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Growth characteristics of *Listeria monocytogenes* as affected by a native microflora in cooked ham under refrigerated and temperature abuse conditions☆

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ABSTRACT

This study examined the growth characteristics of *Listeria monocytogenes* as affected by a native microflora in cooked ham at refrigerated and abuse temperatures. A five-strain mixture of *L. monocytogenes* and a native microflora, consisting of *Brochothrix* spp., isolated from cooked meat were inoculated alone (monocultured) or co-inoculated (co-cultured) onto cooked ham slices. The growth characteristics, lag phase duration (LPD, h), growth rate (GR, $\log_{10}$ cfu/h), and maximum population density (MPD, $\log_{10}$ cfu/g), of *L. monocytogenes* and the native microflora in vacuum-packed ham slices stored at 4, 6, 8, 10, and 12 °C for up to 5 weeks were determined. At 4–12 °C, the LPDs of co-cultured *L. monocytogenes* were not significantly different from those of monocultured *L. monocytogenes* in ham, indicating the LPDs of *L. monocytogenes* at 4–12 °C were not influenced by the presence of the native microflora. At 4–8 °C, the GRs of co-cultured *L. monocytogenes* (0.0114–0.0130 $\log_{10}$ cfu/h) were statistically but marginally lower than those of monocultured *L. monocytogenes* (0.0132–0.0145 $\log_{10}$ cfu/h), indicating the GRs of *L. monocytogenes* at 4–8 °C were reduced by the presence of the native microflora. The GRs of *L. monocytogenes* were reduced by 8–7% with the presence of the native microflora at 4–8 °C, whereas there was less influence of the native microflora on the GRs of *L. monocytogenes* at 10 and 12 °C. The MPDs of *L. monocytogenes* at 4–8 °C were also reduced by the presence of the native microflora. Data from this study provide additional information regarding the growth suppression of *L. monocytogenes* by the native microflora for assessing the survival and growth of *L. monocytogenes* in ready-to-eat meat products.

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Refrigerated ready-to-eat (RTE) meat products contaminated with *Listeria monocytogenes* have been linked to several listeriosis outbreaks (CDC, 1998, 1999, 2002). *L. monocytogenes* is frequently isolated from RTE meat products. In 2300 sliced luncheon meat samples collected from federally-inspected establishments between 1990 and 1999 in the U.S., *L. monocytogenes* was present in 4.2–8.0% of the samples (Levine et al., 2001). *L. monocytogenes* was present in 0.89% (82/9199) RTE luncheon meat samples collected in California and Maryland in 2000–2001 (Gombas et al., 2003). In a study examining the prevalence of *L. monocytogenes* in packages of frankfurters obtained from several commercial manufacturers over a 2-year period, Wallace et al. (2003) reported that 1.6% (532/32,800) of the packages were positive for *L. monocytogenes*. The possible contamination of *L. monocytogenes* in RTE meat, beef and chicken products, such as corned beef, pastrami, and frankfurters, is one of the main reasons for Class I type food recalls in the U.S. (FSIS, 2005). A survey conducted in Belgium between 1997 and 1998 showed that 4.9% (167/3405) of retail cooked meat products were contaminated with *L. monocytogenes*, and a higher incidence rate was found in cooked meat products after slicing (6.65%) compared to meat before slicing (1.56%) (Uyttendaele et al., 1999). In the U.S., a listeriosis outbreak that occurred in 1998 caused 40 illnesses in 10 states and was linked to the consumption of *L. monocytogenes*-contaminated frankfurters (CDC, 1998). Another outbreak in 2002 that caused seven deaths, three stillbirths or miscarriages, and a recall of 12.4 million kg of implicated products was linked to the consumption of turkey deli meat (CDC, 2002). Based on a risk assessment report (U.S. FDA/USDA/CDC, 2003), deli meats had the highest estimated per annum risk of illness and death from *L. monocytogenes* among 20 RTE food categories. While heat processing during the manufacturing of RTE meat products is sufficient to eliminate microorganisms in the meat products,

☆ Mention of trade names or commercial products in this article is solely for the purpose of providing specific information and does not imply recommendation or endorsement by the U.S. Department of Agriculture.

