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Prevalence, Enumeration, Serotypes, and Antimicrobial Resistance Phenotypes of *Salmonella enterica* Isolates from Carcasses at Two Large United States Pork Processing Plants

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The objective of this study was to characterize *Salmonella enterica* contamination on carcasses in two large U.S. commercial pork processing plants. The carcasses were sampled at three points, before scalding (prescald), after dehairing/polishing but before evisceration (preevisceration), and after chilling (chilled final). The overall prevalences of *Salmonella* on carcasses at these three sampling points, prescald, preevisceration, and after chilling, were 91.2%, 19.1%, and 3.7%, respectively. At one of the two plants, the prevalence of *Salmonella* was significantly higher ($P < 0.01$) for each of the carcass sampling points. The prevalences of carcasses with enumerable *Salmonella* at prescald, preevisceration, and after chilling were 37.7%, 4.8%, and 0.6%, respectively. A total of 294 prescald carcasses had *Salmonella* loads of >1.9 log CFU/100 cm^2, but these carcasses were not equally distributed between the two plants, as 234 occurred at the plant with higher *Salmonella* prevalences. Forty-one serotypes were identified on prescald carcasses with *Salmonella enterica* serotypes Derby, Typhimurium, and Anatum predominating. *S. enterica* serotypes Typhimurium and London were the most common of the 24 serotypes isolated from preevisceration carcasses. The *Salmonella* serotypes Johannesburg and Typhimurium were the most frequently isolated serotypes of the 9 serotypes identified from chilled final carcasses. Antimicrobial susceptibility was determined for selected isolates from each carcass sampling point. Multiple drug resistance (MDR), defined as resistance to three or more classes of antimicrobial agents, was identified for 71.2%, 47.8%, and 77.5% of the tested isolates from prescald, preevisceration, and chilled final carcasses, respectively. The results of this study indicate that the interventions used by pork processing plants greatly reduce the prevalence of *Salmonella* on carcasses, but MDR *Salmonella* was isolated from 3.2% of the final carcasses sampled.

Food-borne nontyphoidal *Salmonella enterica* (NTS) is estimated to sicken 1 million people annually in the United States, resulting in approximately 19,000 hospitalizations and 378 deaths (13). In most cases, the disease is self-limiting, but invasive salmonellosis is estimated to occur in 5% of cases. Over 2,500 serotypes of *Salmonella enterica* have been identified, but they vary in their host range and ability to cause disease in humans. Human infections caused by four serotypes commonly isolated from carcass samples from swine, *Salmonella enterica* serotypes Choleraesuis, Heidelberg, Schwarzengrund, and Brandenburg, result in significantly higher proportions of invasive disease than that observed for infections caused by *S. enterica* serotype Typhimurium (44).

In the United States, 5% of illnesses due to NTS are attributed to pork products (23). There were six pork-related *Salmonella* outbreaks in the United States during 2007 that resulted in 208 illnesses and 24 hospitalizations (14, 18). Studies on the prevalence of *Salmonella* in U.S. retail pork products are very limited, and results vary from "less than 2%" in one study (66) to 9.6% in another (27). The prevalence of *Salmonella* on carcasses in the United States after chilling (chilled final) examined between 2001 and 2009 ranged between 2 and 4% according to the annual reports of the Food Safety and Inspection Service (FSIS) testing of U.S. slaughter establishments (33). *Salmonella enterica* serotypes Derby and Typhimurium were the two most commonly detected serotypes in FSIS-tested chilled final carcasses and in both clinical and nonclinical veterinary swine samples (31, 33). While *Salmonella* Derby was not among the 20 most commonly isolated serotypes from human clinical samples, *Salmonella* Typhimurium was the most frequently isolated serotype from all human clinical samples and from invasive infections reported to the Centers for Disease Control from 1996 to 2007 (17, 20, 44).

Expanded-spectrum cephalosporins (ESCs) and fluoroquinolones are commonly prescribed antimicrobial agents for the treatment of invasive salmonellosis (37). However, antimicrobial treatment of invasive salmonellosis has been complicated by an increase in *Salmonella* bacteria that are resistant to antimicrobials (16, 47). Additionally, several studies concluded that infections with *Salmonella* resistant to multiple antimicrobials, known as multidrug-resistant (MDR) *Salmonella*, are more invasive than infections caused by non-MDR *Salmonella* (20, 39, 61, 62). Infants are at higher risk for invasive salmonellosis (63), and the incidence of laboratory-confirmed NTS in infants (children less than 1 year old) is much greater than in other age groups (17, 19). Clinical reports of the isolation of *Salmonella* resistant to ESCs, including the drug of choice, ceftriaxone, are increasing (58, 64). This is a grave concern because ceftriaxone and related ESCs are the only treatment option in children due to the risk of cartilage damage from fluoroquinolone use (48). *Salmonella* bacteria resistant to

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ESCs have been isolated from pork products, demonstrating the need for data on the prevalence of drug-resistant *Salmonella* in the pork processing environment (50, 65).

In spite of the evidence of contamination of pork with *Salmonella*, the increased occurrence and invasiveness of MDR *Salmonella*, and the prevalence of *Salmonella* serotypes associated with invasive infections in samples from swine, few studies, if any, have examined the prevalence, load, serotype, and antimicrobial resistance of *Salmonella* present on swine carcasses at harvest. In particular, *Salmonella* occurrence on the skin of swine upon arrival in the abattoir and during the scalding/singeing/polishing process was identified as specific data gaps by the authors of a recent quantitative risk assessment model of the prevalence of *Salmonella* on swine carcasses (5). In the present study, samples were taken with sponges from three points along the pork processing line at two commercial U.S. pork processing facilities: the prescald carcass (postexsanguination), the preevisceration carcass (postscald, singe, and polish), and the chilled final carcass. The resulting prevalence and enumeration data will aid in improving quantitative risk assessment models and in the formulation of interventions to reduce the occurrence of *Salmonella* on the final swine carcasses.

