

Prediction of *Listeria innocua* survival in fully cooked chicken breast products during postpackage thermal treatment

Min Li,* Abani Pradhan,† Wen Wang,* and Yanbin Li*‡§¹

*College of Biosystems Engineering and Food Science, Zhejiang University, Hangzhou 310058, China;

†Department of Nutrition and Food Science & Center for Food Safety and Security Systems,
University of Maryland, College Park 20742; ‡Department of Poultry Science,

and §Department of Biological and Agricultural Engineering, University of Arkansas, Fayetteville 72701

ABSTRACT The effectiveness of postpackage hot water thermal treatment on survival of *Listeria innocua* in fully cooked chicken breast products was investigated at 60, 70, 80, and 90°C. Primary models based on log-linear and Weibull models were used to fit bacterial survival curves at different temperatures. The prediction plot and fit statistics indicated that the Weibull model provided a better fit than the log-linear model and was selected as the primary model. A secondary model based on linear regression was developed to describe the effect of temperature on the kinetic parameters of *Listeria*

innocua survival derived from the Weibull model. The root mean square error and coefficients of determination indicated a good fit of the secondary model. The models were validated by independent data from pilot plant tests, and the values of bias factor and accuracy factor fell into the acceptable range. The models developed in this study can assist poultry producers and risk managers in designing appropriate thermal treatment regimens to minimize the risk associated with *Listeria* in ready-to-eat poultry products.

Key words: *Listeria*, thermal inactivation, postpackage, predictive model, poultry product

2013 Poultry Science 92:827–835

<http://dx.doi.org/10.3382/ps.2012-02743>

INTRODUCTION

Consumption of ready-to-eat (RTE) meat and poultry products contaminated with *Listeria monocytogenes* has become a major concern of foodborne outbreaks of human listeriosis in the past decades (Zhu et al., 2005). According to the Centers for Disease Control and Prevention (CDC), *L. monocytogenes* causes an estimated 1,591 cases of foodborne illnesses and 255 deaths each year in the United States, with hospitalization and death rates as high as 94.0 and 15.9%, respectively (Scallan et al., 2011). The USDA Food Safety and Inspection Service (FSIS) has imposed a zero-tolerance policy for *L. monocytogenes* in RTE meat and poultry products to prevent the risk of human listeriosis (USDA/FSIS, 2003).

Ready-to-eat poultry products are a common source of *L. monocytogenes* contamination. Even with the most rigorous hygienic operations, fully cooked poultry products may still be subjected to recontamination with *L. monocytogenes* during the postcook procedures (e.g., handling and packaging; Lawrence and Gilmour,

1994; Osaili et al., 2011). As a consequence, bacteria may survive or grow in packaged RTE poultry products at refrigeration temperatures when stored for a long period of time, which may lead to the risk of listeriosis for consumers (Lundén et al., 2003; Zhu et al., 2005). Therefore, it is of vital importance to prevent the growth of *L. monocytogenes* in RTE poultry products by appropriate control measures during poultry processing.

Postpackage thermal treatment has been successfully applied to kill bacteria after the packaging procedure using hot water submersion heating (Murphy et al., 2003b; Muriana et al., 2004). To assess the process lethality of postpackage thermal treatment, it is necessary to develop mathematical models to predict the reduction of *L. monocytogenes* under the combinations of different times and temperatures. In predictive microbiology for bacterial inactivation, primary models describe the survival of bacteria over time, and secondary models describe the change of bacterial inactivation kinetics of primary models with environmental conditions. In response to temperature and other processing or product factors that affect bacterial inactivation kinetics under different treatment conditions, bacterial thermal inactivation curves have displayed several typical types: linear curves, curves with a shoulder, curves with a tailing (biphasic curves), and sigmoidal curves

©2013 Poultry Science Association Inc.

Received September 2, 2012.

Accepted November 25, 2012.

¹Corresponding author: yanbinli@zju.edu.cn

containing both a shoulder and a tailing (Baranyi et al., 1996; Char et al., 2009; Miller et al., 2011). Correspondingly, various primary and secondary predictive models have been developed to describe different types of bacterial survival curves in a variety of food products (Baranyi et al., 1996; Valdramidis et al., 2006; Lori et al., 2007; Huang, 2009; Stone et al., 2009; Juneja et al., 2010).

Predictive models have been used for thermal treatment of *L. monocytogenes* in various meat and poultry products (Juneja, 2003; Lihono et al., 2003; Pradhan et al., 2007; Li et al., 2011). A large quantity of RTE chicken breast products has been recalled due to the potential contamination with *L. monocytogenes* or association with listeriosis outbreaks in the past years (CDC, 2002; USDA/FSIS, 2007, 2011a,b). However, reports on modeling the effectiveness of postpackage thermal treatment on the reduction of *Listeria* in boneless, skinless RTE chicken breast products are very limited. Therefore, the purposes of this study were (i) to determine the effectiveness of postpackage thermal treatment using hot water on the reduction of *Listeria* in fully cooked chicken breast products, and (ii) to develop primary and secondary predictive models to describe *Listeria* survival during postpackage thermal treatment at different temperatures.

MATERIALS AND METHODS

Bacterial Strain

Listeria innocua is close to *L. monocytogenes* in physiological nature and has a higher heat resistance compared with *L. monocytogenes* (Fairchild and Foegeding, 1993; Pradhan et al., 2007, 2012; Li et al., 2011). A single strain of *L. innocua* (ATCC 33090) was used as a nonpathogenic surrogate for *L. monocytogenes*, and the heat tolerance of this *L. innocua* strain has been verified to be higher than *L. monocytogenes* by several previous studies (Char et al., 2009, 2010; Lorentzen et al., 2010). The stock cultures of *L. innocua* were maintained in the brain heart infusion broth (BHI; TEKNOVA, Hollister, CA) with 12% glycerol at a -80°C freezer. For each trial, the stock cultures were transferred to a 10-mL BHI broth and incubated at 37°C for 24 h, and the bacterial concentration in the pure culture was determined to be approximately 10^9 cfu/mL.