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L. monocytogenes and other microorganisms may re-contaminate the products during post-heat processing operations such as slicing, packaging or handling by the manufacturer, or at retail, or by the consumer. *L. monocytogenes* is a psychrotrophic microorganism; therefore it is able to grow at refrigeration temperature. Since RTE meat products are normally consumed without prior cooking, the ability of *L. monocytogenes* to grow at refrigeration temperature is a particular concern for refrigerated RTE meat (Amezquita and Brashears, 2002). Studies have been conducted to examine the survival and growth of *L. monocytogenes* in RTE meat as affected by food additives, product formulations, and storage conditions (Samelis et al., 2001, 2005; Stekelenburg and Kant-Muermans, 2001; Glass et al., 2002; Mbandi and Shelef, 2002; Stekelenburg, 2003; Uhart et al., 2004; Luchansky et al., 2006). In addition, mathematical models have been developed to describe the behavior of *L. monocytogenes* in RTE meat products with intrinsic and extrinsic parameters such as pH/acids, a_w/salt, nitrite, polyphosphate, lactate, and diacetate, package atmosphere, and storage temperature (Devlieghere et al., 2001; Seman et al., 2002; Legan et al., 2004; Hwang and Tamplin, 2007). While data on the inactivation, growth, or survival of *L. monocytogenes* in RTE meat products as affected by food additives, product formulation, and storage conditions are readily available, data on the behavior of *L. monocytogenes* as affected by the native microflora of RTE meat products are limited. Examining the behavior of *L. monocytogenes* in RTE meat products with the presence of native microorganisms is important since these products generally contain native microorganisms. The objectives of this study were to examine and describe the growth characteristics of *L. monocytogenes* as affected by a native microflora in cooked ham at refrigerated and abuse temperatures.

1. Materials and methods

1.1. L. monocytogenes inoculum preparation

Five strains of *L. monocytogenes* from the culture collection of the Microbial Food Safety Research Unit, Eastern Regional Research Center, Agricultural Research Service, U.S. Department of Agriculture, were used in this study. These strains were MFS 2 (serotype 1/2a, an environmental isolate from a pork processing plant), H7776 (4b, frankfurter isolate), Scott A (4b, a clinical isolate from a 1983 Massachusetts outbreak linked to pasteurized milk), 101M (4b, beef and pork sausage isolate), and F2365 (Hispanic-style cheese isolate). Each strain was transferred from a stock culture kept at −80 °C into 10 ml of brain heart infusion (BHI) broth (Difco, Becton, Dickinson and Company, Sparks, Maryland) and incubated at 35 °C overnight. A loopful of the cell suspension of each strain was then transferred into another 10 ml BHI broth and incubated at 35 °C for 24 h. One ml of cell suspension from each strain was mixed together, and the mixture was serially diluted in sterile 0.1% peptone water (PW) to obtain an inoculum with *L. monocytogenes* at 10^{2-3} $\log_{10}$ cfu/ml.

1.2. Native microflora inoculum preparation

Five RTE ham products of different varieties from different manufacturers were obtained from a local retail store. These products included varieties with product descriptions such as water-added, low fat, smoked, cured, or natural flavors. The ingredients used in the five products were similar with ingredients such as pork, salt, phosphate, nitrite, and sugar, which are commonly found in RTE meat products. Duplicate 10-g samples from each product were diluted with 40 ml of PW and three serial 1:10 dilutions were made in PW. Duplicate 0.1 ml aliquots from each dilution were spread-plated onto plate count agar (PCA, Difco), and the plates were incubated at 35 °C for 48 h. Colonies that appeared on the plates with the highest dilution factor were presumed to be the predominant native (background) microorganisms in each ham product. Two isolates which were common to three of the ham products were subcultured in tryptic soy broth (TSB, Difco). Each isolate was inoculated into 10 ml TSB broth, and incubated at 35 °C overnight. One ml of the cell suspension from each isolate was combined, and the mixture was serially diluted in PW to obtain inocula with populations of 10^{2-3} $\log_{10}$ cfu/ml. The isolates were initially identified and later confirmed as *Brochothrix* spp. using the API 50 CHB kit by a commercial laboratory.