MATERIALS AND METHODS

Sample collection. Two large-scale commercial pork processing plants located in the United States were each sampled eight times between summer 2007 and spring 2008. Each plant (designated plant A and plant B) was visited once a season (summer, fall, winter, and spring), and carcass samples were collected over two consecutive days on each trip, totaling eight sampling days per plant for a total of 16 sampling days. On each sampling day, 95 samples were taken from each of three sampling points on the processing line: prescald carcass, preevisceration carcass, and chilled final carcass. Over the course of the study, a total of 4,560 samples were taken, 1,520 at each sample point. All samples were taken with sterile sponges (Whirl Pak; Nasco, Fort Atkinson, WI) prewetted with 20 ml of buffered peptone water (BPW; Becton Dickinson, Franklin Lakes, NJ). To prevent cross contamination, gloves were worn during sampling and were changed following each sample. Prescald carcass samples were obtained by using both sides of the prewetted sponge to swab an area of approximately 1,500 cm^2 along the belly midline. After scalding, singeing, and polishing of the carcass, preevisceration carcass samples were obtained by using both sides of the prewetted sponge to swab approximately 4,000 cm^2 of the carcass surface along the midline from ham to breast, including foreshank and jowl. Final carcass samples were obtained from carcasses that had been chilled at least overnight in coolers, by using both sides of the sponge to swab approximately 4,000 cm^2 of the carcass surface along the split midline from ham collar to jowl and foreshank. No effort was made to match samples taken from each point to specific animals or groups of animals at other points. Prescald and preevisceration samples were collected at the same time. Chilled final carcass samples were collected on the same day, from carcasses harvested previously (after 24 to 72 h of chilling). All samples were transported in coolers with ice packs, received, and processed at the U.S. Meat Animal Research Center within 24 h of collection.

***Salmonella* enumeration.** Enumeration of *Salmonella* present in carcass samples was performed as previously described (9) with the following modification. For prescald carcass samples, 500 μl of liquid from the sponge bag was removed, placed in a 1.5-ml microcentrifuge tube, vortexed briefly, and allowed to settle for 3 min. Fifty microliters was then spiral plated onto xylose lysine desoxycholate medium (Oxoid, Basingstoke, United Kingdom) containing 4.6 ml $liter^{-1}$ Tergitol, 15 mg $liter^{-1}$ novobiocin, and 5 mg $liter^{-1}$ cefsulodin (XLD_{tnc}). For preevisceration and chilled final carcass samples, 3 ml of liquid from the sponge bag was mixed with 4 ml of BPW containing 1% (vol/vol) Tween 80. The resulting 7-ml sample was then filtered through an Iso-Grid hydrophobic grid membrane filter (HGMF) (Neogen Corp., Lansing, MI) using a FiltaFlex HGMF apparatus (FiltaFlex, Almonte, Ontario, Canada). The HGMF was placed on an XLD_{tnc} plate. The XLD_{tnc} plates were incubated at 37°C for 18 to 20 h and at room temperature (23 to 25°C) for an additional 18 to 20 h. For each sample, the number of presumptive *Salmonella* on the plate was recorded. Up to 10 presumptive *Salmonella* colonies from each sample were inoculated into 0.7-ml tryptic soy broth (TSB; Becton Dickinson) cultures contained in 96-well blocks. Inoculated blocks were incubated overnight at 37°C. PCR of the *Salmonella*-specific portion of the *invA* gene was used to confirm the presence of *Salmonella* (52, 53). The number of confirmed *Salmonella* was determined for each sample by multiplying the number of presumptive *Salmonella* colonies by the percentage of overnight cultures confirmed to contain *Salmonella* by *invA* PCR (9). The number of confirmed *Salmonella* was then reported as log CFU per 100 cm^2. The lower limit of detection for prescald carcass samples was 1.4 log CFU/100 cm^2. The lower limit of detection for preevisceration and chilled final carcass samples was −0.8 log CFU/100 cm^2.

***Salmonella* prevalence.** Eighty milliliters of TSB was added to each sample taken with a sponge. The samples were preenriched at 25°C for 2 h, heated to 42°C for 6 h, and then held at 4°C (generally 8 to 10 h) until the samples were processed the next day (4). A 1-ml aliquot of each preenrichment sample was removed and mixed with 20 μl of *Salmonella*-specific immunomagnetic separation beads (Dynal, Lake Success, NY) (21). The bacterium-bead complex was extracted, placed into a Rappaport-Vassiliadis soy peptone broth selective enrichment (Oxoid), and incubated at 42°C for 18 to 20 h (8). The selective enrichment was then swabbed onto XLD_{tnc} and Difco brilliant green agar containing 80 mg $liter^{-1}$ sulfadiazine (Becton Dickinson) (8). Up to four presumptive *Salmonella* colonies from each sample were selected for confirmation by PCR for the presence of the *Salmonella*-specific portion of the *invA* gene as described above (52, 53).

Identification of *Salmonella* serotypes. Serotyping was performed on each *Salmonella* isolate confirmed positive for *invA* by PCR ($n = 6{,}089$). A frozen overnight culture of each of these isolates confirmed to be *Salmonella* was streaked for isolation on an XLD_{tnc} plate and incubated at 37°C for 18 to 20 h. One colony demonstrating typical *Salmonella* morphology from each plate was selected, streaked on tryptic soy agar (TSA; Becton Dickinson), and incubated at 37°C for 18 to 20 h. The resulting pure cultures were subjected to molecular serotyping methods (29, 40, 41) and further confirmed by serologic methods using Wellcolex Color *Salmonella* agglutination (Remel, Lenexa, KS) and traditional slide agglutination serotype O grouping and tube agglutination flagellar H typing, using commercial antisera (Denka-Seken Co. Ltd., Tokyo, Japan) following the manufacturer's guidelines.

Identification of antimicrobial-resistant isolates. Each confirmed *Salmonella* isolate was initially screened for antimicrobial resistance by replica plating onto four 150-mm TSA plates supplemented with either no additional antimicrobial agents, 32 mg $liter^{-1}$ ampicillin, 32 mg $liter^{-1}$ tetracycline, or 64 mg $liter^{-1}$ kanamycin using a 96-pin Boekel microplate replicator (Boekel Scientific, Feasterville, PA). It has been demonstrated that resistances to these antimicrobials are the most frequently observed in *Salmonella* (6, 30, 60). Isolates identified as resistant to one or more of these three antimicrobials were grouped into categories based on their sampling point, serotype, growth on the screened antimicrobials, and sampling day isolated.

Antimicrobial susceptibility determination. At least one isolate from each category of resistant isolates described in the previous section was arbitrarily selected for antimicrobial susceptibility testing (912 isolates tested overall). Antimicrobial susceptibility testing was performed using the Sensititre broth microdilution system (TREK Diagnostic Systems, Toledo, OH) and CMV1AGNF test plates to determine the MIC for each of 15 antimicrobial agents. The antimicrobial agents, their abbreviations (shown in parentheses), and breakpoints for resistance in this panel were as follows: amikacin (AMI), ≥64 μg ml^{-1}; amoxicillin-clavulanic acid

TABLE 1 Prevalence of *Salmonella* on the carcasses of swine[a]

Sample point	Prevalence (%)	No. of serotypes isolated	No. of times the following no. of serotypes was isolated per sample: 1	2	3	4	5	>5
Prescald carcasses	91.2	41	676	563	138	8	1	0
Preeviseration carcasses	19.1	24	263	25	3	0	0	0
Chilled final carcasses	3.7	9	56	0	0	0	0	0

[a] A total of 1,520 samples were taken from carcasses at each sampling point (prescald, preeviseration, and after chilling).

