

# Evaluation of Near-Infrared Pasteurization in Controlling *Escherichia coli* O157:H7, *Salmonella enterica* Serovar Typhimurium, and *Listeria monocytogenes* in Ready-To-Eat Sliced Ham

Jae-Won Ha, Sang-Ryeol Ryu, and Dong-Hyun Kang

Department of Food and Animal Biotechnology, Department of Agricultural Biotechnology, Center for Agricultural Biomaterials, and Research Institute for Agriculture and Life Sciences, Seoul National University, Seoul, South Korea

This study was conducted to investigate the efficacy of near-infrared (NIR) heating to reduce *Salmonella enterica* serovar Typhimurium, *Escherichia coli* O157:H7, and *Listeria monocytogenes* in ready-to-eat (RTE) sliced ham compared to conventional convective heating, and the effect of NIR heating on quality was determined by measuring the color and texture change. A cocktail of three pathogens was inoculated on the exposed or protected surfaces of ham slices, followed by NIR or conventional heating at 1.8 kW. NIR heating for 50 s achieved 4.1-, 4.19-, and 3.38-log reductions in surface-inoculated *S. Typhimurium*, *E. coli* O157:H7, and *L. monocytogenes*, respectively, whereas convective heating needed 180 s to attain comparable reductions for each pathogen. There were no statistically significant ( $P > 0.05$ ) differences in reduction between surface- and internally inoculated pathogens at the end of NIR treatment (50 s). However, when treated with conventional convective heating, significant ( $P < 0.05$ ) differences were observed at the final stages of the treatment (150 and 180 s). Color values and texture parameters of NIR-treated (50-s treatment) ham slices were not significantly ( $P > 0.05$ ) different from those of nontreated samples. These results suggest that NIR heating can be applied to control internalized pathogens as well as surface-adhering pathogens in RTE sliced meats without affecting product quality.

*Salmonella enterica* serovar Typhimurium, *Escherichia coli* O157:H7, and *Listeria monocytogenes* are the main food-borne pathogens involved in numerous outbreaks related to delicatessen meats (2 and <http://www.cdc.gov/foodborneoutbreaks/>). *S. Typhimurium* is the most prevalent pathogen among *Salmonella* serotypes. The most common symptoms caused by *S. Typhimurium* are diarrhea, abdominal pain, mild fever, and chills (1, 21). In 2006, an outbreak of *S. Typhimurium* infection occurred in the United States, which was traced back to contaminated deli meats (<http://www.cdc.gov/foodborneoutbreaks/>). By January 2012, a reported 199 persons had become ill due to the outbreak (<http://www.cdc.gov/foodborneoutbreaks/>). *Salmonella* spp. can proliferate in fresh meats at temperature as low as 2.0°C within 6 to 10 days (5). Thus, refrigeration does not guarantee the inhibition of *Salmonella* spp. in vacuum-packaged ham products. *E. coli* O157:H7 has emerged as an important food-borne pathogen, causing hemorrhagic colitis, which is occasionally complicated by hemolytic uremic syndrome (6, 30), and it requires a very low dose (fewer than 700 organisms) to cause infection (28). Most reported infections with Shiga toxin-producing *E. coli* (STEC) are linked to insufficiently cooked ground meats (20). *Listeria monocytogenes* can cause severe illness or even fatalities among the elderly, pregnant women, persons with weakened immune systems, neonates, and fetuses. Listeriosis has a high rate of mortality (>20%) in this group (18). Vacuum-packaged, refrigerated ham products may also provide a potential growth environment for a psychrotrophic, facultatively anaerobic food-borne pathogen, such as *Listeria monocytogenes* (25, 33). Sporadic food-borne illness outbreaks have been traced to contaminated deli meats in the United States in recent years (<http://www.cdc.gov/foodborneoutbreaks/>). Outbreaks of food-borne listeriosis have resulted in large-scale recalls of ready-to-

eat (RTE) deli meat products ([http://www.fsis.usda.gov/fsis\\_recalls/Recall\\_Case\\_Archive](http://www.fsis.usda.gov/fsis_recalls/Recall_Case_Archive)).

Delicatessen meat, especially precooked sliced ham, is a perishable RTE product. Precooked sliced ham has been used widely in delicatessens and home kitchens due to the convenience it provides. Vacuum-packaged ham should have an extended shelf life, because mild cooking decreases the initial microbial populations and vacuum packaging inhibits the growth of undesirable aerobic microorganisms (11, 14, 27). However, insufficient cooking may happen or postcooking contamination may occur during chilling and before packaging. Furthermore, rehandling at consumer outlets permits the introduction of pathogenic bacteria (19, 26), and this is a major cause of food-borne outbreaks associated with RTE meats (23).

Therefore, a postlethality intervention step, which is a process designed to destroy the postprocessing contaminating organisms immediately before the products are packaged or after unwrapping the vacuum-packaged sliced ham products at delicatessens or other retail outlets, may become necessary to ensure the final microbial safety of the products. Inactivation of bacteria by heat is still one of the most effective methods for food preservation, particularly for RTE meats. As contamination occurs primarily on product surfaces, an additional superficial heat treatment may be needed to inactivate pathogenic bacteria on the products.

Infrared (IR) is an invisible form of electromagnetic energy

Received 25 March 2012 Accepted 26 June 2012

Published ahead of print 6 July 2012

Address correspondence to Dong-Hyun Kang, kang7820@snu.ac.kr.

Copyright © 2012, American Society for Microbiology. All Rights Reserved.

doi:10.1128/AEM.00942-12

emitted from objects at extremely high temperature. By exposing an object to infrared radiation, the heat energy generated can be absorbed by a food product. The intense thermal energy from infrared-emitting sources has been widely applied to various thermal processing operations in the food industry, such as dehydration and pasteurization of a variety of materials and products (24). Infrared heating is also gaining popularity because of its higher thermal efficiency and fast heating rate/response time compared to conventional heating (15). Huang and Sites (8) reported that near-infrared (NIR) treatment of cooked chicken breast meat at 1 kW for 156 s reduced surface-inoculated *Listeria monocytogenes* by 3.4 log CFU/g, and hot water immersion heating at a set temperature (75°C) for 5 min reduced *Listeria monocytogenes* by 3.15 log CFU/g. However, these relatively long treatment times are not practical for use by industry and could adversely affect the organoleptic qualities of the products by increasing denaturation of meat surfaces. Comparing the efficiency of NIR heating to the hot water immersion method also is not reasonable due to the differences in electric power consumed and interference of plastic film packaging (0.4 mm thick) with the conduction of thermal energy from heated water to chicken breast meat. Furthermore, a comparison between the sterilization efficacy of NIR and conventional convective heating (electric resistive heater) has not been reported. Accordingly, inactivation rates between NIR heating and conventional convective heating need to be assessed and statistically evaluated under identical conditions. Also, the investigation and optimization of an NIR treatment system which can quickly reduce pathogens by at least 2 to 3 logs on deli meat products while maintaining product quality is required.