1.3. Sample preparation and storage

A restructured cooked ham product was obtained from a local processing plant. The ham product had a pH of 6.8, a_w 0.98, 75% moisture, 3.1% fat, 17.5% protein, 2.0% salt, and <0.1 μg/g nitrite. The ham was cut into 3 × 3 cm blocks, and the blocks were pasteurized in a water bath at 65 °C (product temperature) for 30 min to inactivate background microorganisms. Pasteurization was performed to inactivate the background microorganisms to ensure that the ham samples had a similar microbial count. The ham was aseptically cut into slices with a thickness of 1–2 mm. One side of the ham slices (3–4 g/slice) was inoculated with 0.05 ml of the *L. monocytogenes* or native microflora inoculum (monoculture), or with a mixture of both microflora (co-culture). Inoculated ham slices were placed into 100-ml stomacher bags (Spiral Biotech, Inc., Norwood, Massachusetts), and the bags were vacuum-sealed to 60 mbar using a Multivac A300 vacuum sealer (Multivac Inc., Kansas, Missouri). The inoculum level of each bacterial preparation on the ham was 10^{1-2} cfu/g. The samples were stored at 4, 6, 8, 10, and 12 °C for up to 5 weeks. During storage, two samples from each storage temperature were enumerated for counts of *L. monocytogenes* and native microflora. The sampling frequency was every 1–2 days at the beginning of storage, and every 2–4 days after growth was observed. The experiment was performed in two separate trials with the whole experiment started with inoculum cultivation, ham preparation, inoculation, storage and sampling.

1.4. Enumeration of L. monocytogenes and native microflora

To enumerate *L. monocytogenes* and native microflora in ham on the sampling days, an equal amount of sterile 0.1% PW (3–4 ml) to the samples was added into sample bags, and the bags were pummeled in a BagMixer 400 (Interscience Laboratories Inc., Weymouth, Massachusetts) for 2 min. The homogenates were serially 10-fold diluted in 0.1% PW. Duplicate 0.1-ml aliquots from appropriate dilutions were spread-plated onto PALCAM agar (Difco) plates for *L. monocytogenes* and plate count agar (PCA, Difco) plates for the native microflora. Typical colonies of *L. monocytogenes* and the native microflora that formed on PALCAM and PCA after 48 h incubation at 35 °C were counted.

1.5. Growth parameters of L. monocytogenes and native microflora in ham

The populations of *L. monocytogenes* and native microflora ($\log_{10}$ cfu/g) in ham during storage at 4, 6, 8, 10, and 12 °C were fitted with the three-phase linear model of Buchanan et al. (1997) to obtain the lag phase duration (LPD, h), growth rate (GR, $\log_{10}$ cfu/h), and maximum population density (MPD, $\log_{10}$ cfu/g):

Lag phase: For $t \leq t_{lag}$, $N_t = N_0$,
Exponential growth phase: For $t_{lag} < t < t_{max}$, GR $= (N_t - N_0)/(t - t_{lag})$,
Stationary phase: For $t \geq t_{max}$, $N_t = N_{max}$,

where N_0 is the initial populations ($\log_{10}$ cfu/g), N_t is the bacterial populations at time t (h), N_{max} is the maximum bacterial populations, t_{lag} is the lag phase duration (h), t_{max} is the time required for bacterial populations to reach the maximum (N_{max}), and GR is the growth rate during the exponential growth phase. The GR is the slope of the data points (counts vs. time) in the exponential growth phase. The points selected for calculating GR were those with a linear regression (correlation) coefficient (r^2) > 0.95. The Tukey mean comparison test (SAS 9.1, SAS Institute, Cary, North Carolina) with 95% significance level was used to compare the means of GRs, LPDs, and MPDs of *L. monocytogenes* and native microflora, monocultured and co-cultured, at each storage temperature.

