure module.** The exposure module from the original FFRA model was modified to include a parameter for \( Gt \) during \( R \) to \( C \) storage (storage between retail and consumption), which was calculated using \( Lt \) and \( St_{PR} \). Because the exposure module from the original FFRA model was written in VBA, modification to the model and program code was done also in VBA. In the original model and the program, the \( Gt \) between \( R \) and \( C \) \( (Gt_{RC}) \) was assumed to be equal to the storage time between \( R \) and \( C \) \( (St_{RC}) \). In this modified model, taking into account two new variables \( Lt \) and \( St_{PR} \), the \( Gt_{RC} \) was defined as follows:

\[
Gt_{RC} = \begin{cases} 
\max[0, St_{RC} - (Lt - St_{PR})] & \text{if } Lt - St_{PR} > 0 \\
St_{RC} & \text{if } Lt - St_{PR} \leq 0
\end{cases}
\]

In our model, a triangular distribution was used to characterize \( Lt \) (Table 1) and a uniform distribution was used for \( St_{PR} \) (as detailed in the section “P to R growth module”). To model the EGR, the original FFRA model considered four distributions (triangular, Weibull, gamma, and beta) with different weights assigned to each distribution (so that the most weight was assigned to the distribution that best described the data) to represent uncertainty in EGR distributions, and each distribution represented the variability in EGR. When the data set for EGR values from the original FFRA model was fitted (using BestFit), the logistic distribution...
had the best fit (based on chi-square goodness of fit test statistics), and the logistic distribution was hence used to describe the EGR distribution in our model, representing only the variability dimension (28, 47).

**Dose-response module.** The dose-response module was used to calculate the number of deaths and cases of listeriosis (i.e., those requiring hospitalization) caused by consumption of deli meats contaminated with *L. monocytogenes*. A separate simulation was constructed for each of the three human subpopulations. The framework and models for the dose-response module were not changed from those used in the FFRA model. Outcomes (dose bins) obtained from exposure assessment were used as inputs for the dose-response model to compute the annual number of listeriosis-associated deaths for each subpopulation. As in the original model, the annual number of listeriosis cases was calculated by multiplying the number of deaths per year by factors of 11.3 (for the intermediate age population), 12.7 (for the neonatal population), and 3.7 (for the elderly population) (45).

The variability, i.e., the stochastic process, is the basis of a risk analysis model, and the uncertainty about parameters can be overlaid onto that model variability by using a two-dimensional probabilistic framework (48). However, many risk assessments are one-dimensional. For example, Yang et al. (49) used a one-dimensional probabilistic framework to model only variability in the growth and cross-contamination of *L. monocytogenes* during food storage and preparation of deli meats. In our revised model, we combined one- and two-dimensional probabilistic frameworks. We used a one-dimensional framework to estimate *L. monocytogenes* growth from *P* to *R* based on the variability in the parameters (i.e., *S*<sub>PR</sub>, *ST*<sub>R</sub>, *L*, and EGR) to avoid model complexity (incorporating uncertainty would make interpretation more difficult) and for computational convenience. In the exposure assessment, for *L. monocytogenes* growth from *P* to *R*, we used the two-dimensional probabilistic framework as in the original FFRA model with the exception of modeling only the variability in EGR.

**Simulation and analysis.** To estimate the growth of *L. monocytogenes* between *P* and *R* in three subcategories of deli meats, the growth model between *P* and *R* was simulated with the Monte Carlo sampling technique, and the growth between *P* and *R* (Growth<sub>R</sub>) was added to the contamination level at *P* to obtain the contamination level at *R*.

**RESULTS**

**Comparison of the revised model with the original FFRA model.** The estimated numbers of deaths due to consumption of deli meats based on the original FFRA model were 228, 52, and 32 for elderly, intermediate age, and perinatal groups, respectively (45). Our initial simulation with the original FFRA model reproduced the data reported in the FFRA. Based on expert opinion (42), RTE ham, turkey, and roast beef represent 35, 20, and 15%, respectively, of all types of deli meats produced in the United States. We thus assumed that these percentages also represented appropriate estimates for relative consumption levels for the three deli meat categories. Hence, considering these consumption levels, simulations with the original FFRA model were performed for three subcategories of deli meats (ham,
turkey, and roast beef; see Tables 2 and 3 for the predicted number of deaths and listeriosis cases, respectively).

