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Decontamination of pork carcasses during scalding and the prevention of *Salmonella* cross-contamination

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SUMMARY

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Aims: The objective of this study was to establish critical temperature limits to prevent cross-contamination of pork carcasses during scalding.

Methods and Results: Mixtures of antibiotic-resistant mutants of *Salmonella* species were heat treated at 50, 55 and 60°C in samples of commercial scald tank water. Surviving cell numbers were estimated by plating treated suspensions on (i) tryptone soya agar (TSA) and (ii) on TSA, overlaid with brilliant green agar plus nalidixic acid and streptomycin sulphate and used to estimate *D*-values for the treated mixed cell suspensions.

Conclusions: A time–temperature combination of 1·4 min at 60°C is required to achieve a 1 log reduction in *Salmonella* in scald tank water. The predicted equivalent at 65°C is 0·18 min.

Significance and Impact of the Study: This study provides data and a model to enable pork processors to identify and apply processing parameters to limit the risks of transfer of *Salmonella* between pig carcasses during commercial scalding operations.

Keywords: *Salmonella*, *D*-values, pork slaughter, critical scalding temperatures, preventing cross-contamination.

INTRODUCTION

The occurrence of undesirable human pathogens such as *Salmonella* in meat animals, and their subsequent transfer to derived meat products during commercial slaughter and dressing procedures, poses specific threats to the general public health, as well as long-term challenges in terms of the commercial future of the meat industry. A number of investigations of outbreaks of human salmonellosis, and the sources of such illness, have highlighted pork as an important and frequent source of this pathogen, within the European Union and beyond. For example, the average incidence of salmonellosis in the Netherlands is reported to be about 450 per 100 000 of the population, with an estimated 15% of such cases being associated with pork consumption (Berends *et al.* 1998). The corresponding figures in Denmark are approx. 95 cases per 100 000 with 10–15% being attributed to pork consumption (Hald and Wegener 1999). Such figures mean that pork is the second highest food source of *Salmonella* in Denmark, surpassed only by eggs. Such summary figures can, to some extent, shroud the human impact of such pathogens in the human food chain. Thus, in Denmark, in 1993 one major outbreak of illness associated with pork contaminated with *Salmonella* involved 550 cases of illness (Bager *et al.* 1995), while a more recent outbreak (1998), involving multi-resistant *Salmonella* Typhimurium DT104 resulted in 25 culture-confirmed cases and two deaths (Baggesen *et al.* 1999). In the latter case, the source of infection was traced back to a local pork abattoir, highlighting the role of abattoir practices in preventing or, more correctly, failure to prevent transfer of *Salmonella* to pig meat during carcass processing. In addition to such direct human illness and suffering, these types of incidents have significant negative economic effects on the scale and

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financial health of the pork production and processing industry. Thus, the Economic Research Service (ERS) of the United States Department of Agriculture (USDA) estimate that in the US alone, food-borne *Salmonella* infections associated with pork, costs $0·1–0·2 billion per annum (Frenzen *et al.* 1999).

Despite wide recognition of the scale and impact of such problems, undesirable human pathogens such as *Salmonella* continue to penetrate the food chain, and frequently persist into retail meat products. For example, a recent survey found that almost 10% of pork samples taken from retail outlets in Ireland were contaminated with *Salmonella* (Cloak 1999). Thus considerable progress remains to be made in understanding and controlling the occurrence, transfer and persistence of *Salmonella* within commercial pork production and processing operations.

