Journal of Food Protection, Vol. 75, No. 5, 2012, Pages 896–905
doi:10.4315/0362-028X.JFP-11-491

Growth of *Listeria monocytogenes* in Different Retail Delicatessen Meats during Simulated Home Storage

LEI ZHANG,[1] SCOTT R. MOOSEKIAN,[1] EWEN C. D. TODD,[2] AND ELLIOT T. RYSER[1,3]*

[1]*Department of Food Science and Human Nutrition;* [2]*Department of Advertising, Public Relations, and Retailing; and* [3]*National Food Safety and Toxicology Center, Michigan State University, East Lansing, Michigan 48824, USA*

MS 11-491: Received 1 November 2011/Accepted 20 January 2012

ABSTRACT

Delicatessen meats are reported to be the leading vehicle of foodborne listeriosis in the United States. *Listeria monocytogenes* can reach high numbers in these products during storage, and the growth rate is largely dictated by product formulation and storage temperature. To assess the impact of product age on *Listeria* growth, five commercial brands each of cured and uncured turkey breast, ham, and roast beef (three lots per brand) were sliced (approximately 25 g per slice) at the beginning of the shelf life, the midpoint, and the last allowable day of sale, surface inoculated with an eight-strain cocktail of *L. monocytogenes* (approximately 40 CFU/g), and then quantitatively examined for *Listeria,* lactic acid bacteria, and mesophilic aerobic bacteria during aerobic storage at 4, 7, or 10°C. As expected, *L. monocytogenes* grew faster in deli meats without rather than with *Listeria* inhibitors (lactate and/or diacetate) and at the highest storage temperature (10°C). Lag-phase durations for *L. monocytogenes* in deli meats with and without *Listeria* inhibitors were 9.21, 6.96, and 5.00 and 6.35, 3.30, and 2.19 days at 4, 7, and 10°C, respectively. Generation times for *L. monocytogenes* in deli meats with and without *Listeria* inhibitors were 1.59, 1.53, and 0.85 and 0.94, 0.50, and 0.36 at 4, 7, and 10°C, respectively. Maximum population densities for *L. monocytogenes* in deli meats with and without *Listeria* inhibitors were 5.26, 5.92, and 5.97 and 8.47, 8.96 and 9.34 log CFU/g at 4, 7, and 10°C, respectively. Although lactate and diacetate suppressed *L. monocytogenes* growth, the extent of inhibition differed, ranging from total inhibition in roast beef to only partial inhibition in ham and cured turkey. *Listeria* growth was also impacted by lot-to-lot variation in the concentrations of *Listeria* inhibitors, product pH, and background microflora. These data will be useful for developing recommendations for ''best consumed by'' dating for deli meats using a risk-based approach.

Listeriosis, a foodborne infection caused by *Listeria monocytogenes,* remains an ongoing concern due to the potentially serious clinical outcomes, which can include meningitis, abortion, and perinatal septicemia. In the 2003 risk assessment produced by the U.S. Food and Drug Administration in cooperation with the U.S. Department of Agriculture Food Safety and Inspection Service (USDA FSIS) *(50),* delicatessen meats were identified as possessing the highest risk among the 23 categories of ready-to-eat (RTE) foods examined. Delicatessen meats also have been implicated in several high profile listeriosis outbreaks *(7–9, 18, 34).* In response, the USDA FSIS issued three alternatives for control of *L. monocytogenes* in RTE meats; postpackaging pasteurization and inclusion of *Listeria* growth inhibitors in the product formulation were the two most useful means for reducing the public health risk associated with *L. monocytogenes (49).* Among the various *Listeria* inhibitors, lactate and diacetate are effective in RTE poultry and meat products at maximum allowable levels of 4.8 and 0.25% (wt/wt), respectively *(48, 49),* and inhibitory activity increases when these two additives are used in combination *(2, 16, 29, 31, 40, 44, 45, 47).*

The ability of *L. monocytogenes* to grow in deli meats, including cured and uncured turkey, ham, bologna, corned beef, roast beef, and salami, varies based on product formulation, storage temperature, and the presence of native microflora *(3, 5, 15, 20, 25, 26).* In the 2003 risk assessment, a single exponential growth rate was used for all RTE meats *(50)*; however, compositional differences between these products greatly affect *Listeria* growth and survival *(5, 15).* Risk profiles for *L. monocytogenes* in RTE meats *(13, 24)* identified the need for additional growth data for *Listeria* in a wider range of RTE meats and times and temperatures during retail and consumer storage to make better informed risk management decisions. Most published studies have examined the survival and growth of *L. monocytogenes* or the efficacy of various antilisterial hurdles during vacuum-packaged storage of RTE meats *(2, 31, 40, 44, 45, 47),* but data on the behavior of the pathogen under home storage conditions are limited. The potential for *Listeria* growth in deli meats of different compositions during retail display, transit to the home, and extended storage has raised ongoing safety concerns regarding the risk that these products pose to consumers at the time of consumption *(10, 11, 21, 32, 54).*

* Author for correspondence. Tel: 517-355-8474, Ext 185; Fax: 517-353-8963; E-mail: ryser@msu.edu.

This study was undertaken to evaluate the growth characteristics of *L. monocytogenes* in four different types of deli meat—uncured turkey, cured turkey, ham, and roast beef, with or without lactate or diacetate—under various aerobic consumer storage scenarios. The objective was to determine the impact of product age, *Listeria* growth inhibitors, and background microflora on *L. monocytogenes* growth at three storage temperatures: 4, 7, and 10°C. These data can then be used to develop recommendations for ''best consumed by'' dating of deli meats as part of a risk-based approach.

MATERIALS AND METHODS

***L. monocytogenes* strains.** Eight *L. monocytogenes* strains were used in this study. Four strains were previously isolated from deli meats during a large-scale survey in Minnesota: Lm5417 from uncured turkey, Lm5426 from roast beef, Lm5772 from cured turkey, and Lm6100 from ham. Four U.S. outbreak strains were provided by Dr. Martin Wiedmann (Cornell University, Ithaca, NY): R2-499 (serotype 1/2a, clinical isolate from the 2000 deli turkey outbreak), N1-227 (serotype 4b, frankfurter isolate from the 1998 to 1999 outbreak), R2-763 (serotype 4b, clinical isolate from a 2002 deli turkey outbreak), and R2-764 (serotype 4b, deli turkey isolate from the same 2002 outbreak). Each strain was preserved at −80°C in Trypticase soy broth containing 0.6% yeast extract (TSBYE; Difco, BD, Sparks, MD) and 15% glycerol (J. T. Baker, Phillipsburg, NJ) and subjected to two consecutive 24-h transfers at 37°C in 10 ml of TSBYE. Cultures were then combined in equal volumes, pelleted by centrifugation at 3,100 × *g* (Sorvall Super T21 Centrifuge, Sorvall Products, L.P., Newtown, CT) for 15 min at 4°C, and washed twice in sterile phosphate-buffered saline (PBS; 7.65 g/liter NaCl, 0.724 g/liter anhydrous Na_2HPO_4, 0.21 g/liter KH_2PO_4, pH 7.4; Mallinckrodt Baker, Inc., Phillipsburg, NJ) to obtain an eight-strain *L. monocytogenes* cocktail containing approximately 1×10^9 CFU/ml. This cocktail was then serially diluted in sterile 0.1% peptone water to a level of approximately 10^4 CFU/ml for meat inoculation.