We also simulated the original FFRA model with EGR characterized as a logistic distribution (\(\alpha = 0.2632, \beta = 0.1064\)), i.e., by giving 100% weight to the logistic distribution (which provided the best fit for the observed data) rather than using four alternative distributions with different relative weights (representing the approach used in the original FFRA model). The data set used to estimate EGR for this simulation represented the same data set used to estimate EGR for deli meats in the original FFRA model. The predicted number of deaths and listeriosis cases with this simulation was highly consistent with the initial simulation for the three deli meat subcategories (see Tables 2 and 3). For example, none of the estimated median number of listeriosis-associated deaths (and the 5th and 95th percentiles) for each subcategory differed by more than 1 from the original estimate. These results confirmed that using only the logistic distribution (which best matched all EGR data used, including PS data and data for products with and without GIs) would provide estimates comparable to those obtained with the original FFRA model. All subsequent simulation runs thus used a single distribution (i.e., the logistic distribution) to characterize the variability in EGR. This approach was necessary to assure comparability between results obtained for food products with and without GIs.

After initial test runs with the models used in the original FFRA, we further performed validation test runs with our model, which included (i) a new \(P\) to \(R\) growth module and (ii) a revised version of the \(R\) to \(C\) growth module, which considers lag phase. Internal validity of the \(R\) to \(C\) growth module in our model was tested by switching off the changes we made to the original FFRA model (e.g., setting lag phase and \(P\) to \(R\) storage time to 0). Results at this setting were the same as those obtained from simulation of the original FFRA model, assuring that differences between estimates from our risk assessment and the original FFRA could be attributed only to the model modifications.

**Effects of PS growth parameters on exposure assessment and predicted number of human listeriosis cases.** The effect of PS EGR data on the number of predicted human listeriosis cases for the three selected deli meat subcategories was initially estimated using the \(R\) to \(C\) growth module and the dose-response module used in the original FFRA (using a logistic distribution for EGR). Mean PS EGRs for ham, turkey, and roast beef were 0.19, 0.28, and 0.27 log CFU/day, respectively (Table 1) compared with a mean generic deli meat EGR of 0.28 log CFU/day used in the original FFRA. Consistent with the fact that the PS EGR for ham was lower than the generic deli meat EGR used in the original FFRA, the estimated numbers of listeriosis deaths and illnesses attributed to ham were 1.3-fold lower in the simulation with the PS EGR compared with those estimated with the simulation using the generic deli meat EGR (Tables 2 and 3). For turkey and roast beef, which had PS EGRs very similar to the generic deli meat EGR used in the original model, the estimated numbers of listeriosis deaths and illnesses were very similar in the simulations with PS EGR and with the generic deli meat EGR (Tables 2 and 3). These data indicate that estimation and use of PS growth parameters may affect our estimates of the number of listeriosis cases attributable to different deli meat subcategories and may be needed for an accurate risk assessment.

**Effects of GIs on exposure assessment and predicted number of human listeriosis cases.** Our revised model was used to estimate the numbers of human listeriosis deaths and illnesses that would be attributed to ham, turkey, and roast beef if (i) all products in these categories were formulated without additional GIs and (ii) all products in these categories were formulated with GIs. Simulations were performed separately for the three deli meat subcategories using PS \(Lt\) and EGR data for products with and without GIs. The simulation results of numbers of listeriosis deaths and cases per annum are shown in Tables 2 and 3, respectively, for products formulated with and without GIs.

The estimated numbers of deaths and illnesses obtained from our revised model (using both PS \(Lt\) and EGR) were generally lower than the numbers of deaths and illnesses estimated with PS EGR only and with the original FFRA. In particular, the estimated numbers of deaths and illnesses for ham and roast beef formulated without GIs were 2.4- and 1.9-fold lower for the revised model (which considers PS lag phase and EGR) than those obtained with the original FFRA model with PS EGR but not lag phase (see Tables 2 and 3), reflecting long mean lag phases for these products (\(Lt = 5.9\) and 5.1 days for ham and roast beef, respectively, formulated without GIs). By comparison, the estimated numbers of deaths and illnesses for turkey (without GIs) were only 1.2-fold lower for the revised model compared with the numbers obtained with the original model and PS EGR (see Tables 2 and 3), consistent with the considerably shorter mean lag phase for turkey (\(Lt = 2.2\) days). Overall, inclusion of lag phase appears to be important in an \(L.\) monocytogenes infection risk assessment, particularly when assessing relative risk of different products that differ in the lag phase they impose on \(L.\) monocytogenes present.