While there are numerous opportunities and processes by which pathogens can enter the human food chain, Boes *et al.* (2001) have suggested that direct slaughter line processes are probably the most significant in terms of carcass contamination with *Salmonella*. This is in agreement with Berends *et al.* (1997) who reported a clear correlation between the frequency and scale of *Salmonella* contamination in pigs at the time of slaughter, and the occurrence of *Salmonella* on derived 'finished' carcasses. Such correlations reflect a number of processes, including (i) auto-contamination, in which *Salmonella* entering the abattoir in/on an animal, are transferred onto the carcass derived from that animal, and (ii) cross-contamination, in which *Salmonella* entering the abattoir in/on one animal, are transferred onto one or more carcasses derived from previously *Salmonella*-free animals, by direct and indirect contact during processing. The development of means to limit, if not prevent, such contamination is more likely to have direct effects in reducing the prevalence of, and risks posed by, *Salmonella* on pork products entering the human food chain. While a number of early stages in the commercial process, by which most animals are converted to carcasses, pose significant risks for the transfer of pathogens, some unique elements of pig processing pose particular problems. In cattle and sheep, the hide, an important source of carcass contamination with *Salmonella* and other undesirable pathogens (Sheridan 1998; Ransom *et al.* 2002) is removed from the carcass at an early stage of processing. However, in pigs the hide remains on the carcass during processing. A range of heating, washing, and abrasive treatments including scalding/dehairing, singeing, polishing, etc. are applied to reduce the visual and microbiological impact of its continued presence. Immediately before singeing, in which the majority of the visible hair is 'burned' from carcasses, carcasses are pulled through a large vat containing hot water at 60–70°C, a process which facilitates subsequent removal of remaining bristles from the hair follicles. A large number of pigs are usually scalded in the same batch of scald tank water, which quickly becomes contaminated with dirt, faeces, ingesta and any bacteria carried by the pigs.

If pathogens, including *Salmonella*, survive in the scald water, the scalding process can significantly increase the risk of contamination of concurrently and subsequently scalded carcasses (Hald *et al.* 1999), involving all accessible surfaces, including meat and non-meat elements such as muscle, lungs and other organs (Jones *et al.* 1984). Effective and reliable means of limiting such contamination, without collateral damage to other commercially important characteristics of treated carcasses, can only be developed by investigation, modelling, and manipulation of the parameters involved in bacterial death/damage during such processes.

The data presented in this paper provides the scientific basis for the development of the minimal time–temperature combinations within commercial scalding operations. Application of such processes should eliminate or significantly reduce the persistence of *Salmonella* in scald tank water, limiting the risks of carcass contamination and the spread of this organism within pork products entering the human food chain.

METHODS AND MATERIALS

Scald tank water

Samples of scald tank water were obtained from a commercial pork slaughterhouse. Samples (5 l) were withdrawn approx. 4 h after the commencement of slaughter, i.e. approximately half way through the days' operations, transported to the laboratory within 4 h and stored at approx. 2°C for 24 h.

Bacterial cultures

Two different *Salmonella* cocktails were used in these experiments. The first (cocktail A) included *S.* Enteritidis (NCTC 5765), *S.* Typhimurium (NCTC 12023), *S.* Bredeney, *S.* Kentucky, *S.* Typhimurium and *S.* Derby. *S.* Bredeney and *S.* Kentucky were both isolated from pork samples 3 years before, while the latter two strains were isolated from pig carcasses at that time.

The second cocktail (cocktail B) included *S.* Typhimurium, *S.* Hadar, *S.* Derby and *S.* Infantis, all of which were isolated from pig carcasses in a commercial pork plant within the 3 months prior to this experiment.

To facilitate recovery of these organisms during the course of these experiments, antibiotic-resistant strains, resistant to 50 μg ml^{-1} of nalidixic acid and 1000 μg ml^{-1} of streptomycin sulphate were prepared for each *Salmonella* strain as described by Blackburn and Davies (1994) and stored on cryo-protective beads (Technical Service Consultanta Ltd., Heywood, UK) at −20°C.

Inoculum preparation

One bead of each doubly antibiotic resistant mutant of *Salmonella* was aseptically transferred to 30 ml of Brain Heart Infusion broth (BHI, Oxoid) and incubated at 37°C for 24 h. Following incubation, a 1-ml aliquot from each culture was transferred to 100 ml BHI and incubated for a further 18 h at 37°C. Each culture was then centrifuged (Eppendorf centrifuge 5403) at 3000 $\times g$ for 10 min at 4°C. The recovered pellet was washed three times with, and resuspended in, maximum recovery diluent (MRD). These suspensions were mixed to form the two composite 'cocktail' inocula.

The numbers of *Salmonella* cells per millilitre in each cocktail mixture were estimated using the membrane epifluorescent staining technique of Walls and Sheridan (1989) and the mixtures were diluted from the inocula containing approx. 5 $\log_{10}$ CFU ml^{-1}.

