



Effects of Reheating against *Listeria monocytogenes* Inoculated on Cooked Chicken Breast Meat Stored Aerobically at 7°C

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SUMMARY

The presence of *Listeria monocytogenes* in domestic and foodservice environments has led to increased attention to handling practices during food preparation and storage. The objective of the present study was to evaluate survival and multiplication of *L. monocytogenes* inoculated on cooked chicken breasts that were stored aerobically at 7°C for 7 days. Reduction of pathogen counts by microwave, domestic oven, and stove top reheating was also evaluated during storage. *L. monocytogenes* populations had increased from 3.7 ± 0.1 to 7.8 ± 0.2 log CFU/g by 7 days. Microwave oven reheating for 90 s, and stove-top and oven reheating to 70°C internal temperature, reduced pathogen populations to < 0.4 – 2.6 , < 0.4 – 4.8 , and 1.4 – 5.9 log CFU/g, respectively; numbers of survivors after reheating were higher ($P < 0.05$) in products stored for more time; up to 7 days. At shorter microwaving times and lower product internal temperatures (stove-top and oven reheating), similar reduction trends were observed, but with higher levels of survivors after the treatment. Although reheating methods in this study reduced *L. monocytogenes* contamination by 2–5 log CFU/g, growth of the pathogen during previous storage allowed high numbers of cells to survive reheating, especially with storage periods more than 2 days. This indicates that storage period and the type and intensity of reheating need to be considered to ensure safe consumption of leftovers.

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INTRODUCTION

Cooked leftover food, both in the domestic environment and at food-service establishments, has reduced levels of background microflora. If cross-contamination occurs during refrigerated storage, leftover foods may harbor and support growth of foodborne pathogens (22), especially psychrotrophic pathogens such as *Listeria monocytogenes* and *Yersinia enterocolitica* (16, 18). Many consumers tend to link foodborne illness to food consumed outside the home (19), but recent epidemiological investigations indicate that poor hygienic practices in the domestic environment is also a major contributing factor to foodborne disease episodes (23). Infrequent handwashing, poor handwashing technique, lack of handwashing prior to food preparation, inadequate cleaning of kitchen surfaces, presence of pets in the kitchen, and touching of the face, mouth, nose and/or hair during preparation of food (17), as well as improper storage and inadequate cooking and reheating (11, 15, 21), are some of the practices that could potentially result in introduction of pathogens of public health significance into food products and allow their survival and multiplication to levels of concern. Contamination in the kitchen environment can be transferred to a food product during preparation and storage. It has been shown that the microbiological profile of cooked food stored in a kitchen refrigerator is very similar to microbial profiles of swabs taken from the environment of the same kitchen (34). In recent years, various studies have detected foodborne pathogens, and specifically *L. monocytogenes*, on kitchen surfaces, dishcloths, sinks, drains, and refrigerators (4, 9, 14, 20), making leftovers potentially hazardous foods because of contamination with *L. monocytogenes*.

Listeriosis caused by *L. monocytogenes* is a severe illness, being responsible for 19% of foodborne disease associated deaths in the United States (26). The very young, the elderly, pregnant women and the immunocompromized are the most susceptible groups (25). Because of the presence of *L. monocytogenes* in a wide array of environments, its halophilic nature, its potential to form biofilms, and its ability to survive and multiply at re-

frigeration temperatures, it has been of special interest in academic and industrial research in recent years (30, 31, 32).

Chicken meat is gaining popularity both among consumers at home and in foodservice establishments; while 30 years ago poultry accounted for approximately 21% of meat consumption in the United States, it now accounts for at least 37%, higher than the consumption of beef, pork, or lamb (3, 13). White muscle tissue (i.e., breast meat) of chicken is considered one of the most popular cuts of meat for both domestic use and commercial processing in the United States (3, 13) and has been involved in many foodborne outbreaks and recalls, including a recall of 10,878 kg of cooked chicken breast contaminated with *L. monocytogenes* (7).

While many studies have targeted inactivation of *L. monocytogenes* in ready-to eat products (22, 24, 29) little work has been designed to investigate inactivation of *L. monocytogenes* by use of domestically available appliances, during storage of leftover foods. Thus, the objective of this study was to investigate the survival and multiplication of background microflora and inoculated *L. monocytogenes* on cooked boneless skinless chicken breasts stored aerobically at 7°C. The effects of three reheating methods, applied at 0, 1, 2, 4, and 7 days of storage, against the pathogen and background microflora were also investigated.

