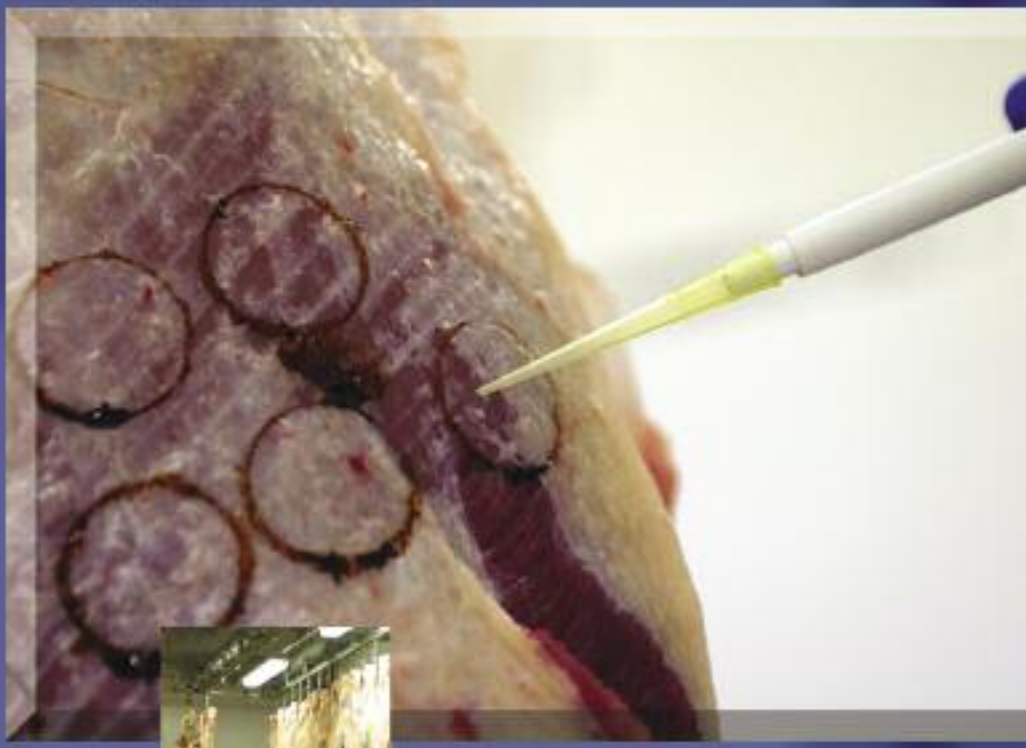
Agriculture and Food Development Authority

**FINAL REPORT**

Project RMIS No. 5185

# Indicator organisms to determine the use of chilling as a critical point in beef slaughter HACCP



Ashtown Food Research Centre

RESEARCH & TRAINING FOR THE FOOD INDUSTRY

RESEARCH REPORT
NO 100

# INDICATOR ORGANISMS TO DETERMINE THE USE OF CHILLING AS A CRITICAL CONTROL POINT IN BEEF SLAUGHTER HACCP

Editor-in-Chief: Dr Gerard Downey

Authors:

Deirdre M. Prendergast BSc PhD
James J. Sheridan MA MSc PhD
**Ashtown Food Research Centre, Ashtown, Dublin 15**

ISBN 1 84170 533 0
November 2008



Teagasc Oak Park Carlow Co. Carlow

# CONTENTS

## SUMMARY

During chilling, temperatures of carcass surfaces at different sites change over time as do other parameters such as water activity ($a_w$), the structure of the muscle and other tissues, as the carcass enters *rigor mortis*. Many of these factors are known to have a major effect on cell survival and growth and must be considered in determining the influence of chilling on bacterial survival on carcass surfaces. This study aimed to determine if chilling could be used as a critical control point (CCP) in beef slaughter in relation to pathogens such as *E. coli* O157:H7 and *L. monocytogenes*, using *E. coli* and *Listeria innocua* as pathogen indicators. The present study was designed to determine the influence of (a) chilling at 10°C for 72 h on the survival of *E. coli* and (b) chilling at 4°C for 72 h on the survival of *L. innocua* inoculated at different sites on beef carcasses. Three sites (neck, outside round and brisket) were inoculated (1) immediately after dressing while hot (*E. coli* and *L. innocua*) and (2) when cold after chilling (*L. innocua*). The influence of changes in surface $a_w$ was also considered and their relationship to the survival of *E. coli* and *L. innocua* over time was assessed. The data are discussed in relation to the use of chilling as a CCP in beef hazard analysis (HACCP) and the monitoring of neck temperature as the most suitable CCP.