Deli meats. Growth of *L. monocytogenes* was assessed in five retail brands each of cured turkey, uncured turkey, ham, and roast beef. These products were selected based on the presence or absence of nitrite as a curing agent and of lactate and diacetate, two *Listeria* growth inhibitors approved for controlling *Listeria* in deli meats *(48, 49)*. Three whole chubs of cured turkey, uncured turkey, ham, or roast beef from the same lot number were purchased from local supermarkets in Lansing, MI, on the day of delivery. To assess the impact of product shelf life on *Listeria* growth, one chub was immediately inoculated with the eight-strain cocktail, and the remaining two chubs were stored at 4°C for inoculation at the midpoint of the shelf life and the last allowable sale date as stated on the wrapper. The time intervals between the day of delivery and last allowable date of sale for each deli meat type and/or brand were approximately 1 month (two brands) and 2 months (three brands) for uncured turkey, 2 to 2.5 months for cured turkey (all five brands), 1.5 to 3.5 months for ham, and 0.5 month (two brands) to 1 month (three brands) for roast beef. All experiments were conducted in triplicate with three different lot numbers of the same product from the same manufacturer. A total of 180 chubs of deli meat were evaluated during the study (5 brands × 4 types of deli meat × 3 inoculation times × 3 replications).

Deli meat inoculation. The outer wrapper of the packaged chub was disinfected with 70% ethanol. After slitting the package with a flame-sterilized knife, a 250-g portion was cut from one end of the chub, placed in a Whirl-Pak bag (Nasco, Modesto, CA), and frozen at −20°C for acetate, lactate, and pH analyses. The cut face of the chub was then sliced with a mechanical delicatessen slicer (Omcan 9-in. [23-cm] meat slicer, model 220F, Food Machinery of America Inc., Niagara Falls, NY) to obtain 70 to 80 slices weighing 25 ± 1 g each. Individual slices were then spot inoculated on one side with 100 μl of the diluted eight-strain *L. monocytogenes* cocktail to obtain approximately 40 CFU/g. After 20 min to allow the inoculum to absorb into the product, each inoculated slice was aseptically transferred to a separate sterile Whirl-Pak bag. Twenty-two slices from each product lot were stored at 4, 7, and 10°C; uninoculated slices of the same product were used as negative controls for quantification of lactic acid and mesophilic aerobic bacteria.

Measurement of pH. Ten-gram deli meat samples were transferred to a Whirl-Pak bag containing 90 ml of sterile distilled water and homogenized for 2 min (Stomacher 400 Circulator, Seward, Worthington, UK), and the pH was measured with a standard combination electrode (HI 221, Hanna Instruments, Inc., Woonsocket, RI).

Microbiological analysis. Duplicate 25-g deli meat slices stored at 4, 7, and 10°C were analyzed at 3-, 2-, and 1-day intervals up to days 33, 22, and 11, respectively. All samples were diluted 1:10 in PBS and homogenized by stomaching for 2 min in the Stomacher 400 at high speed. Appropriate serial dilutions in PBS were then plated on modified Oxford agar (Difco, BD), Trypticase soy agar supplemented with 0.6% yeast extract (Difco, BD), and lactobacilli de Man Rogosa Sharpe agar (Difco, BD) to enumerate *L. monocytogenes,* mesophilic aerobic bacteria (MAB), and presumptive lactic acid bacteria, respectively, after incubation for 48 h at 35°C, 48 h at 30°C, and 72 h at 30°C, respectively.

Lactate and diacetate concentrations in deli meat. Frozen samples from each lot of deli meat containing lactate and/or diacetate as stated on the packaging label were shipped overnight in an ice chest to Dr. Ann Draughon (University of Tennessee, Knoxville). After thawing, 50-g samples were added to 450 ml of deionized water, homogenized in a mechanical blender for 2 min, filtered through no. 113 filter paper (Whatman, Piscataway, NJ), extracted in 100 ml of 0.5 N perchloric acid, and filtered through Whatman no. 4 filter paper and then through a 0.45-μm-pore-size syringe filter (Millipore, Billerica, MA). Standard lactic acid and acetic acid solutions were prepared by dissolving reagent lactic acid (Sigma Chemical Co., St. Louis, MO) and acetic acid (Acros Organics, Morris Plains, NJ) in double-deionized water. A high-performance liquid chromatography system (Dionex Corp., Sunnyvale, CA) consisting of a GP50 gradient pump, PDA-100 photodiode array detector, AS50 auto-sampler, and PeakNet software was used. All separations were carried out on an Aminex HPX-87H ion exclusion column (Bio-Rad Laboratories, Richmond, CA) with a cation H^+ Microguard cartridge (Bio-Rad). The mobile phase consisted of 0.005 M H_2SO_4 at a flow rate of 0.6 ml/min. Filtered samples (20 μl) were injected with an autoinjector. The analytes were detected with a UV detector at 210 nm and then quantified based on the external lactate and acetate standards.

Growth parameters for *L. monocytogenes*. *L. monocytogenes* populations in deli meat were fitted using the modified Gompertz equation *(53, 55)* to obtain the lag-phase duration (LPD), growth rate (generation time, GT), and maximum population density (MPD) at 4, 7, and 10°C using the following formula:

$$y(t) = y_0 + (y_{max} - y_0) \cdot \exp\{-\exp[\mu \cdot \exp(1) \cdot (\text{lag time}) \div (y_{max} - y_0) + 1]\} \quad (1)$$

TABLE 1. *Deli meat pH at delivery, midpoint of shelf life, and the last allowable day of sale*

Deli meat	Brand	Antimicrobial(s)[a]	pH[b]		
			Delivery	Midpoint	Last allowable day
Uncured turkey	A	−	6.44 ± 0.05	6.35 ± 0.10	6.27 ± 0.09
	B	−	6.37 ± 0.06	6.23 ± 0.14	6.22 ± 0.24
	C	−	6.35 ± 0.02	6.29 ± 0.10	6.34 ± 0.06
	D	−	6.40 ± 0.09	6.38 ± 0.14	6.40 ± 0.03
	E	−	6.19 ± 0.04	6.30 ± 0.19	6.34 ± 0.10
	Mean		6.35 ± 0.10	6.31 ± 0.13	6.31 ± 0.12
Cured turkey	F	A	6.17 ± 0.05	6.23 ± 0.11	5.90 ± 0.20
	G	A	6.27 ± 0.06	6.24 ± 0.13	6.31 ± 0.19
	H	A	6.29 ± 0.07	6.30 ± 0.01	6.32 ± 0.09
	I	−	6.43 ± 0.01	6.33 ± 0.06	6.29 ± 0.02
	J	−	6.31 ± 0.07	6.40 ± 0.08	6.34 ± 0.06
	Mean		6.29 ± 0.10	6.30 ± 0.10	6.23 ± 0.21
Ham	K	−	6.35 ± 0.09	6.35 ± 0.09	6.41 ± 0.15
	L	−	6.43 ± 0.08	6.35 ± 0.03	6.44 ± 0.05
	M	A, L	6.42 ± 0.05	6.23 ± 0.14	6.32 ± 0.07
	N	A, L	6.37 ± 0.12	6.48 ± 0.01	6.33 ± 0.10
	O	−	6.27 ± 0.05	6.29 ± 0.06	6.34 ± 0.06
	Mean		6.37 ± 0.09	6.33 ± 0.11	6.32 ± 0.09
Roast beef	P	−	5.69 ± 0.08	5.82 ± 0.10	5.92 ± 0.20
	Q	A, L	6.19 ± 0.03	6.08 ± 0.23	5.81 ± 0.24
	R	−	5.90 ± 0.07	5.81 ± 0.10	5.65 ± 0.36
	S	−	6.15 ± 0.04	6.02 ± 0.15	5.87 ± 0.37
	T	A	5.94 ± 0.11	6.04 ± 0.08	6.03 ± 0.14
	Mean		5.97 ± 0.20	5.95 ± 0.17	5.85 ± 0.27

[a] −, without antimicrobials; A, diacetate; L, lactate.
[b] Mean ± standard deviation ($n = 3$).

where $y(t)$ is the *L. monocytogenes* population (log CFU per gram) at time t (days), y_0 is the initial population (log CFU per gram), y_{max} is the maximum population (log CFU per gram), lag is the lag phase (days), and μ is the maximum growth rate (per day). The Statistical Analysis Systems NLIN procedure (2009, SAS Institute, Cary, NC) for a nonlinear least squares regression was used to obtain the Gompertz parameters.