MATERIALS AND METHODS

Preparation of bacterial inoculum

Five food and human-disease originated strains of *L. monocytogenes*, kindly provided by Dr. Martin Wiedmann (Dept. of Food Science, Cornell University, Ithaca, NY), representing diverse ribotypes, PFGE patterns, lineages, and serotypes, were used in this study (8). These strains were J1-177 (lineage I, Serotype 1/2b), C1-056 (lineage II, Serotype 1/2a), N3-013 (lineage I, Serotype 4b), R2-499 (lineage II, Serotype 1/2a), and N1-227 (lineage I, Serotype 4b). The strains were kept on PALCAM agar (Difco, Becton Dickinson, Franklin Lakes, NJ) at 4°C prior to the study, and were activated individually from a single colony of the stored stock as described

by Yang et al. (38). Each strain was then washed with 10 ml sterile saline (0.85% NaCl) and centrifuged at 4,629 g for 15 min, resuspended in 10 ml of ham homogenate (37), and habituated separately for two days at 7°C before inoculation, to allow acclimatization of *L. monocytogenes* cells to the food environment and low temperature. Before inoculation, the suspensions of the five habituated strains were composited and serially 10-fold diluted in phosphate-buffered saline (PBS, pH 7.4; 0.2 g/liter KH_2PO_4 , 1.5 g/liter $\text{Na}_2\text{HPO}_4 \cdot 7\text{H}_2\text{O}$, 8.0 g/liter NaCl, and 0.2 g/liter KCl) to achieve an initial concentration of 3–4 log CFU per gram of sample. The *L. monocytogenes* counts of the composite inoculum after habituation were 9.2 ± 0.2 CFU/ml.

Preparation of chicken samples, inoculation, and storage

Fresh boneless skinless chicken breast muscles purchased from a local processor were aseptically cut into approximately 100-g pieces and stored in sealed plastic bags (approximately 20 samples in each bag) at -20°C for up to two weeks prior to use. Chicken samples were thawed at refrigeration temperature (4°C) for approximately 48 h and cooked (16-in electric skillet, National Presto Industries, Inc., Eau Claire, WI) to the target internal temperature of 73.8°C. The temperature was recorded every 5 s with k-type thermocouples connected to PicoLog data acquisition software (Pico Technology Ltd., Cambridge, UK). Cooked samples were stored aerobically at 7°C in Pyrex dishes (25 by 35 cm, 5 cm deep) covered with cling paper for no more than 2 h. The surfaces of the cooked and cooled-to-4°C samples were then inoculated with 100 µL per side of the above-mentioned diluted habituated composite of *L. monocytogenes* strains, with a 15 min interval between inoculation of the two sides, to achieve an initial inoculation level of approximately 3–4 log CFU/g. The inoculated samples were placed in Pyrex dishes (15 samples in each dish), covered with cling paper, and stored aerobically in a 7°C incubator. During storage, samples were reheated and analyzed microbiologically and for physiochemical properties on days 0, 1, 2, 4, and 7.

Product reheating

Cooked stored inoculated samples were individually placed in a microwave safe dish (22 cm diameter, 4 cm deep) and subjected to 30, 60 or 90 s of microwave heating at the 100% power level in a domestic microwave oven (Amana, Model Radarange AMC5243, Newton, IA) with 1100 W power output. At the end of the intervention, the surface temperature of each sample was measured and recorded manually with a noncontact infrared thermometer (Oaklon TemoTestr IR, Lane Cove, Australia) from a distance of approximately 15 cm (to cover a reading area of 2.75 cm², based on manufacturer's recommendation). Immediately after microwaving and recording of surface temperature, each sample was aseptically transferred in a sterile filter bag (Whirl-Pak, Modesto, CA), placed into ice-water slush, and prepared for microbiological analyses.

For oven reheating, each sample was placed onto a sterile stainless steel tray with a k-type thermocouple aseptically inserted into its geometric center. A domestic oven (Magic Chef Standard Kitchen Oven, Maytag Group, Newton, IA) was preheated to 148°C (300°F) for approximately 30 min and samples were then reheated to an internal temperature of 50, 60 or 70°C. The temperature of the cooking chamber of the oven was also monitored with a k-type thermocouple suspended approximately in the center of the oven, without any contact with the surroundings.

For reheating by the stove-top method, each sample with inserted thermocouple in its geometric center was placed onto the sterile surface of a nonstick skillet (diameter approximately 30 cm [Farberware Licensing Company, LLC, Berwick, PA]) preheated on a domestic oven stove top (Magic Chef Standard Kitchen Oven, Maytag Group, Newton, IA). Every two min, samples were flipped over for exposure of both sides to the skillet surface. The samples were reheated to an internal temperature of 50, 60, or 70°C, with collection of time/temperature profile data every 5 s as described for oven reheating. Surface temperature of the skillet was also measured by suspending a thermocouple in approximately 5 ml of vegetable oil in a 20 ml glass container placed onto the skillet during the reheating procedure. For both stove top and oven, reheating,

similar to microwave treatment, immediately after the designated internal temperature, was reached, samples were placed in sterile filter bags (Whirl-Pak, Modesto, CA), cooled in ice-water slush, and analyzed microbiologically. Internal temperatures, reheating methods, and times for microwave intervention were selected on the basis of results of a preliminary experiment (data not shown).

