TITLE: Effects of commercial chilling methods for reducing bacteria on pork carcasses

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Abstract:

Previous laboratory studies have demonstrated that snap/blast chilling is known to significantly reduce some bacterial populations on pork surfaces, including *Salmonella*, *Campylobacter*, *E. coli*, and coliforms. However, very little information exists as to whether reductions observed under commercial conditions can be attributed to chilling parameters. In this study, individual carcass sponge samples (n=386) were obtained from commercially processed warm pork carcasses (n=188) and chilled pork carcasses (n=198) subjected to snap/blast chilling or conventional chilling and evaluated for mesophilic aerobic plate counts (APC), generic *E. coli* (EC), coliforms (CF), as well as the presence/absence of *Salmonella* spp., *Campylobacter* spp., and *Listeria monocytogenes*. Carcass samples were obtained from one very small, three small, and three large pork processing establishments. Of these, three establishments used conventional chilling only and four establishments used snap/blast chilling, followed by conventional chilling. Overall, warm carcasses exhibited average APC of approximately 7.09 log_{10} CFU/300 cm^2, average EC counts of 1.93 log_{10} CFU/300 cm^2, and average CF counts of 2.23 log_{10} CFU/300 cm^2. *Salmonella* spp., *L. monocytogenes*, and *Campylobacter* spp. were present on 2.1%, 1.2%, and 7.4% of the warm carcasses, respectively. Chilled carcasses exhibited average APC, EC, and CF of 5.21, 0.25, and 0.50 log_{10} CFU/300 cm^2, respectively. Pathogen prevalence on the chilled carcasses was 4.3% *Salmonella* spp., 0.05% *L. monocytogenes*, and 0.0% *Campylobacter* spp. In addition to microbial populations, chilling parameters, and carcass surface temperature were collected. This study demonstrates that commercial chilling conditions may reduce most bacterial populations associated with pork carcasses. This information may provide processors with an additional means to control microbes in/on freshly slaughtered pork, thereby improving the microbiological safety of pork products.
Introduction
The pork industry is committed to producing a high quality, wholesome product that is safe for human consumption. However, during the slaughtering process, microbial contamination of animal carcasses is unavoidable (Bell, et al., 1986; Chung et al., 1989), and this phenomenon represents one of the most critical quality and safety issues faced by the pork industry. While most of the microflora transferred to carcasses during the slaughtering process is non-pathogenic, there is a possibility that pathogens such as Salmonella spp., Campylobacter spp., and Listeria monocytogenes may be present (Epling et al., 1993).

Campylobacter spp. is the number one cause of acute bacterial gastroenteritis in the United States (Stern et al., 2001). The level of incidence of campylobacteriosis in the United States is approximately 20 diagnosed cases per 100,000 populations resulting in approximately 2.4 million infections each year (Anonymous, 1994). S. Typhimurium and L. monocytogenes also are foodborne pathogens that have been responsible for several foodborne outbreaks related to pork products (Anonymous, 1994). All of these pathogens can cause serious illness or death in susceptible populations. Reducing such pathogens in meat and poultry could save up to $12 billion annually in the U.S in medical costs, lost productivity, recalls, legal fees, and loss of business (Buzby, et al., 1996, and Shallow, et al., 1998). Due to the severity and economic implications of these foodborne pathogens, the food industry utilizes a variety of treatments such as freezing, acidification, and heat treatments to obtain a wholesome product (McCleer and Rowe, 1995). Such treatments are known to adversely affect pathogenic microorganisms on pork surfaces, resulting in either cell death or injury.

Microbial activity is influenced by chemical and enzymatic reactions; therefore, the effect of low temperatures on microorganisms is essentially a growth retardation process. Most microorganisms will not grow at freezing temperatures due to reduced metabolic activities; however, the microorganisms will begin to grow again when placed at warmer temperatures (Ingram and Mackey, 1976; Cunningham and Cox, 1987; Mountney and Gould, 1988). It has been demonstrated that chilling also can improve the quality and shelf life of pork, but very little information exists as to the effect of chilling parameters on the microbial safety of pork.