Simulations, with our modified model, for the three selected deli meats subcategories and PS EGR and \(Lt\) for products formulated with GIs (i.e., lactate and diacetate) were performed to estimate the reductions in deaths and illnesses associated with these products that could be achieved if all products in a category were reformulated with GIs. Overall, our simulations showed a considerable reduction of the estimated number of human listeriosis deaths and illnesses if products were assumed to contain GIs. For example, while our modified model predicted, among the elderly subpopulation, 39 deaths due to RTE turkey if no product contained GIs, only 5 deaths due to RTE turkey were predicted in this subpopulation if all products contained GIs, i.e., we estimated 7.8-fold reduction in the number of listeriosis deaths. Estimated reductions achievable with reformulation of ham and roast beef with GIs were less striking. For example, while our modified model predicted, among the elderly subpopulation, 25
### TABLE 2. Estimated number of deaths per annum in the United States due to listeriosis for each type of deli meat in each subpopulation as obtained from simulation of original and modified models with different scenarios

<table>
<thead>
<tr>
<th>Subpopulation</th>
<th>Deli meat type</th>
<th>Original model with original EGR (c)</th>
<th>Original model with logistic EGR (d)</th>
<th>Original model with PS EGR (e)</th>
<th>Modified model without GI, PS EGR, and (L_t^f)</th>
<th>Modified model with GI, PS EGR, and (L_t^g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elderly</td>
<td>Ham</td>
<td>79.72 (15.44, 103.80)</td>
<td>79.53 (15.5, 102.94)</td>
<td>61.08 (11.42, 80.16)</td>
<td>25.13 (4.04, 38.82)</td>
<td>10.13 (1.52, 16.22)</td>
</tr>
<tr>
<td></td>
<td>Turkey</td>
<td>45.55 (8.82, 59.32)</td>
<td>45.44 (8.86, 58.82)</td>
<td>48.06 (9.30, 62.06)</td>
<td>39.4  (7.29, 52.02)</td>
<td>5.07  (0.78, 8.38)</td>
</tr>
<tr>
<td></td>
<td>Roast beef</td>
<td>34.16 (6.62, 44.49)</td>
<td>34.08 (6.64, 44.12)</td>
<td>35.84 (6.95, 46.25)</td>
<td>18.66 (3.09, 27.09)</td>
<td>5.11  (0.80, 8.08)</td>
</tr>
<tr>
<td>Intermediate age</td>
<td>Ham</td>
<td>18.25 (3.74, 22.81)</td>
<td>18.25 (3.75, 22.62)</td>
<td>13.99 (2.77, 17.61)</td>
<td>5.79  (0.97, 8.58)</td>
<td>2.34  (0.37, 3.62)</td>
</tr>
<tr>
<td></td>
<td>Turkey</td>
<td>10.43 (2.13, 13.03)</td>
<td>10.43 (2.14, 12.92)</td>
<td>11 (2.24, 13.63)</td>
<td>8.99  (1.8, 11.45)</td>
<td>1.18  (0.19, 1.86)</td>
</tr>
<tr>
<td></td>
<td>Roast beef</td>
<td>7.82 (1.6, 9.78)</td>
<td>7.82 (1.61, 9.69)</td>
<td>8.21 (1.67, 10.16)</td>
<td>4.29  (0.73, 5.99)</td>
<td>1.20  (0.19, 1.81)</td>
</tr>
<tr>
<td>Perinatal (h)</td>
<td>Ham</td>
<td>11.14 (3.08, 13.60)</td>
<td>11.12 (3.06, 13.48)</td>
<td>8.55 (2.27, 10.53)</td>
<td>3.62  (0.83, 5.13)</td>
<td>1.42  (0.31, 2.13)</td>
</tr>
<tr>
<td></td>
<td>Turkey</td>
<td>6.36 (1.76, 7.77)</td>
<td>6.35 (1.75, 7.70)</td>
<td>6.72 (1.85, 8.13)</td>
<td>5.55  (1.49, 6.83)</td>
<td>0.72  (0.16, 1.11)</td>
</tr>
<tr>
<td></td>
<td>Roast beef</td>
<td>4.77 (1.32, 5.83)</td>
<td>4.77 (1.31, 5.78)</td>
<td>5.02 (1.38, 6.07)</td>
<td>2.66  (0.64, 3.56)</td>
<td>0.73  (0.16, 1.06)</td>
</tr>
</tbody>
</table>

\(^a\) Values are median, with 5th and 95th percentiles in parentheses.