In this study, the efficacy of NIR treatment and conventional convective heating for reducing the populations of food-borne pathogens, including *S. Typhimurium*, *E. coli* O157:H7, and *L. monocytogenes*, on RTE sliced ham were compared under identical conditions. Also, the inactivation of internalized pathogens was investigated through additional experiments utilizing the same treatments.

## MATERIALS AND METHODS

**Bacterial strains.** Three strains each of *S. Typhimurium* (ATCC 19585, ATCC 43971, and DT 104), *E. coli* O157:H7 (ATCC 35150, ATCC 43889, and ATCC 43890), and *L. monocytogenes* (ATCC 7644, ATCC 19114, and ATCC 19115) were obtained from the School of Food Science bacterial culture collection of Seoul National University (Seoul, South Korea) for this study and were used for all experiments. Stock cultures were stored at -80°C in 0.7 ml of tryptic soy broth (TSB; Difco, Becton, Dickinson, Sparks, MD) and 0.3 ml of 50% glycerol. Working cultures were streaked onto tryptic soy agar (TSA; Difco), incubated at 37°C for 24 h, and stored at 4°C.

**Preparation of pathogen inocula.** Each strain of *S. Typhimurium*, *E. coli* O157:H7, and *L. monocytogenes* was cultured in 5 ml of TSB at 37°C for 24 h, followed by centrifugation (4,000 × g for 20 min at 4°C) and washing three times with buffered peptone water (BPW; Difco, Sparks, MD). The final pellets were resuspended in BPW, corresponding to approximately 10<sup>7</sup> to 10<sup>8</sup> CFU/ml. Subsequently, suspended pellets of each strain of the three pathogenic species were combined to produce mixed-culture cocktails (nine strains). These cocktails at a final concentration of approximately 10<sup>8</sup> CFU/ml were used in this study.

**Sample preparation and inoculation.** Precooked and vacuum-packaged sliced ham (approximately 90 by 90 by 2 mm) was purchased from a local grocery store (Seoul, South Korea). The samples were maintained in a refrigerator (4°C) and were used for experiments within 2 days. For surface inoculation (on the exposed surfaces), 8 ml of prepared mixed-

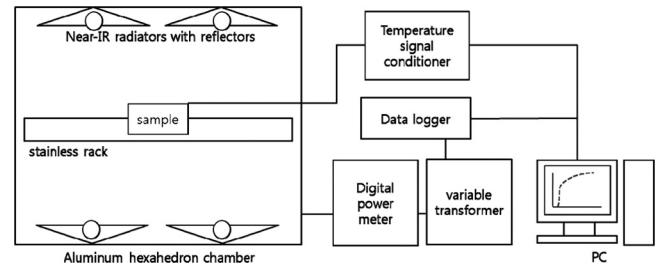


FIG 1 Schematic diagram of the NIR heating system used in this study.

culture cocktail (*S. Typhimurium*, *E. coli* O157:H7, and *L. monocytogenes*) was diluted in 0.8 liters of distilled water. Each ham slice was immersed into the combined pathogen suspension for 10 min at room temperature (22 ± 2°C), drained on a sterilized rack, and dried in a laminar-flow biosafety hood for 20 min with the fan running. Two ham slices (ca. 25 g, inoculum level of 10<sup>5</sup> to 10<sup>6</sup> CFU/sample) were used in each experimental batch.

Internal inoculation (on the protected surfaces) with pathogens was performed as follows. One hour prior to inoculation, sliced ham products were removed from the refrigerator until the temperature of the sample reached that of surface-inoculated samples. Pairs of ham slices were selected, and single slices were placed on a sterilized rack in a biosafety hood with the fan running, and then 0.1 ml of culture cocktail (10<sup>6</sup> to 10<sup>7</sup> CFU/ml) was spread onto one of the paired ham slices, resulting in an inoculum level of 10<sup>5</sup> to 10<sup>6</sup> CFU/sample. Before being overlapped with the other slice, the spread-inoculated ham slice was dried for 10 min.

**Near-infrared heating and conventional convective heating.** A model aluminum chamber (41 by 34 by 29 cm) was used in this study for both NIR and convective heating (Fig. 1). Four quartz halogen infrared heating lamps (NS-104; 350 mm; NSTECH, South Korea), each with a maximum power of 500 W for a 230-V input, were used as the near-infrared emitting source. The four infrared emitters were arranged horizontally in parallel with the four emitting surfaces facing each other, and four aluminum reflectors were installed behind the emitters to redirect the infrared waves and enhance the efficiency of infrared radiation (Fig. 1). Total power consumption of the four emitters was approximately 1.8 kW as measured by a digital power meter (WT-230; Yokogawa, Japan) at standard voltage (220 V). The vertical distance between emitters and sample was 13.5 cm (or 5.3 in) at each side.

For conventional convective heating studies, the four NIR emitters were replaced with four electric resistive emitters (NC420RC; 350 mm; LK, South Korea). The total power of the four resistive heaters was adjusted to match the 1.8 kW used by the NIR system using a variable transformer and data logger (34790A; Agilent Technologies, Palo Alto, CA). The matching wattage was simultaneously verified by means of a digital power meter. The positions and vertical distances of emitters, as well as chamber sizes, were identical to those of the NIR heating system, and the total wattage used was the same.

Surface-inoculated ham slices were placed side by side in the center of a sterilized stainless rack with the long axis parallel to the infrared lamps, whereas the overlapping slices and internally inoculated samples were placed in the center of the treatment rack for the subsequent pasteurization experiment.

