A Predictive Model for the Effect of Temperature and Predrying Treatments in Reducing *Listeria monocytogenes* Populations during Drying of Beef Jerky

YOHAN YOON,1 PANAGIOTIS N. SKANDAMIS,1 PATRICIA A. KENDALL,2 GARY C. SMITH,1 AND JOHN N. SOFOS1*

1Department of Animal Sciences and 2Department of Food Science and Human Nutrition, Colorado State University, Fort Collins, Colorado 80523, USA

ABSTRACT

The objective of this study was to model the effect of drying temperatures (52, 57, and 63°C) and predrying treatments on the inactivation of *Listeria monocytogenes* on beef jerky. Before drying, beef slices were inoculated with a 10-strain composite of *L. monocytogenes* and then treated with the following: (i) nothing (C), (ii) traditional marinade (M), or (iii) dipping in 5% acetic acid solution for 10 min, followed by M (AM). In addition, sequential stresses (exposure to 10% NaCl, followed by an adjustment of the pH to 5.0 and, subsequently, a water bath at 45°C) were applied to the inocula before beef contamination and drying at 63°C. Surviving *L. monocytogenes* were determined on tryptic soy agar plus 0.6% yeast extract (TSAYE) and on PALCAM agar at 0, 2, 4, 6, 8, and 10 h during drying. Data were modeled by a linear regression (treatment AM) and a logistic-based equation capable of fitting biphasic inactivation curves without initial shoulder (treatments C and M). The total log reductions expressed as the CFU per square centimeter of *L. monocytogenes* (3.9 to 5.1) for the samples treated with M (3.5 to 5.4) when compared with C were similar, whereas AM-treated samples had higher (6.1 to 6.8) reductions. All survival curves were characterized by an initial rapid decrease in populations within the first 2 h, which was followed by a secondary death phase at a lower rate. No significant (*P* > 0.05) differences in inactivation were observed due to drying temperatures in the range (52 to 63°C) tested. Inactivation differences between recovered counts of stressed and unstressed cells were significant (*P* < 0.05) in PALCAM but not in TSAYE. The acidified predrying treatment (AM) had higher pathogen inactivation during drying than other treatments, regardless of drying temperature. The models developed may be useful in designing effective drying processes for beef jerky.

Beef jerky is a common snack consumed in the United States because of its stability and nutritional value (8). However, documented outbreaks of microbial illness associated with the consumption of jerky indicate potential concerns. Levine et al. (19) showed that the cumulative prevalence of *Salmonella* and *Listeria monocytogenes* was 0.31 and 0.52%, respectively, from 1990 to 1999 in jerky samples obtained from U.S.-inspected plants. In addition, there have been outbreaks of foodborne illness related to *Salmonella* in beef jerky (11) and outbreaks of *Escherichia coli* O157:H7 in venison jerky (16). Recently, beef jerky products were recalled in New Mexico because of possible *Salmonella* contamination (29). To control *L. monocytogenes*, the Food Safety and Inspection Service of the U.S. Department of Agriculture (USDA/FSIS) enforces a “zero-tolerance” policy for ready-to-eat meat products, including jerky. The USDA/FSIS compliance guideline for jerky processing (30) recommends preheating meat slices in marinade to achieve an internal temperature of 71.1°C or dipping the meat slices in 5% acetic acid for 10 min before marination. Harrison and Harrison (14) reported that 10 h of drying at 60°C was sufficient to cause a 5-log reduction in *E. coli* O157:H7, *Salmonella* Typhimurium, and *L. monocytogenes* in marinated beef jerky. Calicioglu et al. (8, 9) suggested that the use of chemicals (acetic acid and Tween 20) in jerky marinades increased the inactivation of *E. coli* O157:H7 during drying. In addition, Calicioglu et al. (6, 7, 10) studied the effects of acid adaptation and organism variation in marinade composition on the survival of *Salmonella, E. coli* O157:H7, and *L. monocytogenes* for contamination on beef jerky during storage. The results showed that the modified marinades and the low water activity (*a*<sub>W</sub>) of the product provided antimicrobial effects against the pathogens, whereas previous acid adaptation of the inocula did not increase survival of the pathogens.