Microbiological and physiochemical analyses

As indicated, for microbiological analyses, each sample was placed in a sterile filter bag (Whirl-Pak, Modesto, CA); an equal amount of maximum recovery diluent (0.85% NaCl and 0.1% peptone [Difco, Becton Dickinson]), was added immediately after reheating and cooling the samples in ice. Samples were then homogenized (Masticator, IUL Instruments, Barcelona, Spain) for 2 min (6 strokes per s), serially diluted (10-fold) with 0.1% buffered peptone water (Difco, Becton Dickinson, Franklin Lakes, NJ), and spread-plated onto tryptic soy agar (Acumedia, Lansing, MI) with 0.6% yeast extract (Difco, Becton Dickinson) and PALCAM agar (Difco, Becton, Dickinson), for enumeration of total aerobic bacteria and *L. monocytogenes* counts after incubation at 25 and 30°C for 72 and 48 h, respectively. The detection limit for these microbiological analyses was 0.4 log CFU per g of sample. Samples with no detected colonies on plates at the detection level dilution were enriched to evaluate presence/absence of the pathogen by a procedure modified from the USDA-FSIS (35) method, as described by Rodriguez-Marval et al. (24). For all samples with pathogen counts below the detection limit, no pathogen was detected by enrichment. Water activity (AquaLab Instrument, Decagon Devices, Inc., Pullman, WA) and pH (Denver Instrument, Arvada, CO) of samples were measured as described by Byelashov et al. (5).

Experimental design and statistical analyses

The experiment was repeated twice with different ingredients, with three replicates within each of these two repetitions. Mean microbial counts of each

treatment and storage period, after log transformation, as well as cooking time, pH, and water activity values, were compared statistically with ANOVA-based procedures followed by Tukey-adjusted multiple comparison methods for further mean separation at type one error level of 0.05 ($\alpha = 0.05$). Each dataset was analyzed as a randomized complete block design, with each of the two repetition trials considered a blocking factor, using Proc GLM and Proc Mixed commands of SAS 9.2 (SAS, Inc., Chicago, IL). Additionally, to compare the inactivation rates of *L. monocytogenes* by stove-top and oven reheating methods at the three internal temperatures of 50, 60, and 70°C, GlnaFit software, a non-log-linear microbial survivor curve, was used to compare counts at days 7, 4, 2, 1, and 0 (after inoculation). This model, as described by Geeraerd et al. (10), reports specific inactivation rates of K_{\max} and adjusted-R². K_{\max} is the specific inactivation rate for log-linear monophasic curves fitted for each internal temperature with unit of 1/time; thus a longer time required for microbial cell reduction is associated with a smaller K_{\max} value while adjusted-R² value show the proportion of the data described by the model. K_{\max} value obtained for each internal temperature of stove-top reheating were compared statistically to those obtained from oven reheating using Student's *t*-test procedure at $\alpha = 0.05$.

RESULTS AND DISCUSSION

Water activity, pH and temperature measurements

Water activity and pH of reheated stored samples ranged from 0.982 ± 0.001 to 0.992 ± 0.001, and 6.03 ± 0.13 to 6.12 ± 0.12, respectively with no difference ($P \geq 0.05$) among samples of different days of storage and reheating methods. Surface temperatures of the microwave treated samples ranged from 48.8 ± 2.5 to 57.6 ± 4.0, 64.5 ± 4.0 to 69.5 ± 4.0, and 75.7 ± 4.7 to 82.6 ± 3.1°C, for samples reheated for 30, 60, and 90 s, respectively. The pH values for these samples ranged from 6.05 ± 0.07 to 6.13 ± 0.08, 5.88 ± 0.08 to 6.14 ± 0.11, and 5.99 ± 0.18 to 6.19 ± 0.16, for samples reheated for 30, 60, and 90 s, respectively, with no statistical difference ($P \geq 0.05$).

TABLE 1. *Listeria monocytogenes* counts (mean ± standard deviation) of cooked chicken during 7 days aerobic storage at 7°C before (control) and after 30, 60, and 90 seconds of domestic microwave oven reheating (1100 W)

Storage (Day)	<i>Listeria monocytogenes</i> counts (log CFU/g)			
	Control	30 s	60 s	90 s
0	3.7 ± 0.1 D a	3.4 ± 0.1 D b	2.7 ± 0.1 D c	<0.4 ± 0.1 C d
1	3.8 ± 0.1 D a	3.6 ± 0.1 D b	2.8 ± 0.1 D c	0.4 ± 0.1 C d
2	4.6 ± 0.1 C a	4.3 ± 0.1 C b	3.5 ± 0.1 C c	0.4 ± 0.1 C d
4	6.2 ± 0.1 B a	4.8 ± 0.1 B b	4.2 ± 0.1 B c	1.8 ± 0.1 B d
7	7.8 ± 0.2 A a	7.3 ± 0.3 A b	5.6 ± 0.1 A c	2.6 ± 0.2 A d

Values within a column followed by different uppercase letters, and values within a row followed by different lowercase letters, are significantly ($P < 0.05$) different.