2. Results

2.1. Growth of L. monocytogenes and native microflora in ham

The growth curves of monocultures and the *L. monocytogenes* and native microflora co-cultured in ham during storage at 4, 8, 10, and 12 °C are shown in Fig. 1A–D. The pH of samples at the end of storage were 0.2–0.4 lower than the initial sample pH. The growth kinetics of *L. monocytogenes* and native microflora at 4 and 6 °C were similar, therefore, only the growth curves at 4 °C are presented. The initial populations of *L. monocytogenes* and native microflora in ham were approximately 1.5–1.8 $\log_{10}$ cfu/g. The population of monocultured *L. monocytogenes* increased to approximately 6.0 $\log_{10}$ cfu/g after 17, 13, 10, and 8 days at 4, 8, 10, and 12 °C, respectively. When co-inoculated with the native microflora, the times for *L. monocytogenes* counts to increase to 6.0 $\log_{10}$ cfu/g were 22 day at 4 °C, and 18 day at 8 °C. The results showed that the growth of *L. monocytogenes* in ham at 4 and 8 °C was slower when the native microflora was present. Regardless of the storage temperatures, populations of *L. monocytogenes* and the native microflora (monoculture or co-culture) increased during storage. At 4 °C, the population increase of monocultured *L. monocytogenes* was faster than that of monocultured native microflora, whereas the population increase of *L. monocytogenes* was similar to the native microflora when both were co-inoculated in ham (Fig. 1A). At 8 °C, the growth kinetics of *L. monocytogenes* and native microflora were similar in ham when they were inoculated alone, while the growth kinetics of *L. monocytogenes* was slower than that of the native microflora when both were co-inoculated in ham (Fig. 1B). Regardless of whether monocultured or co-cultured the growth of *L. monocytogenes* was similar to the native microflora at both 10 and 12 °C (Fig. 1C and D). These results indicated that the growth of *L. monocytogenes* in ham was influenced by the presence of the native microflora at 4–8 °C.

2.2. Growth characteristics of L. monocytogenes and native microflora in ham

The LPDs of monocultured *L. monocytogenes* were 58, 51, 42, 40, and 38 h in ham stored at 4, 6, 8, 10, and 12 °C, respectively (Table 1).