## INTRODUCTION

As pathogens may not be used in abattoir experiments, a suitable alternative indicator is required to determine their potential presence and possible growth/survival on beef carcass surfaces. There is compelling evidence to show that generic *E. coli* is a suitable indicator of faecal contamination of carcasses and Gram negative pathogens such as *E. coli* O157:H7 and *Salmonella enterica* serotypes which are present in faeces (McEvoy *et al.*, 2004; Tergney and Bolton, 2006). The use of *L. innocua* as an indicator of *L. monocytogenes* growth on beef surfaces has been demonstrated in the past (Duffy *et al.*, 2000). The presence of generic *E. coli* and *L. innocua* may therefore be used as indicators of the possible presence of these pathogens

which are frequently found associated with beef carcasses (McEvoy *et al.*, 2003a, 2003b; Rivera-Betancourt *et al.*, 2004).

During chilling, the temperature of carcass surfaces changes over time at different sites, as do other parameters such as $a_w$ and the structure of the muscle (Gill *et al.*, 1984). Many of these factors are known to have a major effect on cell survival and growth and must be considered in determining the influence of chilling on bacterial survival on carcass surfaces (Beales, 2004). Relative humidity (RH) affects microbial growth during carcass chilling by changing water activity ($a_w$) on the carcass surface by processes which are significantly influenced by carcass characteristics such as site location and subcutaneous fat cover (Kinsella *et al.*, 2006). Survival rates decrease with decreasing $a_w$ (Shadbolt *et al.*, 1999) and $a_w$ interacts with temperature i.e. survival rates are lower at higher temperatures and low $a_w$ values (Clavero and Beuchat, 1996).

The interactions of temperature, $a_w$ and other factors may be significant in relation to the safety of beef carcasses during chilling. The present study was designed to determine the influence of (a) chilling at 10°C for 72 h on the survival of *E. coli* and (b) chilling at 4°C for 72 h on the survival of *L. innocua* inoculated at different sites on beef carcasses. Three sites, the neck, rump and brisket, were inoculated (1) immediately after dressing while carcasses were hot (*E. coli* and *L. innocua*), and (2) after chilling when surfaces were cold (*L. innocua*). The influence of changes in surface $a_w$ was also considered and its relationship to the survival of *E. coli* and *L. innocua* was assessed over time.

## OBJECTIVES

- To determine the influence of chilling beef carcasses at 10°C for 72 h on the survival of *E. coli* and the effect of chilling at 4°C for 72 h on the survival of *L. innocua*.
- To determine the use of chilling as a critical control point in beef HACCP.

## MATERIALS AND METHODS

The experimental abattoir used in this study, located at Ashtown Food Research centre (AFRC), is designed for low-throughput slaughter and processing of cattle, sheep and pigs. In the present study, 18-24 month old grass-fed Hereford x Friesian heifers were processed under normal slaughter conditions and the carcasses produced after slaughter had a mean side weight of 119.5 kg.

Three inoculation sites on the trailing carcass (right side) from each animal were identified as follows: (1) the rump (2) the brisket and (3) the neck. The first two sites are covered by a fibrous membrane of connective tissue or fascia, while the portion of the neck investigated was the lean surface exposed during slaughter which was not covered by fascia.

*Sampling and inoculation of carcasses: E. coli*

Sampling at different times over a 72 h period was facilitated by slaughtering two animals on the same day and inoculating the three sites on carcasses from both animals. The first carcass was slaughtered at 8.00 am and immediately after washing the hot carcass side was transferred to the boning hall for inoculation, approximately 1 h after stunning. At each site, a sterile coring punch with the edges smeared with edible ink was used to delimit twenty 5 $cm^2$ areas. Ten of the marked areas were inoculated by pipette with a 5 ml inoculum containing approximately 3 $\log_{10}$ cfu of *E. coli;* the remaining ten served as uninoculated controls. A time zero sample was taken from each site at approximately 9.30 am. Thereafter, samples were taken from each site at 6, 12, 24, 30, 36, 48, 54, 60 and 72 h. On the same day, a second animal was slaughtered at 4.00 pm and the same sites were inoculated after transfer of the resultant carcass to the boning hall. The same demarcation and inoculation procedure was followed with this second carcass but only 6 areas were marked out i.e. three inoculated and three uninoculated which facilitated sampling at 18, 42 and 66 h. Inoculated carcasses were left for 30 min to facilitate adhesion in an experimental chill at AFRC. The two animals slaughtered on a single day represented a complete treatment over time and there were four

replicate treatments carried out on different days. At each sampling time, an inoculated and uninoculated 5 $cm^2$ area was cut to a depth of approximately 5 mm and excised using a sterile scalpel and tweezers.

*Sampling and inoculation of carcasses: L. innocua*

Six carcasses were inoculated at the above three sites with *L. innocua* (1) immediately after slaughter and washing (hot) and (2) a further six were inoculated in the chill at 4°C after chilling for 24 h (cold), to determine the survival and growth of the organism on different surfaces during and after chilling. The hot carcasses were stored at 4°C and examined at intervals over 72 h while the cold carcasses were first chilled to 4°C before inoculation and storage for the same time.

At each site, a sterile coring punch with the edges smeared with edible ink, was used to delimit eight 5 $cm^2$ areas. Four of the marked areas were inoculated by pipette with a 5 ml inoculum containing approx. 3 $\log_{10}$ cfu of *L. innocua*; the remaining four served as uninoculated controls. Inoculated carcasses were left for 30 min to facilitate attachment. At times 0, 24, 48 and 72 h, an inoculated and uninoculated 5 $cm^2$ area was cut to a depth of approximately 5 mm and excised using a sterile scalpel and tweezers.