The temperatures of the oven cooking chamber for samples reheated to target internal temperatures of 50, 60 and 70°C were 145.9 ± 11.0, 147.6 ± 9.9, and 143.5 ± 1.2°C, respectively. Surface temperatures of the cooking area during stove top reheating were 142.7 ± 12.4, 140.9 ± 9.9, and 148.3 ± 11.3°C for samples reheated to internal temperatures of 50, 60, or 70°C, respectively. The pH values for samples reheated in the oven were in the ranges of 6.05 ± 0.07 to 6.13 ± 0.10, 6.02 ± 0.09 to 6.13 ± 0.05, and 6.03 ± 0.04 to 6.20 ± 0.10 for samples reheated to internal temperatures of 50, 60, and 70°C, respectively, and 6.07 ± 0.07 to 6.13 ± 0.12, 6.09 ± 0.06 to 6.16 ± 0.03, and 5.98 ± 0.12 to 6.10 ± 0.06, for samples reheated on the stove top to internal temperatures of 50, 60, and 70°C, respectively, without significant differences ($P \geq 0.05$) among samples reheated to different internal temperatures.

Survival and growth during storage

Previous studies have isolated *L. monocytogenes* from domestic and industrial refrigerators as well as food preparation and processing environments (27, 36), making leftovers potentially hazardous foods because of contamination with psychrotrophic pathogens, including *L. monocytogenes*. In the present study, initial counts of *L. monocytogenes* on the day of inoculation were 3.7 ± 0.1

log CFU/g and increased to 7.8 ± 0.2 log CFU/g by day 7 of storage at 7°C (Table 1), confirming the concern that if cross-contamination occurs after preparation of foods, *L. monocytogenes* can multiply extensively during refrigerated storage. Similar results were observed for aerobic plate counts, with more than a 4-log increase from the first day to day 7 (data not shown). The extensive multiplication of *L. monocytogenes* in this study could be explained by the ability of cooking to increased hydrolysis of macromolecules and bioavailability of nutrients on the surface of chicken samples (6), which can enhance multiplication of the pathogen as well as background microflora during storage.