GT was calculated from μ as described by the following equation:

$$TTR_{1,000} = LPD + (3 - y_0) \cdot \log_2 10 \cdot GT \quad (2)$$

The LPD, GT, and initial *L. monocytogenes* population were combined to calculate the time in days needed to reach *L. monocytogenes* populations of 100 and 1,000 CFU/g (TTR_{100}, $TTR_{1,000}$) using the following equations:

$$TTR_{100} = LPD + (2 - y_0) \cdot \log_2 10 \cdot GT \quad (3)$$

$$TTR_{1,000} = LPD + (3 - y_0) \cdot \log_2 10 \cdot GT \quad (4)$$

Statistical analysis. The mean values obtained for LPD, MPD, and GT using nonlinear regression were analyzed for significant differences at $P < 0.05$ using the GLM procedure (SAS Institute).

RESULTS

Deli meat pH and antimicrobial concentrations. Uncured turkey, cured turkey, and ham had a similar pH of approximately 6.40, whereas roast beef had a pH close to

TABLE 2. *Acetate and lactate concentrations in deli meats*

Deli meat	Brand	Acetate (mg/g)[a]	Lactate (mg/g)[a]
Cured turkey	F	0.68 ± 0.32 (0.39–1.43)	−[b]
	G	0.62 ± 0.29 (0.32–1.12)	−
	H	0.83 ± 0.15 (0.66–1.02)	−
Ham	M	0.70 ± 0.06 (0.61–0.76)	12.14 ± 1.25 (10.55–14.13)
	N	0.83 ± 0.11 (0.64–0.96)	10.78 ± 1.39 (8.34–12.09)
Roast beef	Q	1.07 ± 0.44 (0.58–1.80)	11.65 ± 1.51 (9.05–13.18)
	T	1.13 ± 0.09 (0.95–1.26)	−

[a] Mean ± standard deviation (range) ($n = 9$).
[b] −, without antimicrobials.

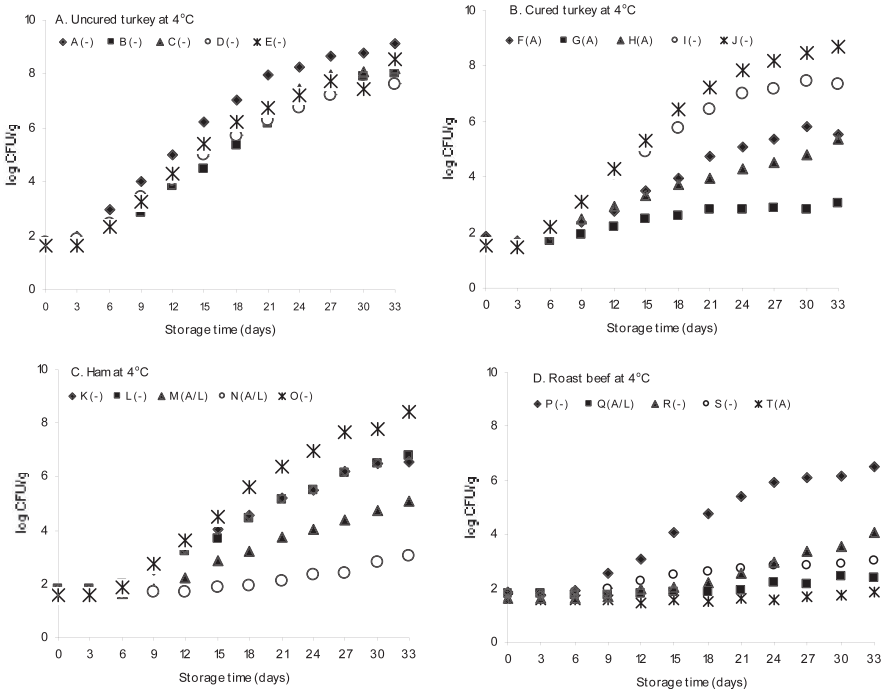

FIGURE 1. *Populations (log CFU per gram) of* L. monocytogenes *on 20 brands (A through T, three lots per brand) of sliced inoculated deli meat without antimicrobials (−) or with diacetate (A) or lactate (L). Results are averages on the date of delivery, the midpoint of shelf life, and the last allowable day of sale. Samples were all stored aerobically at 4°C. (A) Uncured turkey; (B) cured turkey; (C) ham; (D) roast beef.*

6.0 (Table 1). Initial pH values for deli meats with and without lactate and/or diacetate were also generally similar. During extended vacuum-packaged storage at 4°C, product pH remained relatively stable, indicating minimal growth of lactic acid bacteria.

Lactate and/or diacetate concentrations in cured turkey, ham, and roast beef are shown in Table 2. Mean concentrations of lactic and acetic acids ranged from 11 to 12 and 0.6 to 1.1 mg/g respectively, which were below the maximum allowable levels of 48 and 2.5% (mg/g). In many cases, inconsistent concentrations of antimicrobials were found between production lots of the same brand, especially for acetic acid; the acetate levels in brand G cured turkey ranging from 0.32 to 1.12 mg/g (mean, 0.62 mg/g). These variations in antimicrobial concentration may account for the variable growth of *L. monocytogenes* and background microflora in different lots of the same product.

Growth characteristics for *L. monocytogenes* in uncured turkey. Growth of *L. monocytogenes* in each brand of deli meat was unaffected by product age. Therefore, the growth data for products inoculated at delivery, the midpoint of the shelf life, and the last allowable day of sale were combined to calculate the LPD, GT, and MPD. The growth curves for *L. monocytogenes* in uncured turkey during storage at 4°C are shown in Figure 1A. Similar *L. monocytogenes* growth curves were obtained for each brand during storage at 7 and 10°C. The LPD for *L. monocytogenes* in different uncured turkey products ranged from 4.00 to 4.93, 1.62 to 3.42, and 1.31 to 1.88 days in product stored at 4, 7, and 10°C, respectively, with GTs at 4, 7, and 10°C ranging from 0.75 to 0.91, 0.32 to 0.53, and 0.29 to 0.32 days, respectively. During storage at 4 to 10°C, *L. monocytogenes* reached MPDs of 8.42 to 10.29 log CFU/g (Table 3). Although LPD and GT were affected by storage temperature with significantly longer ($P < 0.05$) LPDs and GTs at 4°C than at 10°C, similar MPDs were eventually obtained at all three storage temperatures. At 4°C, similar LPDs, GTs, and MPDs were obtained for *L. monocytogenes* in the five brands of uncured turkey, none of which contained inhibitors. Although some variations in LPDs, GTs, and MPDs were seen when these same products were stored at 7 and 10°C, these differences were generally not significant (Table 3).

TABLE 3. *LPD, GT, and MPD of* L. monocytogenes *in uncured turkey (without antimicrobials) stored at 4, 7, and 10°C*[a]

	LPD (days)			GT (days)			MPD (log CFU/g)		
Brand	4°C	7°C	10°C	4°C	7°C	10°C	4°C	7°C	10°C
A	4.06 A	2.63 A	1.71 AB	0.75 A	0.38 BC	0.29 A	9.08 A	9.50 A	10.12 AB
B	4.93 A	2.68 A	1.88 A	0.89 A	0.53 A	0.29 A	8.84 A	8.96 A	8.98 B
C	4.00 A	1.62 A	1.31 B	0.90 A	0.32 C	0.32 A	8.87 A	8.99 A	9.43 AB
D	4.38 A	3.42 A	1.49 AB	0.91 A	0.49 A	0.31 A	8.42 A	8.70 A	10.09 AB
E	4.55 A	2.75 A	1.80 AB	0.78 A	0.44 AB	0.32 A	9.13 A	9.42 A	10.29 A

[a] LPD, lag-phase duration; GT, generation time; MPD, maximum population density. Within the same column, means followed by different letters are significantly different ($P < 0.05$).