\(^b\) Three major types of deli meats were considered. The consumption level of ham, turkey, and roast beef were assumed to be the same as the production levels, which were 35, 20, and 15%, respectively, of the total amount of deli meat produced in the United States (based on the expert opinion).

\(^c\) FDA-FSIS risk assessment (FFRA) model (the original model) was simulated with the same generic exponential growth rate (EGR) distributions as used in the original model.

\(^d\) Original model was simulated with the generic EGR described by a logistic distribution representing the data set used in the original model.

\(^e\) Original model was simulated with product-specific (PS) EGR (Table 1) for three types of deli meats formulated without addition of growth inhibitors (GIs).

\(^f\) Modified model was simulated with PS EGR and lag time \((L_t)\) for products without addition of GIs (see Table 1).

\(^g\) Modified model was simulated with PS EGR and \(L_t\) for products with added GIs (see Table 1).

\(^h\) Number of perinatal (combined prenatal and neonatal) deaths was calculated by multiplying the neonatal deaths by 2.5 to account for abortions and stillbirths not reported in FoodNet surveillance reports (45).
<table>
<thead>
<tr>
<th>Subpopulation</th>
<th>Deli meat type</th>
<th>Original model with original EGR</th>
<th>Original model with logistic EGR</th>
<th>Original model with PS EGR</th>
<th>Modified model without GI, PS EGR, and Lt</th>
<th>Modified model with GI, PS EGR, and Lt</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elderly</td>
<td>Ham</td>
<td>294.96 (57.13, 384.07)</td>
<td>294.25 (57.35, 380.87)</td>
<td>226.01 (42.25, 296.6)</td>
<td>92.98 (14.94, 143.62)</td>
<td>37.47 (5.62, 60.01)</td>
</tr>
<tr>
<td></td>
<td>Turkey</td>
<td>168.55 (32.65, 219.47)</td>
<td>168.14 (32.77, 217.64)</td>
<td>177.84 (34.42, 229.46)</td>
<td>145.8 (26.96, 192.49)</td>
<td>18.77 (2.9, 30.99)</td>
</tr>
<tr>
<td></td>
<td>Roast beef</td>
<td>126.41 (24.48, 164.40)</td>
<td>126.11 (24.58, 163.23)</td>
<td>132.6 (25.72, 171.13)</td>
<td>69.02 (11.44, 100.23)</td>
<td>18.92 (2.97, 29.88)</td>
</tr>
<tr>
<td>Intermediate age</td>
<td>Ham</td>
<td>206.19 (42.21, 257.74)</td>
<td>206.24 (42.41, 255.55)</td>
<td>158.03 (31.31, 199.04)</td>
<td>65.42 (10.98, 96.92)</td>
<td>26.41 (4.16, 40.87)</td>
</tr>
<tr>
<td></td>
<td>Turkey</td>
<td>117.82 (24.12, 147.28)</td>
<td>117.85 (24.24, 146.03)</td>
<td>124.35 (25.31, 154.06)</td>
<td>101.61 (20.35, 129.42)</td>
<td>13.36 (2.11, 21.03)</td>
</tr>
<tr>
<td></td>
<td>Roast beef</td>
<td>88.37 (18.09, 110.46)</td>
<td>88.39 (18.18, 109.52)</td>
<td>92.72 (18.84, 114.86)</td>
<td>48.50 (8.21, 67.65)</td>
<td>13.51 (2.18, 20.44)</td>
</tr>
<tr>
<td>Perinatal</td>
<td>Ham</td>
<td>56.57 (15.63, 69.11)</td>
<td>56.48 (15.57, 68.49)</td>
<td>43.43 (11.53, 53.4)</td>
<td>18.37 (4.21, 26.06)</td>
<td>7.24 (1.57, 10.82)</td>
</tr>
<tr>
<td></td>
<td>Turkey</td>
<td>32.32 (8.93, 39.49)</td>
<td>32.28 (8.9, 39.14)</td>
<td>34.13 (9.41, 41.31)</td>
<td>28.21 (7.57, 34.7)</td>
<td>3.67 (0.8, 5.62)</td>
</tr>
<tr>
<td></td>
<td>Roast beef</td>
<td>24.24 (6.7, 29.62)</td>
<td>24.21 (6.67, 29.35)</td>
<td>25.52 (7.01, 30.82)</td>
<td>13.54 (3.24, 18.11)</td>
<td>3.69 (0.81, 5.4)</td>
</tr>
</tbody>
</table>