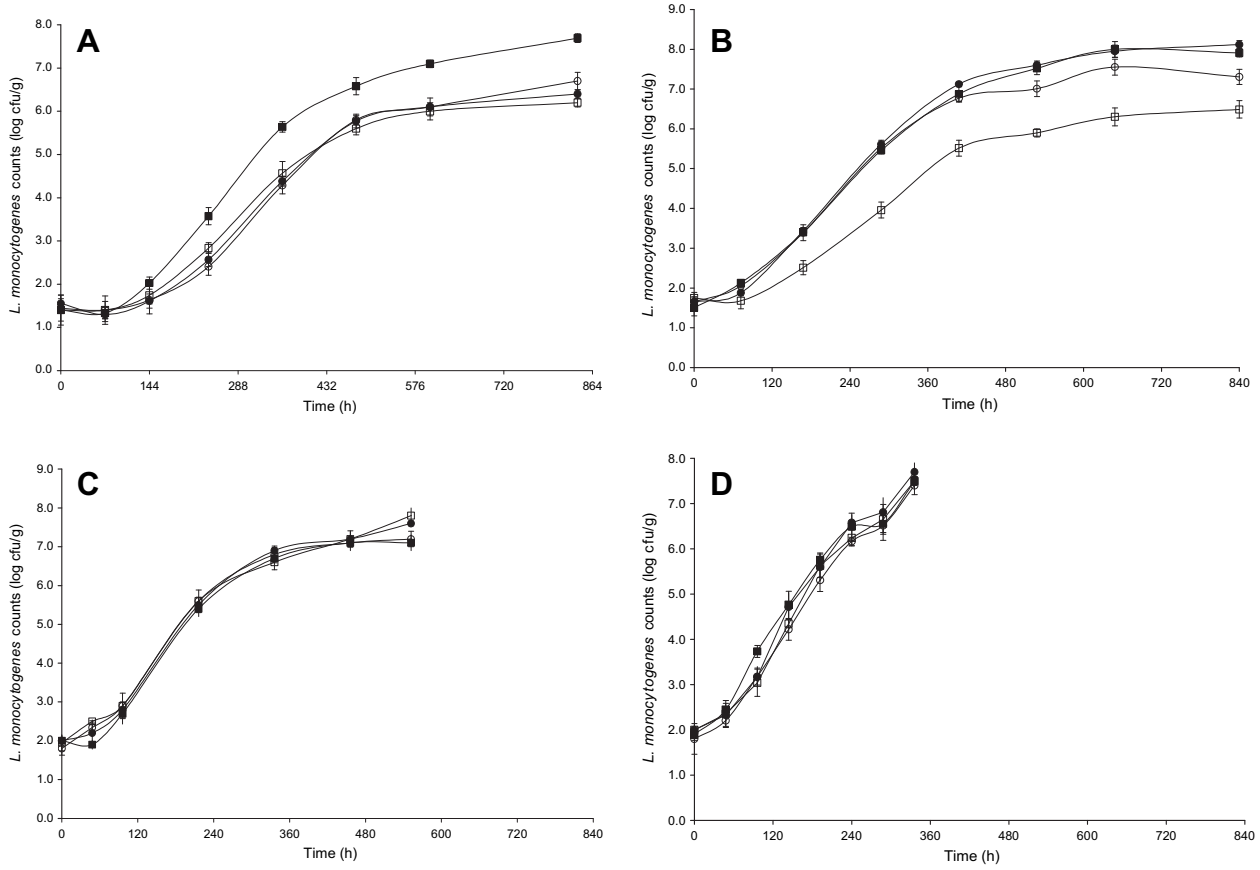

Fig. 1. Growth of monocultured *L. monocytogenes* (■) and native microflora (●), and co-cultured *L. monocytogenes* (□) and native microflora (○) during storage at 4 (A), 8 (B), 10 (C), and 12 °C (D).

Table 1
Means of LPD, GR, and MPD (standard deviation) of monocultured *L. monocytogenes* (*Lm*) and native microflora (NA), and co-cultured *L. monocytogenes* (*Lm*+) and native microflora (NA+) in ham stored at 4–12 °C.

Temperature (°C)	Microflora	LPD	GR	MPD
4	*Lm*	58 (3)	0.0132 (0.0010)	7.7 (0.2)
	Lm+	62 (3)	0.0114 (0.0008)	6.5 (0.1)
6	*Lm*	51 (8)	0.0135 (0.0011)	7.4 (0.2)
	Lm+	56 (7)	0.0121 (0.0009)	6.8 (0.3)
8	*Lm*	42 (2)	0.0145 (0.0010)	8.0 (0.1)
	Lm+	55 (1)	0.0130 (0.0004)	6.7 (0.3)
10	*Lm*	40 (3)	0.0169 (0.0012)	8.0 (0.1)
	Lm+	42 (4)	0.0162 (0.0008)	8.1 (0.2)
12	*Lm*	38 (2)	0.0248 (0.0005)	7.7 (0.2)
	Lm+	40 (1)	0.0249 (0.0003)	7.7 (0.2)
4	NA	75 (5)	0.0146 (0.0011)	6.5 (0.1)
	NA+	76 (8)	0.0136 (0.0019)	6.8 (0.2)
6	NA	64 (2)	0.0149 (0.0004)	7.6 (0.1)
	NA+	69 (8)	0.0140 (0.0003)	6.9 (0.2)
8	NA	50 (4)	0.0160 (0.0011)	8.1 (0.3)
	NA+	55 (1)	0.0153 (0.0007)	7.5 (0.2)
10	NA	31 (2)	0.0166 (0.0003)	8.2 (0.2)
	NA+	35 (3)	0.0165 (0.0003)	7.9 (0.2)
12	NA	37 (8)	0.0260 (0.0002)	7.8 (0.1)
	NA+	36 (6)	0.0257 (0.0004)	7.4 (0.1)

When co-cultured with the native microflora, the LPDs of *L. monocytogenes* were 62, 56, 55, 42, and 32 h. At 4–12 °C, the LPDs of co-cultured *L. monocytogenes* were not significantly different from those of monocultured *L. monocytogenes*. The LPDs of co-cultured native microflora were not different from those of monocultured native microflora. The results indicated that the LPDs of *L. monocytogenes* in ham at 4–12 °C were not influenced by the presence of the native microflora.