*Enumeration of E. coli* and *L. innocua*

Excised samples were transferred into separate sterile 101 x 152 mm stomacher bags (Seward Ltd., Norfolk, UK) containing 10 ml MRD and pulsified for 30 s using a model PUL 100 Pulsifier™ (Filtaflex Ltd., Ontario, Canada). For direct enumeration, 1 ml aliquots of the inoculated and uninoculated tissue samples were plated onto selective agars for *E. coli* (MacConkey agar) and *L. innocua* (*Listeri*a selective agar) and incubated at 37°C (*E. coli*) and 30°C (*L. innocua*) for 24 h.

*Water activity ($a_w$)*

During each experiment, the water activity ($a_w$) of uninoculated excised meat samples from the three sites investigated was measured at each sampling time from time zero to 72 h using an AquaLab model CX-2 (Labcell, Basingstoke,

UK). On each sampling day, before sample measurements, the water activity meter was calibrated using distilled water ($a_w$ = 1.000 +/- 0.003) and a saturated solution of sodium chloride (NaCl, [$a_w$ = 0.755 +/- 0.001 at 20°C]).

*Monitoring temperatures of carcass surfaces and chill temperature and relative humidity (RH)*

The surface temperature on a carcass at each of the sites was measured every 30 min for 72 h by inserting Type T copper-constantan thermocouples directly under the carcass skin. The core temperature of the carcass was measured by inserting a Type T copper-constantan thermocouple (insertion-type) into the aitch bone. The RH and ambient temperature in the chills were measured every 30 min for 72 h using a model HMP 45D Vaisala humidity probe (Vaisala Oyj, Helsinki, Finland) which was placed in the centre of the chill, approximately 1.5 metres above the floor. Temperature and RH data were recorded using a 2040 series Grant Squirrel data logger (Grant Instruments, Cambridge, UK). All temperature and RH data were downloaded from the Squirrel data logger to Microsoft® Office Excel 2003 in which data were viewed in graphical form.

*Statistical Analysis*

All statistical tests examining bacterial counts were performed using log transformations. For all such data, 0.998 was added to the original values prior to log transformations. This gave zero counts a value of -0.0009 on the log scale while making no appreciable difference to the log values of all other (non-zero) counts. This allowed mathematical manipulation of all the data (including zero counts) with minimal significant effect on the overall results obtained. The experiment was designed as a split-split-plot with temperature and sites (three carcass sites) on the main plots and time on the sub-sub-plots. The $a_w$ experiment was designed as a split-plot with carcass sites on the main plots and time on the sub-plots. Analysis of variance (ANOVA) was performed using Genstat 5 statistical package (Rothamsted Experimental Station, Harpenden, United Kingdom).

## Survival of *E. coli* on beef carcasses

In this study the mean ambient temperature of the chill was 10.6°C (range of 9.5 – 12.6°C) and the mean RH was 94.0%. The surfaces of the rump, brisket and neck sites cooled to 10°C in 47, 12 and 9 h respectively while the core carcass temperature at the aitch bone site took 52 h to reach this temperature (Figure 1).

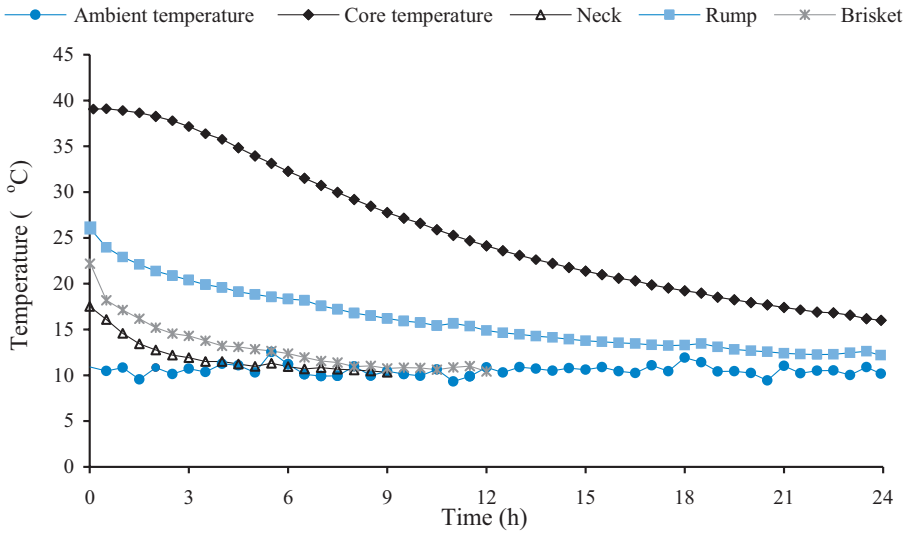



***Figure 1:*** *Mean temperatures (°C) and times (h) required for beef carcass surfaces to reach 10°C*

The survival of *E. coli* ($\log_{10}$ cfu $cm^{-2}$) on beef carcass surfaces after storage at 10°C for 72 h is shown in Table 1. It was observed that growth of *E. coli* took place at the neck while counts on the rump were reduced during the first 24 h (1.42 log) and by 48 h the organism was present only at very low levels (0.14 log). At the brisket, counts remained relatively unchanged throughout storage. When the survival of *E. coli* was compared on different substrates, the neck had higher counts at all times than the rump and a similar observation was made for the neck and brisket; the rump and brisket always had similar counts.