Inoculation, sampling and enumeration

Samples of scald tank water (90 ml) were dispensed into 100 ml screw cap bottles (Duran Schott, Mainz, Germany) and equilibrated to 50, 55 or 60°C by immersion in water-baths (Grant Instruments, Cambridge, UK) at these temperatures, within 20 min. The temperatures of the scald tank water samples were monitored using thermocouples inserted into 'blank' samples, and attached to a temperature microprocessor (Ellab A/S, Oslo, Norway). Immediately after equilibration, duplicate scald tank water samples were inoculated with 10 ml of cocktail A or cocktail B, (each containing approx. 6 $\log_{10}$ CFU ml^{-1} *Salmonella*). Samples of 10 ml were periodically withdrawn (including a sample at time zero to check inoculation levels) pipetted into a sterile thin-walled 30 ml plastic sterile bottle and immediately immersed in an ice-bath at approx. 2°C.

Each cooled sample was serially diluted in MRD. Members of each dilution series were plated onto brilliant green agar (BGA, Oxoid) (supplemented with nalidixic acid (50 $\mu g\ ml^{-1}$) and streptomycin sulphate (1000 $\mu g\ ml^{-1}$) (direct plating) and onto tryptone soya agar (TSA, Oxoid). The BGA plates were incubated at 37°C for 24 h. The TSA plates were incubated at 37°C for 2 h, to allow recovery of injured cells, over-poured with BGA [supplemented with nalidixic acid (50 $\mu g\ ml^{-1}$) and streptomycin sulphate (1000 $\mu g\ ml^{-1}$)] (recovery overlay technique) and incubated for a further 24 h at 37°C.

Calculation of *D*- and *z*-values and statistical analysis

An experiment consisted of obtaining the thermal death data for 50, 55 and 60°C in duplicate (i.e. two inoculated bottles of scald tank water at each temperature) and each experiment was repeated three times using different scald tank water samples. From the data a plot of $\log_{10}$ of surviving cells ml^{-1} against time was drawn and the slope (b) and standard error (S.E.) were obtained for the plot at each temperature using linear regression analysis (Genstat 5, Statistics Department, Rothamsted Experimental Station, Hertfordshire, UK). *D*-values were calculated using the average slope for a given temperature treatment. Differences between *D*-values were determined by analysing differences between the average slopes using the *t*-test $(b_1 - b_2 / v(SE_1)^2 + (SE_2)^2$ with degrees of freedom $(n_1 + n_2) - 4)$.

Each *z*-value was calculated from the slope of an individual curve of a plot of $\log_{10}$ *D*-values against temperature by linear regression of the slopes of the plots. Thermal destruction times were calculated using the formula: $D_x = \log^{-1}(\log D_{60} - ((t_2 - t_1)/z))$.

RESULTS

Table 1 presents *D*-values for both cocktail mixtures of *Salmonella* at the three treatment temperatures used.

The D_{50} values (i.e. the *D*-values for samples treated at 50°C) for *Salmonella* cocktail A was calculated as 55·1 min (from 'direct', BGA counts), and as 54·9 min (from 'recovery overlay', TSA/BGA counts). These results are significantly lower ($P < 0{\cdot}001$) that the D_{50} values for *Salmonella* cocktail B at 83·1 min (from direct, BGA counts) and 83·2 min (from recovery overlay, TSA/BGA counts). For each *Salmonella* cocktail there was no significant difference ($P > 0{\cdot}05$) between the bacterial counts obtained on the different media and the thermal death curve for each cocktail on TSA/BGA is shown in Fig. 1 as an example. Neither of these curves was perfectly linear. The curve for cocktail A displayed tailing after 200 min and the inactivation kinetics for cocktail B

Table 1 The *D*-values for laboratory and pig *Salmonella* isolates in scald tank water

