Thermal Injury and Recovery of *Salmonella enterica* Serovar Enteritidis in Ground Chicken with Temperature, pH, and Sodium Chloride as Controlling Factors†

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ABSTRACT

Cells of *Salmonella enterica* serovar Enteritidis were grown at 25 and 35°C, heat injured (55, 60, and 62.5°C), and recovered in tryptic soy broth (TSB) at various NaCl concentrations (2.0 and 3.5%) and pH levels (5.5 and 6.5). To assess the interactions of growth temperature, heating temperature, NaCl concentration and pH on the thermal injury and recovery of *Salmonella* Enteritidis in ground chicken, a randomized design with each experimental combination was used. When a logistic equation for nonlinear survival curves was used, D-values of cells of *Salmonella* Enteritidis grown at 25°C were 7.60, 5.73, and 4.81 min at 55, 60, and 62.5°C, respectively. For cells grown at 35°C, the D-values were 12.38, 7.45, and 5.70 min at 55, 60, and 62.5°C. The influence of tryptic soy agar and double modified lysine agar (DMLIA) on the recovery of heat-injured cells was determined. Recovery was significantly reduced on DMLIA at increased pH levels and NaCl concentrations. Higher numbers of cells were recovered in TSB with 2.0% NaCl than in TSB with 3.5% NaCl. It was observed that the rate of recovery of heat-injured cells was similar at each pH. Therefore, a pH range of 5.5 to 6.5 does not have a major inhibitory effect on the recovery of *Salmonella* Enteritidis.

*Salmonella* is a leading cause of foodborne illness in the United States and continues to be of public health significance because of its ability to withstand harsh environments. Salmonellosis, the disease caused by this organism, is characterized by symptoms such as diarrhea, fever, and abdominal pain. Foodborne salmonellae are estimated to cause approximately 1.3 million illnesses, 15,000 hospitalizations, and 500 deaths per year in the United States (2). In an effort to reduce the incidence of this pathogen, the U.S. Department of Agriculture Food Safety and Inspection Service has implemented a 7-log relative reduction in population counts of *Salmonella* for fully and partially cooked poultry products (8).

*Salmonella enterica* serovar Enteritidis, a facultative anaerobe, is an etiological agent of gastroenteritis. A recent FoodNet surveillance of foodborne illnesses has shown that the overall incidence of *Salmonella* has decreased by 8% from 1996–1998 to 2004; however, the incidence of *Salmonella* Enteritidis has remained at approximately the same level (3). Chicken had not been previously reported as a cause of sporadic cases of *Salmonella* Enteritidis or as a frequent cause of *Salmonella* Enteritidis outbreaks in the United States until recently. In 2004, a case-control study in five Foodborne Disease Active Surveillance Network surveillance areas identified chicken consumption as a risk factor for sporadic *Salmonella* Enteritidis infections in the United States (10).

Microbial growth in foods is controlled primarily by pH, water activity, and storage temperature, with additional factors such as preservatives, modified atmosphere packaging, or heat treatment also contributing. These factors are usually used in combination because singly they would not be sufficient to control microbial growth (7).

The control and elimination of *Salmonella* by using a heat treatment are a vital method in the safe preparation of many foods. Insufficient processing, cooking, and reheating are major causative factors in foodborne illnesses (9). An effective thermal process is required to control the potential hazard of *Salmonella* in cooked meat products. An important factor of the heating step is to determine the pathogen’s heat resistance. Overestimating the heat resistance will affect product quality, while underestimating increases the possibility that the organism will survive the heat treatment or cooking process. The organism studied in this work has been associated with foodborne illness caused by insufficient heating. A variety of foods have been implicated in *Salmonella* infections, including meat and poultry (6).