TABLE 4. *LPD, GT, and MPD of* L. monocytogenes *in cured turkey stored at 4, 7, and 10°C*[a]

Brand	Inhibitor	LPD (days)			GT (days)			MPD (log CFU/g)		
		4°C	7°C	10°C	4°C	7°C	10°C	4°C	7°C	10°C
F	Yes	7.47 A	3.02 A	2.45 A	1.06 BC	0.52 B	0.35 BC	7.71 BC	9.20 A	8.98 AB
G	Yes	5.66 A	3.42 A	2.36 A	1.31 AB	0.67 B	0.41 B	4.83 D	6.11 B	6.03 C
H	Yes	7.12 A	4.18 A	2.13 A	1.73 A	1.01 A	0.54 A	6.08 CD	6.39 B	6.98 BC
I	No	5.97 A	4.02 A	2.22 A	0.71 C	0.49 B	0.33 C	9.50 A	9.39 A	10.15 A
J	No	5.50 A	3.88 A	2.26 A	0.76 C	0.56 B	0.34 BC	9.30 AB	9.60 A	10.25 A

[a] LPD, lag-phase duration; GT, generation time; MPD, maximum population density. Within the same column, means followed by different letters are significantly different ($P < 0.05$).

TABLE 5. *LPD, GT, and MPD of* L. monocytogenes *in ham stored at 4, 7, and 10°C*[a]

Brand	Inhibitor	LPD (days)			GT (days)			MPD (log CFU/g)		
		4°C	7°C	10°C	4°C	7°C	10°C	4°C	7°C	10°C
K	No	5.96 C	1.85 D	1.80 C	1.05 BC	0.42 C	0.36 C	8.78 A	9.10 A	9.48 A
L	No	6.84 C	2.31 D	2.11 C	1.05 BC	0.54 C	0.37 C	8.02 A	8.52 A	9.76 A
M	Yes	8.87 B	7.21 B	4.74 B	1.55 A	0.90 B	0.55 B	5.05 B	5.74 B	5.92 B
N	Yes	11.13 A	8.83 A	5.91 A	1.43 AB	1.23 A	0.66 A	3.84 B	4.71 B	3.71 C
O	No	6.23 C	3.73 C	2.34 C	0.91 C	0.47 C	0.36 C	9.19 A	9.78 A	9.69 A

[a] LPD, lag-phase duration; GT, generation time; MPD, maximum population density. Within the same column, means followed by different letters are significantly different ($P < 0.05$).

TABLE 6. *LPD, GT, and MPD of* L. monocytogenes *in roast beef stored at 4, 7, and 10°C*[a]

Brand	Inhibitor	LPD (days)			GT (days)			MPD (log CFU/g)		
		4°C	7°C	10°C	4°C	7°C	10°C	4°C	7°C	10°C
P	No	7.54 B	5.03 B	3.39 B	0.80 A	0.62 A	0.45 A	8.04 A	8.66 A	8.45 A
Q	Yes	16.20 AB	12.02 A	13.33 A	−3.72 B	−2.77 B	−2.66 B	1.82 B	1.54 C	1.71 C
R	No	13.96 AB	4.89 B	3.76 B	1.47 A	0.72 A	0.72 A	6.82 A	7.14 A	6.52 AB
S	No	10.02 B	9.46 AB	2.11 B	1.15 A	0.61 A	0.37 A	5.63 A	7.10 A	7.71 AB
T	Yes	20.09 A	7.19 AB	5.05 B	2.11 A	1.19 A	0.57 A	2.51 B	4.67 B	4.50 BC

[a] LPD, lag-phase duration; GT, generation time; MPD, maximum population density. Within the same column, means followed by different letters are significantly different ($P < 0.05$).

Growth characteristics for *L. monocytogenes* in cured turkey. The growth curves for *L. monocytogenes* in cured turkey stored at 4°C are shown in Figure 1B. Similar growth characteristics for each brand were noted for *L. monocytogenes* during storage at 7 and 10°C. However, differences in growth trends were found, ranging from minimal growth in brand G to some growth in brands H and F, all of which contained acetate. *L. monocytogenes* attained the highest populations in brands I and J, which were formulated without *Listeria* growth inhibitors. Significantly longer GTs ($P < 0.05$) and significantly lower MPDs ($P < 0.05$) were found at all three storage temperatures for those brands containing *Listeria* growth inhibitors. Although no significant differences in LPDs were evident among brands, products with antimicrobials had longer *L. monocytogenes* lag times than did those without inhibitors (Table 4).

Growth characteristics for *L. monocytogenes* in ham. The growth curves for *L. monocytogenes* in ham stored at 4°C are shown in Figure 1C. Similar *L. monocytogenes* growth characteristics were seen in ham and cured turkey. The presence of lactate and diacetate significantly extended ($P < 0.05$) the LPDs and GTs and decreased the MPD for *L. monocytogenes*. At 4, 7, and 10°C, LPDs for *L. monocytogenes* were 11.13, 8.83, and 5.91 days in brand N containing *Listeria* growth inhibitors and 5.96, 1.85, and 1.80 days in brand K without inhibitors, respectively. MPDs reached 9 to 10 and 4 to 6 log CFU/g in the absence and presence of *Listeria* growth inhibitors, respectively (Table 5).

Growth characteristics for *L. monocytogenes* in roast beef. The growth curves for *L. monocytogenes* in roast beef during storage at 4°C are shown in Figure 1D. Similar growth curves for each brand were observed for *L. monocytogenes* during storage at 7 and 10°C. Less *L. monocytogenes* growth was seen in roast beef than in uncured turkey, cured turkey, and ham. *L. monocytogenes* populations declined in brand Q, and the pathogen grew poorly in brand T; both of these brands contained *Listeria*

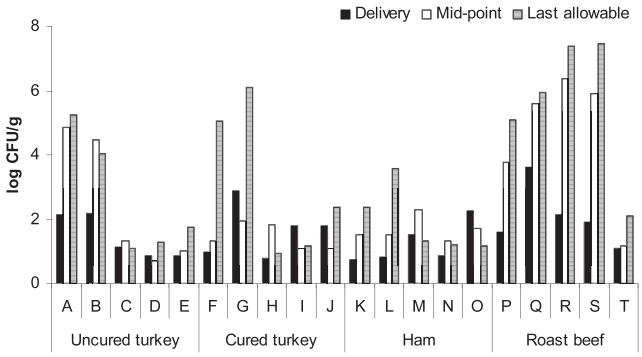

FIGURE 2. *Populations (log CFU per gram) of mesophilic aerobic bacteria (MAB) on five brands of intact chubs of uncured turkey, cured turkey, ham, and roast beef (average of three lots per brand). Vacuum-packaged meats were stored at 4°C, and measurements were taken on the date of delivery, the midpoint of shelf life, and the last allowable day of sale.*

growth inhibitors. Significantly shorter LPDs and GTs ($P < 0.05$) were seen in brands Q, R, and S, which were formulated without *Listeria* growth inhibitors, and these same brands also had significantly higher MPDs ($P < 0.05$) (Table 6).

MAB and lactic acid bacteria. Populations of MAB obtained from intact chubs of deli meat sampled on the day of delivery and after storing at 4°C until the midpoint and last allowable day of sale are shown in Figure 2. MAB increased in some brands of uncured turkey, cured turkey, ham, and in most brands of roast beef during vacuum-packaged storage at 4°C. Most MAB counts were highest at the last allowable day of sale compared with the midpoint and first day of sale (Fig. 2). MAB populations in these deli meats during aerobic storage at 4°C are shown in Figure 3. Similar MAB growth characteristics were observed during storage at 7 and 10°C. Populations of lactic acid bacteria were variable (data not shown), even within samples from the same lot, suggesting that contamination was nonhomogeneous. At any given time point, populations of MAB and presumptive lactic acid bacteria (data not shown) in vacuum-packed and aerobically packaged samples differed, suggesting that some spoilage microflora may not produce acid and therefore may not decrease the product pH. Lactate and diacetate salts were variably effective for controlling MAB, which were a major portion of the background microflora.