Temperature (°C)	*Salmonella* cocktail	Culture medium	*D*-value (min)	*n*	S.E.
50	Laboratory	BGA	55·1	54	0·97
50	Laboratory	TSA/BGA	54·9	54	0·12
50	Pig	BGA	83·1	54	0·12
50	Pig	TSA/BGA	83·2	54	0·14
55	Laboratory	BGA	5·0	36	0·16
55	Laboratory	TSA/BGA	6·6	36	0·14
55	Pig	BGA	6·2	42	0·29
55	Pig	TSA/BGA	7·3	42	0·85
60	Laboratory	BGA	0·9	48	0·13
60	Laboratory	TSA/BGA	1·1	48	0·13
60	Pig	BGA	1·5	48	0·98
60	Pig	TSA/BGA	1·4	48	0·84

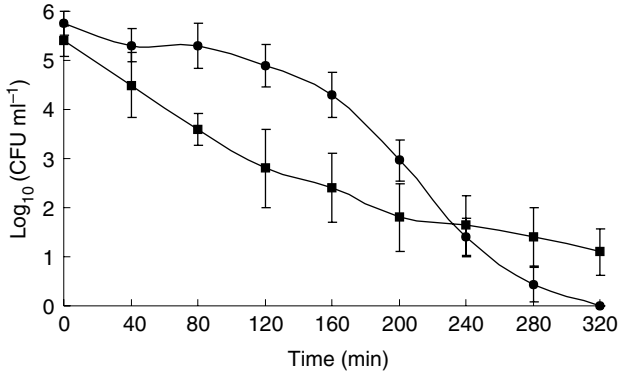

Fig. 1 The thermal death curves for cocktail A (■) and cocktail B (●) of *Salmonella* isolates at 50°C. The experiments were run in duplicate with three replicates. Values are mean, bars indicate the standard deviation

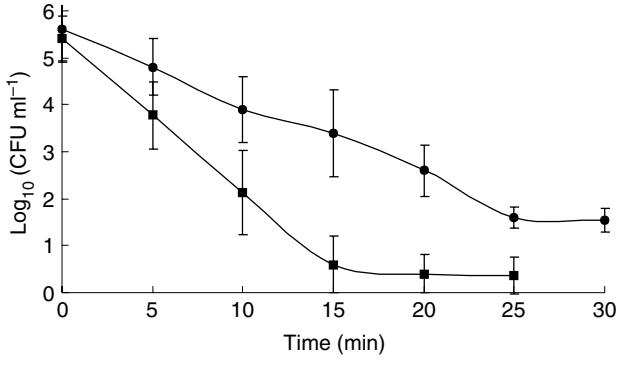

Fig. 2 The thermal death curves for cocktail A (■) and cocktail B (●) of *Salmonella* isolates at 55°C. The experiments were run in duplicate with three replicates. Values are mean, bars indicate the standard deviation

deviated from first order as a lag period or shoulder was observed for the first 80 min and a tail after 240 min.

At 55°C the *D*-values for *Salmonella* cocktail A were 5·0 min and 6·6 min on BGA and TSA/BGA, respectively. The *D*-values for cocktail B were slightly longer but not significantly different at 6·2 and 7·3 min on BGA and TSA/BGA, respectively. Once again the media used did not significantly ($P > 0{\cdot}05$) affect the bacterial counts. Figure 2 presents the thermal death curves for each cocktail derived on TSA/BGA. *Salmonella* cocktail A displayed log linear kinetics for the first 15 min after which a tail was observed. *Salmonella* cocktail B displayed a tailing effect after 25 min.

In scald water samples treated at 60°C the *D*-values for cocktails A and B were 0·9 and 1·1 min, respectively, on TSA/BGA and 1·5 min and 1·4 min, respectively, on BGA. These *D*-values were statistically the same ($P > 0{\cdot}05$) regardless of cocktail or culture media. The thermal death curves for each cocktail on TSA/BGA exhibited tailing after 80 s (Fig. 3).