Destruction of microorganisms by heat may deviate from linear declines in log numbers with time. Survival curves may exhibit an initial lag period followed by exponential decline. In some instances researchers have observed a tailing of more persistent bacteria that decline at a lower rate than do the majority of the cells (12). Selective agents such as bile salts or novobiocin are added to media in order to detect bacteria in foods. However, these agents may inhibit repair of injured bacteria or may kill them.
There has been some research pertaining to the heat resistance of *Salmonella* in chicken meat, and the reported D-values at 55 to 70°C range from 30.1 to 0.238 min. These studies indicate that most *Salmonella* serovars do not have an unusual heat resistance, with the exception of *Salmonella* Senftenberg, which has been shown to be resistant at unusually high temperatures.

The effect of heat on inactivation and destruction of *Salmonella* has been the subject of several investigations, but the effect of sublethal heat or thermal injury has received much less attention. This study seeks to relate this thermal injury to the recovery of *Salmonella* Enteritidis cells in ground chicken breast meat under various NaCl concentrations and pH levels. This investigation had a threefold purpose: (i) to determine any differences in the recovery rates for *Salmonella* Enteritidis cells thermally injured in ground chicken breast meat and grown on selective and nonselective media; (ii) to assess the thermal inactivation of *Salmonella* Enteritidis in ground chicken breast meat by determining D-values; and (iii) to determine the effects of pH and NaCl concentration on the recovery of sublethally injured *Salmonella* Enteritidis cells in ground chicken breast meat. The findings of this study will help to estimate the time required achieving specific log cycle reductions of *Salmonella* Enteritidis at specified temperatures and help the food industry in determining the influence of salts and pH on the recovery of heat-injured *Salmonella* Enteritidis cells.

**MATERIALS AND METHODS**

**Bacterial strain.** *Salmonella enterica* subsp. *enterica* serovar Enteritidis deposited as *Salmonella enteritidis* (Gaertner) (ATCC 13076) was purchased from the American Type Culture Collection (Rockville, Md.) and used in this study. Outbreaks of *Salmonella* Enteritidis have commonly been associated with shell eggs; however, recently chicken consumption has been identified as a risk factor. The heat resistance of *Salmonella* is highly influenced by the strain tested, the type of experiment, culture conditions prior to the experiment, heating menstruum, and recovery conditions. Heat resistance data for *Salmonella* are still scarce in chicken meat.

The strain pellet was rehydrated with 0.5 ml of brain heart infusion (BHI, pH 7.4, containing 200 g of infusion from calf brain, 250 g of infusion from beef heart, 10 g of proteose peptone or gelysate, 5 g of NaCl, 2.5 g of NaHPO₄, 2 g of dextrose, and 1 liter of distilled water) broth. A 10-µl loop of the working slant culture was transferred to 10 ml of BHI broth and incubated for 24 h at the appropriate experimental growth temperature (25 or 35°C). These cultures were not used in the heating studies to eliminate the presence of freeze-damaged cells. A transfer of 0.1 ml of the incubated cultures was made to 10 ml of BHI broth and incubated for 24 h at the appropriate experimental growth temperature (25 or 35°C). The inocula for use in heating studies were prepared by transferring 0.1 ml of the culture free of freeze-damaged cells to 250 ml of BHI broth that was incubated for 24 h at the appropriate experimental growth temperature (25 or 35°C). On the day of each experiment, test cultures were pipetted into 50-ml centrifuge tubes and centrifuged for 15 min at 3,000 × g in a Sorvall RC-5B Refrigerated Super-speed Centrifuge (Dupont Instruments, Newtown, Conn.). Following centrifugation, the supernatants were discarded from each tube and the pellets were each suspended in 2 ml of 0.1% PW containing 1 g of peptone and 1 liter of distilled water. The suspensions were combined in a sterile conical vial to obtain the inocula with a target level of 8 or 9 log CFU/ml prior to inoculation of the ground chicken breasts. The population densities of the inocula were enumerated after diluting in 0.1% PW by spread plating, in duplicate, onto TSA and incubating at 35 ± 1°C for 48 h.