Table 1: Survival of *E. coli* ($log_{10}$ cfu $cm^{-2}$) on beef carcass surfaces after storage at 10°C for 72 h

| Time (h) | Neck | Rump | Brisket |
|---|---|---|---|
| 0 | 2.21 | 2.00 | 1.85 |
| 6 | 2.18 | 1.55 | 1.48 |
| 12 | 2.33 | 1.13 | 0.85 |
| 18 | 2.48 | 0.70 | 0.22 |
| 24 | 2.65 | 0.58 | 0.89 |
| 30 | 2.92 | 0.47 | 1.12 |
| 36 | 2.87 | 0.57 | 1.17 |
| 42 | 2.83 | 0.64 | 1.21 |
| 48 | 3.21 | 0.14 | 1.13 |
| 54 | 1.81 | ND | 1.63 |
| 60 | 2.13 | 0.02 | 0.82 |
| 66 | 2.45 | 0.03 | ND |
| 72 | 3.85 | 0.03 | 1.21 |

Standard error of differences between means: 0.52; degrees of freedom: 143;
ND = not detected

Water activity values decreased at all sites in the first 6 h and then increased up to 24 h. From 24 to 36-42 h, $a_w$ decreased again at all sites and from this time to 72 h remained at about the same levels except on the rump where they increased again. The relationship between the RH of the chill and the surface $a_w$ values over time is shown in Figure 2. The initial $a_w$ on the neck was different to the rump but not the brisket and the latter two sites were similar. In relation to time, $a_w$ values at the neck showed a decrease in the first 6 h after which time there was an increase up to 24 h and thereafter values declined. On the rump, results were similar with a decrease in the first 6 h and an increase to 24 h. On the brisket, $a_w$ decreased in the first 6 h, followed by an increase to 18 h.

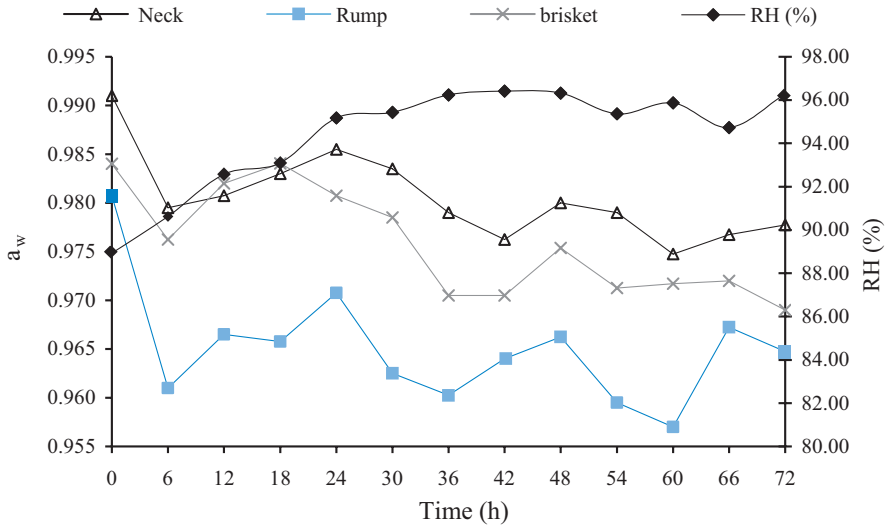



***Figure 2:*** *Relationship between water activity ($a_w$) on beef carcass sites and the relative humidity (RH) of the experimental chill after storage at 10°C for up to 72 h*

When, as in this study, carcasses are chilled at high temperatures (10°C), it is generally to ensure the production of tender meat (Troy, 1999). Surface temperatures during this period and throughout chilling were always highest on the rump, followed by the brisket and the neck. In studies on the survival of *E. coli* and *E. coli* O157:H7, it was demonstrated that survival of these organisms was reduced in conditions where the $a_w$ was low and temperature was high (Shadbolt *et al.*, 1999). Data from the present study suggest that the decline in *E. coli* counts at the three sites is related to a synergistic effect between surface temperature and $a_w$ at each site. The highest level of decline was observed on the rump, which had the highest temperature and lowest $a_w$ in the first 6 h, followed by the brisket, with maximal survival on the neck which had the lowest temperature. The $a_w$ on beef carcass surfaces is generally considered to be controlled by the RH of the chill (Anon, 2003). In the present study, the $a_w$ declined in the first 6 h although the RH increased during this time up to 24 h. The rapid decline in surface $a_w$ of the brisket and rump has been observed previously and was shown to occur in the first 10 h

of chilling (Anon, 2007). It has been observed that the RH of the chill is less important at the beginning of chilling when the hot carcass enters the chill. At this time, the controlling factor is the difference between the vapour pressure at the wet carcass surface and in the air (Levy, 1986). The lack of an association between RH and surface $a_w$ in the first 6 h of chilling is therefore not surprising.