(AMC), ≥32-16 μg ml^{-1}; ampicillin (AMP), ≥32 μg ml^{-1}; cefoxitin (FOX), ≥32 μg ml^{-1}; ceftiofur (TIO), ≥8 μg ml^{-1}; ceftriaxone (AXO), ≥16 μg ml^{-1}; chloramphenicol (CHL), ≥32 μg ml^{-1}; ciprofloxacin (CIP), ≥4 μg ml^{-1}; gentamicin (GEN), ≥16 μg ml^{-1}; kanamycin (KAN), ≥64 μg ml^{-1}; nalidixic acid (NAL), ≥32 μg ml^{-1}; streptomycin (STR), ≥64 μg ml^{-1}; sulfisoxazole (FIS), ≥512 μg ml^{-1}; tetracycline (TET), ≥16 μg ml^{-1}; and trimethoprim-sulfamethoxazole (COT), ≥4-76 μg ml^{-1}. In this study, isolates resistant to three or more classes of antimicrobials were considered MDR. The antimicrobial classes were as follows: aminoglycoside (AMI, GEN, KAN, and STR), β-lactam/β-lactamase inhibitor combination (AMC), cephem (FOX, TIO, and AXO), folate pathway inhibitor (FIS and COT), penicillin (AMP), phenicol (CHL), quinolone (CIP and NAL), and tetracycline (TET).

Statistics. *Salmonella* prevalence and percent enumerable values were evaluated with the Compare2 program of the WinPepi (version 11.7) package (1). Comparisons with *P* values of <0.01 by Pearson's χ^2 test with Bonferroni's correction for multiple comparisons were considered significant.

RESULTS

Prevalence, load, serotype, and antimicrobial resistance of *Salmonella* on prescald carcasses. *Salmonella* was isolated from 1,386 of the 1,520 prescald carcass samples resulting in an overall *Salmonella* prevalence of 91.2% (Table 1). *Salmonella* was enumerated from 573 carcass samples and isolated from enrichment cultures of 1,379 samples. *Salmonella* was not isolated by enrichment culture of seven enumerable samples. At plant A, *Salmonella* prevalence on prescald carcasses was 97.6%, significantly higher ($P < 0.01$) than the 84.7% prevalence observed at plant B (Table 2). *Salmonella* prevalence on prescald carcasses was significantly higher ($P < 0.01$) during spring (100%) and winter (96.3%) than during summer (87.6%) and fall (80.8%) (Table 2). At plant B, the seasonal prevalences of 77.4% in summer and 61.6% in fall were significantly lower ($P < 0.01$) than the 100% prevalence during winter and spring. The plant B summer and fall prevalences were the lowest in this study and account entirely for the overall lower prevalences during summer and fall, since plant A seasonal prevalences ranged between 92.6% and 100% with the lowest ($P < 0.01$) seasonal prevalence occurring during winter (Table 1).

Overall, 279 prescald carcasses were determined to have *Salmonella* loads between 1.4 and 1.9 log CFU/100 cm^2 (Table 3). *Salmonella* loads of 2.0 to 2.9 log CFU/100 cm^2 were recorded for 170 prescald carcasses, and 124 carcasses had loads greater than 2.9 log CFU/100 cm^2. Similar to the prevalence results, the percentage of prescald carcasses with enumerable loads was significantly higher ($P < 0.01$) during spring (56.3%) and winter (47.1%) than during fall (29.7%) and summer (17.6%) (Table 4). *Salmonella* loads of >1.9 log CFU/100 cm^2 were most frequently detected from prescald carcasses during spring ($n = 114$), followed by winter ($n = 111$), fall ($n = 64$), and summer ($n = 5$). The percentage of prescald carcasses with enumerable *Salmonella* was significantly higher ($P < 0.01$) at plant A (48.7%) than at plant B (26.7%). Additionally, enumerable *Salmonella* loads of >1.9 log CFU/100 cm^2 were more frequently detected from prescald carcasses at plant A ($n = 234$) than at plant B ($n = 60$) (Table 4). At plant A, the highest percentages ($P < 0.01$) of prescald carcasses

TABLE 2 *Salmonella* prevalence on carcasses of swine by plant, season, and both plant and season

Prevalence by plant and/or season	Prescald carcass: No. sampled	Prescald carcass: Prevalence (%)[a]	Preevisceration carcass: No. sampled	Preevisceration carcass: Prevalence (%)	Chilled final carcass: No. sampled	Chilled final carcass: Prevalence (%)
Prevalence by plant						
Plant A	760	97.6 A	760	32.0 A	760	6.4 A
Plant B	760	84.7 B	760	6.3 B	760	0.9 B
Prevalence by season						
Summer	380	87.6 C	380	11.1 B	380	6.3 A
Fall	380	80.8 C	380	7.9 B	380	0.0 B
Winter	380	96.3 B	380	26.1 A	380	3.4 A
Spring	380	100.0 A	380	31.6 A	380	5.0 A
Prevalence by season for plant A						
Summer	190	97.9 AB	190	16.8 B	190	12.6 A
Fall	190	100.0 A	190	13.7 B	190	0.0 B
Winter	190	92.6 B	190	42.1 A	190	4.2 A
Spring	190	100.0 A	190	55.3 A	190	8.9 A
Prevalence by season for plant B						
Summer	190	77.4 B	190	5.3 AB	190	0.0 A
Fall	190	61.6 C	190	2.1 B	190	0.0 A
Winter	190	100.0 A	190	10.0 A	190	2.6 A
Spring	190	100.0 A	190	7.9 AB	190	1.1 A

[a] Prevalence values in the same column and subheading that do not have a common letter are statistically different ($P < 0.01$).

TABLE 3 Enumeration of *Salmonella* on swine carcasses

Sample point[a]	% samples with enumerable *Salmonella*	Frequency of enumeration (log CFU/100 cm^2)					
		−0.8 to −0.1	0.0 to 0.9	1.0 to 1.9	2.0 to 2.9	3.0 to 3.9	4.0 to 4.9
Prescald carcasses[b]	37.7	n/a	n/a	279	176	106	12
Preeviseration carcasses[c]	4.8	55	15	3	0	0	0
Chilled final carcasses[c]	0.6	6	1	2	0	0	0

[a] A total of 1,520 samples were taken from carcasses at each sampling point.
[b] The lower limit of detection for prescald carcass samples was 1.4 log CFU/100 cm^2.
[c] The lower limit of detection for preevisceration and chilled final carcass samples was −0.8 log CFU/100 cm^2.

with enumerable *Salmonella* loads occurred during spring (65.3%) and fall (55.8%), followed by winter (47.4%) and summer (26.3%). At plant B, the percentages of prescald carcasses with enumerable *Salmonella* loads were significantly higher ($P < 0.01$) during spring (47.4%) and winter (46.8%) than during summer (8.9%) and fall (3.7%).