**Sample preparation and inoculation.** The inoculum (0.1 ml) was added to 25 g of thawed ground chicken breasts in Whirl-Pak filter bags. Each inoculated sample of meat was pumped with a Stomacher Lab-Blender 400 (Cooke Laboratory Products, Alexandria, Va.) for 2 min to ensure even distribution of *Salmonella* Enteritidis throughout the samples. Filter bags containing ground chicken breasts samples inoculated only with 0.1 ml of 0.1% sterile PW were used as negative controls. After pumping, the samples were formed into uniform sizes (8 cm [diameter] by 0.5 cm [thickness]) by using a circular template. The contact surfaces of the filter bags were pressed firmly against the meat sample within the filter bag, creating a close fit that prevented air pockets within the filter bag.

**Experimental design.** To assess the interactions of growth temperature, heating temperature, NaCl concentration, and pH on the thermal injury and recovery of *Salmonella* Enteritidis in ground chicken breasts, a randomized design with the controlling factors of temperature and media was used. Levels of the factors studied were as follows: growth temperature, 25 and 35°C; heating temperatures, 55, 60, and 62.5°C; NaCl concentration, 2.0 and 3.5%; and pH, 5.5 and 6.5.

Twenty-four different design points of the above factors were studied (Table 1). For each experimental combination, three replicates were performed. A replicate consisted of one sample subjected to the appropriate treatment.

**Heating protocol.** To ensure that a consistent starting temperature was used in each heating study, the sampling bags were closed with tabs folded three times and were placed at 4 ± 2°C for 1 h. Each sample was inoculated with a known concentration...
TABLE 1. Randomized design used to assess the effects of interactions of growth temperature and heating temperature on the heat resistance of Salmonella Enteritidis in ground chicken breast meat to investigate the effects of NaCl concentrations and pH levels on the recovery of heat-injured Salmonella Enteritidis cells

<table>
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<th>pH</th>
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*Salmonella* Enteritidis cells were grown at experimental temperatures of 25 or 35°C and underwent heat treatments at 55, 60, and 62.5°C in a circulating water bath. Heat-injured cells subjected to 55, 60, and 62.5°C were recovered in TSB with various NaCl concentrations (2.0 and 3.5%) and pH levels (5.5 and 6.5). TSB adjusted to pH 5.5 or 6.5 using 1 N HCl.

a Heat-injured salmonellae were grown at experimental temperatures of 25 or 35°C and underwent heat treatments at 55, 60, and 62.5°C in a circulating water bath. Heat-injured cells subjected to 55, 60, and 62.5°C were recovered in TSB with various NaCl concentrations (2.0 and 3.5%) and pH levels (5.5 and 6.5). TSB adjusted to pH 5.5 or 6.5 using 1 N HCl.

b Heat-injured cells subjected to 55, 60, and 62.5°C were recovered in TSB with various NaCl concentrations (2.0 and 3.5%) and pH levels (5.5 and 6.5). TSB adjusted to pH 5.5 or 6.5 using 1 N HCl.

c Thermal injury conditions. The thermal injury conditions were 25°C, 55°C, and 60°C.
d Recovery of thermally injured bacteria. The pulsed samples containing the appropriate experimental TSB medium were incubated aerobically at 35 ± 1°C. Aliquots were taken at predetermined time intervals (6, 12, 18, and 24 h) from each sample. The quantitation of recovery of heat-injured cells was performed only on cells at the time interval at each heat treatment in which the lowest detectable numbers of *Salmonella* Enteritidis bacteria were present on both DMLIA and TSA. This occurred at 45 min at 55°C and 8 min at both 60 and 62.5°C. These cells were diluted into 0.1% PW, and appropriate dilutions (0.1 ml) were surface plated, in duplicate, onto TSA and DMLIA. Also, 0.1-ml aliquots of undiluted suspensions were surface plated where necessary. All plates’ contents were incubated at 35 ± 1°C for at least 48 h prior to counting colonies.

calculation of D-values. The primary thermotolerance response was a switch to nonlinear inactivation kinetics represented by a subpopulation of more resistant bacteria that died at a lower rate. Therefore, D-values (negative reciprocal of the slope) were determined by fitting regression lines to the experimental data points by a logistic equation by using a Data Fit program for Windows, version 7.1 (Oakdale Engineering, Oakdale, Pa.). Two D-values were calculated: D-value of the major population (D1) and the D-value of the subpopulation (D2).