Preparation and Inoculation of Chicken Breast Samples

Frozen boneless, skinless raw chicken breast fillets were obtained from a local grocery store before the experiments and stored at -20°C in the freezer before they were used within 1 wk. Before each trial, chicken breast fillets were thawed at 4°C overnight and cooked in a preheated grill (model 25325, Hamilton Beach/Proctor-Silex Inc., Southern Pines, NC) for 5 to 8 min.

To ensure sufficient cooking, thermocouples were inserted into the centers of random samples at each batch to ensure center temperature to reach 71.1°C during cooking. The fully cooked chicken breast fillets were then cut with a sterile knife into chicken breast strips of similar sizes (51 mm in length, 15 mm in width, 20 mm in thickness, and 14 g in weight, on average).

For inoculation, 1-mL pure culture of *L. innocua* per 100 g of sample was evenly spread using a sterile plastic rod onto all surfaces (top, bottom, and sides) of the samples to obtain an initial level of approximately 10^7 cfu/g. After inoculation, samples were kept on a sterile wire rack at 4°C for 30 min to allow bacterial attachment.

Postpackage Thermal Treatment

The hot water thermal treatment was carried out in a laboratory water bath (model 25, Precision Scientific, Chicago, IL) at 60, 70, 80, and 90°C for predetermined treatment times as obtained from the preliminary tests (data were collected every 60 s at 60°C , 30 s at 70°C , 20 s at 80°C , and 20 s at 90°C). The total holding times for samples were 30, 10, 7, and 5 min at 60, 70, 80, and 90°C , respectively. At each temperature, 2 inoculated fully cooked chicken breast strips not to be thermally treated were used as positive controls to enumerate the initial bacterial load. For each trial (time-temperature combinations), 3 inoculated samples for bacterial sampling and 1 uninoculated sample for temperature measurement were used. The temperature sample was inserted with 5 thermocouples (type T, Omega Engineers, Stamford, CT) at different locations to measure the temperature changes at the top surface, bottom surface, and center of the sample (1 probe went to the top and bottom surfaces, respectively, and the other 3 went to the center). All samples were individually packaged in a 0.05-mm-thick plastic vacuum storage bag (Rival, Milford, MA) and sealed with a vacuum sealer (model VS105, Rival, Milford, MA). The sample packages were placed on a stainless-steel wire rack and quickly dipped in the water bath when the temperature was equilibrated to the treatment temperature, i.e., 60, 70, 80, or 90°C . During the thermal treatment, temperature data were recorded every 2 s by a data acquisition system (model 34970, Hewlett Packard, Loveland, CO). Samples were cooled in an ice water bath immediately after thermal treatment. After being cooled to less than 4°C , bags containing chicken samples were withdrawn from the ice water bath and analyzed within 30 min. Thermocouple probes were maintained in the temperature samples for continuous temperature measurement as they went through the heating and cooling process.

For the purpose of model validation, independent data for thermal inactivation of *L. innocua* conducted in a pilot-plant scale hot water pasteurizer were collected. The hot water pasteurizer was specially designed to simulate commercial hot water processing procedures,

as described by Enns et al. (2007). To allow even heat transfer during thermal treatment, product packages were placed on an expanded metal platform that could move back and forth relative to the continuous hot water sprays from both the top and bottom, which simulated the movement of a continuous belt past a series of spray nozzles down a production line (Enns et al., 2007). Preparation of chicken breast samples and experimental procedures followed the same protocol as in the laboratory tests.

Microbiological Analyses

After the cooling process, the chicken breast packages were removed from the ice water bath and aseptically cut with a sterile knife. Chicken breast pieces in the package were aseptically transferred to a sterile stomacher bag (17.7 × 30.5 cm; Seward, London, UK) added with 50 mL of sterile 0.1% (wt/vol) buffered peptone water (Difco, Becton Dickinson, Sparks, MD). The entire content in the stomacher bag was stomached for 2 min using a stomacher (model 400, Seward, London, UK). The wash fluid was decimally diluted and a 0.1-mL portion of 3 appropriate dilutions was plated in duplicate onto modified Oxford medium (Merck, Darmstadt, Germany) overlaid with tryptic soy agar (Difco, Becton Dickinson) to resuscitate heat-injured bacteria (Murphy et al., 2003b; Li et al., 2011). The plates were incubated at 37°C for 48 to 72 h before the enumeration of colonies. To detect lower levels of *L. innocua* that cannot be determined by direct plating, an enrichment procedure was performed by mixing the entire sample in the stomacher bag with 50 mL of sterile BHI broth and incubating at 37°C for 24 h (Pradhan et al., 2007; McKinney et al., 2009; Latorre et al., 2010). The enriched solution was then plated onto modified Oxford medium-tryptic soy agar plates and checked for bacterial survivors after incubation at 37°C for 48 to 72 h. If *L. innocua* was found to be present in any of the replicate samples, it was recorded as growth at that treatment time; otherwise, the samples were recorded as no-growth, which means approximately 7 log cfu/g bacterial reduction.

Model Development

The survival curves of *L. innocua* under different temperatures were fitted with log-linear model and Weibull model, and their performances were compared in order to choose the model with better fit.

The log-linear model assumes homogeneous bacterial resistance to heat treatment and has been frequently used for thermal inactivation (Lori et al., 2007):

$$\log_{10} N_t = \log_{10} N_0 - \frac{t}{D}, \quad [1]$$

where N_t is the number of bacterial survivors at time t (cfu/g), N_0 is the initial bacterial number (cfu/g), and

D is the decimal reduction time (min) at the specific treatment temperature (°C).