Overall, 41 *Salmonella* serotypes were isolated from the prescald carcass samples, and multiple serotypes were isolated from the same sample more frequently ($n = 710$) than isolation of a single serotype from the same sample ($n = 676$) (Table 1). The three most prevalent serotypes on prescald carcasses were *Salmonella* serotypes Derby, Typhimurium, and Anatum, which were isolated from 437, 412, and 316 samples, respectively. The next most prevalent serotypes isolated from prescald carcass were *Salmonella* serotypes Infantis, Agona, London, and Munster, which were isolated from 178, 170, 166, and 96 samples, respectively. Some serotypes were very narrowly distributed; for example, all 75 *Salmonella* serotype Brandenburg prescald carcass prevalence-positive samples were from the spring 2 sampling day at plant A (Table 5). Interestingly, the most prevalent serotype on the first day of seasonal sampling at a plant was never the most prevalent on the following day. Indeed, eight different serotypes (*Salmonella* serotypes Agona, Anatum, Brandenburg, Derby, Infantis, London, Muenster, and Typhimurium) were the most prevalent prescald carcass serotype on at least 1 day (Table 5).

Limited resources prevented antimicrobial susceptibility testing of all 5,318 prescald carcass *Salmonella* isolates. Replica plating onto media containing AMP, KAN, or TET determined that 4,092 isolates were resistant to at least one of these antimicrobial agents. A total of 697 of these resistant isolates were selected for antimicrobial susceptibility testing as described in Materials and Methods. Of these 697 isolates, 496 (71.2%) were MDR (resistant to 3 or more classes of antimicrobial agents) (Table 6). A total of 255 (36.6%) isolates were resistant to AMP, CHL, STR, FIS, and TET (ACSSuT^r), their serotypes were as follows: *Salmonella* serotypes Typhimurium ($n = 218$), Agona ($n = 27$), Derby ($n = 3$), Ohio ($n = 2$), Heidelberg ($n = 2$), London ($n = 1$), and Rissen ($n = 1$) and not typeable ($n = 1$). Fifty-one isolates (7.3%) were resistant to the expanded-spectrum cephalosporin AXO, and their serotypes were as follows: *Salmonella* serotypes Agona ($n = 27$), Seftenberg ($n = 11$), London ($n = 5$), Ohio ($n = 2$), Heidelberg ($n = 2$), Derby ($n = 1$), Havana ($n = 1$), London ($n = 1$), and Rissen ($n = 1$). Three isolates (2 *Salmonella* Heidelberg isolates and 1 *Salmonella* Derby isolate) were resistant to an antimicrobial agent from all eight classes tested.

Prevalence, load, serotype, and antimicrobial resistance of *Salmonella* on preevisceration carcasses. *Salmonella* was isolated from 291 (19.1%) preevisceration carcasses (Table 1). *Salmonella* was enumerated from 73 samples and isolated from enrichment cultures of 288 samples, but *Salmonella* was not isolated by enrichment culture from three of the enumerable samples. At plant A, *Salmonella* prevalence on preevisceration carcasses was 32.0%, significantly higher ($P < 0.01$) than the 6.3% prevalence observed at plant B (Table 2). *Salmonella* prevalence on preevisceration carcasses was significantly higher ($P < 0.01$) during spring (31.6%) and winter (26.1%) than during summer (11.1%) and fall (7.9%) (Table 2). The seasonal differences at plant A contributed greatly to the overall seasonal differences, since the prevalences were significantly higher at plant A ($P < 0.01$) during winter (42.1%) and spring (55.3%) than during summer (16.8%) and fall (13.7%), while at plant B the seasonal prevalences were all 10.0% or less (Table 2).

At plant A, 7.9% of preevisceration carcasses had enumerable *Salmonella*, significantly higher ($P < 0.01$) than the 1.7% of enumerable preevisceration carcasses at plant B (Table 4). The percentage of preevisceration carcasses with enumerable loads was significantly higher ($P < 0.01$) during spring (9.7%) and winter (7.6%) than during summer (1.6%) and fall (0.3%) (Table 4). The majority of the enumerable samples (80.8%) were from two seasons at plant A, spring and winter. Accordingly, the highest percentages of preevisceration carcasses with enumerable *Salmonella* by season and plant were spring at plant A (16.8%) and winter at plant A (14.2%), while the percentage of preevisceration carcasses with enumerable *Salmonella* was ≤2.6 for all seasons at plant B and during the summer and fall seasons at plant A (Table 4). *Salmonella* loads were <0.0 log CFU/100 cm^2 on 55 of the enumerable preevisceration carcasses. Only 18 preevisceration carcasses had loads of ≥0.0 log CFU/100 cm^2 (Table 3).

Overall, 24 *Salmonella* serotypes were isolated from the preevisceration carcass samples, and multiple serotypes were isolated from the same sample less frequently ($n = 28$) than isolation of a single serotype from the same sample ($n = 263$) (Table 1). The two most prevalent serotypes on preevisceration carcasses were *Salmonella* serotypes Typhimurium and London, isolated from 109 and 70 samples, respectively. The next most prevalent serotypes isolated from preevisceration carcasses were *Salmonella* serotypes Derby, Agona, and Brandenburg, isolated from 28, 27, and 18 samples, respectively. Nonuniform distribution was observed for each of the five most prevalent serotypes from *Salmonella*-positive preevisceration carcasses, >50% of the positive samples were from one sampling day, and >75% of the positive samples were from a single plant (Table 5). Indeed, 59 of the 109 *Salmonella* Typhimurium-positive preevisceration carcasses were from a single day (spring day 1) at plant A, and 103 were from plant A (Table 5). Additionally, 54 of the 70 *Salmonella* London-positive preevisceration carcasses were

TABLE 4 Enumeration of *Salmonella* on swine carcasses by plant, season, and both plant and season