Although the effects on bacterial cells due to chilling or freezing are not completely understod, chilling is known to extend the lag phase of these organisms thereby, reducing the overall microbial load on pork samples (Troller, 1976; Vanderzant et al., 1985). Chilling at temperatures slightly above ice formation or by freezing to 0°C can cause injury or death to bacteria. Generally, freezing may kill cells by formation of ice crystals that penetrate the cell membrane, resulting in release of cellular constituents (Mazur, 1966). When no ice is formed, may cause chemical changes in the lipid bilayer of the cells, resulting in permanent physical damage (Smith, 1995). As such, chilling is still considered the most important preservation method for foods (Smith, 1995).

The pork industry currently utilizes either snap/blast- or conventional-chilling to reduce the temperature of carcasses immediately after slaughter and before fabrication. The conventional-chilling process consists of placing pre-rigor pork carcasses in a chiller at 1 to 4°C with an air velocity of 1.5 to 3 f/s for 18 to 24 h, and prior to fabrication, further processing or shipping (Greer and Dilts, 1987; Huff-Lonergan and Page, 2000). While this chilling method is widely adopted, it has several disadvantages, including substantial evaporative weight loss, considerable cooler space requirements, and increase in the incidence of pale soft exudative (PSE) pork (Greer and Dilts, 1987; Crenwelge, et al., 1984). Conversely, conventional-chilling of pork carcasses has been found to reduce levels of Campylobacter jejuni, presumably due to the drying process, reduced relative humidity, or oxygen exposure that occurs under forced ventilation (Oosterom, et al., 1983: Bracewell et al., 1985). Snap/blast-chilling requires an air temperature of –20 to –40 °C with an air velocity of 10 to 16 f/s for 1 to 3 h (Huff-Lonergan and Page, 2000). This method has been shown to reduce shrinkage, drip loss, incidence of PSE pork, and produce
firmer muscle (Greer and Dilts, 1987; Jones, et al., 1987). To date, the effect of snap/blast-chilling on the microbiological quality of the pork has been debatable. While some researchers have found that snap/blast-chilling reduced levels of carcass contamination and improved keeping quality (Dann, 1972; Price et al., 1976; Wernberg, 1972), others found that snap/blast-chilling did not improve the microbial quality of the pork product (Greer and Dilts, 1987). Other published reports also demonstrate some disparity in the efficacy of snap/blast-chilling as compared with conventional-chilling. Cooper (1968) and James et al. (1983) found no differences using conventional-chilling or snap/blast-chilling to reduce bacterial populations. Greer and Dilts (1987) examined mesophilic and psychrotrophic bacterial populations on carcasses, loins, and loin chops under retail display following conventional- and snap/blast-chilling. These researchers demonstrated that chilling treatment had no effect on odor, appearance, or shelf-life of fresh pork products and that snap/blast-chilling could be applied without compromising the bacterial quality of pork products (Greer and Dilts, 1987). Carr et al (1998) established that populations of Staphylococcus spp. and lactic acid bacteria were not affected by either chilling treatment, except when no fat trimming was used; mesophilic bacteria and coliforms counts were not significantly different when chilling methods were compared. It was hypothesized that the minimum growth temperature of 10°C may contribute to the reduced growth of Staphylococcus spp. and other bacteria on carcasses (Troller, 1976).