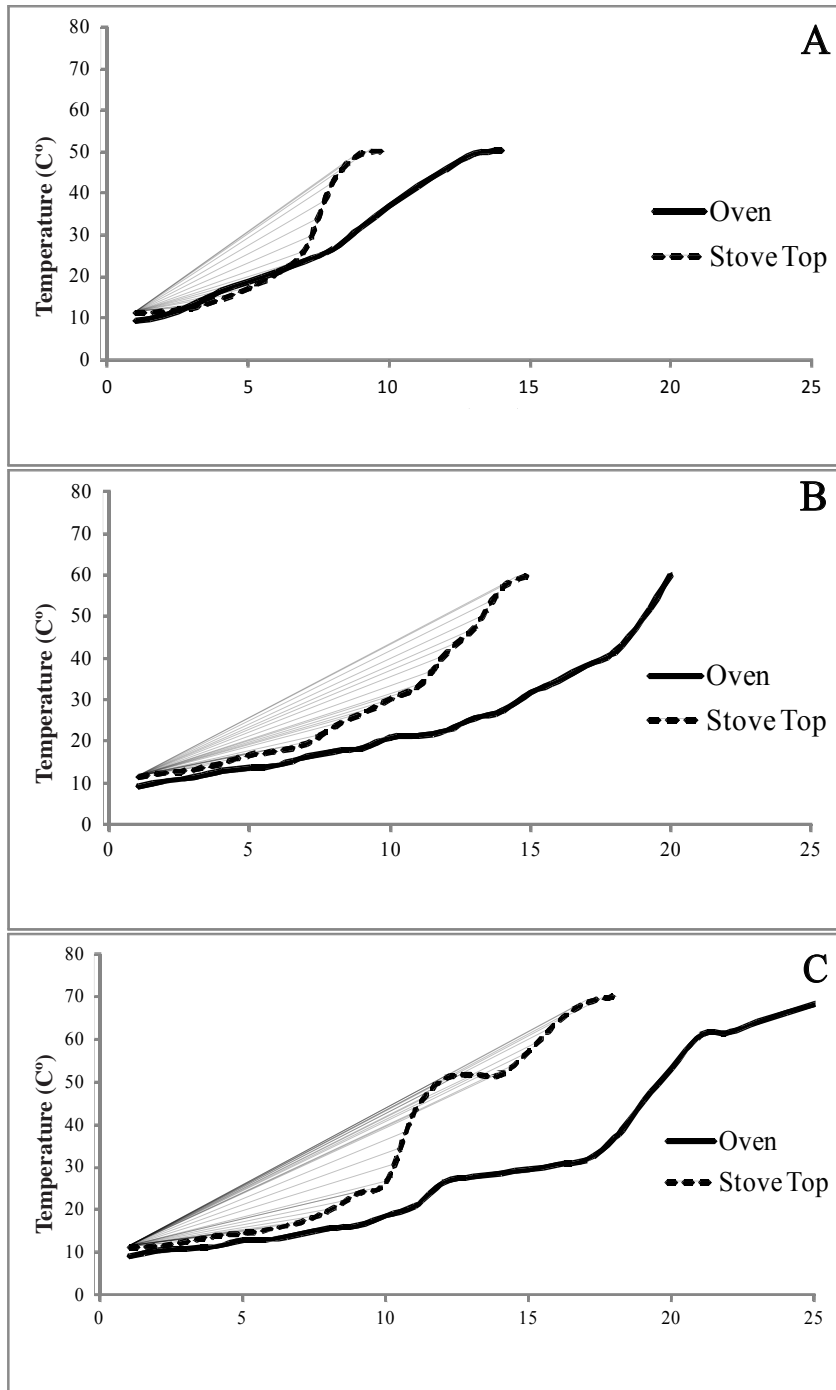
Inactivation by reheating

Microwave oven reheating showed a high potential for reduction of *L. monocytogenes* counts. On day 0, microwaving for 30, 60, and 90 s was responsible for reductions of 0.3, 1.1, and > 3.4 log CFU/g of *L. monocytogenes*, respectively. Similar trends were observed for samples reheated on days 1, 2, 4, and 7 (Table 1). For example, on day 4, counts of the untreated control were 6.2 ± 0.1 and were reduced ($P < 0.05$) to 4.8 ± 0.1, 4.2 ± 0.1 and 1.8 ± 0.8 as the result of microwaving treatment for 30, 60, and 90 s, respectively. As storage days increased, higher number of survivors after reheating were observed. In other words, storage time affected the initial

microbial counts and had a major impact on subsequent survival of reheating. On day 4, as an example, although 90 s of microwaving caused more than a 4 log reduction of the pathogen, the 1.8 log CFU/g of survivors could still be of concern from a food safety standpoint. However, the same intervention (microwaving for 90 s) on day 0 was able to reduce the *L. monocytogenes* counts to undetectable levels. This indicates that intensity of reheating needs to be adjusted based on the storage period (which affects the initial bacterial load) of food in order to ensure reduction of potential pathogen counts to acceptable levels. In general, the microwave treatment compared with other reheating methods evaluated in this study, had high potential for reduction of microbial loads with 3.4, 3.5, 4.2, 4.4, and 5.2 log CFU/g reductions in 90 s after 0, 1, 2, 4, and 7 days of storage, respectively (Table 1).

The effectiveness of microwave treatments against surface inoculated *L. monocytogenes* is in agreement with previous studies. Rodriguez-Marval et al. (24) demonstrated that *L. monocytogenes* inoculated on surface of frankfurters could be reduced by 3.7 log CFU/g by 75 s of microwaving. Similarly, Shen et al. (29) showed that 30 s of microwaving resulted in 0.8 to 1.3 log CFU/g reduction of inoculated *L. monocytogenes*. It is noteworthy that, although microwave treatments showed high pathogen reduction effectiveness, it has been reported that their performance can be considerably affected by size and position of the

FIGURE 1. Time and temperature profiles of samples reheated by domestic oven and stove top methods. (A) samples reheated to internal temperature of 50°C, (B) samples reheated to internal temperature of 60°C, and (C) samples reheated to internal temperature of 70°C



food and by age and power output of microwave (33) all of which must be considered before recommendations can be developed for safe microwave reheating of leftover food by consumers.

Oven reheating on day-0 reduced the pathogen from 3.7 ± 0.1 to 2.9 ± 0.1 , 2.7 ± 0.1 , and 1.4 ± 0.3 log CFU/g

after reheating to internal temperatures of 50, 60, and 70°C, respectively (Table 2). Similar to the results seen with microwave treatment, as storage time between inoculation and reheating increased, microbial multiplication increased and more cells survived on the reheated samples (Table 2). In other

words, storage time affected the initial microbial counts on each day of storage and thus had a major impact on extent of subsequent death due to reheating. For stove-top reheating, on days 0 and 1, *L. monocytogenes* loads of samples reheated to the internal temperature of 70°C were less than one log CFU/g, but, as with other treatments, more survivors were detected as the time interval between inoculation and reheating increased. Similar results have been observed by other investigators for reduction of inoculated *L. monocytogenes* on pork scrapple reheated by pan-frying methods during storage (1).

When comparing the domestic oven and stove-top reheating, methods are compared, the two methods required considerably different lengths of time to reach the same internal temperatures (i.e., 50, 60, and 70°C) in the geometric center of the samples (Fig. 1), and thus, exhibited different pathogen reduction potential. On day 7, as an example, the oven and stove top reheating to internal temperatures of 70°C required 24.2 and 17.1 min (Fig. 1), and were responsible for reductions of 2.0 and 3.0 log CFU/g of the pathogen, respectively (Table 2). These differences could be explained by the different heat transfer principles involved in the two methods: oven reheating involves convection heat transfer whereas stove top reheating involves conduction as the primary mode of heat transfer (2). The other characteristic of oven and stove reheating methods investigated in this study was the relatively high performance variation. For example, domestic oven reheating to internal temperature of 50°C caused 0.8 log reduction on day 0, while the same treatment caused a 0.1 log reduction on day 1. Also, samples reheated to internal temperatures of 50 and 60°C had similar counts ($P \geq 0.05$) on day 7 but not on any other day ($P < 0.05$). The high variation in performance of domestic cooking appliances has also been reported by other investigators (12, 28), and it appears that these variations need to be considered for preparation of safe reheating recommendations for consumers.