* Values are estimated median (with 5th and 95th percentiles in parentheses) number of cases of serious illnesses (i.e., those requiring hospitalization).

b Three major types of deli meats were considered. The consumption level of ham, turkey, and roast beef were assumed to be the same as the production levels, which were 35, 20, and 15%, respectively, of the total amount of deli meat produced in the United States (based on the expert opinion).

c FDA-FSIS risk assessment (FFRA) model (the original model) was simulated with the same generic exponential growth rate (EGR) distributions as used in the original model.

d Original model was simulated with the generic EGR described by a logistic distribution representing the data set used in the original model.

e Original model was simulated with product-specific (PS) EGR (Table 1) for three types of deli meats formulated without addition of growth inhibitors (GIs).

f Modified model was simulated with PS EGR and lag time (Lt) for products without addition of GIs (see Table 1).

g Modified model was simulated with PS EGR and Lt for products with added GIs (see Table 1).

h Number of perinatal (combined prenatal and neonatal) cases of illnesses equals neonatal cases of illnesses. A multiplier of 2.5 to neonatal cases was not used to calculate perinatal cases, because nonlethal infections do not result in prenatal hospitalizations (45).
The risk of foodborne disease associated with *L. monocytogenes* has been the subject of several quantitative risk assessment endeavors over the last few years (22, 36, 37, 40, 44, 45, 49). Over at least the last decade, both the food industry and government agencies have pursued a number of complementary strategies to reduce both *L. monocytogenes* prevalence and contamination levels in deli meats, with the ultimate goal of reducing the number of human listeriosis cases. Key strategies pursued to control *L. monocytogenes* include stringent implementation of and adherence to sanitation standard operating procedures and good manufacturing practices (18, 27), postlethality treatments (2, 32, 50), and product reformulations to control *L. monocytogenes* growth (7, 17, 29, 30). Use of our revised risk assessment models with existing data on growth kinetics parameters (i.e., EGR and Lt) for three selected RTE deli meat subcategories, formulated with and without the GIs (lactate and diacetate), indicated that (i) deli meat product types differ in their ability to support *L. monocytogenes* growth and their estimated per annum risk to human health and thus should be separated into different categories in a risk assessment and (ii) reformulation with GIs will reduce human listeriosis deaths and illnesses, although the public health effects of GIs differ by product type.

**Deli meat product types differ in their ability to support *L. monocytogenes* growth and their estimated per annum risk to human health.** Although the FFRA estimated the public health risk associated with the presence of *L. monocytogenes* in 23 different RTE food categories, one food category, RTE deli meats, was estimated to be responsible for about 90% of human listeriosis cases in the United States (1,598.7 cases per year). In contrast, 14 other RTE food categories (e.g., vegetables, fruits, and fresh soft cheese) each were estimated to be responsible for <1 human listeriosis case per year. RTE deli meats include a number of different products (e.g., bologna, corned, cooked, and roast beef, ham, beef sirloin, sliced chicken, chicken fillets, and sliced turkey), which differ not only in various physicochemical parameters (e.g., pH, water activity, and salt content) (9, 16–18, 25, 29) but also in their ability to support *L. monocytogenes* growth (14, 16). Different types of RTE deli meats also differ in the relative effects that various GIs have on *L. monocytogenes* growth. For example, RTE deli meats with lower pH may support less growth, and the effectiveness of GIs such as lactate and diacetate also differs based on the product pH (7). Thus, we initially used the models from the original FFRA to model the human health risk from *L. monocytogenes* attributable to three selected deli meat types: ham, turkey, and roast beef.

A comprehensive risk assessment model considering different RTE deli meats remains a major challenge because of (i) the limited availability of PS growth parameters for many RTE deli meats and (ii) the limited availability of consumption data. Thus, we selected as an initial model three deli meat subcategories for which sufficient growth kinetics data were available. Consumption of these subcategories in the United States had to be estimated (as a percentage of all deli meats consumed annually in the United States) from expert opinion. Although we appreciate that the estimated consumption may not be correct, the absolute consumption data were needed only for illustration purposes because relative trends (i.e., fold difference in estimated deaths between models using generic deli meat EGR and PS EGR) are independent of total numbers. Improved data on relative consumption of different RTE deli meat subcategories will be needed for development of a risk ranking of different deli meat types.