Mathematical models for the prediction of microbial inactivation in foods can be applied to this organism to determine the effects or interactions among processing parameters (20) and can also be used to estimate the effects of various combinations of variables in food environments (31). Thus, these models can help processors in formulating or reformulating food products that are safe for the consumer after validation in real food environments (23). The objective of this study was to develop models that could be applied by processors of beef jerky to determine lethality and to select appropriate treatments and processing times at various temperatures that would ensure the inactivation of *L. monocytogenes* during the processing of beef jerky.
MATERIALS AND METHODS

Preparation of inoculum. The inoculum consisted of the 10-strain mixture of L. monocytogenes described and prepared as reported by Bedie et al. (4) and Samelis et al. (24, 25). Each strain was incubated in 10 ml of tryptic soy broth (Difco, Becton Dickinson, Sparks, Md.) plus 0.6% yeast extract (TSBYE; Acumedica, Baltimore, Md.) at 30°C for 24 h. Stationary-phase cells were harvested by centrifugation (4,629 × g, 15 min, 4°C) and washed once with 100 ml of phosphate-buffered saline (PBS; pH 7.4; 2.0 g of KH₂PO₄, 1.5 g of Na₂HPO₄·7H₂O, 8.0 g of NaCl, and 0.2 g of KCl in 1 liter of distilled water). The resulting cell pellets were resuspended in PBS and then combined and diluted with PBS to a final volume of 100 ml to yield approximately 7 log CFU/ml. Sublethally stressed cells were obtained as follows: (i) the stationary-phase cell pellet was resuspended in 100 ml of a 10% NaCl solution dissolved in TSBYE to provide NaCl stress for 30 min, (ii) the pH of the 10% NaCl solution was then adjusted with lactic acid to pH 5.0, (iii) the solution (10% NaCl with pH 5.0) was transferred to a water bath (45°C) after 30 min to apply heat stress to L. monocytogenes cells for 30 min, and (iv) the sequentially stressed cells were harvested as described.

Preparation of meat slices. Beef inside rounds (vacuum packaged at 4°C for no longer than 3 months) were obtained from the Colorado State University Meat Science Laboratory (Fort Collins, Colo.). Frozen inside rounds were sliced to 0.6-cm thickness with a food slicer (model 610, Hobar Corp., Troy, Ohio) and cut into 8.7- by 4.0-cm pieces. Meat slices were vacuum packaged (20 by 25 cm, 3 mil std barrier, nylon/polyethylene bags, Koch, Kansas City, Mo.) and stored at −18°C (for 1 to 3 weeks) before thawing (4°C for 24 h) for analysis.

Inoculation procedure. A volume of 0.5 ml of the L. monocytogenes inoculum was applied to the surface of each beef slice and spread evenly over the entire surface area with a flame-sterilized bent glass rod. Beef slices that had been inoculated with L. monocytogenes inoculum was applied to the surface of each beef slice and spread over the entire surface area evenly with a flame-sterilized bent glass rod. Beef slices that had been inoculated with L. monocytogenes cells were kept at 4°C for 15 min to allow attachment; then, they were flipped over to inoculate the other side by the same procedure. The attached inoculum was approximately 6,3 log CFU/cm².

Predrying treatments. Predrying treatments included the following: (i) control (no treatment; C), (ii) marination with a traditional marinade (M), and (iii) dipping into 5% acetic acid solution (pH 2.5) for 10 min, followed by M (AM). The M treatment formulation for 1 kg of meat (2, 8) consisted of 60 ml of soy sauce (Kikkoman Foods, Walworth, Wis.), 15 ml of Worchester sauce (Heinz, Pittsburgh, Pa.), 0.6 g of black pepper (Hellman Seasoning and Ingredients Inc., Chicago, Ill.), 1.25 g of garlic powder (Excalibur Seasoning Co. Ltd., Pekin, Ill.), 1.5 g of onion powder (Excalibur), and 4.35 g of old hickory smoked salt (Tone Brothers Inc., Ankeny, Iowa). A volume of 34 ml of this marinade was spread evenly onto 20 inoculated beef slices with a glass rod. For the AM treatment, inoculated meat slices were dipped into a 5% (vol/vol) acetic acid (Malinckrodt Baker Inc., Paris, Ky.) solution (450 ml per 450 g of meat) for 10 min at ambient temperature, drained for 2 min, and then marinated with the traditional marinade as described for treatment M. The untreated slices (C) and slices treated with M and AM were covered with aluminum foil and held at 4°C for 24 h prior to drying (8).