## Survival of *Listeria innocua* on hot and cold beef carcasses

The mean ambient chill temperature was 4.15°C (range of 2.99 – 7.01°C) and the mean RH was 89.47%. Carcass cooling to 4.0°C varied at the different sites with the rump taking approximately 45 h to reach this temperature, followed by the brisket and the neck at 16 and 5.5 h respectively (Figure 3).

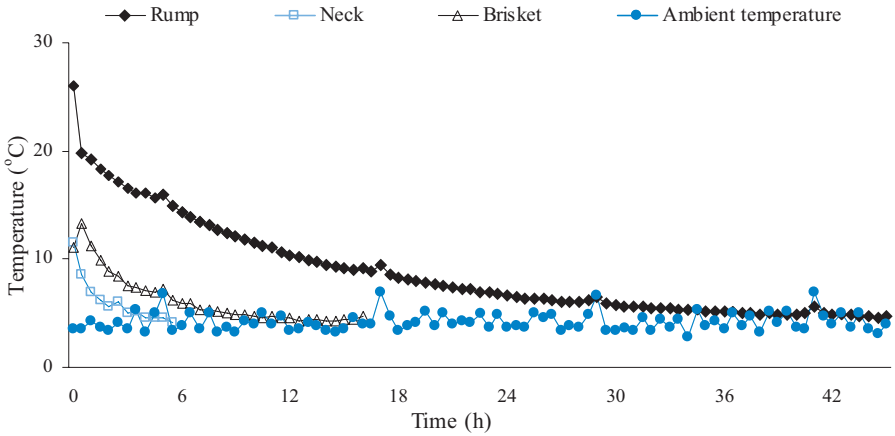



***Figure 3:*** *Mean temperatures (°C) and time (h) required for beef carcass surfaces to reach 4°C*

Following inoculation of hot carcasses, *L. innocua* survived on all three carcass sites but a decrease in counts was observed over time (Table 2). When the survival of *L. innocua* was compared on the different sites, the neck had higher counts than the rump or brisket at all times; rump and brisket always had similar counts. Following inoculation of cold carcasses, a decrease in *L. innocua* counts was observed over time on all three sites. When the survival

of *L. innocua* was compared on the different substrates, all three sites had similar counts. When hot and cold carcasses were compared, *L. innocua* counts were always lower on cold carcasses.

**Table 2:** Survival of Listeria innocua ($log_{10}$ cfu $cm^{-2}$) on beef carcass surfaces after storage at 4°C for 72 h

| Carcass type | Time (h) | Neck | Rump | Brisket |
|---|---|---|---|---|
| Hot | 0 | 3.08 | 2.37 | 2.53 |
| | 24 | 2.47 | 2.10 | 1.82 |
| | 48 | 2.12 | 1.40 | 1.25 |
| | 72 | 2.51 | 1.28 | 1.54 |
| | | | | |
| Cold* | 0 | 1.89 | 2.28 | 2.47 |
| | 24 | 1.92 | 1.49 | 1.11 |
| | 48 | 1.43 | 1.59 | 0.51 |
| | 72 | 1.18 | 0.95 | 0.90 |

*carcasses had been stored for 24 h prior to inoculation; standard error of differences between means: columns: 0.30; rows: 0.34; degrees of freedom: columns: 119; rows: 138

The relationship between the RH of the chill and the surface $a_w$ values over time is shown in Figure 4. Data show that the RH of the chill rose over the first 24 h from about 89 to 96% and stayed at this value for the remainder of the storage period. Carcass $a_w$ values at the different sites all declined in the first 24 h and thereafter remained at about the same value. Within individual sites, when time zero $a_w$ values were compared with all other times, they were higher at the neck and rump while the brisket remained unchanged. For the brisket, differences were observed only at 48 h when surface $a_w$ was higher than at 24, 72 and 96 h. The largest reductions in $a_w$ at individual sites occurred in the first 24 h at the neck (0.009) and rump (0.015) with the brisket remaining almost unchanged (0.003). In general, results revealed that

the neck had higher $a_w$ values than the other three sites while the rump had the lowest values. The magnitude of the differences between sites varied over time.