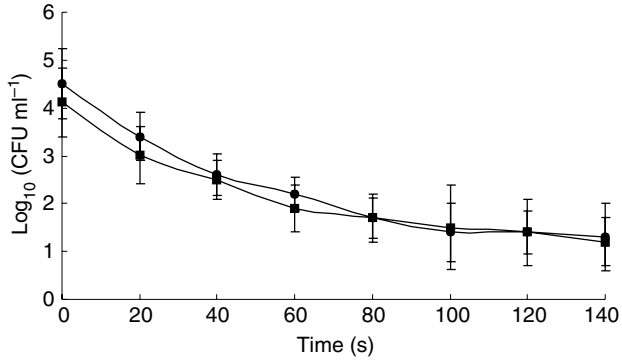

Fig. 3 The thermal death curves for cocktail A (■) and cocktail B (●) of *Salmonella* isolates at 60°C. The experiments were run in duplicate with three replicates. Values are mean, bars indicate the standard deviation

Table 2 The *z*-values for laboratory and pig *Salmonella* isolates in scald tank water

Salmonella cocktail	Culture medium	*z*-value (min)
Laboratory	BGA	5·7
Laboratory	TSA/BGA	5·9
Pig	BGA	5·7
Pig	TSA/BGA	5·7

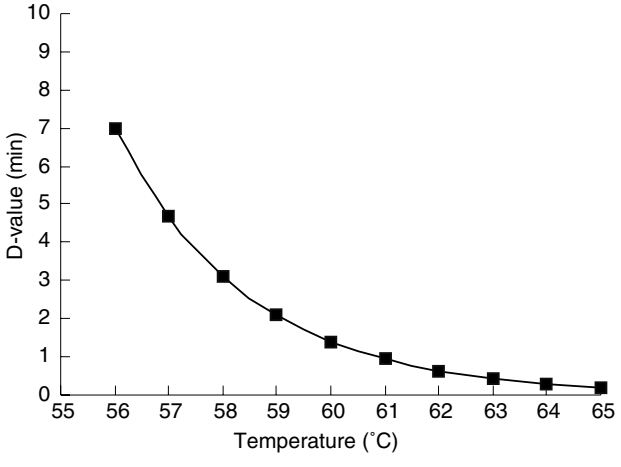

Fig. 4 The time required to achieve a 1 $\log_{10}$ reduction in *Salmonella* in the scald tank and equivalent thermal treatments calculated using the formula $D_x = \log^{-1}(\log D_{60} - ((t_2 - t_1)/z))$

The *z*-values for the cocktail A isolates as determined using BGA counts was 5·7 and as determined using TSA/BGA counts was 5·9 (Table 2). The *z*-value for cocktail B derived from BGA counts and from TSA/BGA counts was 5·7. Figure 4 predicts the thermal destruction times using the formula: $D_x = \log^{-1}(\log D_{60} - ((t_2 - t_1)/z))$.

DISCUSSION

This study did not observe significant differences between *D*-values calculated from estimates of surviving bacterial numbers from direct plating on selective medium (BGA) and *D*-values similarly calculated from estimates from recovery/overall plating on TSA with subsequent BGA overlay. This is in agreement with Bolton *et al.* (2000), who noted the absence of significant differences between direct and recovery/overlay counts in heat-treated samples of a range of other food-borne pathogens including *Listeria monocytogenes* and *Yersinia enterocolitica*. This observation differs from the report of Strantz and Zottola (1989), who obtained significantly higher estimates, and therefore significantly higher *D*-values, from (recovery overlay) TSA/BGA counts, than from (direct) BGA counts in the detection/enumeration of heat stressed *S.* Typhimurium.

In general, procedures using non-selective or recovery/overlay methods give higher estimates of the numbers of bacteria surviving heat treatments than procedures using direct, selective plating (Embarek and Huss 1993; Boziaris *et al.* 1998). This effect is probably due to the reduced capacity of heat-damaged cells to recover and grow on the more inhibitory (selective) media, and leads to the derivation of lower *D*-values (Fain *et al.* 1991) based solely on 'undamaged' cell numbers.

Such inconsistencies i.e. differences, or lack of differences among counts (and derived *D*-values) obtained using selective, non-selective, and recovery/overlay media are probably related to the specifics of the damage incurred by heat-treated organisms, and the capacity of particular media to support the recovery of damaged bacteria. In cases where the nature of the damage is such that it significantly reduces the ability of treated organisms to grow on selective media, but can be repaired during growth on non-selective media, there will be larger differences between selective and non-selective/recovery counts, and consequently, larger differences between *D*-values derived from such counts. However, if most of the damage incurred during heat treatment cannot be repaired during growth on either media type, there will be little or no significant difference between the *D*-values derived from selective and non-selective [overlay] counts. It would therefore appear that, under the particular conditions used in this study, heat-treated cells (i) survived in forms that were capable of growing equally well on selective and non-selective agars, or (ii) were rendered equally non-viable on both agars.