Drying. Inoculated, treated, and refrigerated (4°C for 24 h) meat slices were placed on trays of American Harvest Gardenmaster dehydrators (model FD-1000, Nesco, Chaska, Minn.) and dried for 10 h at 52, 57, or 63°C. Dehydrators were cylindrical and were operated with three drying trays, as recommended by the company for optimal operation. The base unit of the dehydrator was designed to generate hot air blowing upward through the sides and a channel in the middle of the trays. The air temperature was monitored with thermocouples (type K beaded probes, Pico Technology Ltd., Cambridge, UK) inserted into the middle channel of each dehydrator. The empty trays of dehydrators were preheated for approximately 20 min before meat slices were loaded on the trays; approximately 20 meat slices were placed on each tray. The temperature of the meat slice surfaces on the middle trays was monitored with thermocouples (Pico) and recorded with real-time data-recording software (Pico) during dehydration.

Analyses. Two samples per treatment were individually transferred into 0.68-liter-capacity sterile plastic bags (Nasco, Modesto, Calif.). Samples were taken after inoculation and at 0 (after a 24-h marination at 4°C), 2, 4, 6, 8, and 10 h during drying. A volume of 25 ml of 0.1% sterile buffered peptone water (BPW; Difco, Becton Dickinson) was added to each sample bag prior to pummeling (Masticator, U.L. Instruments, Barcelona, Spain) for 2 min (120 strokes per min) at ambient temperature. After serially diluting each sample in BPW, 0.1-ml portions of the two samples were separately plated onto each of two duplicate agar plates. Total bacterial populations were enumerated on tryptic soy agar (Difco, Becton Dickinson) plus 0.6% yeast extract (TSAYE; Acumedica), and PALCAM (Difco, Becton Dickinson) agar was used as a selective medium for L. monocytogenes enumeration. All plates were incubated at 30°C for 48 h. The enumeration detection limit was −0.5 log CFU/cm².

The pH values of samples used for microbial analyses were determined with a pH meter (Denver Instruments, Arvada, Colo.) having a glass pH electrode (Denver Instruments). The aₜ values of samples were measured by cutting one beef jerky slice into small pieces to fit into the plastic aₜ cup of an AquaLab Model Series 3 aₜ meter (Decagon Devices, Inc., Pullman, Wash.).

Statistical analysis and model development. The study was repeated two times with two samples for each replication (n = 4). A complete factorial design ([52, 57, and 63°C] × [C, M, and AM] × [0, 2, 4, 6, 8, and 10 h]) was used in this study. The drying temperatures (52, 57, and 63°C) and marination treatments (C, M, and AM) were used in the development of the models describing the survival of L. monocytogenes. Microbial data from sublethally stressed cells were not included in the model development because this inoculum was tested only at a drying temperature of 63°C. Only microbial data recovered with TSAYE were used to develop the models in order to include injured cells. The predicted bacterial populations of C and M treatments were modeled by the logistic Kamau equation (equation 1) (15). The parameters for this model were obtained with the research data graphic statistical software Fig.P, version 2.5 (12).

\[ \log S = \log \left[ \frac{2f}{1 + \exp^{-a}} \right] + \frac{2(1 - f)}{1 + \exp^{-b}} \]  

where f and (1 − f) represent the two phases of the bacterial survival curve (differing with respect to drying); a and b are the death rates for the two phases of the bacterial survival curve, respectively; and t represents the drying time. The two phases describe the two slopes (rates) for the exponential decline of bacterial populations.

The predicted bacterial populations for AM-treated samples were modeled by an alternative function, which is the piecewise linear regression function (equation 2), by SAS (26). The logistic Kamau et al. (15) equation was not fitted on the microbial survival data of the AM treatment because of a very rapid death rate during drying.
the first death phase. Piecewise linear regression can be used with indicator variables when the regression of $y$ on $x$ follows a particular linear relation in some range of $x$ but follows a different linear relation elsewhere.

$$\log S = \beta_0 + \beta_1 x_1 + \beta_2(x_2 - x_2) x_2 + \epsilon \quad (2)$$

where $\beta_0$ = intercept (initial bacterial populations), $\beta_1$ = slope of first phase, $\beta_2$ = differential effect in the slope in the negative direction from $x_p$ to zero, $\beta_1 + \beta_2$ = slope of second phase, $x_p$ = point where the slope changes, $x_1 = time$, and $x_2 = 1$ if $x_1 > x_p$; otherwise, $x_2 = 0$. Each of the above variables for this equation is an indicator variable. This model also assumes that the two cell fractions are killed exponentially at different independent rates.