The GRs for monocultured *L. monocytogenes* and the native microflora in ham at 4–12 °C were 0.0132–0.0248 $\log_{10}$ cfu/h and 0.0146–0.0260 $\log_{10}$ cfu/g, respectively (Table 1). In co-cultures at 4–12 °C, the GRs for *L. monocytogenes* and native microflora were 0.0114–0.0249 $\log_{10}$ cfu/h and 0.0136–0.0257 $\log_{10}$ cfu/h, respectively. The GRs of monocultured or co-cultured native microflora were significantly higher ($p < 0.05$) than those of *L. monocytogenes* at 4, 6, 8 and 12 °C. At 4–8 °C, the GRs of co-cultured *L. monocytogenes* were significantly (0.0114–0.0130 $\log_{10}$ cfu/h) lower than monocultured *L. monocytogenes* (0.0132–0.0145 $\log_{10}$ cfu/h) in ham, indicating the GRs of *L. monocytogenes* at 4–8 °C were reduced by the presence of the native microflora. The GRs of co-cultured *L. monocytogenes* or the native microflora in ham stored at 4–8 °C were significantly lower than those of monocultured *L. monocytogenes* or native microflora, indicating that the GRs of *L. monocytogenes* or the native microflora were affected by the presence of the other microflora. While the GRs between the co-cultured and the monocultured *L. monocytogenes* and native microflora were statistically different, the differences were marginal.

The LPDs, GRs, and MPDs of *L. monocytogenes* or their transformed values, logarithm ($\log_{10}$), natural logarithm (Ln), or square root, as a function of storage temperature were analyzed using the General Linear Model procedure of SAS 9.1 (SAS Institute). Fig. 2 shows the curves of square root-transformed GRs vs. the storage temperatures. The curves appear to have a curvature nature. Therefore, the square root-transformed GRs of *L. monocytogenes* as a function of storage temperature were fitted with a 2nd-order polynomial regression (Fig. 2). The regression equations are:

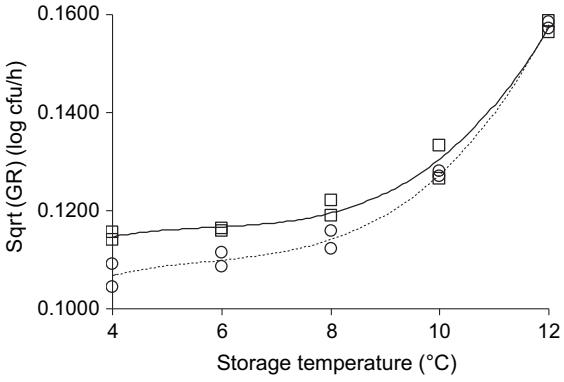

Fig. 2. Regression of $\sqrt{\text{GR}}$ of monocultured *L. monocytogenes* (□) and co-cultured *L. monocytogenes* (○) as a function of storage temperature.

$$\sqrt{\text{GR}}_{\text{monocultured } L.\ monocytogenes} = 0.1457 - 0.011*\text{temperature} + 0.001*(\text{temperature})^2$$

$$\sqrt{\text{GR}}_{\text{co-cultured } L.\ monocytogenes} = 0.13770 - 0.0118*\text{temperature} + 0.0011*(\text{temperature})^2$$

Comparing the estimated GRs obtained using these equations showed that the GRs of *L. monocytogenes* were reduced by 8–2% at 4–12 °C in ham when the native microflora was present.

During storage at 4–12 °C, the MPDs of co-cultured *L. monocytogenes* and native microflora in ham all reached >7.0 $\log_{10}$ cfu/g and >6.5 $\log_{10}$ cfu/g, respectively. The MPDs of co-cultured *L. monocytogenes* were 1.2, 0.6, and 1.3 $\log_{10}$ cfu/g lower ($p < 0.05$) than those of monocultured *L. monocytogenes* at 4, 6, and 8 °C, respectively. The result indicated that the MPDs of *L. monocytogenes* at lower storage temperatures were affected by the presence of the native microflora. The presence of *L. monocytogenes* also affected the LPDs of the native microflora at 6 and 8 °C, at which the MPDs of the co-cultured native microflora were significantly lower than those of the monocultured native microflora.