Overall, our estimates indicate that use of PS *L. monocytogenes* growth parameters will affect estimates of the number of human illnesses attributable to different product categories. For example, for a product such as ham that permits reduced growth and imposes an extended lag phase, the estimated number of attributed human listeriosis deaths will be lower than the estimate obtained with generic deli meat growth parameters. Hence, an accurate risk ranking among different RTE deli meats will require models that use PS growth parameters and estimate deaths and illnesses for different product categories. Turkey permitted the fastest *L. monocytogenes* EGR and the shortest lag phase, which is particularly important because sliced deli meats have been associated with multiple listeriosis outbreaks in the United States over the last several years (10, 11), further emphasizing the public health risk associated with this RTE deli meat category. Our models could be used further to quantify the impact of curing on *L. monocytogenes* growth and listeriosis cases associated with turkey products as long as sufficient data are available to estimate growth parameters for cured or uncured RTE turkey deli meats.

**Reformulation with GIs will reduce human listeriosis deaths and illnesses, although the public health effects of GIs differ by product type.** We focused on two GIs, lactate and diacetate, because (i) they are commonly used, (ii) they are approved for use in RTE deli meats, and (iii) data are available on *L. monocytogenes* growth parameters for RTE deli meats formulated with these GIs (12, 13, 16, 29, 30, 32). Our models can easily be used to assess the impact of other GIs (e.g., nisin, acetic acid, lactic acid, sorbate, benzoate, and propionate) (2, 16, 17) as long as sufficient data are available to estimate growth parameters for foods formulated with a given GI. Overall, our models assumed that use of GIs would not affect other parameters associated with either the exposure assessment or the dose-response curve. For example, use of GIs could suppress the growth of spoilage microflora, thus increasing product shelf life and reducing competitive microflora, both factors that could affect *L. monocytogenes* contamination levels at the time of consumption.
Reductions in the number of human listeriosis cases achievable as a result of reformulation of RTE deli meats will vary for different product types; reductions were estimated to range from 7.8-fold reduction for cases caused by turkey (if all products in this category consumed in the United States were to be reformulated with GIs) to a 2.5-fold reduction for cases caused by ham. However, in all cases the 5th and 95th percentiles range for the number of listeriosis deaths and illnesses for a given product category overlap for products formulated with and without GIs, indicating that these predicted reductions should be interpreted with caution. This overlap in model results in part reflects the overlapping growth kinetic parameters (i.e., EGR and Lt) for products formulated with and without GIs (Table 1). Overall, however, the results from our simulations indicate that reformulation of RTE deli meats with GIs has the potential to lead to considerable reduction in the numbers of listeriosis deaths and illnesses, even though the magnitude of this effect may differ between different deli meat categories. Reformulation of RTE deli meat products with GIs thus is likely to have a positive public health impact (if reformulation does not affect other parameters such as shelf life). However, reformulation, even if used for all RTE deli meat products, clearly will not reduce the number of listeriosis cases attributable to RTE deli meats to numbers close to zero. Even with reformulation, control of deli meat–associated listeriosis will require a comprehensive strategy, including shelf life limitation, reduction of contamination at the manufacture (9, 27, 29) and retail (18, 19, 21, 27, 31) levels (e.g., through stringently enforced sanitation standard operating procedures and good manufacturing practices), and consumer education (27, 35).

The risk assessment models presented here, which are freely available to others, and the initial results generated with our model provide new quantitative estimates of the effects of RTE deli meat PS parameters and the possible public health impact of product reformulation with GIs. Further use and refinement of our models will provide an opportunity for improved risk rankings and prioritization of different intervention strategies for L. monocytogenes contamination in RTE deli meats. Although a number of data needs have already become apparent through our risk assessment (e.g., consumption data for different types of deli meats, PS growth kinetics data for products with and without GIs, and contamination data at manufacturing), future sensitivity analyses with our models will help to identify the most pressing data needs, which will have the greatest impact on the confidence associated with modeling outcomes. Because our model includes separate production to retail (P to R) and retail to consumption (R to C) growth modules, it also will permit modeling of the relative contributions of contamination at manufacturing and retail to the health impact of human listeriosis, a particularly critical need considering the emerging evidence that contamination at retail represents a considerable concern (19, 27, 31).

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REFERENCES


34. Pathogen Reduction Laboratory, Center for Meat Safety and Quality (Colorado State University, Fort Collins). Unpublished data.


42. Thippareddi, H. (Department of Food Science and Technology, University of Nebraska–Lincoln). Personal communication.