Enumeration by plant and/or season	Prescald carcass						Preevisceration carcass					Chilled final carcass				
	No. sampled	% with enumerable *Salmonella*[a]	Frequency of enumeration (log CFU/100 cm^2)				No. sampled	% with enumerable *Salmonella*	Frequency of enumeration (log CFU/100 cm^2)			No. sampled	% with enumerable *Salmonella*	Frequency of enumeration (log CFU/100 cm^2)		
			1.4 to 1.9	2.0 to 2.9	3.0 to 3.9	4.0 to 4.9			−0.8 to −0.1	0.0 to 0.9	1.0 to 1.9			−0.8 to −0.1	0.0 to 0.9	1.0 to 1.9
Enumeration by plant																
Plant A	760	48.7 A	136	115	101	18	760	7.9 A	46	13	1	760	0.5 A	3	1	0
Plant B	760	26.7 B	143	55	5	0	760	1.7 B	9	2	2	760	0.7 A	3	0	2
Enumeration by season																
Summer	380	17.6 C	62	5	0	0	380	1.6 B	3	1	2	380	0.3 A	1	0	0
Fall	380	29.7 B	49	46	18	0	380	0.3 B	1	0	0	380	0.0 A	0	0	0
Winter	380	47.1 A	68	79	29	3	380	7.6 A	24	5	0	380	1.3 A	3	0	2
Spring	380	56.3 A	100	40	59	15	380	9.7 A	27	9	1	380	0.8 A	2	1	0
Enumeration by season for plant A																
Summer	190	26.3 C	48	2	0	0	190	0.5 B	1	0	0	190	0.5 A	1	0	0
Fall	190	55.8 AB	42	46	18	0	190	0.0 B	0	0	0	190	0.0 A	0	0	0
Winter	190	47.4 B	21	41	25	3	190	14.2 A	22	5	0	190	0.0 A	0	0	0
Spring	190	65.3 A	25	26	58	15	190	16.8 A	23	8	1	190	1.6 A	2	1	0
Enumeration by season for plant B																
Summer	190	8.9 B	14	3	0	0	190	2.6 A	2	1	2	190	0.0 A	0	0	0
Fall	190	3.7 B	7	0	0	0	190	0.5 A	1	0	0	190	0.0 A	0	0	0
Winter	190	46.8 A	47	38	4	0	190	1.1 A	2	0	0	190	2.6 A	3	0	2
Spring	190	47.4 A	75	14	1	0	190	2.6 A	4	1	0	190	0.0 A	0	0	0

[a] Percent enumerable *Salmonella* values in the same column and subheading that do not have a common letter are statistically different ($P < 0.01$).

TABLE 5 *Salmonella* serotype prevalences on swine carcasses

Plant, season, and sampling day	Serotype (no. of samples isolated from carcasses)[a]		
	Prescald carcass	Preevisceration carcass	Chilled final carcass
Plant A			
Summer			
Day 1	MNS (67), TYP (17), ANA (17), MVD (10), SEN (8), DER (6), AGN (4), INF (4), KEN (2), CUB (1), DJU (1), NT (1)	LDN (9), MNS (7), TYP (2), DER (1), II (1)	JOH (14), CER (1), TYP (1)
Day 2	DER (47), TYP (42), MNS (25), LDN (15), CER (14), KEN (13), SEN (5), ANA (4), THO (4), WOR (2), AGN (1), MBA (1), MVD (1), NT (1)	ALT (5), DER (4), LDN (3), WOR (1)	JOH (8)
Fall			
Day 1	TYP (94), ANA (30), MBA (12), DER (12), LDN (8), MVD (6), KEN (3), SEN (2), INF (1), MNS(1), MO7 (1), NT (1)	TYP (8), MBA (6), INF (2), DER (1)	–[b]
Day 2	DER (43), TYP (40), ANA (21), JOH (18), MVD (16), KEN (12), MBA (9), LDN (8), CER (3), SEN (2), MNS (1), TOU (1)	MBA (7), DER (2), TYP (1)	–
Winter			
Day 1	ANA (35), DER (34), TYP (25), UGA (8), LDN (7), BRD (3), AGN (2)	TYP (15), INF (1)	TYP (1)
Day 2	LDN (92), ANA (42), DER (5), TYP (1), NT (1)	LDN (54), TYP (10), ANA (3), SEN (2), DER (1), MNC (1), UGA (1), NT (1)	DER (3), TYP (2), ANA (1), MNC (1)
Spring			
Day 1	TYP (95), PUT (55), DER (24), OHI (8), LDN (4), BRD (4), ANA (1), MNS (1)	TYP (59), AGN (6), LDN (4), PUT (2), SEN(2)	INF (2), TYP (1)
Day 2	BDB (75), TYP (35), LDN (26), DER (15), BRD (14), PUT (6), OHI (4), ANA (2), CUB (2), SPA (2), INF (1), NT (1)	BDB (18), DER (15), TYP (8), SPA (1), INF (1), SWZ (1)	TYP (12), PUT (2)
Plant B			
Summer			
Day 1	DER (58), AGN (22), SWZ (10), TYP (4), HAD (2), OHI (2), LEX (1), LDN (1), SEN (1), NT (1)	RIS (2), NT (2), DER (1)	–
Day 2	AGN (30), DER (21), SPA (19), TYP (10), SWZ (3), NT (2), HEI (1), MVD (1), MNC (1)	OHI (6), TYP (3), BUK (1), NT (1)	–
Fall			
Day 1	ANA (28), DER (23), MIN (7), TYP (7), HAV (4), AGN (3), MBA (3), SWZ (3), KRE (2), BRD (1), JOH (1)	AGN (3), DER (1)	–
Day 2	TYP (24), DER (22), OHI (7), MBA (3), KRE (2), ANA (1), RIS (1), SEN (1), NT (1)	HEI (1)	–
Winter			
Day 1	DER (71), KRE (40), INF (19), HEI (7), TYP (5), ADE (5), LDN (4), BER (2), JAM (2), BOV (1), MEL (1), MNS (1)	TYP (1), MO6 (1)	–
Day 2	AGN (86), ANA (56), DER (25), INF (21), TYP (3), MEL (1), UGA (1)	AGN (15), DER (1), INF (1)	MO6 (5)
Spring			
Day 1	ANA (73), INF (39), DER (24), AGN (18), TYP (9), IDK (1), LDN (1)	INF (4), AGN (3), TYP (2), ANA (1), DER (1), SWZ (1)	TYP (1)
Day 2	INF (93), DER (7), ANA (6), AGN (4), MBA (2), JOH (1), MNC (1), TYP (1)	INF (4)	DER (1)

[a] *Salmonella* serotype abbreviations: ADE, Adelaide; AGN, Agona; ALT, Altona; ANA, Anatum; BER, Berta; BOV, Bovismorbificans; BDB, Brandenburg; BRD, Bredeney; BUK, Bukuru; CER, Cerro; CUB, Cubana; DER, Derby; DJU, Djugu; HAD, Hadar; HAV, Havana; HEI, Heidelberg; IDK, Idikan; II, II (3,10;lv;enx); INF, Infantis; JAM, Jamaica; JOH, Johannesburg; KEN, Kentucky; KRE, Krefeld; LEX, Lexington; LDN, London; MBA, Mbandaka; MEL, Meleagridis; MIN, Minnesota; MO6, monophasic (6,7:−:1,5); MO7, monophasic (7:z10:−); MVD, Montevideo; MNC, Muenchen; MNS, Muenster; NT, not typeable; OHI, Ohio; PUT, Putten; RIS, Rissen; SPA, Saintpaul; SWZ, Schwarzengrund; SEN, Senftenberg; THO, Thompson; TOU, Tounouma; TYP, Typhimurium; UGA, Uganda; WOR, Worthington.