DISCUSSION

Although the deli meat category includes a wide range of products, four major types were selected for this study: uncured turkey, cured turkey, ham, and roast beef. These four products, which are the most popular deli meats in the United States, represent a range of formulations, and higher pH products (e.g., uncured turkey, cured turkey, and ham at pH 6.2 to 6.4) allow more rapid growth than do those products with a lower pH (e.g., roast beef at pH 5.4 to 6.0) *(5, 15)*. In this study, the pH of uncured turkey, cured turkey, and ham was close to 6.40, whereas that of roast beef was less than 6.00. The initial pH values for deli meats with and without growth inhibitors were similar.

Delicatessen meats were inoculated on the day of delivery, at the midpoint of the shelf life, and on the last allowable day of sale to evaluate the influence of background microflora on the growth of *L. monocytogenes*. In earlier work, background microflora inhibited growth of *L. monocytogenes (1, 4, 14, 20, 41, 51),* as also found in the present study. Although *Listeria* growth inhibitors were not present in roast beef of brands P, R, and S, slower *L. monocytogenes* growth rates were seen in roast beef than in uncured turkey, cured turkey, and ham manufactured without preservatives. Decreased growth of *L. monocytogenes* in roast beef could be the result of the higher populations of native microflora and lower pH. In previous

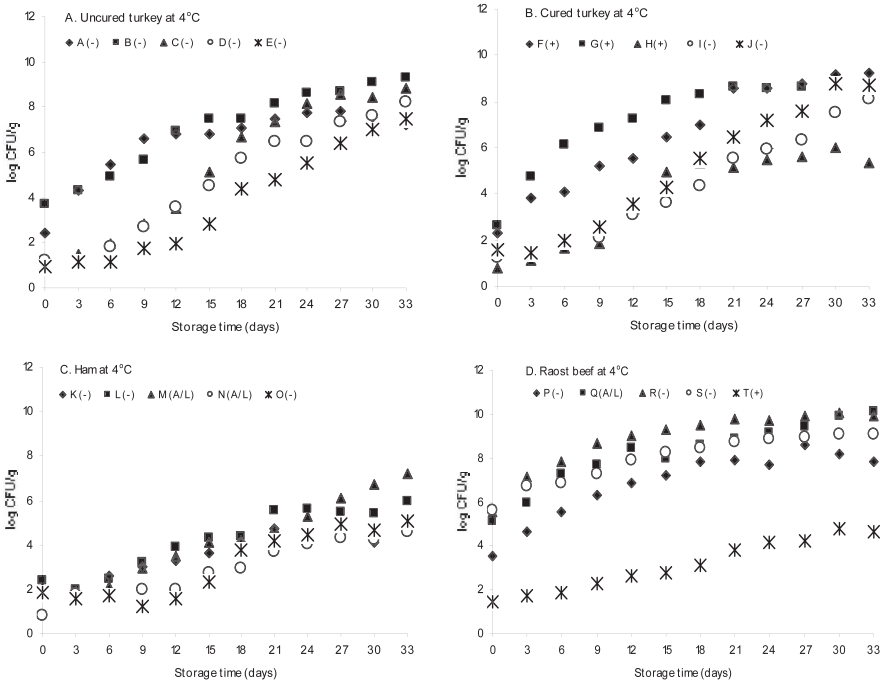

FIGURE 3. *Populations (log CFU per gram) of MAB on 20 brands (A through T, three lots per brand) of sliced inoculated deli meat without antimicrobials (−) or with diacetate (A) or lactate (L). Results are averages on the date of delivery, the midpoint of shelf life, and the last allowable day of sale. Samples were all stored aerobically at 4°C. (A) Uncured turkey; (B) cured turkey; (C) ham; (D) roast beef.*

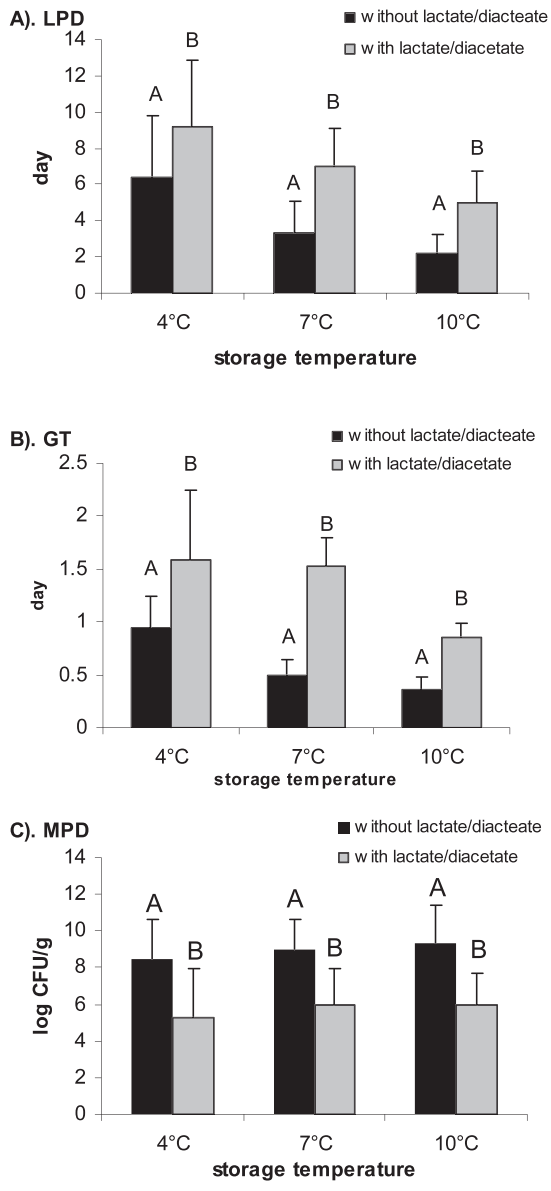

FIGURE 4. *Mean (±standard deviation) LPD, GT, and MPD for* L. monocytogenes *in sliced deli meats with or without lactate and/or diacetate that were stored aerobically at 4, 7, and 10°C.*

studies, lactate and/or diacetate effectively inhibited *L. monocytogenes* growth in RTE foods *(2, 16, 29, 31, 38, 40, 44, 45, 47),* and our results agree with those of these earlier reports. However, we found considerable variation when different production lots of the same product were analyzed for concentrations of lactate and/or diacetate. For example, the concentration of acetate in brand G cured turkey ranged from 0.32 to 1.12 mg/g, with an average of 0.62 mg/g (Table 2). This variation may explain the differences in *L. monocytogenes* growth rates in different lots of the same brand of deli meat. Other researchers have found that these antimicrobials impact the growth of both *L. monocytogenes* and the background microflora *(25, 35, 41)*. In the present study, lactate and diacetate were less inhibitory for the background microflora than for *L. monocytogenes*.

Regardless of the type of deli meat, an increase in storage temperature decreased GT and LPD as expected (Tables 3 through 6). Regardless of storage temperature, LPDs and GTs were also significantly longer ($P < 0.05$) and MPDs were significantly lower ($P < 0.05$) in deli meats with growth inhibitors, indicating that lactate and diacetate effectively limited the growth of *L. monocytogenes* (Fig. 4). However, the extent of inhibition differed among the different types of deli meat (Table 7). Although *L. monocytogenes* declined in roast beef with growth inhibitors, *L. monocytogenes* grew in ham and cured turkey (Table 5). Many factors including the concentration of antimicrobials, storage temperature, product pH, and initial levels/types of background microflora affect the growth characteristics of *L. monocytogenes*. The lower pH values and higher populations of native microflora were likely at least partially responsible for decreased growth of *Listeria* in roast beef.