In general, acetic acid (dissociation constant $K = 1.86 \times 10^{-5}$) may cause greater inactivation of L. monocytogenes than

$$y = y_0 + \frac{y_{end} - y_0}{1 + e^{-(x-x_0)}} \quad (3)$$

where $y = temperature$ or $aw$, $y_{end} = maximum temperature$ or $aw$ plateau, and $x = slope of the curve$; when $x > 0$, the curve increases with time, and $y_0 = minimum temperature or aw plateau$, $\mu = time$ at maximum slope of the curve, and $t = time$.

Model validation. The fitted parameters (mean values of each parameter, regardless of temperature) were used to estimate the survival of L. monocytogenes under given conditions and at given times in accordance with the logistic Kamau et al. (15) and piecewise linear equations. The modified bias and accuracy factors ($B$ and $A$, respectively) (22, 28) were used to evaluate the performance of the developed predictive model:

$$B = \exp \left[ \frac{\sum{\ln y_{predicted} - \ln y_{observed}}}{n} \right] \quad (4)$$

$$A = \exp \left[ \frac{\sum{\ln y_{predicted} - \ln y_{observed}}^2}{n} \right] \quad (5)$$

where $y$ is the response variable, and $n$ is the number of observations. Perfect agreement between predictions and observations leads to bias and accuracy equal to 1. A value higher than 1 for the bias factor indicates that predicted values are larger than observed values. The $B$ and $A$ values were separately obtained only for the C and M treatments. Because the natural logarithm in equations 4 and 5 does not account for negative integers, the calculation of $B$ and $A$ in the AM treatment, which had negative log CFU per square centimeter values, was not possible.

RESULTS AND DISCUSSION

Effect of temperature and aw. The average temperature of beef slices reached the target (52, 57, and 63°C) after 2.5 h of dehydration (Fig. 1A). Changes in $aw$ for the samples of the various treatments showed similar trends, and no differences in initial aw values among treatments were observed; thus, only aw data for treatment M are presented for each of the different drying temperatures (Fig. 1B). The aw of beef jerky samples decreased considerably during dehydration, and the final aw of products dried at 63°C was lower than that of products dried at 52 and 57°C, except for AM-treated samples (Fig. 1B). As the temperature of the beef jerky surface increased, aw and bacterial populations decreased; data for treatment M at 57°C are shown in Figure 1C.

The four-parameter logistic function used to describe the trends of aw and drying temperatures showed good coefficients of determination ($R^2 = 0.900$ to 0.992 for aw; $R^2 = 0.978$ to 0.995 for drying temperatures) (Fig. 1A and 1B); and residuals were normally distributed (Fig. 2A and 2B); this equation could be applied to model aw and drying temperature in other studies.

pH values were constant during drying. The pH values of samples from treatments C and M were similar to the initial values during the dehydration process (5.35 to 5.55); however, the pH of samples treated with AM ranged from 4.34 to 4.65 during the dehydration process (data not shown). Even though the pH value of the 5% acetic acid solution was approximately 2.5, the pH was buffered by the beef tissue. However, the lower pH of the AM treatment may have contributed to cell inactivation in the dry environment.

Acetic acid solution sensitizes L. monocytogenes cells. Bacterial populations were reduced by 0.1 to 0.7 log CFU/cm² (C and M) and by 0.8 to 1.5 log CFU/cm² (AM) after 24 h of marination at 4°C, depending on the agar media used (time 0) (Fig. 3). After 10 h of drying, the AM-treated samples had higher (6.1 to 6.8 log CFU/cm²) reductions of L. monocytogenes than the samples of the C (3.9 to 5.1 log CFU/cm²) and M (3.5 to 5.4 log CFU/cm²) treatments ($P < 0.05$). Differences between recovered counts of stressed and unstressed cells were significant ($P < 0.05$) in PALCAM but not in TSAYE (Fig. 3). The AM treatment caused greater cell injury (cell injury = log CFU/cm² of TSAYE − log CFU/cm² of PALCAM) than in samples subjected to C and M predrying treatments, especially within the first 2 to 4 h of drying (Fig. 4). The injury of cells caused by dipping the beef jerky slices in acetic acid (treatment AM) for 10 min prior to drying may have contributed to the more extensive destruction observed for these cells due to low aw and the heat during drying. A similar conclusion was reached by Calcioglu et al. (9). In general, acetic acid (dissociation constant $K = 1.86 \times 10^{-5}$) may cause greater inactivation of L. monocytogenes than
other organic acids, such as lactic acid (dissociation constant \( K_a = 8.00 \times 10^{-4} \)) and citric acid (dissociation constant \( K_a = 1.38 \times 10^{-4} \)), due to its lower dissociation constant \( (I) \).