**Temperature measurement.** A fiber optic temperature sensor (FOT-L; FISO Technologies Inc., Quebec, Canada) connected to a signal conditioner (TMI-4; FISO Technologies Inc., Quebec, Canada) was used to measure real-time temperatures in samples during NIR and convective heating. The sensor was placed directly on the surface of the treated ham slices or inserted between two ham slices, and the temperature was manually recorded every 5 s. In the case of convective heating, the temperature was recorded every 10 s after 50 s of treatment. Since the fiber optic sensors were coated with electrical insulating material, they did not interfere with

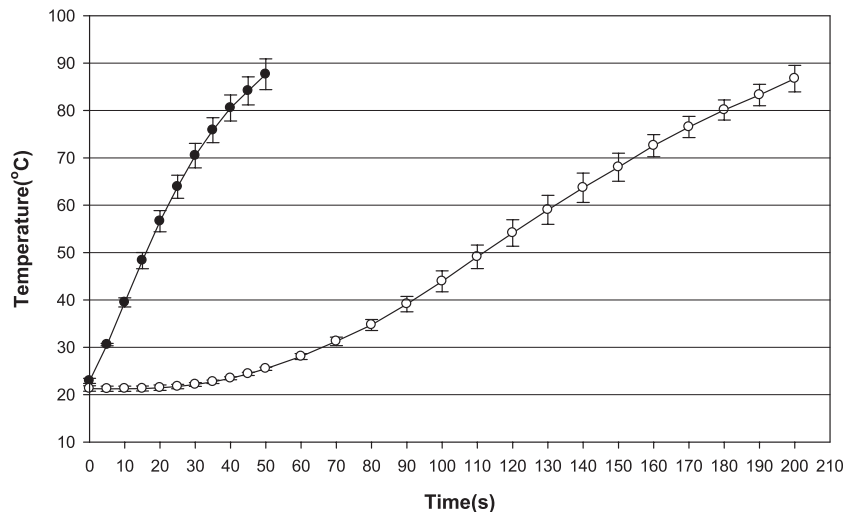


FIG 2 Average temperature-time histories of ham slice surfaces during conventional convective and NIR heating. The error bars indicate standard deviations calculated from triplicates. ○, convective heating at 1.8 kW; ●, NIR heating at 1.8 kW.

the temperature profile of the treated sample (31). All experiments were repeated three times, and averages and standard deviations of sample temperatures for NIR and convective heating were compared to determine the heating rate of samples.

**Bacterial enumeration.** For enumeration of pathogens, each of two treated ham slices (ca. 25 g) was placed at room temperature for 30 s and then immediately transferred into a sterile stomacher bag (Labplas Inc., Sainte-Julie, Quebec, Canada) containing 225 ml of BPW (detection limit, 10 CFU/g) and homogenized for 2 min with a stomacher (Easy mix; AES Chemunex, Rennes, France). After homogenization, 1-ml aliquots of sample were 10-fold serially diluted in 9-ml blanks of BPW, and 0.1 ml of sample or diluent was spread plated onto each selective medium. Xylose lysine desoxycholate agar (XLD; Difco), sorbitol MacConkey agar (SMAC; Difco), and Oxford agar base with Bacto Oxford antimicrobial supplement (MOX; Difco) were used as selective media for the enumeration of *S. Typhimurium*, *E. coli* O157:H7, and *L. monocytogenes*, respectively. Where low numbers of surviving cells were anticipated, 1 ml of undiluted stomacher bag contents was divided between four plates to lower the detection limit. All agar media were incubated at 37°C for 24 to 48 h before counting. To confirm the identity of the pathogens, random colonies were selected from the enumeration plates and subjected to biochemical and serological tests. These tests consisted of the *Salmonella* latex agglutination assay (Oxoid, Ogdensburg, NY), *E. coli* O157:H7 latex agglutination assay (RIM; Remel, Lenexa, KS), and API Listeria (bioMérieux, Hazelwood, MO) for *S. Typhimurium*, *E. coli* O157:H7, and *L. monocytogenes*, respectively.

**Enumeration of heat-injured cells.** Phenol red agar base with 1% sorbitol (SPRAB; Difco) was used to enumerate heat-injured cells of *E. coli* O157:H7 (22). After incubation at 37°C for 24 h, typical white colonies characteristic of *E. coli* O157:H7 were enumerated. Randomly selected isolates from SPRAB plates were subjected to serological confirmation as *E. coli* O157:H7 (RIM *E. coli* O157:H7 latex agglutination test; Remel, Lenexa, KS), because SPRAB is not typically used as a selective agar for enumerating *E. coli* O157:H7. The overlay (OV) method was used to enumerate injured cells of *S. Typhimurium* and *L. monocytogenes* (16). TSA was used as a nonselective medium to repair and resuscitate heat-injured cells. One hundred microliters of appropriate dilutions were spread onto TSA medium, and plates were incubated at 37°C for 2 h to allow injured microorganisms to repair and resuscitate (12). The plates were then overlaid with 7 to 8 ml of selective medium (XLD or OAB agar). After solidification, plates were further incubated for an additional 24 to 48 h at 37°C. Following incubation, typical black colonies were enumerated.

**Color and texture measurement.** To evaluate the effect of NIR heating on the color of ham slices, a Minolta colorimeter (model CR300; Minolta Co., Osaka, Japan) was used to measure the color changes of NIR-treated surfaces. The color attributes were quantified by the value of  $L^*$ ,  $a^*$ , and  $b^*$  and measured at random locations on ham slices.  $L^*$ ,  $a^*$ , and  $b^*$  values indicate color lightness, redness, and yellowness of the sample, respectively. All measurements were taken in triplicate.

Changes in texture of NIR-treated ham slices were evaluated with a TA-XT2i texture analyzer (Texture Technology Corp., Scarsdale, NY) with a blade set probe. After cooling the treated samples, six stacked slices (45 by 45 mm) were placed onto the press holder, and a blade was moved down at 2 mm/s. Maximum force was recorded using Texture Expert software (version 1.22; Texture Technology Corp.). These experiments were replicated three times.

**Statistical analysis.** All experiments were repeated three times with duplicate samples. Data were analyzed by analysis of variance (ANOVA) using the ANOVA procedure of SAS (SAS Institute, Cary, NC), and mean values were separated using Duncan's multiple-range test.  $P < 0.05$  was used to indicate significant difference.