[b] −, *Salmonella* was not isolated.

from a single day at plant A, winter day 2 (Table 5). All 18 of the *Salmonella* Brandenburg-positive preevisceration carcass samples were obtained from plant A during spring day 1.

Replica plating on media containing AMP, KAN, or TET determined that 454 of the 623 preevisceration *Salmonella* isolates were resistant to at least one of these antimicrobial agents. One hundred thirteen of the resistant isolates were selected for more detailed antimicrobial susceptibility testing, and 54 (47.8%) isolates were MDR (Table 6). Twenty-six (23.0%) isolates were ACSSuT^r, their serotypes were as follows: *Salmonella* serotypes Typhimurium (n = 19), Bukuru (n = 6), and Agona (n = 1). Only one preevisceration isolate tested was AXO resistant, and its serotype was *Salmonella* Agona (Table 6).

Prevalence, load, serotype, and antimicrobial resistance of *Salmonella* on chilled final carcasses. *Salmonella* was isolated

TABLE 6 Antimicrobial susceptibilities of selected *Salmonella* isolates

Sample point and *Salmonella* serotype	No. of isolates tested	% MDR[a]	% ACSSuT resistant[b]	% resistant isolates by class and antimicrobial agent: Aminoglycoside				β-Lac/inhib. combo[e]	Cephem			Folate pathway inhibitor		Penicillin	Phenicol	Quinolone		Tetracycline
				AMI	GEN	KAN	STR	AMC	FOX	TIO	AXO	FIS	COT	AMP	CHL	CIP	NAL	TET
Prescald carcasses	697	71.2	36.6	0.3	1.1	7.6	61.5	14.1	7.7	6.5	7.3	72.6	5.0	51.8	44.3	0.0	0.6	82.5
Typhimurium	274	98.2	79.6	0.0	0.0	0.4	82.8	15.7	0.4	0.0	0.0	98.9	0.0	97.8	96.4	0.0	0.0	98.5
Derby	138	92.8	2.2	0.7	0.7	1.4	94.2	1.4	1.4	1.4	0.7	92.0	2.2	4.3	2.2	0.0	1.4	99.3
London	49	16.3	2.0	0.0	0.0	0.0	4.1	10.2	10.2	10.2	10.2	26.5	0.0	14.3	2.0	0.0	0.0	28.6
Anatum	42	2.4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	2.4	2.4	0.0	0.0	40.5
Agona	38	84.2	71.1	0.0	0.0	68.4	81.6	71.1	71.1	57.9	71.1	92.1	71.1	73.7	71.1	0.0	0.0	97.4
Infantis	31	9.7	0.0	0.0	0.0	0.0	9.7	3.2	3.2	3.2	3.2	9.7	6.5	3.2	6.5	0.0	0.0	64.5
Putten	24	95.8	0.0	0.0	0.0	29.2	0.0	8.3	0.0	0.0	0.0	100.0	0.0	87.5	0.0	0.0	0.0	100.0
Senftenberg	17	88.2	0.0	0.0	11.8	5.9	82.4	64.7	64.7	58.8	64.7	88.2	5.9	76.5	5.9	0.0	0.0	94.1
Others[c]	84	20.2	7.1	1.2	6.0	19.0	26.2	8.3	8.3	6.0	7.1	21.4	2.4	19.0	11.9	0.0	2.4	47.6
Preevisceration carcasses	113	47.8	23.0	0.0	0.9	12.4	46.0	8.8	2.7	1.8	0.9	75.2	1.8	33.6	33.6	0.0	0.9	81.4
London	32	3.1	0.0	0.0	0.0	0.0	3.1	0.0	0.0	0.0	0.0	93.8	0.0	0.0	0.0	0.0	0.0	93.8
Typhimurium	25	88.0	76.0	0.0	0.0	8.0	88.0	16.0	0.0	0.0	0.0	96.0	0.0	84.0	84.0	0.0	0.0	88.0
Ohio	8	100.0	0.0	0.0	0.0	25.0	100.0	37.5	0.0	0.0	0.0	62.5	0.0	62.5	62.5	0.0	0.0	100.0
Agona	6	50.0	16.7	0.0	0.0	16.7	50.0	16.7	16.7	0.0	16.7	100.0	16.7	16.7	16.7	0.0	0.0	100.0
Bukuru	6	100.0	100.0	0.0	0.0	100.0	100.0	0.0	0.0	0.0	0.0	100.0	0.0	100.0	100.0	0.0	0.0	100.0
Derby	6	100.0	0.0	0.0	0.0	0.0	100.0	0.0	0.0	0.0	0.0	100.0	0.0	0.0	0.0	0.0	0.0	100.0
Infantis	4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Senftenberg	4	75.0	0.0	0.0	0.0	0.0	50.0	0.0	0.0	0.0	0.0	75.0	0.0	25.0	50.0	0.0	0.0	75.0
Others[d]	22	22.7	0.0	0.0	4.5	13.6	18.2	9.1	9.1	9.1	0.0	22.7	4.5	18.2	13.6	0.0	4.5	50.0
Chilled final carcass	102	77.5	13.7	0.0	0.0	1.0	73.5	2.9	0.0	0.0	0.0	95.1	0.0	23.5	18.6	0.0	0.0	98.0
Johannesburg	66	74.2	0.0	0.0	0.0	0.0	74.2	0.0	0.0	0.0	0.0	100.0	0.0	0.0	0.0	0.0	0.0	100.0
Typhimurium	18	94.4	77.7	0.0	0.0	5.6	83.3	16.7	0.0	0.0	0.0	100.0	0.0	94.4	94.4	0.0	0.0	94.4
monophasic (6,7:−:1,5)	5	100.0	0.0	0.0	0.0	0.0	100.0	0.0	0.0	0.0	0.0	100.0	0.0	100.0	0.0	0.0	0.0	100.0
Cerro	4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	100.0
Derby	4	75.0	0.0	0.0	0.0	0.0	75.0	0.0	0.0	0.0	0.0	75.0	0.0	0.0	0.0	0.0	0.0	75.0
Infantis	2	100.0	0.0	0.0	0.0	0.0	100.0	0.0	0.0	0.0	0.0	100.0	0.0	0.0	100.0	0.0	0.0	100.0
Putten	2	100.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	100.0	0.0	100.0	0.0	0.0	0.0	100.0
Muenchen	1	100.0	0.0	0.0	0.0	0.0	100.0	0.0	0.0	0.0	0.0	100.0	0.0	0.0	0.0	0.0	0.0	100.0