3. Discussion

This study showed that the growth characteristics of *L. monocytogenes* in ham were influenced by the presence of the native microflora at 4–8 °C. The presence of the native microflora reduced the GRs and lowered the MPDs of *L. monocytogenes* in ham at lower storage temperatures, and the influence diminished as the temperature increased. The results from this study were in agreement with those examining microflora/competitive microflora on the growth of *L. monocytogenes* in various food products. The growth of *L. monocytogenes* was inhibited by *Lactobacillus sakei* in cooked, sliced, vacuum-packaged meat (Bredholt et al., 2001) and by *Carnobacterium piscicola* in vacuum-packaged meat (Schobitz et al., 1999). In a study screening 91 strains of lactic acid bacteria (LAB) originating from meat products, 91% of the strains were able to inhibit the growth of *L. monocytogenes* (Vermeiren et al., 2004). *Pediococcus acidilactici*, *Lactobacillus casei*, and *Lactobacillus paracasei*, isolated from commercial RTE meat products, were shown to have bacteriostatic activity in cooked ham and bactericidal activity in frankfurters against *L. monocytogenes* at 5 °C (Amezquita and Brashears, 2002). Studies have also reported that the growth of *L. monocytogenes* was inhibited by LAB in cold-smoked salmon (Nilsson et al., 1999; Vescovo et al., 2006; Tome et al., 2008) and Italian marinated seafood salad (Andrighetto et al., 2009). In fermented sausage, LAB were shown to

inhibit the growth of pathogenic microorganisms and spoilage microorganisms (Chikthimmah et al., 2001; Calicioglu et al., 2001; Benkerroum et al., 2003; Hwang et al., 2009). However, there were studies reported that native microflora had no or limited effect on the growth of *L. monocytogenes*. Barakat and Harris (1999) reported that the growth of *L. monocytogenes* on cooked modified atmosphere-packaged leg quarters was not influenced by *Carnobacterium*, *Leuconostoc*, *Lactococcus*, and *Brochothrix* spp. at 3.5–10 °C for up to 5 weeks, although the population of the microflora was 1–2 log higher than that of *L. monocytogenes*. Guillier et al. (2008) studied the co-growth of *L. monocytogenes* and an un-identified biofilm microflora on smeared cheese wooden shelves. They reported that the lag phase and growth rate of *L. monocytogenes* were not affected by the biofilm microflora, and only the levels of *L. monocytogenes* reached at the stationary phase were reduced due to the Jameson effect (1962). A probable reason for the discrepancy of the reported results may be attributed to the nature of the native/competitive microflora used in each study, which consisted mainly of various strains of LAB. The antilisterial activity of LAB has been attributed primarily to the production of antimicrobial compounds such as organic acids, hydrogen peroxide, and bacteriocins (Barakat and Harris, 1999; Nilsson et al., 1999; Amezquita and Brashears, 2002). Therefore, the specific LAB strains and their ability to grow in a particular environment and produce antilisterial compounds would determine the effect of the LAB on *L. monocytogenes*. For example, the growth of a lactic acid bacterium that is capable of producing a higher level of antimicrobial compounds under a certain condition would show a greater inhibition on the growth of *L. monocytogenes* than a strain that produced less antimicrobial compounds under less optimal conditions. This is the basis of numerous studies in which LAB were screened and characterized (strains and growth conditions) to select high antimicrobial-producing LAB for biopreservation purposes (Nilsson et al., 1999; Amezquita and Brashears, 2002; Vermeiren et al., 2004; Wilderdyke et al., 2004; Tome et al., 2008). In addition to the antimicrobial compounds, the growth inhibition of *L. monocytogenes* by LAB may also due to the rapid growth of the LAB population that results in depletion of the available nutrients for *L. monocytogenes*. Buchanan and Bagi (1997) examined the co-growth of *L. monocytogenes* and bacteriocin-producing *C. piscicola* in BHI broth. They reported that the suppression of growth of *L. monocytogenes* by *C. piscicola* was affected by the relative growth rates of the two microorganisms. A culture condition that favored the growth of *C. piscicola* increased the suppression of growth of *L. monocytogenes*. They concluded that the suppression of growth of *L. monocytogenes* by LAB was partially attributed to the nutrient depletion by the fast growing LAB. The effect of a high population of competitive microflora inhibiting the growth of pathogens was also reported by Malakar et al. (2003). They examined growth models from predictive microbiology and suggested that the growth-inhibitory effect of the competitive microflora on pathogens only occurred when the populations of the competitive microflora reached $\sim 10^8$ cfu/ml. The population effect was studied and described by a differential form of the simple logistic model (Gimenez and Dalgaard, 2004; Mejlholm and Dalgaard, 2007). The model stipulated that the specific growth rate of a microflora was reduced when the cell populations of the microflora or a competitive microflora increased. Therefore, the inhibition of growth of a microflora by a competitive microflora is also influenced by the populations of the competitive microflora in the co-growth environment.