Death rates as affected by drying temperatures. Regardless of treatment, no significant \((P \geq 0.05)\) differences in death rates were observed among drying temperatures (Table 1). Once individual survival responses (curve fitting) were expressed, by the use of four curve fittings \((n = 4)\) in each predrying treatment, the variation among samples resulted in large standard errors in death rates during drying (Table 1). Therefore, the large standard error allowed no detection of significant \((P \geq 0.05)\) differences in death rates among drying temperatures.

It has been reported \((3, 13)\) that the \(\sigma^B\) factor and the Ctc protein, which belongs to a family of unknown proteins, confer resistance of \(L.\) monocytogenes to osmotic stress. The \(\sigma^B\) factor and Ctc protein of \(L.\) monocytogenes may be activated at the beginning of drying and may lead to a resistance to low \(a_w\) after 2 to 3 h of drying. This phenomenon may be the reason that all survival curves were characterized by an initial rapid decrease in populations within the first 2 to 3 h of drying, which was followed by what can be described as a secondary, slower decline in the death rate (tailing) (Table 1 and Fig. 5). Once \(L.\) monocytogenes is exposed to osmotic stress, it accumulates osmoprotectants, such as glutamate, proline, trimethyl ammonium compound glycine betaine, and the structurally related trimethyl amino acid \(\gamma-N\)-trimethyl aminobutyrate \((5, 18, 21)\). The accumulation of these osmoprotectants occurs through transportation rather than through de novo synthet-
FIGURE 3. Listeria monocytogenes counts on beef jerky recovered on tryptic soy agar with the addition of 0.6% yeast extract (TSAYE) and PALCAM following inoculation and marination at 4°C for 24 h and during drying at 52 (A), 57 (B), and 63°C (C) for 10 h and the survival of sublethally stressed L. monocytogenes (prior to inoculation) on beef jerky following inoculation and marination and during drying at 63°C (D). Al, after inoculation; C, control; M, traditional marination; AM, dipping into 5% acetic acid, followed by M.
FIGURE 4. Illustration of the differences observed between tryptic soy agar with the addition of 0.6% yeast extract (TSAYE) and PALCAM agar counts of *Listeria monocytogenes* during drying of beef jerky at 52 (A), 57 (B), and 63°C (C) for 10 h and the survival of sublethally stressed *L. monocytogenes* (prior to inoculation) on beef jerky during marination and drying at 63°C (D). AI, after inoculation; C, control; M, traditional marination; AM, dipping into 5% acetic acid, followed by M.

TABLE 1. Parameter estimates (standard deviations) obtained by fitting survival data with the logistic Kamau et al. (15) equation (C, M) and piecewise linear regression (AM)

<table>
<thead>
<tr>
<th>Temp (°C)</th>
<th>Treatment</th>
<th>( \beta_0 )</th>
<th>( \beta_1 ) (log CFU/cm²)</th>
<th>( \beta_2 ) (log CFU/cm²)</th>
<th>( (\beta_1 + \beta_2) ) (log CFU/cm²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>52</td>
<td>C</td>
<td>5.485 (0.163)</td>
<td>−2.463 (0.350)</td>
<td>2.298 (0.505)</td>
<td>−0.165</td>
</tr>
<tr>
<td></td>
<td>M</td>
<td>5.485 (0.163)</td>
<td>−2.535 (0.177)</td>
<td>2.426 (0.216)</td>
<td>−0.110</td>
</tr>
<tr>
<td>57</td>
<td>AM</td>
<td>5.553 (0.079)</td>
<td>−2.524 (0.391)</td>
<td>2.750 (0.653)</td>
<td>−0.249</td>
</tr>
<tr>
<td>63</td>
<td>AM</td>
<td>5.553 (0.079)</td>
<td>−2.524 (0.391)</td>
<td>2.750 (0.653)</td>
<td>−0.249</td>
</tr>
</tbody>
</table>

\( a \) Death rate of first phase.

\( b \) Death rate of second phase.