[a] Percentage of isolates resistant to three or more classes of antimicrobials.
[b] Percentage of isolates resistant to AMP, CHL, STR, FIS, and TET.
[c] Other serotypes (number of isolates tested) were as follows: Muenster (11), Bredeney (9), Kentucky (7), Montevideo (6), Brandenburg (5), Saintpaul (5), Ohio (4), Cerro (3), Heidelburg (3), Krefeld (3), Mbandaka (3), nontypeable (3), Cubana (2), Johannesburg (2), Schwarzengrund (2), Uganda (2), Worthington (2), Adelaide (1), Berta (1), Bovismorbificans (1), Havana (1), Idikan (1), Jamaica (1), Meleagridis (1), Minnesota (1), monophasic [7:z10:−] (1), Muenchen (1), Rissen (1), and Tounouma (1).
[d] Other serotypes (number of isolates tested) were as follows: Altona (3), Mbandaka (3), nontypeable (3), Rissen (2), Saintpaul (2), Anatum (1), Brandenburg (1), monophasic [6,7:−: 1,5] (1), Muenchen (1), Muenster (1), Putten (1), II (1), Uganda (1), and Worthington (1).
[e] β-Lac/inhib. combo, β-lactam/β-lactamase inhibitor combination.

from 56 (3.7%) of the chilled final carcasses (Table 1). *Salmonella* was isolated from enrichment cultures of 56 samples, including all nine samples enumerated. Plant A chilled final carcasses accounted for 49 of the prevalence positive carcasses. Accordingly, the 6.4% prevalence of *Salmonella* on final carcasses from plant A was significantly higher ($P < 0.01$) than the 0.9% prevalence from plant B (Table 2). *Salmonella* prevalence on chilled final carcasses was significantly lower ($P < 0.01$) during fall (0.0%) than during summer (6.3%), spring (5.0%), and winter (3.4%) (Table 2). At plant A, the seasonal prevalence of *Salmonella* from final carcasses was significantly higher ($P > 0.01$) during summer (12.6%), winter (4.2%), and spring (8.9%) than during fall (0.0%). At plant B, the prevalence of *Salmonella* from final carcasses ranged from 2.6% in winter to 0.0% in summer and fall but did not differ among seasons ($P > 0.01$). *Salmonella* was enumerated from only nine final carcass samples, and significant differences were not observed between the plants or seasons (Table 4).

Nine serotypes were isolated from the chilled final carcasses, and only one serotype was isolated from each of the positive samples (Table 1). The serotypes (the number of final carcasses each serotype was isolated from is shown in parentheses) were as follows: *Salmonella* serotypes Johannesburg (22), Typhimurium (18), monophasic variant with the antigenic formula of 6,7:−:1,5 (5), Derby (4), Infantis (2), Putten (2), Anatum (1), Cerro (1), and Muenchen (1). Only the *Salmonella* serotypes Johannesburg, Typhimurium, and Derby were isolated on more than one sampling day (Table 5).

Replica plating of the 150 chilled final carcass *Salmonella* isolates obtained during this study determined that 147 resistant were to AMP, KAN, or TET. One hundred two final carcass *Salmonella* isolates were selected for antimicrobial susceptibility testing, and 79 (77.5%) were MDR (Table 6). MDR *Salmonella* was isolated from 3.2% of the 1,520 chilled final carcasses sampled in this study. Fourteen chilled final carcass isolates, all *Salmonella* serotype Typhimurium, were ACSSuT^r (Table 6). None of the final carcass isolates were AXO resistant.

DISCUSSION

This study demonstrated that *Salmonella* was present on the prescald carcasses of a large majority (91.2%) of hogs entering two large U.S. commercial pork processing plants. The prescald carcass samples obtained in this study represent the *Salmonella* present on the hides of swine entering the processing plant, since no interventions were performed prior to sampling of prescald carcasses nor were the animals skinned. Known *Salmonella* contamination sources prior to entering the processing plant include swine production farms, transport trailers, and lairage pens (25, 26, 35, 42, 43, 55). Transportation and lairage are likely sources of contamination, since exposure of uninfected swine to contaminated transport trailers or lairage pens results in rapid infection of lymph nodes, cecal contents, and rectal contents (26, 28, 45, 46, 55, 57, 59). Other potential sources of prescald carcass contamination include belts or elevators colonized by *Salmonella*. The significantly higher ($P < 0.01$) *Salmonella* prevalence on prescald carcasses sampled at plant A (Table 2) could be caused by any of the afore mentioned contamination sources, but the sampling protocol used in this study did not account for any of these factors, so conclusions on the sources of contamination were not made. Sampling was performed each season to control for reported seasonal variation of *Salmonella* prevalence (34, 38). *Salmonella* prevalence on prescald carcasses was statistically lower ($P < 0.01$) during summer and fall (Table 2). However, conclusions relating to the seasonal prevalence of *Salmonella* should not be made, since each plant was sampled only on two consecutive days each season, and additional sampling throughout each season would be required to determine whether seasonal variations existed.

In a study published in 1995, Saide-Albornoz et al. (56) observed a 4.4% *Salmonella* prevalence on preevisceration carcasses by sampling 100 cm^2 on 270 carcasses at three large U.S. pork processing plants. Bolton et al. (7), in a study published in 2002, found no *Salmonella* when they sampled 50 cm^2 on 60 carcasses at a small Irish pork processing plant. We found that 19.1% of preevisceration carcasses were contaminated with *Salmonella* (Table 1). Our observation of a higher prevalence than previously reported could be due to changes in U.S. slaughter practices since 1995 or differences between U.S. and Irish practices. Alternatively, the higher prevalence could also be attributed to different culture methods (including the incorporation of immunomagnetic separation in the *Salmonella* prevalence method), increased sampling breadth and depth (1,520 samples taken on 16 days), or sampling of a much larger carcass surface area (4,000 cm^2). Indeed, our intensive sampling scheme not only revealed the higher than expected overall prevalence but also revealed large daily variations in the *Salmonella* preevisceration carcass prevalence at both plants. On 13 of the 16 sampling days, the *Salmonella* preevisceration carcass prevalence ranged from 1.1 to 20.0%, but on three sampling days (all at plant A), the *Salmonella* preevisceration carcass prevalence ranged from 40.0 to 70.5% (data not shown).