Cross-contamination of RTE foods with *L. monocytogenes* may occur both at the production and retail levels *(27)*. In a large-scale survey of RTE foods collected from retail markets in the United States, Gombas et al. *(17)* found that in-store–packaged deli meats were more likely to harbor *L. monocytogenes* than were manufacturer-packaged products. Based on more recent retail contamination rates *(12, 38)* and consumer behavior data for deli meats *(6, 37),* approximately 83% of those listeriosis cases and deaths attributed to deli meats were associated with products sliced at retail, where the potential for slicers and other utensils to serve as vehicles for transfer of *L. monocytogenes* has been well established *(19, 22, 23, 28, 37, 42, 43, 52)*.

Various studies have been conducted to evaluate the fate of *L. monocytogenes* during vacuum-packaged storage of RTE foods, but data on the behavior of this pathogen in opened packages during home refrigerated storage conditions are limited *(25, 36)*. Risk assessment data indicate that the incidence of listeriosis can be best reduced by minimizing contamination and growth of *L. monocytogenes* in deli meats *(11, 33)*. In this study, we simulated home use of retail-sliced deli meats to assess the levels of *L. monocytogenes* to which consumers could be exposed. Two and 5 days were needed for *L. monocytogenes* to reach 100 CFU/g in uncured turkey (without lactate and/or acetate) when stored at 10 and 4°C, respectively (Table 7). The risks surrounding products formulated with lactate and diacetate are lower; however, *L. monocytogenes* still can grow in these products, particularly under conditions of temperature abuse. In cured turkey containing *Listeria* inhibitors, 3 to 4 days of storage at 7 to 10°C were required for *L. monocytogenes* to reach 100 CFU/g compared with 8 days when stored at 4°C.

In this study, *L. monocytogenes* growth in retail deli meats varied based on the presence of *Listeria* growth inhibitors, storage temperature, and product type. Diacetate and lactate increased the GTs and lowered the MPDs of *L. monocytogenes*. However, the antimicrobial compounds used, depending on the specific inhibitor(s) and their concentration(s), may be insufficient to completely inhibit *L. monocytogenes* growth *(46)*. For example, growth of *L. monocytogenes* was completely suppressed in one roast beef product (brand Q) that contained both sodium diacetate and potassium lactate (Table 6), whereas *L. monocytogenes*

TABLE 7. *Effect of* Listeria *inhibitors on LPD, GT, MPD, TTR_{100}, and $TTR_{1,000}$ of* L. monocytogenes *in deli meats stored at 4, 7, and 10°C*[a]

Temp (°C)	Deli meat	LPD (days)		GT (days)		MPD (log CFU/g)		TTR_{100} (days)		$TTR_{1,000}$ (days)	
		N[b]	Y[b]	N	Y	N	Y	N	Y	N	Y
4	Uncured turkey[c]	4.34 A		0.74 A		8.86 A		5.11		7.55	
	Cured turkey	5.72 AB	6.39 A	0.83 A	1.52 A	9.38 A	6.68 A	6.76	8.30	9.50	13.31
	Ham	6.34 B	9.54 B	1.00 B	1.30 A	8.66 A	4.56 B	7.33	10.83	10.63	15.12
	Roast beef	10.48 C	16.98 C	1.14 B	2.88 B	7.20 B	1.92 C	11.50	NG[d]	15.26	NG
7	Uncured turkey	2.64 A		0.43 A		9.10 A		3.09		4.51	
	Cured turkey	3.95 B	3.51 A	0.53 B	0.73 A	9.50 A	7.62 A	4.61	4.43	6.36	6.83
	Ham	2.62 A	7.97 A	0.47 BC	1.06 A	9.13 A	5.12 B	3.07	8.98	4.62	12.48
	Roast beef	5.67 C	10.41 B	0.65 C	1.45 B	7.89 B	2.71 C	6.25	NG	8.39	NG
10	Uncured turkey	1.63 A		0.31 A		9.80 A		1.97		2.99	
	Cured turkey	2.24 B	2.31 A	0.34 A	0.43 A	10.21 A	7.45 A	2.68	2.86	3.80	4.28
	Ham	2.10 AB	5.10 B	0.37 A	0.59 A	9.67 A	5.32 B	2.48	5.71	3.70	7.66
	Roast beef	3.34 C	11.26 C	0.53 B	1.74 B	7.86 B	2.41 C	3.81	NG	5.56	NG

[a] LPD, lag-phase duration; GT, generation time; MPD, maximum population density; TTR_{100}, time to reach 100 CFU/g; $TTR_{1,000}$, time to reach 1,000 CFU/g. Within the same column, means followed by different letters are significantly different ($P < 0.05$).
[b] N, *Listeria* growth inhibitors absent; Y, *Listeria* growth inhibitors present.
[c] None of the uncured turkey products contained antimicrobials.
[d] NG, no growth observed.

populations increased in cured turkey, ham, and another brand of roast beef containing either diacetate or diacetate plus lactate. As confirmed by Marklinder et al *(30), L. monocytogenes* growth in beef bologna was delayed during aerobic storage at 5°C when either 2.5% sodium lactate or 0.2% sodium diacetate was added and was completely inhibited when 2.5% sodium lactate and 0.2% sodium diacetate were used in combination. According to Lianou et al. *(26), L. monocytogenes* populations on uncured turkey containing 0.05% sodium diacetate and 1.5% potassium lactate increased from 1.6 to 1.7, to 3.2 to 3.5 log CFU/cm^2 after 12 days of aerobic storage at 7°C. Although significantly lower populations and longer generation times ($P < 0.05$) were observed in deli meats containing growth inhibitors compared with antimicrobial-free products, in most cases *L. monocytogenes* growth was not completely inhibited. Therefore, consumer exposure to potentially hazardous levels of *Listeria* may not be completely avoided under home storage conditions. The National Advisory Committee on Microbiological Criteria for Foods *(32)* has acknowledged this fact by suggesting safety-based date labeling for the control of *L. monocytogenes* in refrigerated RTE products and has stated that use of a safety-based date label for products that support rapid growth of *L. monocytogenes* may have a positive impact on public health. Given the extent of *L. monocytogenes* growth observed in the present study, deli meats without antimicrobials may become potentially hazardous within 5 to 7 days of storage at 4°C and within 2 to 4 days at more abusive temperatures, indicating that the development of safety oriented ''consume by'' dates by deli meat manufacturers should be considered when no antimicrobial agents are added. Given the partial inhibition of *L. monocytogenes* in some deli meats containing preservatives, reliance on these antimicrobial agents to suppress *L. monocytogenes* growth is not without some risk. Repeated opening and closing of the package by consumers during refrigerated storage can also introduce *Listeria* into the product, thereby increasing exposure to levels able to cause infection *(25, 26, 54)*. In one consumer-based study, the majority of participants in Sweden reportedly stored opened packages of ham for 3 to 7 days, with a few individuals storing the product for as long as 2 weeks *(30)*. However, because the infectious dose for *L. monocytogenes* is strongly associated with strain virulence and host susceptibility *(35),* relatively low doses can still cause listeriosis in high-risk populations *(11, 27, 39)*. Because consumers are typically unaware of the presence of *Listeria* growth inhibitors, safety-based date labels should be devised based on worst-case scenarios for growth to levels of no more than 1,000 CFU/g.

Overall, the growth characteristics for *L. monocytogenes* in the deli meats examined during simulated home storage (aerobic storage at 4 to 10°C) depended on the presence of antimicrobials in the formulation, the type of deli meat, the level of background bacteria, and the storage temperature. In general, *L. monocytogenes* grew faster in deli meats without *Listeria* inhibitors (lactate and/or diacetate) than in those with inhibitors, with the growth rates independent of product age but dependent on the type of deli meat and the level of background bacteria. Based on these findings, a ''best consumed by'' date of no more than 5 days after purchase may be advisable for deli meats prepared without *Listeria* growth inhibitors.