## RESULTS

**Average temperature-time histories of ham slices.** Average surface temperatures of ham slices during conventional convective and NIR heating, both performed at 1.8 kW, are shown in Fig. 2. The rate of NIR heating was much higher than that of conventional convective heating, especially the initial rate of increase. The surface temperature rose immediately in response to infrared waves when the ham slice samples were exposed to NIR radiation, while the surface temperature of ham slices began to increase approximately 30 s after convective heating was initiated (Fig. 2). After 50 s of NIR heating, the surface temperature of ham slices reached ca. 87°C. To raise the surface temperature from room temperature to 87°C, convective heating for a maximum of ca. 200 s was required. Figure 3 shows temperature histories of the insides of two contiguous ham slices during conventional convective and NIR heating. The patterns of temperature growth were similar to those for surface treatment. During 50 s of NIR treatment, the internal temperature reached ca. 74°C. For convective heating, the maximum time required for the inside to increase from room

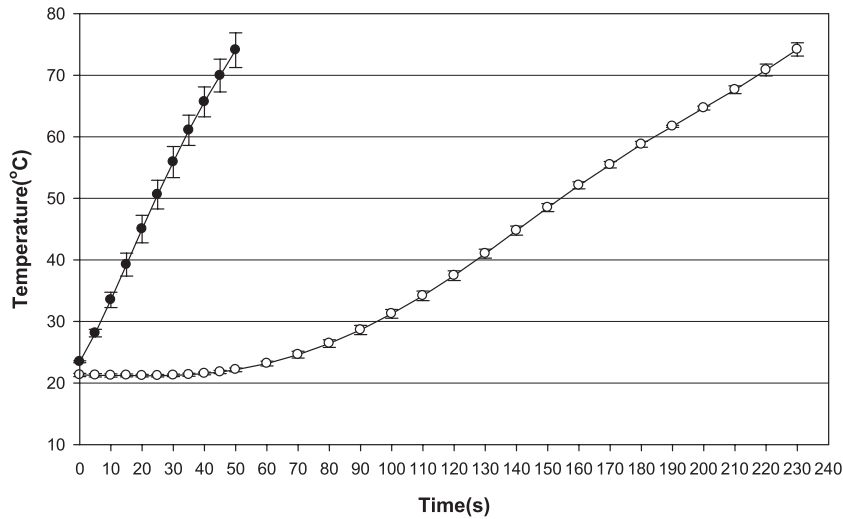


FIG 3 Average temperature-time histories of insides of two contiguous ham slices during conventional convective and NIR heating. The error bars indicate standard deviations calculated from triplicates. ○, convective heating at 1.8 kW; ●, NIR heating at 1.8 kW.

temperature to 74°C was ca. 230 s; furthermore, ca. 50 s was needed for the initiation of temperature increase (Fig. 3).

**Survival curves of food-borne pathogens.** Populations (log CFU/g) of *S. Typhimurium*, *E. coli* O157:H7, and *L. monocytogenes* on ham slice surfaces during NIR and convective heating are shown in Fig. 4. Significant ( $P < 0.05$ ) log reductions of the three pathogens were observed after 30 s of NIR heating and 150 s of convective heating. NIR heating for 50 s achieved 4.1-, 4.19-, and 3.38-log reductions in *S. Typhimurium*, *E. coli* O157:H7, and *L. monocytogenes*, respectively, whereas convective heating required 180 s to attain comparable reductions for each pathogen (Fig. 4).

Figure 5 shows the different effects of NIR and convective heating on inactivation of internally inoculated pathogens. Log reductions of 4.69, 4.56, and 4.16, respectively, were observed in *S. Typhimurium*, *E. coli* O157:H7, and *L. monocytogenes* after NIR

heating for 50 s. The overall reduction patterns of *S. Typhimurium*, *E. coli* O157:H7, and *L. monocytogenes* were similar to those of the surface-inoculated test group; however, the initiation time of significant ( $P < 0.05$ ) reduction was delayed about 10 and 30 s for NIR and convective heating, respectively. Especially for convective heating, 190 s of treatment time was required to reduce each pathogen by ca. 4 log CFU/g (Fig. 5).

**Comparison of pathogen populations between surface- and internally inoculated ham slices.** The results of the surface and internal inoculation methods following NIR and convective heating were arranged for comparison of penetration efficiency (Table 1). Table 1 shows surviving bacterial cells from exposed or protected surfaces of ham slices treated with NIR. There were no statistically significant ( $P > 0.05$ ) differences between surface or internal populations of *S. Typhimurium*, *E. coli* O157:H7, and *L.*

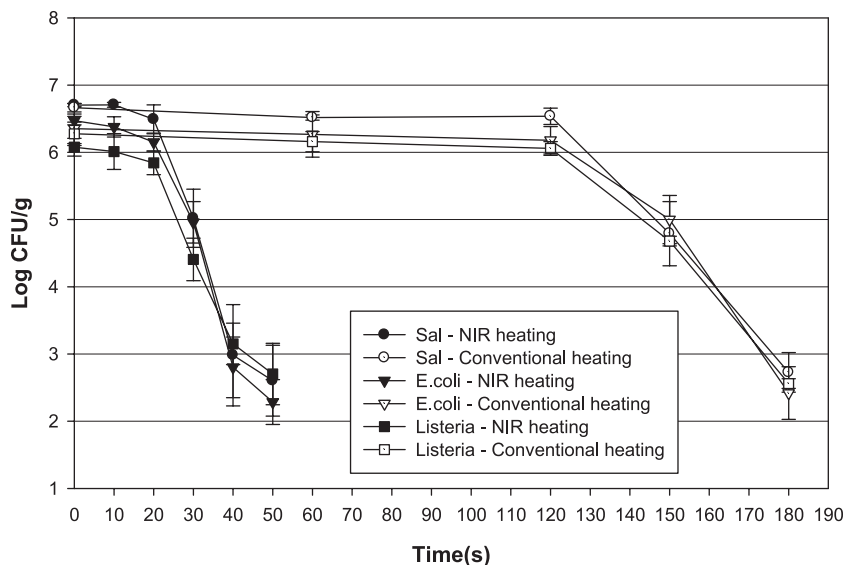


FIG 4 Survival curves of *Salmonella* Typhimurium, *Escherichia coli* O157:H7, and *Listeria monocytogenes* on ham slice surfaces treated with NIR or conventional convective heating. The error bars indicate standard deviations calculated from triplicates.