\( c \) Effect of drying temperature (within a column) was not significant (\( P > 0.05 \)). C, control; M, traditional marination; AM, dipping into 5% acetic acid, followed by M; \( f \), first phase of bacterial survival curves; \( a \), \( b \), death rates for the first and second phases of bacterial survival curves, respectively; \( \beta_0 \), intercept (initial bacterial populations); \( \beta_1 \), slope of the first phase in bacterial survival curves; \( \beta_2 \), differential effect in the slope in the negative direction from the point where the slope changes to zero; \( \beta_1 + \beta_2 \), slope of the second phase in bacterial survival curves.
TABLE 2. Predicted and observed populations of *Listeria monocytogenes* on beef jerky during 10 h of dehydration at 60°C

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Time (h)</th>
<th>Predicted (SD)</th>
<th>Observed (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>C</td>
<td>0</td>
<td>6.4 (0.2)</td>
<td>6.4 (0.2)</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>4.4 (0.3)</td>
<td>4.3 (0.4)</td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>3.6 (0.3)</td>
<td>2.7 (0.4)</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>2.8 (0.3)</td>
<td>2.2 (0.4)</td>
</tr>
<tr>
<td>M</td>
<td>0</td>
<td>6.2 (0.6)</td>
<td>6.2 (0.6)</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>4.2 (0.3)</td>
<td>3.7 (0.2)</td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>3.5 (0.3)</td>
<td>2.9 (0.5)</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>2.7 (0.3)</td>
<td>2.2 (0.7)</td>
</tr>
<tr>
<td>AM</td>
<td>0</td>
<td>5.6 (0.4)</td>
<td>5.6 (0.4)</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>0.2 (0.5)</td>
<td>0.5 (0.4)</td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>−0.3 (0.5)</td>
<td>0.2 (0.5)</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>−0.8 (0.0)</td>
<td>−0.5 (0.0)</td>
</tr>
</tbody>
</table>

C, control; M, traditional marinade; AM, dipping into 5% acetic acid, followed by M.

Development of the models. Because the estimated parameters of the logistic Kamau et al. (15) and piecewise linear equations were not significantly (*P* ≥ 0.05) different among drying temperatures (52, 57, and 63°C), it was not necessary to further express each parameter as a function of drying temperature. Therefore, the average values of each parameter for all drying temperatures (52, 57, and 63°C) were further expressed as a function of *a*, *f*, and *b* for C and M and *β*1 and *β*2 for the AM treatment by the logistic Kamau et al. (15) and piecewise linear equations, respectively, to predict the response of *L. monocytogenes* during 10 h of dehydration at 60°C. Consequently, the final models predicting the survival of *L. monocytogenes* in beef jerky are as follows:

C treatment:

\[
\log S = \log \left[ \frac{2.0916}{1 + e^{(3.473t)}} + \frac{2(1 - 0.916)}{1 + e^{(0.660t)}} \right]
\]

M treatment:

\[
\log S = \log \left[ \frac{2.0949}{1 + e^{(3.020t)}} + \frac{2(1 - 0.949)}{1 + e^{(0.573t)}} \right]
\]

AM treatment:

\[
\log S = \beta_0 - 2.508X_1 + 2.333(X_1 - 2)X_2 \begin{cases} X_2 = 1, & X_1 > 2 \\ otherwise & X_2 = 0 \end{cases}
\]

Validation of the models. To validate the models, the *L. monocytogenes* data obtained by Calicioglu et al. (9) were compared to the predicted populations, which were estimated by the logistic Kamau et al. (15) equation (C and M) and piecewise linear equation (AM). The observed and predicted counts of *L. monocytogenes* are shown in Table 2. Moreover, the entire validation is represented in Figure 6, where predicted values are plotted against observed values. Linear regression was then fitted to estimate the correlation between predicted and observed values (*R^2* = 0.979; standard error of estimate = 0.4893 log CFU/cm^2). The bias (*B*) and accuracy (*A*) factors were 1.226 (C) and 1.137 (M) and 1.267 (C) and 1.164 (M), respectively (perfect agreement would be 1.0). The bias factor of 1.226 indicates that the predicted *L. monocytogenes* and may cause *L. monocytogenes* resistance to osmotic stress (5).
ACKNOWLEDGMENTS

This work was funded by the Cooperative State Research, Education and Extension Service (CSREES) of the U.S. Department of Agriculture and by the Colorado State University Agricultural Experiment Station.

REFERENCES

12. Fig.P Software Cooperation. 1995. Fig.P®, version 2.5. Fig.P Software Cooperation, Durham, N.C.
20. Lihono, M. A., A. F. Mendonca, J. S. Dickson, and P. M. Dixon. 2003. A predictive model to determine the effects of temperature, acidification, and extension service (CSREES) of the U.S. Department of Agriculture and the Colorado State University Agricultural Experiment Station.