Statistical analyses. The bacterial counts were transformed to log counts and analyzed by analysis of variance by using SigmaStat (Systat Software, Point Richmond, Calif.) to determine if there were significant differences among treatments.

results and discussion

Heat treatment is one of the most common methods used in food processing to destroy or inactivate microorganisms. The higher the treatment temperature or the longer the treatment time, the greater the efficiency of the process becomes. However, heat treatments do not cause the same degree of injury to all cells within a population. Some cells may die; others may survive and recover from the thermal injury. The ability of heat-injured salmonellae to repair sublethal damage in different environments is of great significance to the food industry and consumers. Sublethally injured cells exhibit a longer lag phase than do uninjured bacteria. Consequently, there is an increased time for the organisms to attain a critical level in foods (7). These injured organisms may not be detected in postproduction microbiological testing and may recover within the food product and pose a direct consumer risk. Recently, chicken consumption has been identified as a risk factor for sporadic...
illness caused by *Salmonella* Enteritidis (10). If the conditions that inhibit growth and survival of this organism in chicken products are identified, they may be utilized by processors and consumers to reduce the incidence of foodborne illness. Therefore, the present study examined the sublethal injury and the rate of recovery of *Salmonella* Enteritidis cells in ground chicken breast meat.

Some components present in foods can protect bacterial cells from thermal injury by stabilizing membranes or other cellular structures (6). Fat levels in meat have been shown to influence bacterial heat resistance (10). Chicken breasts with 1 to 4% fat were used in the present study. It is possible that the level of fat in the ground chicken meat affected the heat resistance of the *Salmonella* Enteritidis cells; however, a study by Juneja and others (8) revealed that fat levels below 9% did not give cells a significantly higher heat resistance. In chicken products, enhanced survival of *Salmonella* Enteritidis cells may be of great concern if the resistance is sustained by the altered cells.

BAX analyses of uninoculated samples revealed that *Salmonella* was not initially present in the meat samples used in this study. The initial inoculum range of 8 or 9 log (CFU/ml) was obtained in all the trials. *Salmonella* Enteritidis cells heated at 55, 60, and 62.5 ± 0.2°C exhibited nonlinear declines in surviving cells with time. This switch to nonlinear inactivation kinetics observed in the present study may be due to an adaptive survival mechanism brought on by physiological and biochemical changes in the heat-injured *Salmonella* Enteritidis cells. Within the population of cells, there were inevitably different degrees of heat resistance due to differences in stages of the microbial life cycle. Cells in stationary phase are more resistant than those in lag or exponential phase (8). The heterogeneity of heat resistance levels within the population probably caused the nonlinearity of the survivor curves at each heating temperature. The numbers of survivors were determined by plating heated cells onto selective and nonselective media. Survivor curves were constructed by plotting the number of log CFU per milliliter against heating time.

A representative example of a survival curve at 55°C is depicted in Figure 1. The curves differed in slope, depending upon the growth temperature. It was observed that cells grown at 35°C prior to the heat treatments exhibited a greater heat resistance than did cells grown at 25°C. After heat treatment at 55°C for 90 min, the viable population of *Salmonella* Enteritidis cells grown at 35°C, as determined by its ability to grow on nonselective media (TSA), decreased from 8 log to 3 log CFU/ml (Fig. 1). After 45 min, the uninjured cell population, as determined by its ability to grow on selective media (DMLIA), decreased from 8 log to 3 log CFU/ml, and colonies were not detected at later time intervals. The populations of *Salmonella* Enteritidis cells grown at 25°C exhibited a more rapid decrease than that of the cells grown at 35°C. After heat treatment at 55°C for 90 min, the viable population decreased from 8 log to <2 log CFU/ml. After 30 min, the uninjured cell population decreased from 8 log to <2 log CFU/ml, and colonies were not detected at later time intervals, as determined by plating onto DMLIA.