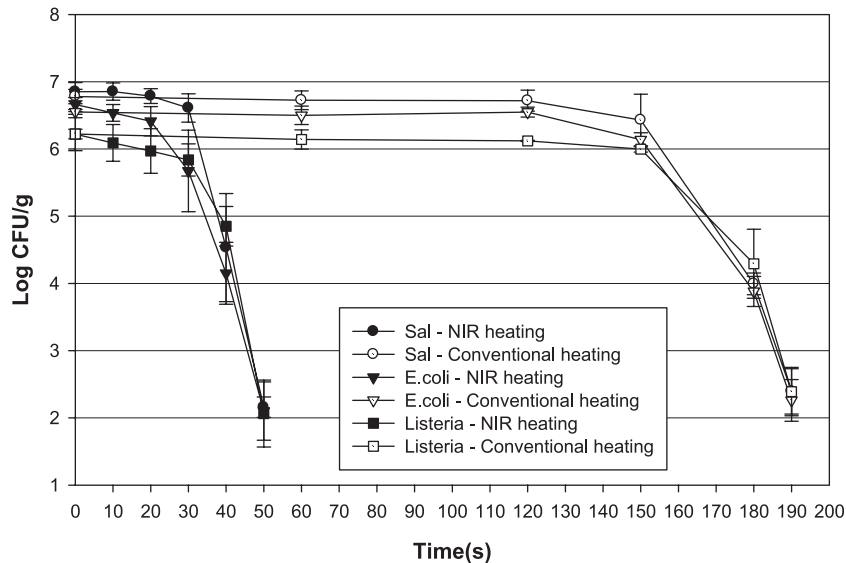


FIG 5 Survival curves of *Salmonella* Typhimurium, *Escherichia coli* O157:H7, and *Listeria monocytogenes* inside two contiguous ham slices treated with NIR or conventional convective heating. The error bars indicate standard deviations calculated from triplicates.

*monocytogenes* at the end of NIR treatment (50 s). However, when conventional convective heating was applied, significant ( $P < 0.05$ ) differences between levels of surface- or internally inoculated cells of the three pathogens were observed during the final part of the treatment (150 and 180 s).

**The recovery of heat-injured cells.** Table 2 shows surviving cells, including heat-injured *S. Typhimurium*, *E. coli* O157:H7, and *L. monocytogenes*, on the surfaces of ham slices following NIR or conventional convective heating. When surface-inoculated ham slices were treated with NIR heating (Table 2), slightly higher numbers of the three pathogens were detected by the agar OV method (SPRAB in the case of *E. coli* O157:H7) than on selective agar. However, there were no significant ( $P > 0.05$ ) differences between the levels of cells enumerated on the appropriate selective

agar (XLD, SMAC, and OAB) versus the agar for resuscitation (OV-XLD, SPRAB, and OV-OAB) during the entire treatment time. In conventional convective heating (Table 2), however, statistically significant ( $P < 0.05$ ) differences between levels of surviving cells, including sublethally injured *S. Typhimurium*, *E. coli* O157:H7, and *L. monocytogenes* cells, were observed after 150 and 180 s of treatment.

**The effect of near-infrared heating on product quality.** Color values of ham slices after NIR treatment are summarized in Table 3.  $L^*$ ,  $a^*$ , and  $b^*$  values of NIR-treated (50 s) ham slices were not significantly ( $P > 0.05$ ) different from those of nontreated samples. Although the  $L$  values (lightness) slightly decreased in accordance with prolonged treatment time, statistically significant differences were not detected during the entire heating interval

TABLE 1 Comparison of pathogen populations between surface- and internally inoculated ham slices following NIR or conventional convective heating<sup>a</sup>

Treatment type and time point (s)	Population (log <sub>10</sub> CFU/g) by organism and inoculation site					
	<i>S. Typhimurium</i>		<i>E. coli</i> O157:H7		<i>L. monocytogenes</i>	
	Surface	Internal	Surface	Internal	Surface	Internal
NIR						
0	6.70 ± 0.02 Aa	6.85 ± 0.14 Aa	6.47 ± 0.06 Aa	6.66 ± 0.10 Aa	6.08 ± 0.13 Aa	6.22 ± 0.25 Aa
10	6.71 ± 0.04 Aa	6.85 ± 0.13 Aa	6.38 ± 0.15 Aa	6.54 ± 0.12 Aa	6.01 ± 0.27 Aa	6.09 ± 0.27 Aa
20	6.49 ± 0.22 Aa	6.79 ± 0.11 Aa	6.15 ± 0.13 Aa	6.41 ± 0.22 Aa	5.84 ± 0.18 Aa	5.97 ± 0.33 Aa
30	5.02 ± 0.43 Ba	6.61 ± 0.21 Ab	4.96 ± 0.31 Ba	5.67 ± 0.61 Ba	4.41 ± 0.32 Ba	5.84 ± 0.24 Ab
40	2.98 ± 0.75 Ca	4.53 ± 0.80 Ba	2.80 ± 0.45 Ca	4.15 ± 0.46 Cb	3.15 ± 0.31 Ca	4.85 ± 0.29 Bb
50	2.60 ± 0.53 Ca	2.16 ± 0.15 Ca	2.29 ± 0.33 Da	2.10 ± 0.43 Da	2.70 ± 0.46 Ca	2.06 ± 0.50 Ca
Conventional heating						
0	6.66 ± 0.06 Aa	6.78 ± 0.11 Aa	6.35 ± 0.22 Aa	6.55 ± 0.04 Aa	6.28 ± 0.17 Aa	6.22 ± 0.07 Aa
60	6.52 ± 0.04 Aa	6.73 ± 0.14 Aa	6.27 ± 0.34 Aa	6.50 ± 0.14 Aa	6.16 ± 0.15 Aa	6.14 ± 0.14 Aa
120	6.54 ± 1.12 Aa	6.72 ± 0.16 Aa	6.18 ± 0.20 Aa	6.55 ± 0.07 Ab	6.06 ± 0.10 Aa	6.12 ± 0.01 Aa
150	4.79 ± 0.48 Ba	6.43 ± 0.39 Ab	5.00 ± 0.36 Ba	6.14 ± 0.10 Bb	4.68 ± 0.07 Ba	6.00 ± 0.04 Ab
180	2.73 ± 0.29 Ca	3.99 ± 0.16 Bb	2.42 ± 0.39 Ca	3.88 ± 0.22 Cb	2.56 ± 0.07 Ca	4.29 ± 0.51 Bb

<sup>a</sup> Means ± standard deviations from three replications. Means with the same capital letter in the same column are not significantly different ( $P > 0.05$ ). Means with the same lowercase letter in the same row are not significantly different ( $P > 0.05$ ).