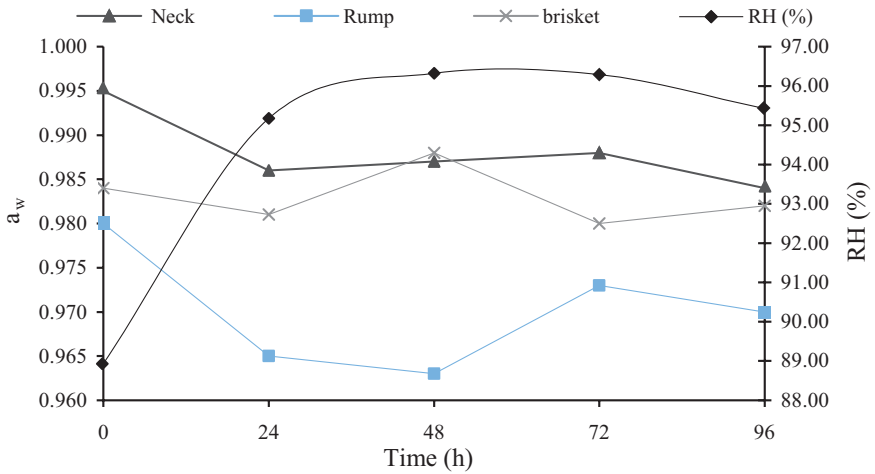



***Figure 4:*** *Relationship between water activity ($a_w$) on beef carcass sites and the relative humidity (RH) of the chill after storage for 96 h*

This study showed that *L. innocua* survived but did not grow after inoculation at different sites on hot and cold beef carcass surfaces, chilled at 4°C and stored for 72 h, with the possible exceptions of the neck at 72 h and brisket at 48 h on hot carcasses. *L. innocua* survived but did not grow on hot beef carcasses at the different sites. As this organism is psychrotrophic, growth could have occurred on hot carcasses during chilling. Although there are many models that predict lag and generation times or growth rates of *Listeria*, few of these have been carried out on meat (Augustin and Carlier, 2000). On sterile beef inoculated with two strains of *L. monocytogenes*, predicted lag phases at $a_w$ 1.0, temperature 4°C and pH 5.8, were 107 and 139 h with generation times of 34 and 41 h respectively (Lebert *et al.*, 1998).

On cold carcasses, which had been stored for 24 h prior to inoculation, reductions in counts occurred over time at all sites. Cells on cold surfaces

would have been subjected to the combined stresses of low $a_w$ and low temperature. This indicates that the decrease in *Listeria* on cold surfaces may be related to both these stresses rather than just one of them. As outlined above, a synergy between $a_w$ and temperature seems unlikely as high temperatures were not available under these conditions. When cells were inoculated on hot and cold carcasses, the initial process taking place was attachment to meat surfaces. This is important in relation to enhancing bacterial survival, particularly in adverse conditions (Frank, 2001; Yu *et al.*, 2001). The surfaces of hot carcasses are wet from washing and *Listeria* cells, which are initially present in a biofilm, become firmly attached within 30 min (Yu *et al.*, 2001). During rigor, structural changes occur on meat surfaces in the form of gaps due to shrinkage of muscle fiber and other structures, which aid bacterial survival due to penetration of cells into these gaps (Frank, 2001). Temperature can also have an effect on attachment as demonstrated for *Salmonella* for which attachment to meat surfaces reduced as temperature decreased from 37 to 4°C (Walls *et al.*, 1993). In contrast, cold carcasses are dry and biofilms and gaps in the meat surface are not present (Gill *et al.*, 1984). The interstitial and surface fat solidifies giving the carcass a hard outer surface at which penetration cannot take place.

Significant differences in $a_w$ between sites on hot carcasses at time zero could not be related to changes in initial *Listeria* counts and, despite significant reductions in $a_{w,}$ after storage for 24 h counts were similar at all sites. Reductions in $a_w$ after 24 h chilling would be expected, due to maximal weight loss of 80 to 90% at this time (Sheridan, 1990). After 24 h, reductions in $a_w$ were small, with the exception at 48 h on the brisket. This would be expected after chilling for periods longer than 24 h as evaporative losses decrease with time (Anon, 2003; Sheridan 1990). During the first 4 h, cells on carcass surfaces were subjected to the maximum $a_w$ stress and declining temperatures but no reductions in counts occurred at any of the sites.

This study has shown that the survival of Gram negative pathogens such as *E. coli* O157:H7 and Gram positive pathogens such as *L. monocytogenes* on beef

carcass surfaces may be controlled by monitoring the temperature of specific sites. This is based on the use of *E. coli* as an indicator of the survival and growth of these pathogens and such a monitoring process is currently in operation in Australia where chilling is used as a CCP in beef slaughter HACCP (Anon, 2007). The present study suggests that the most appropriate monitoring site is the neck because the temperature at this site during chilling is reduced rapidly, irrespective of the regime used. At this site, pathogen growth is controlled by the rapid fall in temperature and while low surface $a_w$ may play a part in this process, there is no synergistic action between the two at low temperatures. At other sites, where the temperature declines slowly in the presence of low $a_w$, the synergy between these will ensure a reduction in pathogen counts. The neck is therefore the most suitable site for monitoring because if proper temperature control is not in place, growth of *E. coli* and *L. monocytogenes* may take place, albeit very slowly.