ACKNOWLEDGMENT

Financial support for this work was provided by U.S. Department of Agriculture, National Integrated Food Safety Initiative grant 2005-51110-02354.

REFERENCES

1. Amézquita, A., and M. M. Brashears. 2002. Competitive inhibition of *Listeria monocytogenes* in ready-to-eat meat products by lactic acid bacteria. *J. Food Prot.* 65:316–325.
2. Bedie, G. K., J. Samelis, J. N. Sofos, K. E. Belk, J. A. Scanga, and G. C. Smith. 2001. Antimicrobials in the formulation to control *Listeria monocytogenes* postprocessing contamination on frankfurters stored at 4°C in vacuum packages. *J. Food Prot.* 64:1949–1955.
3. Beumer, R. R., M. C. te Giffel, E. de Boer, and F. M. Rombouts. 1996. Growth of *Listeria monocytogenes* on sliced cooked meat products. *Food Microbiol.* 13:333–340.
4. Bredholt, S., T. Nesbakken, and A. Holck. 2001. Industrial application of an antilisterial strain of *Lactobacillus sakei* as a protective culture and its effect on the sensory acceptability of cooked, sliced, vacuum-packaged meats. *Int. J. Food Microbiol.* 66: 191–196.
5. Burnett, S. L., E. L. Mertz, B. Bennie, T. Ford, and A. Starobin. 2005. Growth or survival of *Listeria monocytogenes* in ready-to-eat meat products and combination deli salads during refrigerated storage. *J. Food Sci.* 70:M301–M304.
6. Cates, S. C., R. A. Morales, S. A. Karns, L.-A. Jaykus, K. M. Kosa, T. Teneyck, C. M. Moore, and P. Cowen. 2006 Consumer knowledge, storage, and handling practices regarding *Listeria* in frankfurters and deli meats: results of a web-based survey. *J. Food Prot.* 69:1630–1639.
7. Centers for Disease Control and Prevention. 1999. Update: multistate outbreak of listeriosis—United States, 1998–1999. *Morb. Mortal. Wkly. Rep.* 47:1117–1118.
8. Centers for Disease Control and Prevention. 2000. Multistate outbreak of listeriosis—United States, 2000. *Morb. Mortal. Wkly. Rep.* 49:1129–1130.
9. Centers for Disease Control and Prevention. 2002. Public health dispatch: outbreak of listeriosis—northeastern United States, 2002. *Morb. Mortal. Wkly. Rep.* 51:950–951.
10. Chen, Y., W. H. Ross, M. J. Gray, M. Wiedmann, R. Whiting, and V. N. Scott. 2006. Attributing risk to *Listeria monocytogenes* subgroups: dose response in relation to genetic lineages. *J. Food Prot.* 69:335–344.
11. Chen, Y., W. H. Ross, V. N. Scott, and D. E. Gombas. 2003. *Listeria monocytogenes*: low levels equal low risk. *J. Food Prot.* 66:570–577.
12. Draughon, A. F. 2006. A collaborative analysis/risk assessment of *Listeria monocytogenes* in ready-to-eat processed meat and poultry collected in four FoodNet states. Symposium S-16. Presented at the 93rd Annual Meeting of the International Association for Food Protection, Calgary, Alberta, Canada, 13 to 16 August 2006.
13. Gilbert, S., R. Lake, A. Hudson, and P. Cressey. 2009. Risk profile: *Listeria monocytogenes* in processed ready-to-eat meats. New Zealand Food Safety Authority report. Available at: http://www.foodsafety.govt.nz/elibrary/industry/Risk_Profile_Listeria_Monocytogenes_Processed-Science_Research.pdf. Accessed 24 August 2011.
14. Giménez, B., and P. Dalgaard. 2004. Modelling and predicting the simultaneous growth of *Listeria monocytogenes* and spoilage micro-organisms in cold-smoked salmon. *J. Appl. Microbiol.* 96:96–109.
15. Glass, K. A., and M. P. Doyle. 1989. Fate of *Listeria monocytogenes* in processed meat products during refrigerated storage. *Appl. Environ. Microbiol.* 55:1565–1569.
16. Glass, K. A., D. A. Granberg, A. L. Smith, A. M. McNamara, M. Hardin, J. Mattias, K. Ladwig, and E. A. Johnson. 2002. Inhibition of *Listeria monocytogenes* by sodium diacetate and sodium lactate on wieners and cooked bratwurst. *J. Food Prot.* 65:116–123.
17. Gombas, D. E., Y. Chen, R. S. Clavero, and V. N. Scott. 2003. Survey of *Listeria monocytogenes* in ready-to-eat foods. *J. Food Prot.* 66:559–569.
18. Gottlieb, S. L., E. C. Newbern, P. M. Griffin, L. M. Graves, R. M. Hoekstra, N. L. Baker, S. B. Hunter, K. G. Holt, F. Ramsey, and M. Head. 2006. Multistate outbreak of listeriosis linked to turkey deli meat and subsequent changes in US regulatory policy. *Clin. Infect. Dis.* 42:29–36.
19. Humphrey, T. J., and D. M. Worthington. 1990. *Listeria* contamination of retail meat slicers. *PHLS Microbiol. Dig.* 7(3):57.
20. Hwang, C. A., and S. Sheen. 2010. Growth characteristics of *Listeria monocytogenes* as affected by a native microflora in cooked ham under refrigerated and temperature abuse conditions. *Food Microbiol.* 28:350–355.
21. International Life Science Institute Research Foundation, Risk Science Institute. 2005. Achieving continuous improvement in reductions in foodborne listeriosis—a risk-based approach. *J. Food Prot.* 68:1932–1994.
22. Keskinen, L. A., E. C. D. Todd, and E. T. Ryser. 2008. Transfer of surface-dried *Listeria monocytogenes* from stainless steel knife blades to roast turkey breast. *J. Food Prot.* 71:176–181.
23. Keskinen, L. A., E. C. D. Todd, and E. T. Ryser. 2008. Impact of bacterial stress and biofilm-forming ability on transfer of surface-dried *Listeria monocytogenes* during slicing of delicatessen meats. *Int. J. Food Microbiol.* 127:298–304.
24. Lake, R., A. Hudson, P. Cressey, and G. Nortje. 2002. Risk profile: *Listeria monocytogenes* in processed ready-to-eat meats. New Zealand Food Safety Authority report. Available at: http://www.scribd.com/doc/29277860/Listeria-in-Rte-Meat. Accessed 24 August 2011.
25. Lianou, A., I. Geornaras, P. A. Kendall, K. E. Belk, J. A. Scanga, G. C. Smith, and J. N. Sofos. 2007. Fate of *Listeria monocytogenes* in commercial ham, formulated with or without antimicrobials, under conditions simulating contamination in the processing or retail environment and during home storage. *J. Food Prot.* 70:378–385.
26. Lianou, A., I. Geornaras, P. A. Kendall, J. A. Scanga, and J. N. Sofos. 2007. Behavior of *Listeria monocytogenes* at 7°C in commercial turkey breast, with or without antimicrobials, after simulated contamination for manufacturing, retail and consumer settings. *Food Microbiol.* 24:433–443.
27. Lianou, A., and J. N. Sofos. 2007. A review of the incidence and transmission of *Listeria monocytogenes* in ready-to-eat products in retail and food service environments. *J. Food Prot.* 70:2172–2198.
28. Lin, C. M., K. Takeuchi, L. Zhang, C. B. Dohm, J. D. Meyer, P. A. Hall, and M. P. Doyle. 2006. Cross-contamination between processing equipment and deli meats by *Listeria monocytogenes*. *J. Food Prot.* 69:71–79.
29. Luchansky, J. B., G. Cocoma, and J. E. Call. 2006. Hot water postprocess pasteurization of cook-in-bag turkey breast treated with and without potassium lactate and sodium diacetate and acidified sodium chlorite for control of *Listeria monocytogenes*. *J. Food Prot.* 69:39–46.
30. Marklinder, I. M., M. Lindblad, L. M. Eriksson, A. M. Finnson, and R. Lindqvist. 2004. Home storage temperatures and consumer handling of refrigerated foods in Sweden. *J. Food Prot.* 67:2570–2577.
31. Mbandi, E., and L. A. Shelef. 2002. Enhanced antimicrobial effects of combination of lactate and diacetate on *Listeria monocytogenes* and *Salmonella* spp. in beef bologna. *Int. J. Food Microbiol.* 76:191–198.
32. National Advisory Committee on Microbiological Criteria for Foods. 2005. Considerations for establishing safety-based consume-by date labels for refrigerated ready-to-eat foods. *J. Food Prot.* 68:1761–1775.
33. Nørrung, B., and J. K. Andersen. 2000. Variations in virulence between different electrophoretic types of *Listeria monocytogenes*. *Lett. Appl. Microbiol.* 30:228–232.
34. Olsen, S. J., M. Patrick, S. B. Hunter, V. Reddy, L. Kornstein, W. R. MacKenzie, K. Lane, S. Bidol, G. A. Stoltman, D. M. Frye, I. Lee, S. Hurd, T. F. Jones, T. N. LaPorte, W. Dewitt, L. Graves, M. Wiedmann, D. J. Schoonmaker-Bopp, A. J. Huang, C. Vincent, A. Bugenhagen, J. Corby, E. R. Carloni, M. E. Holcomb, R. F. Woron, S. M. Zansky, G. Dowdle, F. Smith, S. Ahrabi-Fard, A. R. Ong, N. Tucker, N. A. Hynes, and P. Mead. 2005. Multistate outbreak of *Listeria monocytogenes* infection linked to delicatessen turkey meat. *Clin. Infect. Dis.* 40:962–967.
35. Pal, A., T. P. Labuza, and F. Diez-Gonzalez. 2008. Evaluating the growth of *Listeria monocytogenes* in refrigerated ready-to-eat frankfurters: influence of strain, temperature, packaging, lactate and diacetate, and background microflora. *J. Food Prot.* 71:1806–1816.
36. Pal, A., T. P. Labuza, and F. Diez-Gonzalez. 2008. Shelf life evaluation for ready-to-eat sliced uncured turkey breast and cured