**TABLE 2** Levels of surviving cells and cells including heat-injured *Salmonella* Typhimurium, *Escherichia coli* O157:H7, and *Listeria monocytogenes* on the ham slices following NIR or conventional convective heating<sup>a</sup>

Treatment type and time point (s)	Population (log <sub>10</sub> CFU/g) by organism and selection medium					
	<i>S. Typhimurium</i>		<i>E. coli</i> O157:H7		<i>L. monocytogenes</i>	
	XLD <sup>b</sup>	OV-XLD	SMAC	SPRAB	OAB	OV-OAB
<b>NIR</b>						
0	6.70 ± 0.02 Aa	6.99 ± 0.21 Aa	6.47 ± 0.06 Aa	6.69 ± 0.21 Aa	6.08 ± 0.13 Aa	6.22 ± 0.07 Aa
10	6.71 ± 0.04 Aa	6.83 ± 0.12 Aa	6.38 ± 0.15 Aa	6.28 ± 0.25 ABa	6.01 ± 0.27 Aa	6.04 ± 0.15 Aa
20	6.49 ± 0.22 Aa	6.60 ± 0.18 Aa	6.15 ± 0.13 Aa	6.14 ± 0.15 Ba	5.84 ± 0.18 Aa	5.91 ± 0.17 Aa
30	5.02 ± 0.43 Ba	5.54 ± 0.15 Ba	4.96 ± 0.31 Ba	5.37 ± 0.08 Ca	4.41 ± 0.32 Ba	4.66 ± 0.47 Ba
40	2.98 ± 0.75 Ca	4.06 ± 0.66 Ca	2.80 ± 0.45 Ca	3.50 ± 0.45 Da	3.15 ± 0.31 Ca	3.54 ± 0.20 Ca
50	2.60 ± 0.53 Ca	3.18 ± 0.61 Da	2.29 ± 0.33 Da	2.83 ± 0.16 Ea	2.70 ± 0.46 Ca	3.00 ± 0.36 Da
<b>Conventional heating</b>						
0	6.60 ± 0.18 Aa	6.69 ± 0.18 Aa	6.06 ± 0.06 Aa	5.98 ± 0.16 Aa	6.23 ± 0.07 Aa	6.49 ± 0.16 Aa
60	6.53 ± 0.16 Aa	6.75 ± 0.06 Aa	5.91 ± 0.23 Aa	5.99 ± 0.16 Aa	6.33 ± 0.24 Aa	6.43 ± 0.20 Aa
120	6.36 ± 0.11 Aa	6.60 ± 0.12 Aa	5.79 ± 0.10 Aa	5.94 ± 0.08 Aa	6.13 ± 0.11 Aa	6.41 ± 0.18 Aa
150	4.34 ± 0.56 Ba	5.78 ± 0.24 Bb	4.34 ± 0.36 Ba	5.44 ± 0.12 Bb	4.01 ± 0.28 Ba	4.97 ± 0.45 Bb
180	2.71 ± 0.12 Ca	4.15 ± 0.25 Cb	2.24 ± 0.41 Ca	3.47 ± 0.47 Cb	2.73 ± 0.25 Ca	3.01 ± 0.56 Ca

<sup>a</sup> Means ± standard deviations from three replications. Means with the same capital letter in the same column are not significantly different ( $P > 0.05$ ). Means with the same lowercase letter in the same row are not significantly different ( $P > 0.05$ ).

<sup>b</sup> XLD, xylose lysine desoxycholate; OV-XLD, overlay XLD agar on TSA; SMAC, sorbitol MacConkey agar; SPRAB, phenol red agar base with 1 % sorbitol; OAB, Oxford agar base; and OV-OAB, overlay OAB agar on TSA.

(Table 3). Table 4 shows the texture parameters of ham slices following NIR treatment. There were no significant ( $P > 0.05$ ) differences in maximum load values of texture measurements among all tested samples, indicating treatment with NIR for 50 s did not significantly ( $P > 0.05$ ) change the quality of ham slices.

## DISCUSSION

In recent years, large-scale outbreaks related to consumption of RTE meats have occurred, in addition to the recall and destruction of massive quantities of products. Because of the high risk potential to the general public, the U.S. Department of Agriculture Food Safety and Inspection Service (USDA-FSIS) recommends that food manufacturers adopt postlethality intervention treatments to control the growth of pathogens in these products (33).

Infrared heating has been explored as a means for pasteurizing meat products to kill pathogens or other spoilage microorganisms. Infrared surface pasteurization has been investigated by several researchers for inactivation of *L. monocytogenes* on RTE meats. Gande and Muriana (7) reported on the use of a radiant heat oven for surface pasteurization of various meat products. The heating time varied from 60 to 120 s, and *L. monocytogenes* levels were reduced by about 1.25 to 3.5 logs after treatment. Huang and

Sites (10) conducted surface pasteurization processing of turkey frankfurters using a ceramic infrared heater. The temperature reached the set point (70, 75, or 80°C) during 82, 92, and 103 s of treatment, respectively, and 3.5- to 4.5-log reductions in *L. monocytogenes* were achieved. However, far- or medium-infrared (F/MIR) heating, which is generated by a quartz tube or ceramic heater, has much lower thermal energy and penetrating capability than NIR. Therefore, the long heating time was shortened by using an NIR lamp-type emitter in a previous study (8).

NIR radiation, with its short wavelength, has a relatively higher energy level than F/MIR radiation, which has a longer wavelength, because total energy decreases as the peak wavelength increases (15). The response of the NIR lamps to electric power was very fast; high-intensity, bright, visible light was emitted from the lamps almost as soon as the power was switched on. The maximum wavelength ( $\lambda_m$ ) generated from the infrared heater used in this study was about 1,210 nm and was measured by the Choice spectral light measurement system (LMS 7600; Labsphere, NH). This wavelength was located in the near-infrared wave range (4). According to Wien's displacement law (13), the absolute temperature of the emitting source can be calculated from the peak wavelength emitted from the infrared heater [ $\lambda_m = (2.898 \times 10^{-3})/T_k$ ].