Peleg and Cole (1998) introduced the Weibull model based on the assumption that bacterial resistance to heat treatment varied from one cell to the other and followed a Weibull distribution. The model has gained popularity for its simplicity and flexibility and is displayed in equation [2]:

$$\log_{10} \frac{N_t}{N_0} = -bt^n, \quad [2]$$

where N_t is the number of bacterial survivors at time t (cfu/g), N_0 is the initial bacterial number (cfu/g), and b and n are temperature-dependent coefficients that represent the scale and shape factors of the model, respectively. $n = 1$ indicates a linear survival curve; $n > 1$ indicates the survival curve is concave downward, and $n < 1$ is concave upward.

Secondary models based on the linear regression equation were established to describe the effect of heating temperature on the parameters of b and n obtained from Weibull model. A square-root transformation was performed to homogenize the variance of b and n .

$$\sqrt{b} = a_0 + a_1T; \quad [3]$$

$$\sqrt{n} = b_0 + b_1T, \quad [4]$$

where T is heating temperature, and a_0 , a_1 , b_0 , and b_1 are regression parameters of the model.

Evaluation of Model Performance

The performances of primary models were evaluated using the root mean square error (RMSE), Akaike information criterion (AIC), and plots of predicted versus observe values.

Akaike information criterion attempts to find the best-fitted model and imposes a penalty for increasing the number of parameters (Akaike, 1981). Models with lower AIC values are preferred to fit the data.

$$AIC = 2k - 2 \ln(L) = 2k + n \left[\ln \left(\frac{2\pi \times RSS}{n} \right) + 1 \right], \quad [5]$$

where k is the number of parameters in the model, n is the number of data points on the survival curves, L is the maximum value of the likelihood function, and RSS is the residual sum of squares in the model.

The secondary model was externally validated using pilot plant test data under different experimental conditions not included in the model, and the indices of bias factor (B_f) and accuracy factor (A_f) were calculated accordingly (Ross, 1996).

B_f measures the extent of under- or overprediction and indicates the average bias of the model:

$$B_f = 10^{\frac{\sum \log(P/O)}{n}} \quad [6]$$

A_f measures how close the predicted values are to the observed values and indicates the average accuracy of the predictions:

$$A_f = 10^{\frac{\sum |\log(P/O)|}{n}}, \quad [7]$$

where P is the predicted kinetic parameters, O is the observed kinetic parameters, and n is the number of observations.

Data Analyses

Data analyses were conducted through the Microsoft Excel 2007 software (Microsoft Corp., Redmond, WA), and the model fitting and parameter estimate were performed using the JMP statistical software version 9.0 (SAS Institute Inc., Cary, NC). The means and SD of the bacterial survivor data were calculated from the independent replicate trials for each treatment, and data were presented as means \pm SD. An ANOVA was conducted to compare the means of RMSE and AIC values among different models ($\alpha = 0.05$).

RESULTS AND DISCUSSION

Temperature Profile

A typical pattern of temperature profiles was exemplified by a chicken breast strip heated at 70°C for the total holding time of 10 min in a hot water bath and subsequently cooled in an ice water bath (Figure 1). Temperature profiles at other treatment temperatures (60, 80, and 90°C) for various times followed a similar pattern as in Figure 1. As indicated in Figure 1, the hot water bath temperature immediately equilibrated to the set temperature of 70°C after chicken breast strips were placed in the water bath. In the heating period (the first 10 min of the treatment), the top and bottom surface temperatures increased faster and remained higher than the center temperatures; therefore, heat conduction from the surface to the center produced a substantial temperature gradient that drove the change of temperature within the chicken product (Pradhan et al., 2007). When the ice cooling process was initiated (after 10 min), heat transfer within the chicken product changed in the opposite direction (i.e., from the center to the surface); therefore, the top and bottom surface temperatures began to decrease rapidly and remained lower than that of the center temperature during the cooling process.

It is noteworthy to indicate that the top and center temperatures of the product did not reach the target treatment temperature of 70°C even for the total holding time of 10 min (Figure 1), indicating quite a long

period of warm-up during the experiment. The same observation was noticed for all tested time-temperature combinations for 80 and 90°C, as indicated in Table 1, which shows the maximum product temperatures reached for the total holding times at each treatment temperature. The only exception was at 60°C, where the top surface, bottom surface, and center temperatures reached the treatment temperature at 18.3, 20.2, and 21.8 min, respectively, which might be attributed to the longer heating time at 60°C than those at other temperatures. It can be observed that the difference between the top and bottom temperatures at 90°C for 5 min were larger than those at other temperatures for their respective holding times. A possible reason can be attributed to the phenomena that the top temperature increased faster than the bottom temperature at the beginning of the heating process (Figure 1), which enlarged the difference between the top and bottom temperatures. However, with the increase of heating time, the increasing rate of top temperature slowed down, which decreased the temperature difference (Figure 1). Therefore, the large discrepancy between the top and bottom temperatures at 90°C would become smaller if given a longer heating time beyond 5 min.

Some previous studies reported much shorter come-up times than in this study. Murphy et al. (2002, 2003a) reported that the come-up times were <20 s for postprocess pasteurization of *Listeria* in fully cooked chicken or turkey breast meat at 55 to 70°C. The major reason for the difference might be that those studies were designed to measure the D - and z -values for bacterial thermal inactivation, and for this purpose the chicken samples were grounded and flattened in the package to make the thickness very small (0.5 to 1 mm) to ensure the target temperature was reached immediately. Nevertheless, Murphy et al. (2003b) reported a longer come-up time for fully cooked chicken breast