Due to the inherent differences between the protocols of the previous studies, we conducted a study to determine the individual and interactive effects of several variables for reducing microbes on pork surfaces (Chang et al, 2003b). The research compared snap/blast-chilling to conventional-chilling for reducing specific pathogens on skin-on and skin-off pork surfaces and determined the effect of chilling against different levels of specific pathogens on pre-rigor pork carcass surfaces. Specifically, fecal slurries at inoculation levels of 3 and 5 log_{10} CFU/cm², with or without pathogens (Listeria monocytogenes, Salmonella Typhimurium and Campylobacter coli), were inoculated onto skin-on and skin-off pork surfaces, and subjected to snap/blast- and conventional-chilling using a thin agar layer (TAL) method for recovery of injured cells (Chang et al, 2003b). Results indicated that there were no statistically significant (P > 0.05) differences between snap/blast- and conventional-chilling treatments in reducing high and low inoculation levels of mesophilic bacteria, total coliforms, or Escherichia coli. Furthermore, treatments were not different for reducing L. monocytogenes, S. Typhimurium or C. coli at low inoculation levels (Chang et al, 2003b). Conversely, both snap/blast- and conventional-chilling treatments were more effective against all bacterial populations when pork samples where inoculated with high inoculation levels (5 log_{10} CFU/cm²). The data also indicated that surface type was a significant factor in reducing S. Typhimurium and C. coli at low inoculation levels. More importantly, pork samples inoculated with low levels of C. coli and subjected to snap/blast-chilling were reduced to undetectable levels (< 1.3 log_{10} CFU/cm²). We concluded that the effects of chilling technology on microbial populations could provide pork processors with an additional intervention for pork slaughter or information to modify procedures to improve the chilling process (Chang et al, 2003b). However, it was concluded that additional data derived from commercial processing is needed to support the laboratory findings.

**Project Objective:**

To determine the effects of commercial (industry) chilling procedures (snap/blast or conventional) for reducing bacterial populations on pork carcasses.

**Materials and Methods:**

One very small, three small, and three large pork processing establishments participated in the sampling study. Of these seven establishments, three used conventional chilling and four used snap/blast chilling
parameters (Table 1). Approximately 357 samples were obtained from the seven plants from May 2003 to August 2003.

Temperature, air flow velocity and pH monitoring

Air temperatures (ºC) of the chillers were monitored with a mercury thermometer (H. O. Trerice Co., Detroit, MI) placed in close proximity to the samples. To monitor surface and internal temperatures (ºC) of conventional- or snap/blast-chilled samples, pork surfaces were measured using a thermocouple thermometer (Model No. 92000-00, Bench top 115V, Cole Palmer Instruments Co, Veron Hills, IL). Each Probe K (1/5000 cm thick, high response; Datapag, Veron Hills, IL) was inserted just under the surface of the tissue (about 1 mm deep). Air velocity at the carcass surface (top, middle, bottom) was determined using a thermoanemometer (Model DAFM2, Beaverton, OR).

Before and after the samples were subjected to snap/blast- and conventional-chilling, the surface pH was also measured on all pork surfaces using a surface electrode (Accumate, Model 25 pH/ion meter, Fisher Scientific, Pittsburgh, PA). When available, processing establishments shared chilling parameters.

Sampling

Sponge samples from pork carcasses were obtained before (on slaughter floor) and again after snap/blast- or conventional-chilling using the 3-site sponge sampling procedure as outlined by USDA-FSIS in the Final Rule (USDA-FSIS, 1996). Briefly, sterile sponges (Specisponge; Nasco, Fort Atkinson, WI) were hydrated with 25 ml of buffered peptone water (BPW; Difco Laboratories, Detroit, MI). Residual moisture was expelled from the sponge inside a Whirlpak bag (Nasco), and the sponge was removed from the bag with sterile gloved hands. Using a sterile template, a 100 cm² area was rubbed with the sponge 10 times in each horizontal and vertical directions. Sponge samples were returned to the sterile Whirlpak bag and transported to the laboratory at on blue ice (temperatures ranged from 4-10°C) within 24 h for microbiological evaluation. Upon receipt, sponge samples were stomached for 2 min in a Stomacher Lab Blender 400 (Tekmar, Inc., Cincinnati, OH) and serially diluted in buffered peptone water (BPW) prior to plating.

Media

APC PetriFilm (3M, Inc., St. Paul, MN, USA) was used for enumeration of mesophilic aerobic bacteria. Total coliforms and E. coli biotype 1 were determined using E. coli/coliform 3M Petrifilm™ (3M).