Table 3 provides inactivation rates of *L. monocytogenes* on days 7, 4, 2, 1, and 0 by stove-top and oven reheating to internal temperatures of 50, 60, and 70°C. Based on adjusted R^2 values, 87% to 89% of samples reheated on the stove-top, and 92 to 97% of oven-reheated

TABLE 2. *Listeria monocytogenes* counts (mean ± standard deviation) of cooked chicken during 7 days aerobic storage at 7°C before (control) and after reheating to internal temperatures of 50, 60, and 70°C, using domestic oven and stove-top methods

Storage (Day)	<i>Listeria monocytogenes</i> counts (log CFU/g)			
	Control	50°C	60°C	70°C
Domestic Oven				
0	3.7 ± 0.1 D a	2.9 ± 0.1 E b	2.7 ± 0.1 E b	1.4 ± 0.3 E c
1	3.8 ± 0.1 D a	3.7 ± 0.1 D b	3.1 ± 0.0 D c	1.8 ± 0.2 D d
2	4.6 ± 0.1 C a	4.5 ± 0.4 C a	4.0 ± 0.1 C b	2.6 ± 0.2 C c
4	6.2 ± 0.1 B a	5.7 ± 0.3 B b	4.9 ± 0.1 B c	4.0 ± 0.1 B d
7	7.8 ± 0.2 A a	6.7 ± 0.1 A b	6.6 ± 0.4 A b	5.9 ± 0.1 A c
Stove-top Reheating				
0	3.7 ± 0.1 D a	1.7 ± 0.1 E b	1.1 ± 0.4 D c	<0.5 ± 0.4 D d
1	3.8 ± 0.1 D a	1.8 ± 0.1 D b	1.3 ± 0.2 D c	<0.4 ± 0.1 D d
2	4.6 ± 0.1 C a	2.6 ± 0.1 C b	2.1 ± 0.1 C c	1.5 ± 0.1 C d
4	6.2 ± 0.1 B a	4.6 ± 0.1 B b	3.8 ± 0.1 B c	3.0 ± 0.1 B d
7	7.8 ± 0.2 A a	6.8 ± 0.0 A b	5.6 ± 0.1 A c	4.8 ± 0.1 A d

Values within a column (for domestic oven and stove-top methods separately) followed by different uppercase letters, and values within a row followed by different lowercase letters, are significantly ($P < 0.05$) different.

TABLE 3. Non-linear microbial survivor analysis for comparing inactivation rates of *L. monocytogenes* by stove-top and oven reheating, comparing counts of days 7, 4, 2, 1, and 0 (after inoculation)

Reheating Method	Internal Temperature (°C)	<i>L. monocytogenes</i> inactivation	
		K_{max}	R^2
Stove-top ^a	70	2.57 ± 0.18	0.87
	60	2.65 ± 0.17*	0.89
	50	3.04 ± 0.20*	0.89
Domestic Oven	70	2.58 ± 0.14	0.92
	60	2.20 ± 0.11	0.93
	50	2.27 ± 0.08	0.97

^a K_{max} values associated with stove-top reheating followed by a * are significantly larger ($P < 0.05$) than values of oven reheating for each internal temperature. K_{max} values (parameter ± standard error) are specific inactivation rates for log-linear monophasic curves fitted for each internal temperatures with unit of 1/time; thus, longer time required for microbial cell reductions is associated with smaller K_{max} values.

samples were described by the utilized GInaFiT model, a log-linear monophasic curve fitted for each internal temperature. At internal temperatures of 50 and

60°C, stove-top reheating had higher ($P < 0.05$) K_{max} values (K_{max} parameter ± SE), indicating that this method required less time than oven reheating to reach an internal temperature of 50 and 60°C.

For the 70°C internal temperature, K_{max} values of stove-top and oven reheating were 2.57 ± 0.18 and 2.58 ± 0.14, respectively, with no statistical difference ($P \geq 0.05$).

In summary, because of different heat transfer mechanisms (i.e., primarily convection-based transfer for oven and conduction-based transfer for stove-top reheating) these two methods demonstrated different pathogen reduction potentials and times for reaching the same designated internal temperature. This indicates the need for method-specific recommendations for assuring the safety of leftover food reheated in domestic and foodservice environments by different reheating methods.