## CONCLUSIONS

- Generic *E. coli* and *L. innocua* may be used as indicators of the possible presence of *E. coli* O157:H7 and *L. monocytogenes* on beef carcass surfaces.
- The survival of Gram negative and Gram positive pathogens such as *E. coli* O157:H7 and *L. monocytogenes* on beef carcass surfaces may be controlled during chilling by monitoring the temperature of specific sites on beef carcasses.
- The interactions of temperature, $a_w$ and other factors may be significant in relation to the safety of beef carcasses during chilling.
- *L. innocua* survived on hot carcasses while on cold carcasses a decrease in survival occurred at all sites. This suggests that reductions on cold carcasses may be related to low $a_w$ and temperature stresses acting individually or together, and that chilled carcasses will not be a major vehicle in the spread of *Listeria* in the abattoir environment.

- The most appropriate site for monitoring carcass temperature is the neck because temperature at this site is rapidly reduced during chilling, irrespective of the regime used.

- Therefore, monitoring the neck carcass temperature during chilling may be used as a CCP during beef slaughter HACCP.

## RECOMMENDATIONS TO INDUSTRY

Food hygiene legislation states that it is the responsibility of every food business to produce safe food by applying good hygienic practices and food safety management procedures based on HACCP principles. The new hygiene regulations which came into force in January 2006 demand full traceability within the food chain and the implementation of a HACCP plan. EU Regulation 853/2004 provides a legal basis to permit the use of a substance other than potable water to remove surface contamination from products of animal origin. Previously, such a legal basis did not exist in the EU legislation for red meat. This present study has shown that the survival of pathogens such as *E. coli* O157:H7 and *Listeria monocytogenes* on beef carcass surfaces may be controlled by monitoring the temperature of specific sites on beef carcasses during chilling. This is based on the use of *E. coli* and *L. innocua* as indicators of the survival and growth of these pathogens and such a monitoring process is currently in operation in Australia where chilling is used as a CCP in beef slaughter HACCP. The present study suggests that the most appropriate monitoring site is the neck because the temperature at this site during chilling is reduced rapidly, irrespective of the regime used. At this site, pathogen growth is controlled by the rapid fall in temperature and while low surface $a_w$ may play a part in this process, there is no synergistic action between the two at low temperatures. At other sites, where the temperature declines slowly, in the presence of low $a_w$, the synergy between these will ensure a reduction in *E. coli* counts. The neck is therefore the most suitable

site for monitoring because if the proper temperature control is not in place, growth of pathogens may take place, albeit very slowly.

## REFERENCES


**Anon.** 2003. Evaporative weight losses during processing. *Food Science Australia*, Meat Technology update, Newsletter 1/03 – February.

**Anon.** 2007. Beef carcass chilling – opportunities for customising programs. *Food Science Australia*, Meat Technology Update. Newsletter 2/07 – April.

**Augustin, J.C. and Carlier, V.** 2000. Modelling the growth rate of *Listeria monocytogenes* with a multiplicative type model including interactions between environmental factors. *International Journal of Food Microbiology* **56**, 53-70.

**Beales, N.** 2004. Adaptation of microorganisms to cold temperatures, weak acid preservatives, low pH, and osmotic stress: A review. *Comprehensive Reviews in Food Science and Food Safety* **3**, 1-20**.**

**Clavero, M.R. and Beuchat, L.R.** 1996. Survival of *Escherichia coli* O157:H7 in broth and processed salami as influenced by pH, water activity, and temperature and suitability of media for its recovery. *Applied and Environmental Microbiology* **62**, 2735-2740.

**Duffy, G., Walsh, D., Sheridan, J.J., Logue, C.M., Harrington, D., Blair, I.S. and McDowell, D.A.** 2000. Behaviour of *Listeria monocytogenes* in the presence of *Listeria innocua* during storage of minced beef under vacuum or in air at 0°C and 10°C. *Food Microbiology* **17**, 571-578.

**Frank, J.F.** 2001. Microbial attachment to food and food contact surfaces. *Advances in Food and Nutrition Research* **43**, 320-370.

**Gill, C.O., Leet, N.G. and Penney, N.** 1984. Structural changes developing with rigor that facilitate bacterial invasion of muscle tissue. *Meat Science* **10**, 265-274.

**Kinsella, K.J., Sheridan, J.J., Rowe, T.A., Blair, I.S. and McDowell, D.A.** 2006. Impact of a novel spray chilling system on surface microflora, water activity and weight loss during beef carcass chilling. *Food Microbiology* **23**, 483-490.

**Lebert, I., Bégot, C. and Lebert, A.** 1998. Development of two *Listeria monocytogenes* growth models in a meat broth and their application to beef meat. *Food Microbiology* **15**, 499-509.

**Levy, F.L.** 1986. Measuring the convective heat transfer coefficient while chilling carcasses. *International Journal of Refrigeration* **9**, 84-88.