For isolation and recovery of Campylobacter spp., Brucella agar, BA (Difco) was chosen as a non-selective medium, while Campylobacter Blood-free selective medium (modified CCDA-Preston) agar (mCCDA; Oxoid, Ltd., Basingstoke, England) was used as a selective medium. Trypticase soy agar (TSA, Difco) was used as the non-selective medium, whereas Xylose Lysine Decarboxylase agar (XLD, Difco) and Modified Oxford agar (MOX, Difco) were chosen as the selective media for S. Typhimurium and L. monocytogenes, respectively.

Thin Agar Layer (TAL) Method

The TAL method was used for recovery and selective enumeration of pathogens. The TAL method consisted of pre-pouring one layer of selective media (20 ml) into a petri dish, allowing for solidification, followed by the addition of two layers, 7 ml each, of non-selective media (Chang et al., 2003a; Kang and Fung, 1999, 2000; Wu et al., 2001). By pouring two separate layers of non-selective media onto the selective medium, the diffusion of the selective agents is retarded by a physical barrier generated by the
two separate layers. This process also allows injured cells to recover before being subjected to selective agents. Sponge samples containing chill- or freeze-injured cells were inoculated directly onto the non-selective media layer and incubated for 24 to 48 h at 37°C for isolation of S. Typhimurium and L. monocytogenes, or for 48 h at 42°C under microaerophilic conditions for isolation of C. coli (Chang et al., 2003a).

**Enrichment**

To ensure detection of low levels of pathogens following chilling procedures, samples were also enriched. For S. Typhimurium, 1 ml of the stomachate was added to 9 ml of lactose broth (Difco) and incubated 24 h at 37°C. Following incubation, 1 ml of this pre-enrichment was transferred to 9 ml of selenite cysteine (SC; Difco) medium or 1 ml to 9 ml of tetrathionate broth (TT; Difco). The broths were incubated for 24 h at 37°C. After incubation, samples from TT and SC were streaked for isolation onto XLD agar. Typical colonies were verified serologically using *Salmonella* agglutination latex test (Oxoid, Ogdensburg, NY). For *L. monocytogenes*, 1 ml of the stomachate was added to 9 ml of Fraser broth (Difco) and incubated for 24 h at 37°C. The enriched culture was streaked onto MOX agar and incubated for 24-48 h at 37°C. Typical colonies were verified using Visual Immunoassay for *Listeria* (TECRA Diagnostics, Roseville, Australia) according to the manufacturer’s instructions. For *Campylobacter* spp., 1 ml of the stomachate was added to 9 ml of *Campylobacter* enrichment broth (Bolton’s formula, Oxoid) with lysed horse blood and antibiotic supplement (Oxoid) and incubated for 4 h at 37°C, followed by 20 h at 42°C under microaerophilic conditions. The enriched culture was streaked onto mCCDA agar and incubated for 24 h at 42°C, under microaerophilic conditions. Typical colonies were verified serologically using *Campylobacter* agglutination test kit (Oxoid).

**Results**

**Chilling parameters**

The chilling parameters of the seven establishments used in the study are depicted in Table 1. Snap/blast chilling, followed by conventional chilling was performed in 1 small and 4 large establishments. Conventional chilling was performed in one very small and two small establishments. Most snap/blast chilling occurred between 90 and 150 min with air velocities ranging from 3.5 to 16 m/sec. Typical conventional chilling parameters ranged from 0 to 6°C for up to 24 h. In some establishments, snap/blast chilling was conducted in zones where different temperatures and air velocities were applied. Average carcass surface temperatures were obtained for warm (22.5°C) and chilled carcasses (4.62°C; Table 2). In very small and small plants, warm carcasses exhibited surface temperatures from 16.3 to 24.2°C. Surface temperatures from large plants ranged from 22.1 to 25.7°C. Carcass surface temperatures ranged from 3.70 to 7.90°C in the very small and small establishments using conventional chilling; whereas surface temperatures ranged from 0.30 to 3.70°C on carcasses subjected to snap/blast chilling, followed by a conventional chilling procedure. The larger establishments using snap/blast chilling exhibited the lowest carcass surface temperatures in this study.