Along with increasing evidence of *L. monocytogenes* presence in domestic and food service environments in recent years, it appears that safe management of leftover food, especially for people at risk for listeriosis, is of particular importance. In our study, initial counts of *L. monocytogenes* increased ($P < 0.05$) by over 4.0 log CFU/g during 7 days of storage at 7°C, confirming the concern that if cross-contamination occurs after preparation of foods, *L. monocytogenes* can multiply extensively during refrigerated storage. Although the reheating methods investigated in this study reduced *L. monocytogenes* contamination by 2–5 log CFU/g, growth of the pathogen during storage allowed survival of high numbers of cells after reheating, especially after two days of storage. In other words, storage time affected the before reheating initial microbial counts and thus had a major impact on the extent of subsequent microbial inactivation due to reheating. This indicates that storage period, as well as type and intensity of reheating need to be considered for safe consumption of leftover food. Regardless of reheating method, high numbers of survivors after reheating, especially after two days of storage, indicated a need to reconsider utilization of leftover food, especially by at-risk populations.

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REFERENCES

1. Adekunle, A. O., A. C. S. Porto-Fett, J. E. Call, B. Shoyer, K. Gartner, L. Tufft, and J. B. Luchansky. 2009. Effect of storage and subsequent reheating on viability of *Listeria monocytogenes* on pork scrapple. *J. Food Prot.* 72:2530–2537.
2. Adler, J. M., I. Geornaras, K. E. Belk, G. C. Smith, and J. N. Sofos. 2009. Thermal inactivation of *Escherichia coli* O157:H7 at different depth of panbroiled and roasted non-intact steak. 96th annual meeting of the International Association for Food Protection, Grapevine, TX. Abstract No. T8–02.
3. [AMI] American Meat Institute Fact Sheet. 2009. U.S. Meat and Poultry Production and Consumption: an overview. Available at: <http://www.meatami.com/ht/a/GetDocumentAction/i/48781>. Accessed January 26, 2012.
4. Beumer, R. R., M. C. te Giffel, E. Spoorenberg, and F. M. Rombouts. 1996. *Listeria* species in domestic environment. *Epidemiol. Infect.* 117:437–442.
5. Byelashov, O. A., J. M. Adler, I. Geornaras, K. Y. Ko, K. E. Belk, G. C. Smith, and John Sofos. 2010. Evaluation of brining ingredients and antimicrobials for effects on thermal destruction of *Escherichia coli* O157:H7 in a meat model system. *J. Food Sci.* 75:209–217.
6. Damodaran, S., K. L. Parkin, and O. R. Fennema (ed.). 2008. Fennema's food chemistry fourth edition. CRC Press Taylor and Francis Group, New York.
7. [FDA] Food and Drug Administration. 2010. Cooked chicken breasts recalled for *Listeria* contamination. Available at: http://www.fda.gov/Safety/Recalls/ArchiveRecalls/2007/default.htm?fragment25_NextRow=51. Accessed January 26 2012.
8. Fugett, E., E. Fortes, C. Nnoka, and M. Wiedmann. 2006. International Life Sciences Institute North America *Listeria monocytogenes* strains collection: Development of standard *Listeria monocytogenes* strain sets for research and validation studies. *J. Food Prot.* 69:2929–2938.
9. Gandhi, M., and M. L. Chikindas. 2007. *Listeria*: A foodborne pathogen that knows how to survive. *Int. J. Food Microbiol.* 113:1–15.
10. Geeraerd, A. H., V. P. Valdramidis, and J. F. Van Impe. 2005. GlnaFit, a freeware tool to assess non-log-linear microbial survivor curves. *Int. J. Food Microbiol.* 102:95–105.
11. Griffith, C. J., K. A. Mathias, and P. E. Price. 1994. The mass media and food hygiene. *British Food Journal.* 96:16–21.
12. Gupta, S., I. Geornaras, K. E. Belk, G. C. Smith, and J. N. Sofos. 2010. Thermal inactivation of *Escherichia coli* O157:H7 in different parts of beef roasts moisture-enhanced with different brining formulations and cooked to rare and very-rare degrees of doneness. 70th Annual Meeting of the Institute of Food Technologists, Chicago, IL, Abstract No. 038–57.
13. Harley, M. M. 2000. Changing consumer demand for meat: The U.S. example, 1970–2000. The U.S. Dept. of Agriculture Economic Research center. Available at: <http://www.ers.usda.gov/publications/wrs011/wrs011g.pdf>. Accessed January 26, 2012.
14. Jackson, V., I. S. Blair, D. A. McDowell, J. Kennedy, and D. J. Bolton. 2007. The incidence of significant foodborne pathogens in domestic refrigerators. *J. Food Control* 18:346–351.
15. James, S. J., and J. Evans. 1992. Consumer handling of chilled foods: Temperature performance. *Int. J. Refrig.* 15:299–306.
16. Jay, M. J., M. J. Loessner, and D. A. Golden. 2005. Modern food microbiology. Springer Inc., New York, N.Y.
17. Jay, L. S., D. Comar, and L. D. Govenlock. 1999. A video study of Australian domestic food-handling practices. *J. Food Prot.* 62:1285–1296.
18. Juneja, V. K., and J. N. Sofos (ed.). 2010. Pathogens and toxins in foods: challenges and interventions. ASM Press, Washington, D.C.
19. Kennedy, J., V. Jackson, I. S. Blair, D. A. McDowell, C. Cowan, and D. J. Bolton. 2005. Food safety knowledge of consumers and the microbiological and temperature status of their refrigerators. *J. Food Prot.* 68:1421–1430.
20. Kilonzo-Nthenge, A., F. Chen, and S. Godwin. 2008. Occurrence of *Listeria* and *Enterobacteriaceae* in Domestic Refrigerators. *J. Food Prot.* 71:608–612.