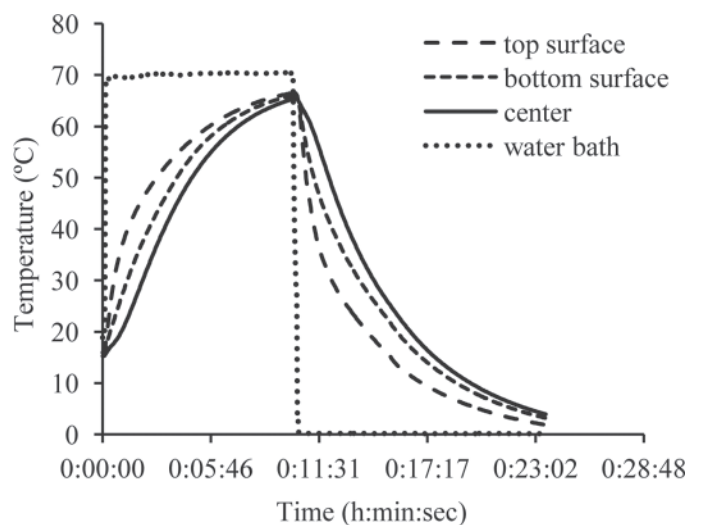


Figure 1. A temperature profile of a chicken breast strip package heated in a 70°C hot water bath for 10 min, and subsequently cooled in a 0°C ice water bath.

Table 1. Maximum product temperatures reached for the total holding times at each treatment temperature

Temperature (°C)	Total holding time (min)	Maximum temperature (°C)		
		Top surface	Bottom surface	Center
60	30	60	60	60
70	10	67.5	66.9	65.8
80	7	74.6	71.9	68.3
90	5	86.7	69.5	59.0

strips heated at 90°C compared with our study, probably because the chicken samples used in their study had greater weight and thickness than those in our study. In general, greater thickness inhibited the heat transfer in chicken products during the heating process and therefore prolonged the warm-up time. The come-up time is of significance in that it reflected the heat transfer rate and consequently the bacterial inactivation rate of the heating process. Longer come-up time implied that the product temperature was elevated progressively rather than immediately; therefore, the bacterial inactivation rate would increase gradually, leading to less thermal lethality during the same period of time compared with the heating process with a shorter come-up time.

The sensory qualities of the thermally treated chicken samples were beyond the scope of our study. However, keeping in mind that high temperature pasteurization for long holding time (e.g., 7 and 5 min at 80 and 90°C, respectively) may affect the appearance of the samples tested, we monitored the surface color change visually. No obvious surface color changes before and after the thermal treatments were found. In the preliminary tests, we also measured the change of moisture content in chicken samples before and after postpackage cooking using an oven drying method (Pradhan et al., 2007). The results indicated that the moisture loss was 7.03, 3.0, 2.31, and 2.63% for cooked chicken breasts heated at 60°C for 30 min, 70°C for 10 min, 80°C for 7 min, and 90°C for 5 min. The reduction of moisture content in the samples was believed to affect the tenderness of the cooked chicken breast samples.

Bacterial Inactivation

The effect of temperature in postpackage thermal inactivation on the survivors of *L. innocua* in cooked chicken breast is shown in Figure 2. Because treatment at higher temperature required less time to inactivate the bacteria, bacterial inactivation rates increased with the increase in treatment temperatures from 60 to 90°C (Figure 2). The 7-log reduction of *L. innocua* survivors occurred at 30, 10, 7, and 5 min at 60, 70, 80, and 90°C determined by the enrichment procedure. At the relatively moderate treatment temperature of 60°C, bacterial survivors did not decrease significantly at the beginning and the survival curve displayed an initial “shoulder” period followed by a stage of rapid bacterial reduction. The shoulder effect has been explained by

several factors such as the clumps of microorganisms, more heat-resistant subpopulations, or poor heat transfer through the heating medium (Baranyi et al., 1996; Xiong et al., 1999; Huang, 2009). In this study, the shoulder period at 60°C was associated with the heat transfer rate. Because 60°C was a relatively moderate treatment temperature and the chicken samples had a large thickness, sufficient time was needed for heat transfer from hot water to the product surface and then to the product geometric center. As such, during the first several minutes of the treatment process, the surface and center temperatures of chicken products did not increase significantly, and bacteria were not substantially killed at the beginning. With the increase of treatment temperatures from 70 to 90°C, the product temperatures increased faster because the heat transfer rate became accelerated; therefore, bacteria in chicken products were subjected to higher initial temperature than at 60°C and were more vulnerable to heat treatment, which could explain the absence of the shoulder effect at these temperatures.

Previous studies addressing the postpackage thermal treatment of *Listeria* in various RTE poultry or meat products have reported varied results. Some studies obtained less bacterial inactivation than in the present study. For instance, Murphy et al. (2003b) examined the thermal lethality of *L. monocytogenes* in cooked chicken breast fillets and strips during postcook pasteurization at 90°C and achieved a 7-log bacterial reduction at approximately 5, 25, and 35 min for single-packaged fillets, 227-g package strips, and 454-g package strips, respectively. Muriana et al. (2002) evaluated the postpackage pasteurization of *L. monocytogenes* in large-sized RTE deli-style turkey products (2.3 to 5.4 kg per package) and obtained 2- to 4- log reductions when heated at 90.6, 93.3, or 96.1°C for 2 to 10 min. The lesser extent of bacterial reduction in those studies might be attributed to the larger size of the chicken samples than those used in this study because the thermal reduction of *Listeria* depends largely on the size and thickness of the products and the efficacy of thermal treatment decreases as product size or thickness increases. Nevertheless, several investigations reported greater bacterial reduction efficiency than our study. Porto et al. (2004) reheated frankfurters (56 g per package) in a hot water bath to a surface temperature of 60, 70, 80, and 90°C after refrigeration at 4°C or frozen storage at -18°C, and found that a 5-log reduction of *L. monocytogenes*