When the recovery medium was TSA, the *D*-value of the major population (*D*₁) increased from 7.6 to 12.38 min and the *D*-value of the subpopulation (*D*₂) from 7.45 to 41.88 min as the growth temperature prior to heating increased from 25 to 35°C (Table 2). Similar increases in *D*₁ and *D*₂ were observed at 60 and 62.5°C. When the recovery medium was DMLIA, the increase in *D*-values with the increase in growth temperature from 25 to 35°C

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**Table 2. Heat resistance (expressed as D-values in minutes) for *Salmonella Enteritidis* in ground chicken breast meat heated at 55 to 62.5°C and subsequently plated onto selective and nonselective media**

<table>
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<tr>
<th>Plating medium</th>
<th>Temp (°C)</th>
<th>Growth temp (°C)</th>
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<th>CMD (r²)</th>
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<td>7.60 ± 0.38</td>
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<td>35</td>
<td>12.38 ± 1.04</td>
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<td>60.0</td>
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<td>5.73 ± 0.45</td>
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<td>7.45 ± 1.53</td>
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<td></td>
<td>62.5</td>
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<td>4.81 ± 0.79</td>
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<td></td>
<td>62.5</td>
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<td>DMLIA</td>
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<td>1.00 ± 0.20</td>
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<td>62.5</td>
<td>35</td>
<td>0.21 ± 0.01</td>
<td>0.93</td>
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</tbody>
</table>

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* D-value of the major population (*D*₁) and the *D*-value of the subpopulation (*D*₂) of three replicate experiments expressed as mean ± standard error.

"Survivors of thermal treatments were plated onto both nonselective (TSA) and selective (DMLIA) agar.

Samples of ground chicken breast meat were heated at 55, 60, or 62.5°C.

Two different growth temperatures (25 or 35°C) were used to provide the inocula used to inoculate the ground chicken breast meat samples.

Coefficient of multiple determination.

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**Figure 1. Thermal injury of *Salmonella Enteritidis* cells grown at 25 and 35°C and enumerated onto TSA and DMLIA after heat treatment at 55°C. Each point represents the mean ± standard error of the mean of the log CFU per milliliter from three replicates.**
was observed only when the heating temperature was 55°C; the $D_1$ increased from 1.00 to 1.29 min and $D_2$ from 38.13 to 44.26 min (Table 2).

When comparing the results in this study with those reported from other studies, it should be kept in mind that meat species, muscle type, pH, fat content, and other environmental factors including the method of enumeration may influence the bacterial heat resistance. Also, certain strains of *Salmonella* are less resistant to and less tolerant of changes in temperature (17). The thermal inactivation data in this study were inconsistent with those reported elsewhere (9, 16). However, the medium conditions for enumeration of bacteria were different from previous studies (9, 16). In addition, the current study used a single strain of *Salmonella*, while other studies in the published literature conducted thermal inactivation of *Salmonella* cocktails in chicken products (9, 16). *Salmonella Enteritidis* was used as the target pathogen in this study due to its recent recognition as a risk factor for sporadic infection through chicken consumption (10). In comparing the results of this study with previous literature, Juneja and others (9) tested an eight-strain *Salmonella* cocktail in chicken (7% fat) at 58 to 65°C and obtained $D$-values of 7.08 min at 58°C, 5.20 min at 60°C, and 1.36 min at 62.5°C. If one were using these estimates of $D$-values and assuming zero lag time, the times needed to obtain a 7-log relative reduction would be about 49.56 min at 58°C, 36.4 min at 60°C, and 9.52 min at 62.5°C. In a study by Murphy and others (16), $D$-values in chicken breast meat of 30.1, 5.88, and 2.51 min were obtained at 55, 60, and 62.5°C, respectively. If one were using these estimates of $D$-values and assuming zero lag time, the times needed to obtain a 7-log relative reduction would be about 210.7 min at 55°C, 41.16 min at 60°C, and 17.57 min at 62.5°C. In the present study, the times needed to obtain a 7-log reduction would be 86.66 min at 55°C, 52.15 min at 60°C, and 39.9 min at 62.5°C.