21. Marklinder, I. M., M. Lindblad, L. M. Erikson, A. M. Finnson, and R. Lindqvist. 2004. Home storage temperatures and consumer handling of refrigerated foods in Sweden. *J. Food Prot.* 67:2570–2577.
22. Murphy, R. Y., L. K. Dumcan, E. R. Johnson, M. D. Davis, R. E. Wolfe, and H. G. Brown. 2001. Thermal lethality of *Salmonella* Senftenberg and *Listeria innocua* in fully cooked and packaged chicken breast strips via steam pasteurization. *J. Food Prot.* 64: 2083–2087.
23. Redmond, E. C., and C. J. Griffith. 2003. Consumer food handling in the home: a review of food safety studies. *J. Food Prot.* 66:130–161.
24. Rodriguez-Marval, M., I. Geornaras, P.A. Kendall, J. A. Scanga, K. E. Belk, and J. N. Sofos. 2009. Microwave oven heating for inactivation of *Listeria monocytogenes* on frankfurters before consumption. *J. Food Microbiol.* 74: 453–460.
25. Ryser, E. T., and E. H. Marth. 2007. *Listeria*, listeriosis, and food safety (ed.). 2007. CRC Press Taylor and Francis Group, New York.
26. Scallan, E., R. M. Hoekstra, F. J. Angulo, R. V. Tauxe, M. A. Widdowson, S. L. Roy, J. L. Jones, and P. M. Griffin. 2011. Foodborne illness acquired in the United States—major pathogens. *Emerg. Infect. Dis.* 17:7–15.
27. Sergelidis, D., A. Abraham, A. Sarimvei, C. Panoulis, Pr. Karaioannoglou, and C. Genigeorgis. 1997. Temperature distribution and prevalence of *Listeria* spp. in domestic, retail and industrial refrigerators in Greece. *Int. J. Food Microbiol.* 34:171–177.
28. Shen, C., J. M. Alder, I. Geornaras, K. E. Belk, G. C. Smith, and J. N. Sofos. 2010. Inactivation of *Escherichia coli* O157:H7 in nonintact beefsteaks of different thicknesses cooked by pan broiling, double pan broiling, or roasting using five types of cooking appliances. *J. Food Prot.* 73:461–469.
29. Shen, C., I. Geornaras, P. A. Kendall, and J. N. Sofos. 2009. Antilisterial activities of salad dressing, without or with prior microwave oven heating, on frankfurters during simulated home storage. *Int. J. Food Microbiol.* 132:9–13.
30. Sofos, J. N. 2008. Challenges to meat safety in the 21st century. *J. Meat Sci.* 78:3–13.
31. Sofos, J. N. 2009. Developments and future outlook for postslaughter food safety. *J. Anim. Sci.* 87:2448–2457.
32. Sofos, J. N., and I. Geornaras. 2010. Overview of current meat hygiene and safety risks and summary of recent studies on biofilms, and control of *Escherichia coli* O157:H7 in nonintact, and *Listeria monocytogenes* in ready-to-eat, meat products. *J. Meat Sci.* 86:2–14.
33. Swain, M. J., S. J. James, and M. V. L. Swain. 2008. Effect of power output reduction of domestic microwave ovens after continuous (intermittent) use on food temperature after reheating. *J. Food. Eng.* 87:11–15.
34. Toule, G., and O. Murphy. 1978. A study of bacteria contaminating refrigerated cooked chicken; their spoilage potential and possible origin. *J. Hyg. Camb.* 81:161–169.
35. [USDA-FSIS] U.S. Dept. of Agriculture, Food Safety and Inspection Service. 2008. Laboratory guidebook notice of change. Isolation and identification of *Listeria monocytogenes* from red meat, poultry, eggs, and environmental samples. Available at: http://www.fsis.usda.gov/Science/Microbiological_Lab_Guidebook/index.asp. Accessed January 26 2012.
36. Walker, S. J., P. Archer, and J. G. Banks. 1990. Growth of *Listeria monocytogenes* at refrigerated temperatures. *J. Appl. Microbiol.* 68:157–162.
37. Yang, H., P. A. Kendall, L. C. Medeiros, and J. N. Sofos. 2009a. Efficacy of sanitizing agents against *Listeria monocytogenes* biofilms on high-density polyethylene cutting boards surfaces. *J. Food Prot.* 72:990–998.
38. Yang, H., P. A. Kendall, L. C. Medeiros, and J. N. Sofos. 2009b. Inactivation of *Listeria monocytogenes*, *Escherichia coli* O157:H7, and *Salmonella* Typhimurium with compounds available in households. *J. Food Prot.* 72:1201–1208.