As mentioned before, this study was not designed to identify the source of *Salmonella* contamination, so the causes of the significantly higher ($P < 0.01$) preevisceration *Salmonella* prevalence at plant A (Table 1) could not be definitively identified. However, a possible explanation is that plant A interventions were less effective than those at plant B. Alternatively, the interventions employed at both plants could be equally effective but the *Salmonella* loads present on plant A prescald carcasses were higher than those at plant B and these higher loads could be above the level that the interventions can remove. Indeed, enumeration of *Salmonella* on prescald carcasses revealed that the 2 days (plant A, winter, sampling day 2 and plant A, spring, sampling day 1) with the highest *Salmonella* enumerable loads on prescald carcasses were the 2 days with the highest preevisceration carcass prevalences (data not shown). Additionally, on these 2 days, the serotypes most responsible for the elevated *Salmonella* loads on prescald carcasses (data not shown) were the predominate serotype on the preevisceration carcasses (Table 5). These results suggest that elevated concentrations of *Salmonella* on prescald carcasses may overwhelm interventions and carry over to an increased prevalence on preevisceration carcasses. The prescald and preevisceration samples were obtained concurrently, but no attempts were made to sample the same carcasses at each point. Therefore, samples taken at each point in this study were considered independent, and this prevented us from conclusively linking increased load on plant A prescald carcasses to increased prevalence on plant A preevisceration carcasses. These results demonstrate the need for comprehensive research, including enumeration of *Salmonella* on carcasses, to evaluate the currently poorly defined antimicrobial abilities, limitations, and potential for cross contamination of each step in the scalding, singeing, and polishing processes. These results seem intuitive, since the carcasses were not skinned, and

they agree with comparable studies of beef harvest indicating that incoming hide load is positively correlated with subsequent carcass contamination (3, 10, 51). As has been hypothesized for beef harvest, final carcass contamination may result from an incoming pathogen load high enough to overwhelm the capabilities of the processing interventions in place.

The overall prevalence of *Salmonella* on chilled final carcasses sampled in this study was 3.7%, similar to the 2 to 4% yearly prevalence from 2001 to 2009 reported by FSIS (33). In our study, final carcasses that were chilled at least overnight were randomly sampled in the cooler. As observed for prescald and preevisceration carcasses, the prevalence of *Salmonella* on chilled final carcasses was significantly higher ($P < 0.01$) at plant A (Table 2). The final carcasses sampled were independent of the prescald and preevisceration carcass samples. However, there are several possible explanations for the significantly higher *Salmonella* prevalences on plant A carcasses at each of the points sampled in this study (Table 2); possible explanations include greater contamination of hides entering the plant A, cross contamination at plant A, and/or differences in antimicrobial impact of different processing steps, such as blast chilling in plant B. Identification of the source(s) of *Salmonella* contamination was beyond the scope of this study, but we note that the largest difference in *Salmonella* prevalence between plants was on preevisceration carcasses (Table 3).

Salmonella Typhimurium was either the most prevalent or second most prevalent serotype isolated from each of the sampling points examined in this study. *Salmonella* Typhimurium is among the most prevalent serotypes isolated from both swine veterinary samples and human clinical samples (16, 31). We identified the ACSSuT^r phenotype from >76% of the *Salmonella* Typhimurium isolates tested from all three sampling points (Table 6), which is higher than the 48.9% prevalence of the ACSSuT^r phenotype among veterinary *Salmonella* Typhimurium isolates tested by the National Antimicrobial Resistance Monitoring System (NARMS) from 1998 to 2009 (15). This discrepancy raises the possibility that swine carcasses are more frequently contaminated with ACSSuT^r *Salmonella* Typhimurium than NARMS testing of veterinary isolates indicates. However, we note that the isolates we selected for antimicrobial susceptibility testing were first screened for resistance, and this may have increased the percentage of the ACSSuT^r phenotype.

The ACSSuT^r phenotype has been linked to the presence of either of two genetic elements: *Salmonella* genomic island I or IncA/C MDR-AmpC-encoding plasmids (36). IncA/C MDR-AmpC-encoding plasmids may also harbor the bla_{CMY-2} gene that encodes resistance ESCs, including AXO (11, 12, 22). AXO resistance was identified in 1.9% of veterinary swine *Salmonella* isolates tested by NARMS from 1998 to 2009 (15) but was identified from 5.7% of the isolates tested in this study. The serotypes with an AXO-resistant isolate identified in this study were *Salmonella* serotypes Agona, Havana, Heidelburg, Ohio, Rissen, and Senftenberg (Table 6). *Salmonella* serotypes Agona and Heidelburg have previously been identified as serotypes contributing significantly to the total amount of ESC-resistant *Salmonella* (32). None of the final carcass isolates tested were AXO resistant. This result was not unexpected, since only 3.7% of final carcasses were positive for *Salmonella*, the final carcass samples were independent of the prescald and preevisceration samples, and the serotypes of AXO-resistant prescald and preevisceration isolates (*Salmonella* serotypes Agona, Havana, Heidelburg, Ohio, Rissen, and Senftenberg) were not among the final carcass serotypes identified (Table 5).

The *Salmonella* prevalences and number of serotypes detected at each sampling point in this study are likely underreported, since only one method of selective enrichment was used in this study. Indeed, the direct plating method used in this study for enumeration demonstrated shortcomings of selective enrichment, since there were 10 samples (7 prescald samples and 3 preevisceration samples) from which *Salmonella* was enumerated by direct plating but *Salmonella* was not isolated from selective enrichment. Studies of swine fecal samples subjected to multiple methods of selective enrichment for isolation of *Salmonella* have demonstrated that no culture method is 100% sensitive and that the serotypes isolated from the same sample differed by the culture method used, suggesting that serotypes differ in their susceptibilities to selective agents in enrichment media (24, 49, 54). The *Salmonella* serotype Choleraesuis, which is associated with swine, has been demonstrated to be sensitive to selective enrichment methods (2, 54). *Salmonella* Choleraesuis was not isolated from any of the 4,560 carcasses sampled during this study, suggesting that the selective enrichment used in this study may not be suitable for the isolation of this serotype.

In summary, the results of this study indicate that hogs entering processing plants have a very high incidence of *Salmonella* on their skins at sometimes high levels. The interventions used by pork processing plants greatly reduced the prevalence of *Salmonella* on final carcasses, but *Salmonella* was found on a low percentage of finished, chilled carcasses. The presence of MDR *Salmonella* on the finished carcasses, albeit at low prevalence, demonstrates that additional interventions should be considered in pork processing plants to further reduce the risk of invasive salmonellosis caused by pork products.

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