**Microbiological analyses**

Microbiological populations were determined for warm and chilled carcasses from the seven establishments (Table 3). APC from warm carcasses ranged from 3.33 (very small) to 7.85 (small), with an average of 7.09 log\(_{10}\) CFU/300 cm\(^2\). *E. coli* counts from warm carcasses ranged from -0.01 (very small) to 2.60 (small) with an average of 1.93 log\(_{10}\) CFU/300 cm\(^2\). Coliform counts on warm carcasses ranged from -0.67 (large) to 2.91 (small), with an average of 2.23 log\(_{10}\) CFU/300 cm\(^2\).
Chilling (conventional or snap/blast) resulted in significant reductions in APC, *E. coli* and coliform populations. APC from chilled carcasses ranged from 1.88 (very small) to 5.96 (small), with an average of 5.21 log$_{10}$ CFU/300 cm$^2$. *E. coli* counts from chilled carcasses ranged from -1.11 (small) to 0.19 (small) with an average of -0.25 log$_{10}$ CFU/300 cm$^2$. Coliform counts on warm carcasses ranged from -0.63 (small) to 1.09 (large), with an average of 0.50 log$_{10}$ CFU/300 cm$^2$. Based on this information, chilling reduced APC, *E. coli*, and coliform populations >1.80, >1.90, and >1.73 log$_{10}$ CFU/300 cm$^2$, respectively.

*Salmonella* spp., *L. monocytogenes*, and *Campylobacter* spp. were found on warm or chilled carcasses from all seven establishments evaluated in this study (Table 4). *Salmonella* spp., *L. monocytogenes*, and *Campylobacter* spp. were found on warm carcasses at prevalence rate of 2.1, 1.2, and 7.4%, respectively. On chilled carcasses, *Salmonella* spp., *L. monocytogenes*, and *Campylobacter* spp. were found at 4.3, 0.05, and 0.0%. These data indicated that while the incidence of *L. monocytogenes* and *Campylobacter* spp. was reduced following chilling, *Salmonella* spp. increased, especially in carcasses processed in very small and small establishments.
<table>
<thead>
<tr>
<th>Plant Identification and Chill Type</th>
<th>Establishment Size</th>
<th>Length of chill</th>
<th>Air Temperature</th>
<th>Air velocity</th>
</tr>
</thead>
<tbody>
<tr>
<td>A Conventional</td>
<td>Very Small</td>
<td>24 h</td>
<td>0-4°C</td>
<td>2.5 m/s</td>
</tr>
<tr>
<td>B Snap/blast followed by conventional chill (CC)</td>
<td>Small</td>
<td>110 min</td>
<td>Zone 1: -15 to -10.5°C</td>
<td>Zone 1: 3.5 m/sec</td>
</tr>
<tr>
<td>C Snap/blast followed by conventional chill (CC)</td>
<td>Small</td>
<td>2 hrs, 30 min</td>
<td>Zone 2: 2.2°C</td>
<td>Zone 2: 1.02 to 5.7 m/sec</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Zone 3: 2.2°C</td>
<td>Zone 3: 1.02 to 5.7 m/sec</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>CC: 0-4°C</td>
<td>CC: n/a</td>
</tr>
<tr>
<td>D Conventional</td>
<td>Small</td>
<td>24 h</td>
<td>0-4°C</td>
<td>n/a</td>
</tr>
<tr>
<td>E Snap/blast followed by conventional chill (CC)</td>
<td>Large</td>
<td>90 min</td>
<td>-18 to -21°C</td>
<td>n/a</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>CC: 0-4°C</td>
<td>CC: n/a</td>
</tr>
<tr>
<td>F Snap/blast followed by conventional chill (CC)</td>
<td>Large</td>
<td>110 min</td>
<td>Zone 1: -15°C</td>
<td>13 m/s</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Zone 2: -10°C</td>
<td>CC: n/a</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Zone 3: -10°C</td>
<td>CC: n/a</td>
</tr>
<tr>
<td>G Snap/blast followed by conventional chill (CC)</td>
<td>Large</td>
<td>2 h</td>
<td>Zone 1: -15°C</td>
<td>Zone 1: 16 m/s</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Zone 2: -10°C</td>
<td>Zone 2: 13-16 m/s</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Zone 3: 0 to 5°C</td>
<td>Zone 3: 12-14 m/s</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>CC: 0-4°C</td>
<td>CC: n/a</td>
</tr>
</tbody>
</table>
Table 2. Carcass surface temperature.