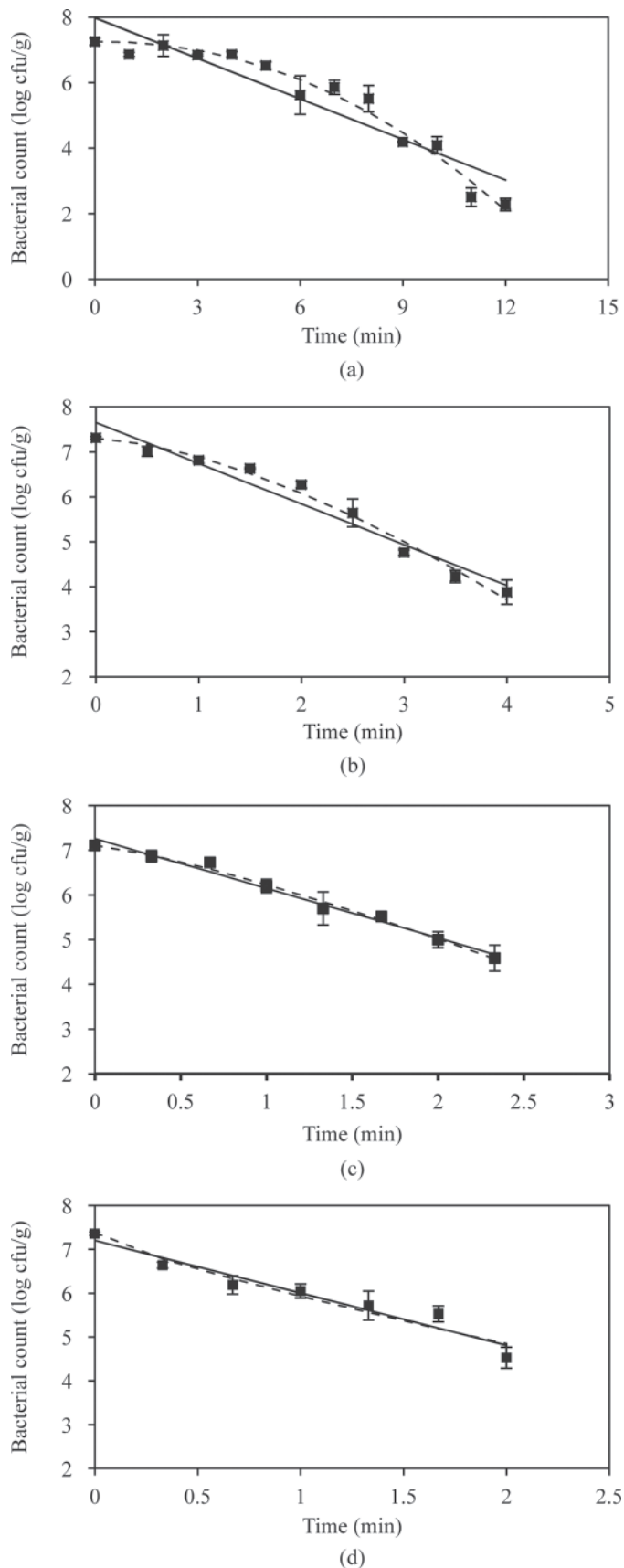


Figure 2. Survival data and primary model fitting for *Listeria innocua* in fully cooked chicken breast strips thermally treated at 60°C (a), 70°C (b), 80°C (c), and 90°C (d) for different times in a hot water bath. Data points represent observed bacterial survival data, and solid and dotted lines indicate the fitting of log-linear and Weibull models, respectively.

was achieved at 70°C for 2 min and at 80 and 90°C for 0.6 min, respectively. McCormick et al. (2003) studied the *D*- and *z*-values of *L. monocytogenes* in low-fat RTE turkey bologna (4 cm² and 2.2 mm thick per package) subjected to a surface thermal treatment and obtained *D*-values of 124 and 16.2 s at 61 and 65°C, which corresponded to a 7-log reduction at 61°C for 14.5 min and at 65°C for 1.9 min, respectively. A possible explanation for the different bacterial reduction could be the greater heat diffusion rate in the product during the heating process, either because of the smaller thickness of the products that facilitates heat transfer or shorter come-up time achieved by moving the products backward and forward in the water bath to ensure homogeneous temperature distribution (McCormick et al., 2003; Porto et al., 2004). In general, it is difficult to compare the differences in *Listeria* reduction in this study with previous studies because various factors may affect the bacterial thermal lethality, such as meat types, product formulations, bacterial strains, physiological state of bacteria, thickness of package bags, and folds, crevices, or wrinkles on product surfaces that may protect bacteria from heat injury.

Primary Models

The goodness-of-fit of primary models was evaluated by the RMSE and AIC values, as presented in Table 2. The RMSE values of Weibull model were consistently less than those of the log-linear model for all treatment temperatures from 60 and 90°C. Both models, log-linear and Weibull, generally showed smaller RMSE values at higher temperature than at lower temperatures. The AIC values of Weibull model were less than those of the log-linear model at 60, 70, and 80°C, except at 90°C, where the log-linear model showed a relatively smaller AIC value than Weibull model. Overall, the analysis of RMSE and AIC statistics indicated that Weibull model performed better in comparison with the log-linear model to describe the inactivation data at tested temperatures of 60, 70, 80, and 90°C, although at 90°C only, the AIC statistics value was slightly better for log-linear than for the Weibull model (−0.42 vs. 1.65).

From the graphs showing observed data and predicted values (Figure 2), it can be seen that Weibull model agreed reasonably well with observed data at all treatment temperatures; at 60 and 70°C, the Weibull model even provided a better fit than the log-linear model to the downwardly concave curve (Figure 2a). Although the Weibull model fitted well at all 4 treatment temperatures, as the treatment temperature increased, the log-linear model also reasonably fitted well at higher temperatures (e.g., 80 and 90°C). This is not unexpected because the log-linear model assumes a homogeneous bacterial die-off rate and therefore a linear curve is applied to describe bacterial survivors, which is not suitable for the description of survival curves at lower treatment temperatures (e.g., 60°C) that present a downward concavity (Lori et al., 2007). In com-

Table 2. Statistical analysis of primary models fitted to *Listeria innocua* survival curves at different treatment temperatures

Primary model	Fit statistics	Temperature (°C)			
		60	70	80	90
Log-linear	AIC ¹	25.01	3.6	-10.36	-0.42
	RMSE ²	0.638	0.301	0.129	0.243
Weibull	AIC	8.14	-4.95	-13.14	1.65
	RMSE	0.308	0.167	0.096	0.242

¹AIC = Akaike information criterion.