**TABLE 3** Surface color values of NIR-treated ham slices<sup>a</sup>

Treatment time (s)	Parameter <sup>b</sup>		
	L*	a*	b*
0	68.10 ± 0.56 a	11.69 ± 0.24 a	11.38 ± 0.39 a
10	68.45 ± 0.95 a	11.35 ± 0.99 a	11.41 ± 0.30 a
20	68.04 ± 0.47 a	11.32 ± 0.30 a	12.03 ± 0.37 a
30	67.55 ± 1.10 a	11.36 ± 1.04 a	11.95 ± 0.53 a
40	66.63 ± 0.80 a	11.41 ± 0.54 a	12.55 ± 0.80 a
50	66.64 ± 0.75 a	11.04 ± 0.47 a	12.74 ± 0.80 a

<sup>a</sup> Means ± standard deviations from three replications. Values followed by the same letters within the column per parameter are not significantly different ( $P > 0.05$ ).

<sup>b</sup> Color parameters are lightness (L\*), redness (a\*), and yellowness (b\*).

**TABLE 4** Maximum load values for quantifying texture of ham slices following treatment with NIR<sup>a</sup>

Treatment time (s)	Maximum load (g)
0	1,171.47 ± 51.21 a
10	1,158.90 ± 13.24 a
20	1,135.27 ± 24.45 a
30	1,175.87 ± 58.02 a
40	1,110.57 ± 8.80 a
50	1,141.70 ± 59.58 a

<sup>a</sup> Means ± standard deviations from three replications. Values followed by the same letters within the column per parameter are not significantly different ( $P > 0.05$ ).

At 1,210 nm, the theoretical maximum temperature attainable by the emitter was 2,122°C (3,852°F).

A study by Bolshakov et al. (3) suggested that a maximum transmission of IR radiation was affected by spectral wavelength. The penetration depths into cooked pork were 2.3, 1.0, and 0.7 mm at a  $\lambda_m$  of 1.07, 2.4, and 4.2  $\mu\text{m}$ , respectively. Since the wavelength used in this study was 1,210 nm, the penetration depth at this wavelength could be almost 2 mm below the surface of ham slices.

Bacterial cells are very small and are never completely located on ham slice surfaces due to the uneven microscopic topography of meat products. Some bacteria may survive during heat treatment if they harbor in the crevices and cracks under the surface (9) and potentially present a risk to the consumer. As conduction of heat from the surface to the interior by F/MIR or conventional convective heating is a very slow process, these heating types could contribute to greater deterioration in product quality. Actually, browning was visually observed on meat products which were treated with F/MIR for a slightly excessive time (9, 10). In contrast, with NIR heating, thermal energy transfers through both direct penetration and conduction at the same time (Fig. 3). Furthermore, similar bactericidal effects were observed between pathogens located on the surface and located internally between two contiguous ham slices following NIR heating (Table 1). These results imply that NIR radiation used in this study sufficiently penetrated the thickness ( $1.82 \pm 0.05$  mm, measured by a digital micrometer) of ham slices and delivered thermal energy to the inoculated interface between the slices. NIR heating was more efficient in inactivating pathogens which were internalized or hidden under the surface of ham slices than was convective heating (Table 1).

The average bacterial concentrations of *S. Typhimurium*, *E. coli* O157:H7, and *L. monocytogenes* inoculated on the exposed or the protected surfaces of ham slices were 6 to 7 log CFU/g. A high-inoculum concentration was used to make enumeration of surviving bacteria easier. These inoculation levels were extremely high for precooked meat product, far higher than would be encountered in commerce, since the USDA-FSIS has established a zero-tolerance level for *L. monocytogenes* in RTE foods (29). Even in noncooked meats, usually less than 3 logs of pathogens are detected (32). Therefore, bearing in mind the low numbers of pathogens present on vacuum-packaged precooked ham products, the application of a treatment capable of achieving a 2- to 3-log reduction in pathogen numbers would be more than sufficient to render the treated product pathogen free. These 2- to 3-log reductions were achieved with just 40 s of NIR treatments both on surface- and internally inoculated samples (Table 1). Also, this treatment time is much shorter (40 to 50 s) than that of an experiment conducted by Huang and Sites (156 s) (8) for reducing *Listeria monocytogenes* on the surface of chicken meat using NIR heating.

Following heating treatment, sublethally injured food-borne pathogens could assume added significance, because they are potentially as dangerous as their uninjured counterparts (16, 17). Under favorable environmental conditions, heat-injured cells usually undergo repair and become functionally normal. Therefore, the cell numbers enumerated on selective media probably are not representative of the total surviving populations in the samples. In this study, the occurrence of sublethally injured pathogens on ham slices was assessed by plating on selective agars with and

without a resuscitation step. More sublethally injured cells were produced in conventional convective heating than in NIR heating (Table 2). This suggests that NIR heating effectively inactivated *S. Typhimurium*, *E. coli* O157:H7, and *L. monocytogenes* on ham slices without generating many heat-injured cells which could recover and grow.

It is essential and advantageous to investigate the quality changes occurring during NIR heat treatment for commercial practical application of this intervention. In a previous NIR study, the statistical measurements of quality changes were insufficient and limited to visual examination (8). Therefore, in our study, the experimental conditions for investigating the quality change of ham products were focused on treatment with the NIR rather than conventional heating. After the maximum treatment of 50 s, color values ( $L^*$ ,  $a^*$ , and  $b^*$ ) and cutting maximum load values of samples were not significantly ( $P > 0.05$ ) different from those of the control (Tables 3 and 4). These results suggest that NIR heating can be applied to control pathogens in RTE sliced meats without affecting product quality.

NIR heating on an industrial scale for controlling postprocessing contamination can be performed on a continuous basis. Sliced deli meats moving on a stainless steel conveyor belt could be exposed to banks of NIR heaters oriented horizontally on both sides of the belt. Treatment times could be adjusted by altering the speed of the belt. This step is essential in order to evaluate the efficacy and capital costs of NIR processing compared to conventional heat treatment. In addition, further studies to shorten the NIR treatment time for minimization of quality changes or enhancing the effect of inactivation are required.

## ACKNOWLEDGMENTS

This research was supported by the WCU (World Class University) program through the National Research Foundation of Korea (NRF) funded by the Ministry of Education, Science and Technology (R32-2008-000-10183-0). This research was also supported by the Agriculture Research Center program of the Ministry for Food, Agriculture, Forestry and Fisheries, South Korea.