Recovery of *Salmonella Enteritidis* was performed on cells subjected to 55°C for 45 min, 60°C for 8 min, and 62.5°C for 8 min. The selective medium used in this study was DMLIA, which contains novobiocin, bile salts, lactose, and sucrose to enhance the selectivity and differentiation capacity of the medium (17). The high degree of selectivity in the plating and recovery media probably resulted in lower numbers of colonies present on the DMLIA plates after heat treatments and a decreased rate of recovery of heat-injured *Salmonella Enteritidis* cells. The cells plated onto DMLIA were not able to reach 8 or 9 log CFU/ml within the 24-h recovery period, unlike cells plated on TSA.

In comparing the effects of media on survival and growth of microorganisms with previous literature, Brasher and others (4) found that *Escherichia coli* O157:H7 and *Salmonella* subjected to stress conditions exhibited significant reductions in populations when plating was done on nonselective media, indicating that the cells had died. Injured *E. coli* O157:H7 cells were not recovered on selective media, unlike the case with TSA. The findings of the present study seem to agree with this previous study. The heat stress that was placed on *Salmonella Enteritidis* cells in ground chicken reduced the populations by at least 5 log CFU/ml. Injured cells were not able to recover on the selective media of DMLIA. These findings are also in agreement with a study by Kobayashi and others (11) in which the count determined by the plating assay with TSA, a non-selective medium, was nearly 1,000-fold higher than that determined by the assay with a selective medium.

NaCl was used as a selective agent, and the effect of NaCl concentrations (2.0 and 3.5%) in the recovery medium (TSB) was investigated (Figs. 2 through 7). Following incubation at 35°C in TSB, heat-treated cells were plated onto DMLIA and TSA. There was a substantial decrease in the number of cells recovered on DMLIA. The recovery of heat-injured cells was inhibited by increasing the level of NaCl in the recovery medium. It was observed at 55 and 62.5°C that, with 3.5% NaCl added to the recovery medium, there was a marked decrease in the growth rate of the heated organisms and that a longer time was required for colonies plated on DMLIA to recover from the heat stress. At 60°C, there was not a statistically significant difference in the rate of recovery of heat-injured *Salmonella Enteritidis* cells at the various NaCl concentrations and pH levels of the recovery medium for cells grown at either 25°C ($P = 0.13$) or 35°C ($P = 0.15$) (Figs. 3 and 4). The recovery medium (TSB) contained various concentrations of NaCl and enhanced the selectivity of the plating medium. It has been reported that the addition of NaCl to the recovery media decreased the heat resistance of some microorganisms probably by impairing heat damage repair mechanisms.

It was observed that the rate of recovery of heat-in-

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**FIGURE 2.** Recovery of *Salmonella Enteritidis* cells grown at 25°C, injured in ground chicken at 55.0°C, recovered in TSB with various NaCl concentrations and pH levels, and enumerated onto (a) TSA and (b) DMLIA.
FIGURE 3. Recovery of Salmonella Enteritidis cells grown at 35°C, injured in ground chicken at 55.0°C, recovered in TSB with various NaCl concentrations and pH levels, and enumerated onto (a) TSA and (b) DMLIA.