ham under probable storage conditions based on *Listeria monocytogenes* and psychrotroph growth. *Int. J. Food Microbiol.* 126:49–56.
37. Pradhan, A. K., R. Ivanek, Y. T. Gröhn, R. Bukowski, I. Geornaras, J. N. Sofos, and M. Wiedmann. 2010. Quantitative risk assessment of listeriosis-associated deaths due to *Listeria monocytogenes* contamination of deli meats originating from manufacture and retail. *J. Food Prot.* 73:620–630.
38. Pradhan, A. K., R. Ivanek, Y. T. Gröhn, I. Geornaras, J. N. Sofos, and M. Wiedmann. 2009. Quantitative risk assessment for *Listeria monocytogenes* in selected categories of deli meats: impact of lactate and diacetate on listeriosis cases and deaths. *J. Food Prot.* 72:978–989.
39. Quesenberry, H. H., D. Gallagher, S. Endrikat, D. LaBarre, E. Ebel, C. Schroeder, and J. Kause. 2010. Final FSIS comparative risk assessment for *Listeria monocytogenes* in ready-to-eat meat and poultry deli meats report. Available at: http://www.fsis.usda.gov/PDF/Comparative_RA_Lm_Report_May2010.pdf. Accessed 24 August 2011.
40. Samelis, J., G. K. Bedie, J. N. Sofos, K. E. Belk, J. A. Scanga, and G. C. Smith. 2005. Combinations of nisin with organic acids or salts to control *Listeria monocytogenes* on sliced pork bologna stored at 4°C in vacuum packages. *Lebensm.-Wiss. Technol.* 38:21–28.
41. Samelis, J., A. Kakouri, and J. Rementzis. 2000. Selective effect of the product type and the packaging conditions on the species of lactic acid bacteria dominating the spoilage microbial associated of cooked meats at 4°C. *Food Microbiol.* 17:329–340.
42. Sheen, S. 2008. Modeling surface transfer of *Listeria monocytogenes* on salami during slicing. *J. Food Sci.* 73:E304–E311.
43. Sheen, S., and C.-A. Hwang. 2008. Modeling transfer of *Listeria monocytogenes* from slicer to deli meat during mechanical slicing. *Foodborne Pathog. Dis.* 5:135–146.
44. Stekelenburg, F. K. 2003. Enhanced inhibition of *Listeria monocytogenes* in frankfurter sausage by the addition of potassium lactate and sodium diacetate mixtures. *Food Microbiol.* 20:133–137.
45. Stekelenburg, F. K., and M. L. T. Kant-Muermans. 2001. Effects of sodium lactate and other additives in a cooked ham product on sensory quality and development of a strain of *Lactobacillus curvatus* and *Listeria monocytogenes*. *Int. J. Food Microbiol.* 66:197–203.
46. Thompson, R. L., C. E. Carpenter, S. Martini, and J. R. Broadbent. 2008. Control of *Listeria monocytogenes* in ready-to-eat meats containing sodium levulinate, sodium lactate, or a combination of sodium lactate and sodium diacetate. *J. Food Sci.* 73:M239–M244.
47. Uhart, M., S. Ravishankar, and N. D. Maks. 2004. Control of *Listeria monocytogenes* with combined antimicrobials on beef franks stored at 4°C. *J. Food Prot.* 67:2296–2301.
48. U.S. Department of Agriculture, Food Safety and Inspection Service. 2000. Food additives for use in meat and poultry products: sodium diacetate, sodium acetate, sodium lactate and potassium lactate. *Fed. Regist.* 65:3121–3123.
49. U.S. Department of Agriculture, Food Safety and Inspection Service. 2004. Control of *Listeria monocytogenes* in ready-to-eat poultry and meat products. *Fed. Regist.* 69:70051–70053.
50. U.S. Food and Drug Administration, Center for Food Safety and Applied Nutrition. 2003. Quantitative assessment of relative risk to public health from foodborne *Listeria monocytogenes* among selected categories of ready-to-eat foods. Available at: http://www.fda.gov/downloads/food/scienceresearch/researchareas/riskassessmentsafetyassessment/ucm197330.pdf. Accessed 24 August 2011.
51. Vermeiren, L., F. Devlieghere, and J. Debevere. 2004. Evaluation of meat born lactic acid bacteria as protective cultures for the biopreservation of cooked meat products. *Int. J. Food Microbiol.* 96:149–164.
52. Vorst, K. L., E. C. D. Todd, and E. T. Ryser. 2006. Transfer of *Listeria monocytogenes* during mechanical slicing of turkey breast, bologna, and salami. *J. Food Prot.* 69:619–626.
53. Wijtzes, T., J. C. de Wit, J. H. J. Huis in't Veld, K. van't Riet, and M. H. Zwietering. 1995. Modelling bacterial growth of *Lactobacillus curvatus* as a function of acidity and temperature. *Appl. Environ. Microbiol.* 61:2533–2539.
54. Yang, H., A. Mokhtari, L. A. Jaykus, R. A. Morales, S. C. Cates, and P. Cowen. 2006. Consumer phase risk assessment for *Listeria monocytogenes* in deli meats. *Risk Anal.* 26:89–103.
55. Zwietering, M. H., I. Jongenburger, F. M. Rombouts, and K. van't Riet. 1990. Modeling of the bacterial growth curve. *Appl. Environ. Microbiol.* 56:1875–1881.