Refrigerated RTE meat products are heat processed, so most vegetative microorganisms are killed. Background/native microflora in RTE meat are mainly spore-forming bacteria that survive the heat process and/or microorganisms that contaminate the product after heat processing. Samelis et al. (2000) reported that the initial microbial populations in RTE meat after slicing and packaging were at 0.5–2 $\log_{10}$ cfu/g with LAB as the majority, and the predominated LAB were *Lactobacillus*, *Leuconostoc*, and *Carnobacterium* spp. In the present study, the native microflora isolated from the commercial RTE meat was identified as *Brochothrix* spp. This microorganism is a Gram-positive, rod-shaped facultative anaerobe that is capable of growing at refrigeration temperature and is closely related to the genera of *Lactobacillus* and *Listeria*. It is commonly isolated from raw meat and processed meat. *Brochothrix thermosphacta* is the most well-known among the species of *Brochothrix* spp. In modified atmosphere- or vacuum-packaged meat stored at refrigeration temperature, *B. thermosphacta* frequently become predominant spoilage microorganisms over Enterobacteriaceae, *Lactobacillus*, *Leuconostoc*, and *Pseudomonas* (Pin et al., 2002). Under anaerobic conditions, *Brochothrix* spp. was capable of producing lactate, acetate, ethanol, bacteriocins, and short chain fatty acids (Grau, 1983; Pin et al., 2002; Greer and Dilts, 2006), some are known antimicrobials that are similar to metabolites produced by LAB. The ability of *Brochothrix* spp. to produce antimicrobial compounds and its influence on the growth of *L. monocytogenes* has been less studied than LAB such as *Lactobacillus*, *Leuconostoc*, or *Carnobacterium* spp. This study showed that the influence of this microflora on the growth characteristics of *L. monocytogenes* was not as significant as that of *L. sake*, *Lactobacillus plantarum*, *L. casei*, or *C. piscicola*. In this study, the growth of *L. monocytogenes* occurred at 4–12 °C, with populations increasing to >6.0 $\log_{10}$ cfu/g if co-cultured with *Brochothrix* spp, while studies have reported that there was no or little growth of *L. monocytogenes* in frankfurters, cooked ham (Amezquita and Brashears, 2002), vacuum-packaged cooked meat (Bredholt et al., 2001), and cold-smoked salmon (Nilsson et al., 1999; Vescovo et al., 2006; Tome et al., 2008) when *L. monocytogenes* was co-inoculated with LAB.

To compare the GRs of *L. monocytogenes* when it was cultured along or co-cultured with *Brochothrix* spp, the square root-transformed GRs of *L. monocytogenes* were plotted vs. the storage temperatures and modeled. The curves were non-linear and a 2nd-order polynomial regression was found to well describe the GRs as a function of storage temperature (Fig. 2). The resulted models were different from 1st-order linear models such as square root models (Ratkowsky et al., 1982; Wijtzes et al., 2001) and cardinal models (Augustin and Carlier, 2000; Le Marc et al., 2002) that describe the effect of temperature, water activity, pH, salt, and/or acid concentration on the growth rates of microorganisms. A biphasic relationship between growth rate and temperature has also been observed for some strains of *L. monocytogenes* (Bajard et al., 1996) and one strain of *Listeria innocula* (Le Marc et al., 2002).

This study demonstrated the effect of a non-lactic acid bacterium on the growth of *L. monocytogenes* in cooked ham at refrigeration and abuse temperatures. The data from this study add to the vast growth/survival data of *L. monocytogenes* in RTE meat as affected by the product's intrinsic and extrinsic parameters, such as pH, a_w, food additives and preservatives, and product handling and storage conditions. Results from this study add to the understanding of the behavior of *L. monocytogenes* in RTE meat products, and hence further enhance the food safety of these products.

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