## REFERENCES

- Baird-Parker AC. 1990. Foodborne salmonellosis. *Lancet* 336:1231–1235.
- Belongia EA, MacDonald KL, Parham GL, Kasale KA, Osterholm MT. 1991. An outbreak of *Escherichia coli* O157:H7 colitis associated with consumption of precooked meat patties. *J. Infect. Dis.* 164:339–343.
- Bolshakov AS, et al. 1976. Effects of infrared radiation rates and conditions of preliminary processing of quality index on baked products. Paper 15, 22nd European Meeting of Meat Research Workers, Malmo, Sweden.
- Dagerskog M, Osterstrom L. 1979. Infra-red radiation for food processing. I. A study of the fundamental properties of infra-red energy. *Lebensmittel-Wissenschaft Technol.* 12:237–242.
- D'Aoust JY. 1991. Psychrotrophy and foodborne *Salmonella*. *Int. J. Food Microbiol.* 13:207–215.
- Doyle MP. 1991. *Escherichia coli* O157:H7 and its significance in foods. *Int. J. Food Microbiol.* 12:289–302.
- Gande N, Muriana P. 2003. Prepackage surface pasteurization of ready-to-eat meats with a radiant heat oven for reduction of *Listeria monocytogenes*. *J. Food Prot.* 66:1623–1630.
- Huang L, Sites J. 2009. Elimination of *Listeria monocytogenes* on cooked chicken breast meat surfaces by near-infrared surface pasteurization prior to final packaging. *J. Food Process Eng.* 35:1–15.
- Huang L, Sites J. 2008. Elimination of *Listeria monocytogenes* on hotdogs by infrared surface treatment. *J. Food Sci.* 73:M27–M31.
- Huang L, Sites J. 2004. Infrared surface pasteurization of turkey frankfurters. *Innov. Food Sci. Emerg. Tech.* 5:345–351.
- Jones SL, Carr TR, McKeith FK. 1987. Palatability and storage characteristics of precooked pork roasts. *J. Food Sci.* 52:279–285.

12. Kang DH, Siragusa GR. 1999. Agar underlay method for recovery of sublethally heat-injured bacteria. *Appl. Environ. Microbiol.* **65**:5334–5337.
13. Knudsen JG, et al. 1984. Heat transmission by radiation, p 10–52–10–68. In Perry RH, Green S, Maloney JO (ed), Perry's chemical engineers' handbook, 16th ed. McGraw-Hill, New York, NY.
14. Kotzekidou P, Bloukas JG. 1996. Effect of protective cultures and packaging film permeability on shelf-life of sliced vacuum-packed cooked ham. *Meat Sci.* **42**:333–345.
15. Krishnamurthy K, Khurana HK, Jun SJ, Irudayaraj J, Demirci A. 2008. Infrared heating in food processing: an overview. *Compr. Rev. Food Sci. Saf.* **7**:2–12.
16. Lee SY, Kang DH. 2001. Suitability of overlay method for recovery of heat-injured *Listeria monocytogenes* and *Salmonella* Typhimurium. *Food Sci. Biotechnol.* **10**:323–326.
17. McCleery DR, Rowe MT. 1995. Development of a selective plating technique for the recovery of *Escherichia coli* O157:H7 after heat stress. *Lett. Appl. Microbiol.* **21**:252–256.
18. Mead PS, et al. 1999. Food-related illness and death in the United States. *Emerg. Infect. Dis.* **5**:7–25.
19. Mol JHH, Hietbrink JEA, Mollen HW, Van Tinteren J. 1971. Observations on the microflora of vacuum-packed sliced cooked cured meats. *J. Appl. Bacteriol.* **34**:377–379.
20. Rangel JM, Sparling PH, Crowe C, Griffin PM, Swerdlow DL. 2005. Epidemiology of *Escherichia coli* O157:H7 outbreaks, United States, 1982–2002. *Emerg. Infect. Dis.* **11**:603–609.
21. Rhee MS, Lee SY, Dougherty RH, Kang DH. 2003. Antimicrobial effects of mustard flour and acetic acid against *Escherichia coli* O157:H7, *Listeria monocytogenes*, and *Salmonella enterica* serovar Typhimurium. *Appl. Environ. Microbiol.* **69**:2959–2963.
22. Rhee MS, Lee SY, Hillers VN, McCurdy SM, Kang DH. 2003. Evaluation of consumer-style cooking methods for reduction of *Escherichia coli* O157:H7 in ground beef. *J. Food Prot.* **66**:1030–1034.
23. Ryser ET, Marth ET. 1991. *Listeria*, listeriosis, and food safety. Marcel Dekker Inc., New York, NY.
24. Sakai N, Fujii A, Hanzawa T. 1993. Heat transfer analysis in a food heated by far infrared radiation. *Nippon Shokuhin Kogyo Gakkaishi* **40**:469–477.
25. Schwartz B, et al. 1988. Association of sporadic listeriosis with consumption of uncooked hot dogs and undercooked chicken. *Lancet* **i**:779.
26. Steele JE, Stiles ME. 1981. Microbial quality of vacuum-packaged sliced ham. *J. Food Prot.* **44**:435–439.
27. Stites CR, McKeith FK, Bechtel PJ, Carr TR. 1989. Palatability and storage characteristics of precooked beef roasts. *J. Food Sci.* **54**:3–6.
28. Tuttle J, et al. 1999. Lessons from a large outbreak of *Escherichia coli* O157:H7 infections: insights into the infectious dose and method of widespread contamination of hamburger patties. *Epidemiol. Infect.* **122**:185–192.
29. USDA. 1989. Revised policy for controlling *Listeria monocytogenes*. *Fed. Reg.* **54**(98):22345–22346.
30. Wang G, Zhao T, Doyle MP. 1996. Fate of enterohemorrhagic *Escherichia coli* O157: H7 in bovine feces. *Appl. Environ. Microbiol.* **62**:2567–2570.
31. Wang Y, Wig TD, Tang J, Hallberg LM. 2003. Sterilization of foodstuffs using radio frequency heating. *J. Food Sci.* **68**:539–543.
32. Zaika L, et al. 1990. Destruction of *Listeria monocytogenes* during frankfurter processing. *J. Food Prot.* **53**:18–21.
33. Zhu M, DU M, Cordray J, Ahn DU. 2005. Control of *Listeria monocytogenes* contamination in ready-to-eat meat products. *Compr. Rev. Food Sci. Food Saf.* **4**:34–42.