²RMSE = root mean square error.

parison, the Weibull model assumes that the heat resistance of bacterial cells is different and the survival curves exhibit a cumulative form of the distribution of lethal events, and thus is more accurate to describe the shoulder effect or concave downward or upward curves (Peleg and Cole, 1998; Huang, 2009). Because of overall better fitting of data with Weibull model at all treatment temperatures compared with log-linear model (Table 2 and Figure 2), in this study the Weibull model was selected as the primary model to fit *L. innocua* survival curves at different treatment temperatures and estimated parameters of Weibull model were used further for secondary modeling.

The kinetic parameters estimated from the Weibull model are summarized in Table 3. With the increase of treatment temperatures from 60 to 90°C, the values of scale factor *b* increased and the values of shape factor *n* decreased. Because the parameter *n* signifies the heat resistance of bacteria at different temperatures, downward concavity at 60, 70, and 80°C with *n* > 1 reflected that these temperatures were relatively moderate to eliminate the bacteria, whereas the upward concavity at 90°C with *n* < 1 indicated that this harsher temperature increased the bacterial sensitivity to heat treatment and killed the bacteria at a faster rate.

Secondary Models

The performance of secondary models to describe the effect of heating temperature on primary model parameters is shown in Figure 3. It is evident that model predictions agreed well with observed data, indicating a close linear relationship between treatment temperatures and the square roots of *b* and *n*. The coefficients of determination (*R*²) were 0.98 and 0.99 for the linear relationship of temperature between \sqrt{b} and \sqrt{n} , respectively. In addition, the RMSE of model were 0.00671 and 0.0696, suggesting a high agreement between predictions and observations.

Evaluation of Model Performance

The bias factor (*B_f*) and accuracy factor (*A_f*) of the predictive model were calculated from the validation test in the pilot plant. *B_f* = 1 suggests that model prediction agrees perfectly with experimental data. In this

study, the *B_f* values were 1.03 and 1.08 for the parameters \sqrt{b} and \sqrt{n} , respectively, which were slightly greater than 1 and were thus considered acceptable because they were within the acceptable range of 0.7 to 1.15 proposed by Ross (1999). As for the accuracy factor, a larger *A_f* value indicates less accuracy of the model prediction. The acceptable *A_f* value for a single variable should be no greater than 1.10 to 1.15 (Ross et al., 2000). The *A_f* values were 1.13 and 1.10 for \sqrt{b} and \sqrt{n} , respectively, which fell into this acceptable range. The *B_f* and *A_f* values in this study indicated that the predictive models were not only fit for laboratory tests, but also applicable to the pilot-plant scale processing conditions. In addition, the *B_f* and *A_f* values were consistent with those reported by other researchers concerning the bacterial inactivation kinetics (Fernández et al., 2001; Álvarez et al., 2003; Gómez et al., 2005). Overall, the predictive models generated in this study are reasonable and can provide satisfactory predictions for *L. innocua* inactivation in RTE chicken breast products during postpackage hot water treatment at different heating temperatures.

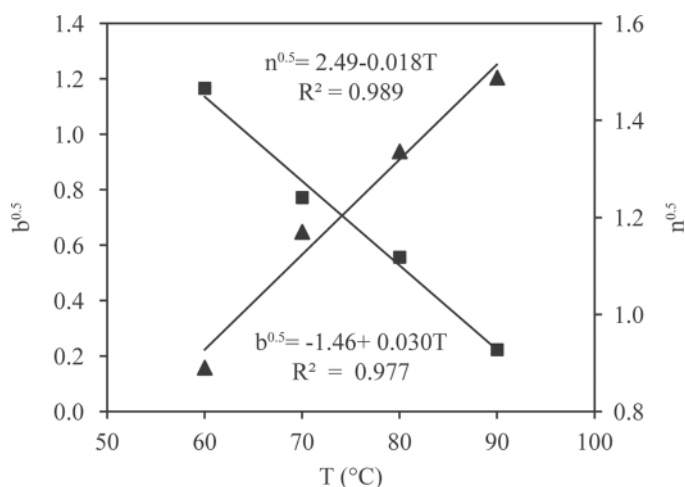


Figure 3. Effect of treatment temperatures (*T*) on the kinetic parameters of the Weibull model. Dotted points represent observed values, and solid lines represent model fitting. ▲: \sqrt{b} values obtained from Weibull model at each temperature. ■: \sqrt{n} values obtained from Weibull model at each temperature.

Table 3. Estimates of kinetic parameters for the Weibull model¹

Parameter	Temperature (°C)			
	60	70	80	90
<i>b</i>	0.025 ± 0.011	0.42 ± 0.063	0.88 ± 0.051	1.45 ± 0.089
<i>n</i>	2.15 ± 0.19	1.54 ± 0.12	1.25 ± 0.084	0.86 ± 0.11

¹Data were represented as mean ± SD.

The predictive models developed in this study can provide a useful tool for the poultry industry to design effective thermal processing regimens to reduce or eliminate *L. monocytogenes* contamination in poultry products. The models can also assist risk managers in reducing parameter uncertainties in risk assessment models and developing risk management strategies to minimize risk associated with *L. monocytogenes* in RTE poultry products.