While there is a dearth of comparable thermal resistance data in the literature, Sorquist and Danielsson-Tham (1990) have reported D_{60} values for *S.* Derby, *S.* Montevideo and *S.* Typhimurium in commercial pork scald tank water. The D_{60} values obtained in this study (0·9–1·5 min) (Table 1) were considerably higher than those reported (*S.* Derby, 0·37 min; *S.* Montevideo, 0·39 min; *S.* Typhimurium, 0·29 min) (Sorquist and Danielsson-Tham 1990). The reasons for such differences are not clear, but may include real interspecies differences, and/or artefactual differences such as phenotypic variation induced by recent growth conditions, differences in the relative extent of protective effects of the heating media, or artefactual effects related to experimental design, plating medium, etc. (Embarek and Huss 1993; Doyle and Mazzotta 2000).

The thermal death curves obtained in this study (Figs 1–3) showed deviation from first order kinetics, i.e. a lag period or shoulder was observed with cocktail B at 50°C while tailing was observed with both *Salmonella* cocktails at 55 and 60°C. The shoulder effect may be attributed to a lag period before the cells sustain sufficient thermal damage to render them non-viable and/or a protective effect of the organic matter in the scald tank water (Juneja and Eblen 2000). Tailing may be due to cell density, the clumping of cells and the protection of cells in the core of such clumps (Stumbo 1973), the synthesis of heat shock proteins (Lindquist 1986; Humpheson *et al.* 1998) and/or the presence of a more heat-resistant subpopulation (Peleg and Cole 1998; Murphy *et al.* 1999).

The temperature equivalence calculations predict that changing the temperature of the scald tank water by one or two degrees would greatly influence the survival of *Salmonella*. For example, the equivalence calculations suggest that decreasing the temperature from 60 to 58°C would increase the *D*-values for cocktail B 2·2-fold. Such calculations are in agreement with Sorquist and Danilesson-Tham (1990) who estimated that similar minor changes in scald tank temperature induced significant changes in the *D*-values for *S.* Derby (2·5-fold), *S.* Montevideo (2·0-fold) and *S.* Typhimurium (2·3-fold).

Differences in immersion intervals, thermal resistance and the numbers of *Salmonella* entering the scald tank will all affect the numbers of such organisms which will transfer between, and survive on, scalded carcasses. However, the risk of contamination via the scald water can be minimized if the water temperature is sufficiently high. While Davies et al. (1999), Hald *et al.* (1999) and Pearce *et al.* (2002) suggest that *Salmonella* survival in the scald tank and cross-contamination of the carcasses may be prevented when the water temperature is above 61°C, these studies ignore any time component. Time is an important consideration when developing thermal destruction models. For example, 1 kg of polluting material containing 10^5 *Salmonella* per gram, would be required to give the scald tank water 10 *Salmonella* per ml when suspended in a 10 000 l scald tank (the capacity of the tank where the scald water was obtained). Using the *D*-values/*z*-values calculated, a minimum treatment time temperature combination of 1·4 min at 60°C, 0·94 min at 61°C, 0·62 min at 62°C, etc. would be required to ensure the

destruction of these 10 Salmonella ml^{-1} of scald tank water. In reality the high dilution effect of the scald water volume would ensure that these levels of *Salmonella* are never attained. However, it is often useful to set a target level of destruction thus ensuring the incorporation of a satisfactory safety margin. In the food processing industry, for example, this is frequently set at a 5 log reduction in the target organism.

The application of the D and z value models developed in this study should allow the consistent suppression of auto- and cross-contamination of pork carcasses with *Salmonella* during scalding, reliably reducing the risks posed to consumers of commercially produced pork and pork products. Such an evidence-based approach should form an effective and verifiable control point [CCP1] within improved good manufacturing practice (GMP) and hazard analysis and critical control point (HACCP) food safety management programmes.

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