<table>
<thead>
<tr>
<th>Plant Identification</th>
<th>Establishment Size</th>
<th>Warm Carcass Temperature (°C)</th>
<th>Chilled Carcass Temperature (°C)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Very Small</td>
<td>16.3* (23)**</td>
<td>6.00 (23)</td>
</tr>
<tr>
<td>B</td>
<td>Small</td>
<td>22.9 (30)</td>
<td>3.70 (30)</td>
</tr>
<tr>
<td>C</td>
<td>Small</td>
<td>24.2 (30)</td>
<td>7.90 (45)</td>
</tr>
<tr>
<td>D</td>
<td>Small</td>
<td>22.7 (30)</td>
<td>3.70 (40)</td>
</tr>
<tr>
<td>E</td>
<td>Small</td>
<td>22.1 (30)</td>
<td>0.30 (30)</td>
</tr>
<tr>
<td>F</td>
<td>Large</td>
<td>25.7 (15)</td>
<td>2.10 (15)</td>
</tr>
<tr>
<td>G</td>
<td>Large</td>
<td>22.5 (15)</td>
<td>3.70 (15)</td>
</tr>
<tr>
<td>Average carcass surface temperatures for all samples</td>
<td></td>
<td>22.5 (173)</td>
<td>4.62 (198)</td>
</tr>
</tbody>
</table>

*Average temperature
**(n)= number of samples
Table 3. Bacterial populations from warm and chilled carcasses obtained from individual establishments.

<table>
<thead>
<tr>
<th>Plant Identification</th>
<th>Establishment Size</th>
<th>Warm APC</th>
<th>Warm E. coli</th>
<th>Warm Coliforms</th>
<th>Chilled APC</th>
<th>Chilled E. coli</th>
<th>Chilled Coliforms</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Very Small</td>
<td>3.33*</td>
<td>-0.01</td>
<td>0.28</td>
<td>1.88</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(23)**</td>
<td>(23)</td>
<td>(23)</td>
<td>(23)</td>
<td>(23)</td>
<td>(23)</td>
</tr>
<tr>
<td>B</td>
<td>Small</td>
<td>7.85</td>
<td>2.60</td>
<td>2.91</td>
<td>5.96</td>
<td>0.01</td>
<td>0.59</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(30)</td>
<td>(30)</td>
<td>(30)</td>
<td>(30)</td>
<td>(30)</td>
<td>(30)</td>
</tr>
<tr>
<td>C</td>
<td>Small</td>
<td>3.73</td>
<td>0.92</td>
<td>1.13</td>
<td>4.70</td>
<td>-1.11</td>
<td>-0.63</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(30)</td>
<td>(30)</td>
<td>(30)</td>
<td>(45)</td>
<td>(45)</td>
<td>(45)</td>
</tr>
<tr>
<td>D</td>
<td>Small</td>
<td>4.73</td>
<td>1.37</td>
<td>1.45</td>
<td>3.70</td>
<td>0.19</td>
<td>0.41</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(30)</td>
<td>(30)</td>
<td>(30)</td>
<td>(40)</td>
<td>(40)</td>
<td>(40)</td>
</tr>
<tr>
<td>E</td>
<td>Large</td>
<td>4.42</td>
<td>-0.14</td>
<td>0.47</td>
<td>4.19</td>
<td>-0.78</td>
<td>1.09</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(30)</td>
<td>(30)</td>
<td>(30)</td>
<td>(30)</td>
<td>(30)</td>
<td>(30)</td>
</tr>
<tr>
<td>F</td>
<td>Large</td>
<td>3.54</td>
<td>-0.32</td>
<td>-0.67</td>
<td>4.05</td>
<td>-0.12</td>
<td>-0.12</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(15)</td>
<td>(15)</td>
<td>(15)</td>
<td>(15)</td>
<td>(15)</td>
<td>(15)</td>
</tr>
<tr>
<td>G</td>
<td>Large</td>
<td>4.44</td>
<td>1.84</td>
<td>2.02</td>
<td>4.15</td>
<td>-0.33</td>
<td>0.22</td>
</tr>
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<td></td>
<td></td>
<td>(15)</td>
<td>(15)</td>
<td>(15)</td>
<td>(15)</td>
<td>(15)</td>
<td>(15)</td>
</tr>
<tr>
<td>Average counts for all samples</td>
<td></td>
<td>7.09</td>
<td>1.93</td>
<td>2.23</td>
<td>5.21</td>
<td>-0.25</td>
<td>0.50</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(173)</td>
<td>(173)</td>
<td>(173)</td>
<td>(198)</td>
<td>(198)</td>
<td>(198)</td>
</tr>
</tbody>
</table>