ACKNOWLEDGMENTS

This research was supported by grants from Cooperative State Research, Education, and Extension Service, USDA, Washington, DC and the Food Safety Consortium, Fayetteville, AR. The authors thank Lisa C. Kelso, Department of Poultry Science, and Eddie Stiles, Department of Food Science, University of Arkansas, Fayetteville, for their technical assistance during the experiments.

REFERENCES

- Akaike, H. 1981. Likelihood of a model and information criteria. *J. Econom.* 16:3–14.
- Alvarez, I., R. Virto, J. Raso, and S. Condón. 2003. Comparing predicting models for the *Escherichia coli* inactivation by pulsed electric fields. *Innov. Food Sci. Emerg. Technol.* 4:195–202.
- Baranyi, J., A. Jones, C. Walker, A. Kaloti, T. P. Robinson, and B. M. Mackey. 1996. A combined model for growth and subsequent thermal inactivation of *Brochothrix thermosphacta*. *Appl. Environ. Microbiol.* 62:1029–1035.
- CDC. 2002. Public health dispatch: Outbreak of listeriosis—North-eastern United States, 2002. *Morb. Mort. Week. Rep.* 51:950–951.
- Char, C., S. Guerrero, and S. M. Alzamora. 2009. Survival of *Listeria innocua* in thermally processed orange juice as affected by vanillin addition. *Food Contr.* 20:67–74.
- Char, C., S. Guerrero, and S. M. Alzamora. 2010. Mild thermal process combined with vanillin plus citral to help shorten the inactivation time for *Listeria innocua* in orange juice. *Food Biol. Technol.* 3:752–761.
- Enns, D. K., P. G. Crandall, C. A. O'Bryan, C. L. Griffis, and E. M. Martin. 2007. A 2-step cooking method of searing and hot water pasteurization to maximize the safety of refrigerated, vacuum packaged, chicken breast meat. *J. Food Sci.* 72:M113–M119.
- Fairchild, T. M., and P. M. Foegeding. 1993. A proposed nonpathogenic biological indicator for thermal inactivation of *Listeria monocytogenes*. *Appl. Environ. Microbiol.* 59:1247–1250.
- Fernández, A., M. J. Ocio, P. S. Fernández, and A. Martínez. 2001. Effect of heat activation and inactivation conditions on germination and thermal resistance parameters of *Bacillus cereus* spores. *Int. J. Food Microbiol.* 63:257–264.
- Gómez, N., D. García, I. Álvarez, S. Condón, and J. Raso. 2005. Modelling inactivation of *Listeria monocytogenes* by pulsed electric fields in media of different pH. *Int. J. Food Microbiol.* 103:199–206.
- Huang, L. 2009. Thermal inactivation of *Listeria monocytogenes* in ground beef under isothermal and dynamic temperature conditions. *J. Food Eng.* 90:380–387.
- Juneja, V. K. 2003. Predictive model for the combined effect of temperature, sodium lactate, and sodium diacetate on the heat resistance of *Listeria monocytogenes* in beef. *J. Food Prot.* 66:804–811.
- Juneja, V. K., A. C. S. Porto-Fett, J. E. Call, H. B. Marks, M. L. Tamplin, and J. B. Luchansky. 2010. Thermal inactivation of *Bacillus anthracis* Sterne in irradiated ground beef heated in a water bath or cooked on commercial grills. *Innov. Food Sci. Emerg. Technol.* 11:123–129.
- Latorre, A. A., J. S. Van Kessel, J. S. Karns, M. J. Zurakowski, A. K. Pradhan, K. J. Boor, B. M. Jayarao, B. A. Houser, C. S. Daugherty, and Y. H. Schukken. 2010. Biofilm in milking equipment on a dairy farm as a potential source of bulk tank milk contamination with *Listeria monocytogenes*. *J. Dairy Sci.* 93:2792–2802.
- Lawrence, L. M., and A. Gilmour. 1994. Incidence of *Listeria* spp. and *Listeria monocytogenes* in a poultry processing environment and in poultry products and their rapid confirmation by multiplex PCR. *Appl. Environ. Microbiol.* 60:4600–4604.
- Li, M., A. Pradhan, L. Cooney, A. Mauromoustakos, P. Crandall, M. Slavik, and Y. Li. 2011. Predictive model for the inactivation of *Listeria innocua* in cooked poultry products during postpackage pasteurization. *J. Food Prot.* 74:1261–1267.
- Lihono, M. A., A. F. Mendonca, J. S. Dickson, and P. M. Dixon. 2003. A predictive model to determine the effects of temperature, sodium pyrophosphate, and sodium chloride on thermal inactivation of starved *Listeria monocytogenes* in pork slurry. *J. Food Prot.* 66:1216–1221.
- Lorentzen, G., E. Ytterstad, R. L. Olsen, and T. Skjerdal. 2010. Thermal inactivation and growth potential of *Listeria innocua* in rehydrated salt-cured cod prepared for ready-to-eat products. *Food Contr.* 21:1121–1126.
- Lori, S., R. Buckow, D. Knorr, V. Heinz, and A. Lehmacher. 2007. Predictive model for inactivation of *Campylobacter* spp. by heat and high hydrostatic pressure. *J. Food Prot.* 70:2023–2029.
- Lundén, J. M., T. J. Autio, A. M. Sjöberg, and H. J. Korkeala. 2003. Persistent and nonpersistent *Listeria monocytogenes* contamination in meat and poultry processing plants. *J. Food Prot.* 66:2062–2069.
- McCormick, K., I. Y. Han, J. C. Acton, B. W. Sheldon, and P. L. Dawson. 2003. *D*- and *z*-values for *Listeria monocytogenes* and *Salmonella* Typhimurium in packaged low-fat ready-to-eat turkey bologna subjected to a surface pasteurization treatment. *Poult. Sci.* 82:1337–1342.
- McKinney, J., R. C. Williams, G. D. Boardman, J. D. Eifert, and S. S. Sumner. 2009. Dose of UV light required to inactivate *Listeria monocytogenes* in distilled water, fresh brine, and spent brine. *J. Food Prot.* 72:2144–2150.
- Miller, F. A., B. F. Ramos, M. M. Gil, T. R. S. Brandão, P. Teixeira, and C. L. M. Silva. 2011. Heat inactivation of *Listeria innocua* in broth and food products under non-isothermal conditions. *Food Contr.* 22:20–26.
- Muriana, P., N. Gande, W. Robertson, B. Jordan, and S. Mitra. 2004. Effect of prepackage and postpackage pasteurization on postprocess elimination of *Listeria monocytogenes* on deli turkey products. *J. Food Prot.* 67:2472–2479.
- Muriana, P. M., W. Quimby, C. A. Davidson, and J. Grooms. 2002. Postpackage pasteurization of ready-to-eat deli meats by submersion heating for reduction of *Listeria monocytogenes*. *J. Food Prot.* 65:963–969.