Recovery of injured cells was similar at pH 5.5 and 6.5. Therefore, a pH of 5.5 to 6.5 does not have a major effect on the recovery of Salmonella Enteritidis. The time of recovery was greatest for colonies plated onto nonselective media for both pH 5.5 and pH 6.5. It was observed that cells that were grown at 35°C prior to the heat treatments exhibited a higher rate of recovery in the recovery medium at each NaCl concentration and pH combination.

FIGURE 4. Recovery of Salmonella Enteritidis cells grown at 25°C, injured in ground chicken at 60.0°C, recovered in TSB with various NaCl concentrations and pH levels, and enumerated onto (a) TSA and (b) DMLIA.

Growth temperature affects lipid biosynthesis, com-

FIGURE 5. Recovery of Salmonella Enteritidis cells grown at 35°C, injured in ground chicken at 60.0°C, recovered in TSB with various NaCl concentrations and pH levels, and enumerated onto (a) TSA and (b) DMLIA.

FIGURE 6. Recovery of Salmonella Enteritidis cells grown at 25°C, injured in ground chicken at 62.5°C, recovered in TSB with various NaCl concentrations and pH levels, and enumerated onto (a) TSA and (b) DMLIA.
position of membranes, and protein synthesis and thereby influences the ability of *Salmonella* to withstand thermal inactivation (6) and to recover from sublethal heat injury. In this study, *Salmonella* Enteritidis cells were grown at 25 and 35°C. It was found that, when grown at 35°C, *Salmo-
nella* Enteritidis had a greater heat resistance and that in-
jured cells recovered at a higher rate than did cells grown at 25°C. Cells grown at higher temperatures are more heat resistant than those grown at lower temperatures (6), presumably because of differences in gene expression and membrane fluidity.

The rate at which a population of injured cells under-
goes repair will vary with incubation temperature, pH, and salt concentration of the medium. In general, nutritionally rich media allow a relatively rapid repair for a high proportion of injured cells. Cells with very little injury are less demanding, and repair may occur under a wider range of conditions than for cells with a high degree of injury (19). In the present study, *Salmonella* Enteritidis cells were able to repair heat injury with nonselective (TSA) enumeration agar. However, at increased pH levels and NaCl concentra-
tions, the rate of recovery was lower. These findings agree with an investigation by Chawla and others (5) in which repair of injured *L. monocytogenes* occurred more slowly at higher NaCl concentrations and at higher pH values.

The present study found that the addition of NaCl to the recovery medium negatively impacted the ability of *Sal-
monella* Enteritidis cells to recover from heat damage at 55 and 62.5°C. These results are in agreement with those of other authors, who also found that increasing NaCl in the recovery medium inhibited the recovery of heat-injured cells (13, 14, 18).

In a previous study by McKay and Peters (15), the effect of NaCl concentration and pH on the growth of *Sal-
monella* Typhimurium was assessed. It was found that in-
creasing the NaCl concentration and decreasing the pH had little effect on colony growth. This was true in the present study for cells that underwent a heat treatment at 60°C for 8 min. There was not a statistically significant difference found between the various NaCl concentrations and pH values of the recovery media.

A good understanding of sublethal heat injury and re-
covery is important. The growth conditions of the *Sal-
monella* Enteritidis cells prior to heat treatment influenced the survival of the organisms during and after the process. The presence of sublethally injured cells after a heat treatment may lead to an underestimation of the numbers of surviv-
vors, particularly if selective media are used.

The results of the present study can be used to predict the time required at the specified temperatures to achieve 7-log reductions of *Salmonella* Enteritidis when it is heated in ground chicken. Based on the *D*-values determined in this study, contaminated ground chicken should be heated to an internal temperature of 60°C for at least 52.15 min. *D*-values from this study determined for ground chicken will assist food processors in designing acceptance limits on critical control points that ensure safety against *Salmo-
nella* Enteritidis in cooked chicken meat.

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