*Log_{10} CFU/300 cm²

**(n) = number of samples taken at an individual establishment.
Table 4. Presence/Absence of pathogens from warm and chilled carcasses.

<table>
<thead>
<tr>
<th>Plant Identification</th>
<th>Establishment Size</th>
<th>Warm Salmonella spp. (%)</th>
<th>Warm L. monocytogenes (%)</th>
<th>Warm Campylobacter spp. (%)</th>
<th>Chilled Salmonella spp. (%)</th>
<th>Chilled L. monocytogenes (%)</th>
<th>Chilled Campylobacter spp. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Very Small</td>
<td>0/23* (0.0)**</td>
<td>1/23 (4.3)</td>
<td>0/23 (0.0)</td>
<td>4/23 (17)</td>
<td>0/23 (0.0)</td>
<td>0/23 (0.0)</td>
</tr>
<tr>
<td>B</td>
<td>Small</td>
<td>0/30 (0.0)</td>
<td>1/30 (3.3)</td>
<td>4/30 (13)</td>
<td>2/30 (6.7)</td>
<td>0/30 (0.0)</td>
<td>0/30 (0.0)</td>
</tr>
<tr>
<td>C</td>
<td>Small</td>
<td>2/45 (4.4)</td>
<td>0/45 (0.0)</td>
<td>6/45 (13)</td>
<td>2/45 (4.4)</td>
<td>0/45 (0.0)</td>
<td>0/45 (0.0)</td>
</tr>
<tr>
<td>D</td>
<td>Small</td>
<td>0/30 (0.0)</td>
<td>0/30 (0.0)</td>
<td>2/30 (6.7)</td>
<td>0/40 (0.0)</td>
<td>0/40 (0.0)</td>
<td>0/40 (0.0)</td>
</tr>
<tr>
<td>E</td>
<td>Large</td>
<td>1/30 (3.3)</td>
<td>0/30 (0.0)</td>
<td>2/30 (6.7)</td>
<td>0/15 (0.0)</td>
<td>0/15 (0.0)</td>
<td>0/15 (0.0)</td>
</tr>
<tr>
<td>F</td>
<td>Large</td>
<td>1/15 (6.7)</td>
<td>0/15 (0.0)</td>
<td>0/15 (0.0)</td>
<td>0/15 (0.0)</td>
<td>0/15 (0.0)</td>
<td>0/15 (0.0)</td>
</tr>
<tr>
<td>G</td>
<td>Large</td>
<td>0/15 (0.0)</td>
<td>0/15 (0.0)</td>
<td>0/15 (0.0)</td>
<td>1/15 (6.7)</td>
<td>0/15 (0.0)</td>
<td>0/15 (0.0)</td>
</tr>
<tr>
<td>% incidence for all samples</td>
<td></td>
<td>4/188 (2.1)</td>
<td>2/188 (1.2)</td>
<td>14/188 (7.4)</td>
<td>8/184 (4.3)</td>
<td>1/184 (0.05)</td>
<td>0/184 (0.0)</td>
</tr>
</tbody>
</table>