- Murphy, R. Y., L. K. Duncan, B. L. Beard, and K. H. Driscoll. 2003a. *D* and *z* values of *Salmonella*, *Listeria innocua*, and *Listeria monocytogenes* in fully cooked poultry products. *J. Food Sci.* 68:1443–1447.
- Murphy, R. Y., L. K. Duncan, M. E. Berrang, J. A. Marcy, and R. E. Wolfe. 2002. Thermal inactivation *D*- and *z*-values of *Salmonella* and *Listeria innocua* in fully cooked and vacuum packaged chicken breast meat during postcook heat treatment. *Poult. Sci.* 81:1578–1583.
- Murphy, R. Y., L. K. Duncan, K. H. Driscoll, B. L. Beard, M. B. Berrang, and J. A. Marcy. 2003b. Determination of thermal lethality of *Listeria monocytogenes* in fully cooked chicken breast fillets and strips during postcook in-package pasteurization. *J. Food Prot.* 66:578–583.
- Osaili, T. M., A. R. Alaboudi, and E. A. Nesiari. 2011. Prevalence of *Listeria* spp. and antibiotic susceptibility of *Listeria monocytogenes* isolated from raw chicken and ready-to-eat chicken products in Jordan. *Food Contr.* 22:586–590.
- Peleg, M., and M. B. Cole. 1998. Reinterpretation of microbial survival curves. *Crit. Rev. Food Sci. Nutr.* 38:353–380.
- Porto, A. C. S., J. E. Call, and J. B. Luchansky. 2004. Effect of reheating on viability of a five-strain mixture of *Listeria monocytogenes* in vacuum-sealed packages of frankfurters following refrigerated or frozen storage. *J. Food Prot.* 67:71–76.
- Pradhan, A. K., M. Li, Y. Li, L. C. Kelso, T. A. Costello, and M. G. Johnson. 2012. A modified Weibull model for growth and survival of *Listeria innocua* and *Salmonella* Typhimurium in chicken breasts during refrigerated and frozen storage. *Poult. Sci.* 91:1482–1488.
- Pradhan, A. K., Y. Li, J. A. Marcy, M. G. Johnson, and M. L. Tamplin. 2007. Pathogen kinetics and heat and mass transfer based predictive model for *Listeria innocua* in irregular-shaped poultry products during thermal processing. *J. Food Prot.* 70:607–615.
- Ross, T. 1996. Indices for performance evaluation of predictive models in food microbiology. *J. Appl. Bacteriol.* 81:501–508.
- Ross, T. 1999. Predictive Food Microbiology Models in the Meat Industry. Meat and Livestock Australia, Sydney, Australia.
- Ross, T., P. Dalgaard, and S. Tienungoon. 2000. Predictive modeling of the growth and survival of *Listeria* in fishery products. *Int. J. Food Microbiol.* 62:231–245.
- 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.
- Stone, G., B. Chapman, and D. Lovell. 2009. Development of a log-quadratic model to describe microbial inactivation, illustrated by thermal inactivation of *Clostridium botulinum*. *Appl. Environ. Microbiol.* 75:6998–7005.
- USDA/FSIS. 2003. Control of *Listeria monocytogenes* in ready-to-eat meat and poultry products; final rule. Fed. Regist. 68:9 CFR Part 430.
- USDA/FSIS. 2007. South Carolina firm recalls chicken breast strips for possible *Listeria* contamination. Accessed Oct. 16, 2012. http://www.fsis.usda.gov/News_&_Events/Recall_012_2007_Release/index.asp.
- USDA/FSIS. 2011a. North Carolina firm recalls oven roasted chicken breast for possible *Listeria* contamination. Accessed Oct. 16, 2012. http://www.fsis.usda.gov/News_&_Events/Recall_097_2011_Release/index.asp.
- USDA/FSIS. 2011b. Texas firm recalls ready-to-eat chicken products for possible *Listeria* contamination. Accessed Oct. 16, 2012. http://www.fsis.usda.gov/News_&_Events/Recall_052_2011_Release/index.asp.
- Valdramidis, V. P., A. H. Geeraerd, J. E. Gaze, A. Kondjoyan, A. R. Boyd, H. L. Shaw, and J. F. V. Impe. 2006. Quantitative description of *Listeria monocytogenes* inactivation kinetics with temperature and water activity as the influencing factors; model prediction and methodological validation on dynamic data. *J. Food Eng.* 76:79–88.
- Xiong, R., G. Xie, A. S. Edmondson, R. H. Linton, and M. A. Sheard. 1999. Comparison of the Baranyi model with the modified Gompertz equation for modelling thermal inactivation of *Listeria monocytogenes* Scott A. *Food Microbiol.* 16:269–279.
- Zhu, M., M. Du, J. Cordray, and D. U. Ahn. 2005. Control of *Listeria monocytogenes* contamination in ready-to-eat meat products. *Comp. Rev. Food Sci. Food Safety* 4:34–42.