**McEvoy, J.M., Doherty, A.M., Sheridan, J.J., Blair, I.S. and McDowell, D.A.** 2003a. The prevalence of *Salmonella* spp. in bovine faecal, rumen and carcass samples at a commercial abattoir. *Journal of Applied Microbiology* **94**, 693-700.

**McEvoy, J.M., Doherty, A.M., Sheridan, J.J., Thomson-Carter, F.M., Garvey, P., McGuire, L., Blair, I.S. and McDowell, D.A.** 2003b. The prevalence and spread of *Escherichia coli* O157:H7 at a commercial beef abattoir. *Journal of Applied Microbiology* **95**, 256-266.

**McEvoy, J.M., Sheridan, J.J., Blair, I.S. and McDowell, D.A.** 2003a. Microbial contamination on beef in relation to hygiene assessment based on criteria used in EU Decision 2001/471/EC. *International Journal of Food Microbiology* **92**, 217-225.

**Rivera-Betancourt, M., Shackelford, S.D., Arthur, T.M., Westmoreland, K.E., Bellinger, G., Rossman, M.O., Reagan, J. and Koohmaraie, M.** 2004. Prevalence of *Escherichia coli* O157:H7, *Listeria monocytogenes* and *Salmonella* in two geographically distant commercial beef processing plants in the United States. *Journal of Food Protection* **67**, 295-302.

**Shadbolt, C.T., Ross, T. and McMeekin, T.A.** 1999. Non-thermal death of *Escherichia coli*. *International Journal of Food Microbiology* **49**, 129-138.

**Sheridan, J.J.** 1990. The ultra- rapid chilling of lamb carcasses. *Meat Science* **28**, 31-50.

**Tergney, A. and Bolton, D.J.** 2006. Validation studies on an online monitoring system for reducing faecal and microbial contamination on beef carcasses. *Food Control*, **17**, 378-382.

**Troy, D.J.** 1999. Enhancing the tenderness of beef. The National Food Centre, Teagasc, Ashtown, Dublin 15. Research Report No. 11, 1-28. ISBN 1 84170 010 4.

**Walls, I., Cooke, P.H., Benedict, R.C., Buchanan, R.L.** 1993. Sausage casings as a model for attachment of *Salmonella* to meat. *Journal of Food Protection* **56**, 390-394.

**Yu, S.L., Cooke, P.H. and Tu, S.I.** 2001. Effects of chilling on sampling of bacteria attached to swine carcasses. *Letters in Applied Microbiology* **32**, 205-210.

## PUBLICATIONS FROM THIS PROJECT

**Prendergast, D.M., Rowe, T.A. and Sheridan, J.J.** 2007**.** Survival of *Listeria innocua* on hot and cold beef carcass surfaces. *Journal of Applied Microbiology* **103**, 2721-2729.

**Crowley, K.M., Prendergast, D.M., Sheridan, J.J., McDowell, D.A. and Blair, I.S.** 2005. The effects of aerobic and vacuum packed storage conditions on the shelf life of minced beef stored at 5°C. *Proceedings of the International Conference: The Science of Food Safety and Nutrition*, Croke Park Stadium, Dublin, 1st – 2nd December p:132

**Sheridan, J.J. and Prendergast, D.M.** 2005. Food safety microbiology: Research priorities for the future. *Proceedings of the International Conference: The Science of Food Safety and Nutrition*, Croke Park Stadium, Dublin, 1st – 2nd December p: 66-73.

**Sheridan, J.J. and Prendergast, D.M.** 2005. Food Safety Microbiology: some research priorities for the future. *International workshop: "Quality management of the chill chain"* 16th December, Training Centre of the National Bank of Greece, Poseidonos Avenue 41-43, 16675, Glyfada, Athens, Greece, p: 4

**Crowley, K.M., Prendergast, D.M., Sheridan, J.J., McDowell, D.A. and Blair, I.S.** 2005. Comparison of the survival of *E. coli* on beef carcass surfaces with that of excised beef pieces. *Proceedings of 35th Research Conference Food, Nutrition and Consumer Sciences,* University College Cork

**Sheridan, J.J. and Prendergast, D.M.** 2005. Growth of microorganisms on meat: surface versus submerged [broth] growth, microbial interactions and the future of microbiological criteria. *Safety and shelf-life assessment of meat products in Europe.* International symposium 18th February. 'De Reehorst' Ede, the Netherlands.

**Prendergast, D.M., Rowe, T.A. and Sheridan, J.J.** 2004. Survival of *Pseudomonas fluorescens* on hot and cold beef carcass surfaces. *Proceedings of the International Food Conference: thinking beyond tomorrow*, Dublin, 17th – 18th June p:170.

**Prendergast, D.M., Rowe, T.A. and Sheridan, J.J.** 2003. Attachment of *Pseudomonas fluorescens* to cold and hot beef surfaces. The National Food Centre Annual Research Report p: 56-57.

# Ashtown Food Research Centre

RESEARCH & TRAINING FOR THE FOOD INDUSTRY

Ashtown, Dublin 15, Ireland.

Telephone: (+353 1) 805 9500

Fax: (+353 1) 805 9550