*Represents number of samples positive for pathogen/number of samples
**(%) = % positive for pathogen
Discussion

This study clearly demonstrates that overall microbial populations (APC, \textit{E. coli}, and coliforms), \textit{L. monocytogenes}, and \textit{Campylobacter} spp. were reduced as a result of chilling carcasses. These findings confirm previous laboratory studies conducted by Chang et al (2003b). In this particular study, both snap/blast- and conventional-chilling treatments were more effective against all bacterial populations when pork samples where inoculated with high inoculation levels \((5 \log_{10} \text{CFU/cm}^2)\) of pathogens. More importantly, pork samples inoculated with low levels of \textit{C. coli} and subjected to snap/blast-chilling were reduced to undetectable levels \(< 1.3 \log_{10} \text{CFU/cm}^2\). In the present study, a slight increase in \textit{Salmonella} spp. prevalence on chilled carcasses processed in very small and small establishments was observed. After chilling, carcass temperatures processed in very small and small establishments that employ conventional chilling were higher than carcasses processed under snap/blast chill conditions. It can be concluded that higher air temperatures observed in very small and small establishments do affect carcass chilling, thereby explaining the increase in \textit{Salmonella} spp. prevalence. This study demonstrates that commercial chilling conditions may reduce most bacterial populations associated with pork carcasses. This information may provide processors with an additional means to control microbes in/on freshly slaughtered pork, thereby improving the microbiological safety of pork products.

Lay Interpretation

Previous laboratory studies have demonstrated that snap/blast chilling is known to significantly reduce some bacterial populations on pork surfaces, including \textit{Salmonella}, \textit{Campylobacter}, \textit{E. coli}, and coliforms. However, very little information exists as to whether reductions observed under commercial conditions can be attributed to chilling parameters. In this study, individual carcass sponge samples from 386 carcasses were obtained; 188 samples from warm carcasses and 198 samples from chilled carcasses. Chilled carcasses were subjected to snap/blast chilling (followed by conventional chilling) or conventional chilling and evaluated for mesophilic aerobic plate counts (APC), generic \textit{E. coli} (EC), coliforms (CF), as well as the presence/absence of \textit{Salmonella} spp., \textit{Campylobacter} spp., and \textit{Listeria monocytogenes}. Carcass samples were obtained from one very small, three small, and three large pork processing establishments. Of these, three establishments used conventional chilling only and four establishments used snap/blast chilling, followed by conventional chilling. Overall, warm carcasses sampled with the USDA three-site sampling procedure exhibited approximately 10 million APC, 85 EC, and 170 CF in a 300 square cm area. \textit{Salmonella} spp., \textit{L. monocytogenes}, and \textit{Campylobacter} spp. also were present on 2.1%, 1.2%, and 7.4% of the warm carcasses, respectively. Chilled carcasses exhibited approximately 160,000 APC, 2 EC, and 3 CF in a 300 square cm area. Pathogen prevalence on the chilled carcasses was 4.3% \textit{Salmonella} spp., 0.05% \textit{L. monocytogenes}, and 0.0% \textit{Campylobacter} spp. In addition to microbial populations, chilling parameters, and carcass surface temperature data were collected. Based on these findings, the use of snap/blast- or conventional chilling processes that reduce pathogenic bacteria may provide processors with an additional means to control pathogens in/on freshly slaughtered pork, thereby improving the microbiological safety of pork products.

References:


