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Effect of Early Post-mortem Cooling on Temperature, pH Fall and Meat Quality in Pigs

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ABSTRACT

The aim of this study was to investigate how early cooling of carcasses after slaughter by showering with cold water affected the rate of the pH fall post mortem, *protein denaturation and drip loss. Eighty pigs were selected in pairs at debleeding according to sex and farm of origin. All pigs were halothane genotyped and glycolytic potential in LD analysed. One of each pair was cooled 30 min* post mortem *by showering with 10–12°C water for 12 min. The control pig was treated normally except for the same delay before batch chilling commenced. The initial pH fall in LD and BF did not depend on the glycolytic potential in LD but at 5 to 6 and 24 hr* post mortem *pigs with the lowest glycolytic potential had the highest pH. Weight and lean meat content did not affect the cooling curve, i.e. the temperature fall. The results showed that it was possible to reduce the temperature in BF and LD by cooling at slaughter. The maximum difference in temperature between control and cooled carcasses 2 hr* post mortem *was 2 and 1°C in LD and BF respectively. The lowering of the muscle temperature early* post mortem *resulted in a reduced rate of the pH fall and a higher pH from 2 to 6 hr in the cooled carcasses. The rate of the pH fall in LD and BF seemed to be independent of temperature at levels above approximately 37°C, but decreased linearly as the temperature dropped below approximately 37°C. The cooling procedure used here did not result in a significant reduction in protein denaturation or drip loss, although there was a tendency towards lower drip loss in LD and BF in cooled carcasses. © 1998 Elsevier Science Ltd. All rights reserved*

INTRODUCTION

Reduced drip loss and improved colour is of major concern for both economic output and optimising meat quality in pork. Previous work, mainly associated with PSE (pale, soft

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and exudative) meat, has shown that increased drip formation is caused by the denaturation of both myofibrillar and sarcoplasmic proteins, which predominantly occurs early *post mortem* when the pH is low (<5.8) and the temperature is still high ($>38°C$) (Honikel and Kim, 1986).

The rate at which pH falls in muscles *post mortem* is therefore critical for protein denaturation and drip loss in the meat the day after slaughter (Bendall, 1973; Offer, 1991). If the rate of pH fall is high (0·1 unit min^{-1}), denaturation is significant and will lead to development of PSE meat, but lower rates of the pH fall (0·01 unit min^{-1}) can also lead to increased drip loss (Offer, 1991) and paler colour (Honikel, 1987) although without development of PSE meat. Denaturation of muscle proteins leads to defects in the membranes around the myofibrillar bundles and shrinkage of the filamentous net in the muscle, which again leads to accumulation of drip between the muscle fibre bundles (Honikel and Kim, 1986; Offer and Knight, 1988). The degree of denaturation is dependent on the rate of the pH fall up to the onset of rigor. When rigor is complete the bonding between actin and myosin will prevent further denaturation of the myosin heads (Offer, 1991). In pork with a good water holding capacity, rigor is complete 3–6 hr *post mortem* (Taylor and Dant, 1971).

The rate of anaerobic metabolic processes, and thereby the accumulation of lactate leading to a pH fall in the muscles after exsanguination is dependent on the temperature in the muscle (Pearson and Young, 1989). At slaughter the carcass produces metabolic heat (Bendall, 1973), and the slaughter process adds energy to the carcass during scalding (van der Wal *et al.*, 1994). This energy is removed from the carcass by evaporation. The temperature fall in the carcass normally starts at gut removal and carcass splitting and continues during chilling, so that 24 hr after slaughter temperatures are below 7°C throughout the carcass.

Apart from severe PSE, Offer (1991) suggested there was a benefit in reducing the carcass temperature immediately *post mortem* in order to decrease the rate of the pH fall. This will diminish the total protein denaturation and improve water holding capacity but prolong the time to rigor. Earlier work has shown that hot boning and chilling of meat from PSE prone pigs resulted in a reduction of muscle temperature, rate of pH fall, and drip loss compared to traditional batch chilling (Honikel *et al.*, 1986; Woltersdorf and Troeger, 1986). Other investigations showed that the skinning process resulted in lower muscle temperatures and higher pH values at various times *post mortem* (up to 45 min) as well as improvement in water holding capacity (Troeger and Woltersdorf, 1987; Maribo, 1996).

The aim of this study was to investigate how early cooling of carcasses on the slaughter line, by showering with cold water affected the rate of pH fall *post mortem*, protein denaturation and drip loss.

MATERIALS AND METHODS

Slaughter and chilling process

A total of 80 gilts and castrates were selected after exsanguination in 40 pairs which originated from the same farm and were of the same sex. Thus both pigs in each pair had similar treatment prior to slaughter and were of equal fatness. The abattoir used a traditional scalding-singeing process. After CO_2-stunning the carcasses were shackled by the right leg, exsanguinated for 5 min before scalding for 7 min in 60°C hot water. Subsequently, dehairing with cold water (10–12°C), singeing (approx. 1200°C for 10 s) and black scraping with cold water (10–12°C) took place. Evisceration and removal of plucks

took place 23 and 25 min *post mortem* respectively. In this trial both carcasses were then taken off the line 30 min *post mortem* and one of each pair was put through a cooling procedure by showering for 12 min with cold water (10–12°C). The six sprinklers were placed with four on the outside of the lumbar region of the loin and the ham and two on the inside of the carcass spraying on the lumbar region of the loin. The carcasses were then returned to the slaughter line, split, checked by the veterinary control and classified before entering the chill room. The chilling process at the factory was batch chilling at 2–4°C (0·2 m s^{-1}) until 24 hr *post mortem*. The time from start of exsanguination to chilling was prolonged from about 45 to 65 min for all carcasses due to the cooling procedure.

Measurements and analysis

Temperature and pH were measured in the left *m. longissimus dorsi* (LD) between the 2nd and 4th lumbar vertebra and in the centre of *m. biceps femoris* (BF) both at a depth of 4 cm. The measurements were carried out after exsanguination (5 min *post mortem*), at evisceration (23 min *post mortem*), after the cooling procedure (54 min *post mortem*), after entering the chilling room (approx. 1 hr *post mortem*) and 2, 3, 4, 5 and 6 hr *post mortem* in the chilling room. The pH meters were calibrated regularly (buffers: pH = 4·00, pH = 7·00) and adjusted to the temperature of the measured carcasses. The day after slaughter pH was measured in LD, BF, *m. semimembranosus* (SM), *m. semispinalis capitis* (SC) and samples of LD between 2–4 lumbar vertebra and centre of BF were cut from the left side of the carcass for estimation of protein denaturation (water and salt soluble proteins) (Barton-Gade *et al.*, 1996), determination of drip loss (Rasmussen and Anderson, 1996) and analysis of glycolytic potential (Monin and Sellier, 1985). Finally, a sample of LD was taken for genotyping for the halothane gene (ryanodine receptor gene) (Fujii *et al.*, 1991).

Statistical analysis

The course of the temperature (T, °C), pH fall and rate of pH fall (ΔpH hr^{-1} units per hour) were described by a statistical model based on repeated measurements (Rutter and Elashof, 1994). A mean curve was estimated for each treatment as an average of the curves for the individual carcasses. The curves describe the change of pH or temperature in LD or BF the first 6 hr *post mortem*. The curves were polynomials up to 5th degree and the coefficients were considered as random coefficients. In the description of the curve for pH fall, glycolytic potential was included as covariate, and in the description of the temperature fall, carcass weight and lean meat content were included as covariates. The pH and temperature curve was described using following basic models:

$$\text{Temperature}: Y_{ijk} = treat_k + B_{1k}t_{ij} + B_{2k}t_{ij}^2 + B_{3k}t_{ij}^3 + B_{4k}t_{ij}^4 + B_{5k}t_{ij}^5 + \gamma_1 \text{ carcass weight }_i + \gamma_2 \text{ lean meat }_i + \epsilon_{ijk}$$

$$\text{pH}: Y_{ijk} = \text{treat}_k + B_{1k}t_{ij} + B_{2k}t_{ij}^2 + B_{3k}t_{ij}^3 + B_{4k}t_{ij}^4 + B_{5k}t_{ij}^5 + \gamma gly - pot_i + \epsilon_{ijk}$$

where y_{ijk} is measurement No. j of pH value or temperature at the ith carcass from treatment k, where $j = 1,2,...,9$; $i = 1,2,..., n$ and $k = 1,2$; where t_{ij} is time *post mortem* for the jth registration at the ith carcass at treatment 1 or 2; where treat_k describes the water cooling or control and the gly-pot the potential, carcass weight and lean meat content are

covariates. t_{ij} is the time and B_{1k}, B_{2k}, B_{3k}, B_{4k}, B_{5k} are stochastic variables with means β_{1k}, β_{2k}, β_{3k}, β_{4k}, β_{5k}, and covariance Σ, independent of ϵ_{ijk}, the random errors. To investigate the effect of glycolytic potential on the slope of the pH fall the stochastic coefficients were made dependent on the glycolytic potential in LD, where the pigs were divided in four groups according to their glycolytic potential.

Drip loss and protein denaturation were analysed using analysis of covariance (GLM procedure SAS, 1990):

$$Y_{ik} = \text{treat}_k + \beta_1 \text{ carcass weight}_i + \beta_1 \text{ lean meat content}_i + e_{ik}$$

where treat_k describes the treatment, and carcass weight and lean meat content are considered as covariates.

Differences in temperature and pH before cooling were tested using a Student's *t*-test.

RESULTS

Experimental material

Among the selected animals were found eight halothane carriers (six and two in the control and cooled group respectively). Pairs with one or two pigs carrying the halothane gene were excluded from the primary statistical analysis and analysed separately. Therefore, the two groups of pigs free of the halothane gene consisted of 32 pigs each.

There was no difference in carcass weight, lean meat content or glycolytic potential between carcasses in the two treatments (Table 1). The average temperature and pH level in LD and BF before cooling were equal in the two treatments (Table 2), and from 5 to 30 min *post mortem* the pH and temperature fell similarly.

Temperature change

Carcass weight and lean meat content were included in the model as covariates, but these factors did not influence the course of the temperature change and were therefore discarded. The course of the temperature change in LD and BF for the two treatments was estimated and can be described by the following polynomials, where t is any selected time from 5 min to 6 hr *post mortem*:

$$\text{Control} - \text{LD } T = 38{\cdot}9239 + 4{\cdot}2087t - 3{\cdot}7915t^2 + 0{\cdot}0818t^3 + 0{\cdot}1453t^4 - 0{\cdot}0147t^5$$

$$\text{Cooled} - \text{LD } T = 38{\cdot}6427 + 5{\cdot}8737t - 8{\cdot}1452t^2 + 2{\cdot}1151t^3 - 0{\cdot}2272t^4 + 0{\cdot}0089t^5$$

TABLE 1
Means and Standard Deviation for Carcass Weight, Lean Meat Content and Glycolytic Potential in Control or Cooled Pigs

	Control (n = 32)	*Cooled* (n = 32)	p-*value*
Glycolytic potential (μg g^{-1})	180 (44)	185 (54)	0·67
Lean meat (%)	59·5 (2·9)	59·1 (2·3)	0·55
Carcass weight (kg)	75·8 (5·6)	76·1 (6·2)	0·83

TABLE 2
Means and Standard Deviation for pH and Temperature in LD and BF, 5 and 30 min *Post mortem* in Control or Cooled Pigs

Muscle	*Time pm*	*Temperature*			*pH*		
		Control (n = 32)	*Cooled* (n = 32)	p-*value*	*Control* (n = 32)	*Cooled* (n = 32)	p-*value*
m. longissimus dorsi							
	5 min	39·5 (0·6)	39·2 (0·7)	0·052	6·81 (0·14)	6·81 (0·17)	0·993
	30 min	39·7 (0·7)	39·6 (0·7)	0·856	6·73 (0·17)	6·67 (0·17)	0·250
m. biceps femoris							
	5 min	39·7 (0·5)	39·6 (0·3)	0·126	6·88 (0·14)	6·85 (0·17)	0·600
	30 min	39·6 (0·6)	39·6 (0·6)	0·555	6·80 (0·15)	6·77 (0·16)	0·701

$$\text{Control} - \text{BF}\ T = 39{\cdot}5045 + 1{\cdot}5131t - 1{\cdot}9888t^2 + 0{\cdot}2538t^3 - 0{\cdot}0087t^4$$

$$\text{Cooled} - \text{BF}\ T = 39{\cdot}4810 + 1{\cdot}5609t - 2{\cdot}7090t^2 + 0{\cdot}4933t^3 - 0{\cdot}0295t^4$$

As expected, the cooled pigs had a significantly lower temperature than control pigs in LD and BF after the cooling process from 1 to 6 hr *post mortem*. The maximum temperature difference between the control and the cooled carcasses was 2°C for LD and 1°C for BF and occurred 2 to 4 hr *post mortem* [Fig. 1(a) and (b)]. At 24 hr *post mortem* the temperature was 2–3°C and the same for both treatments.

pH change

The glycolytic potential was included in the model as a covariate, but had no significant effect on the average pH curve (i.e. the rate and extent of the pH fall) within treatment, and was therefore discarded from the basic model. The course of the pH fall was described for LD and BF for each treatment by the following polynomials where t is any selected time from 5 min to 6 hr *post mortem*:

$$\text{Control} - \text{LD} : \text{pH} = 6{\cdot}8827 - 0{\cdot}4442t + 0{\cdot}0591t^2 - 0{\cdot}0028t^3$$

$$\text{Cooled} - \text{LD} : \text{pH} = 6{\cdot}8353 - 0{\cdot}3390t + 0{\cdot}0468t^2 - 0{\cdot}0028t^3$$

$$\text{Control} - \text{BF} : \text{pH} = 6{\cdot}9604 - 0{\cdot}4193t + 0{\cdot}0332t^2$$

$$\text{Cooled} - \text{BF} : \text{pH} = 6{\cdot}9291 - 0{\cdot}3366t + 0{\cdot}0232t^2$$

The cooled carcasses had a higher pH in LD and BF, by 0·1 to 0·2 units respectively from 3 to 6 hr *post mortem* [Fig. 2(a) and (b)], but were equal at 24 hr *post mortem*.

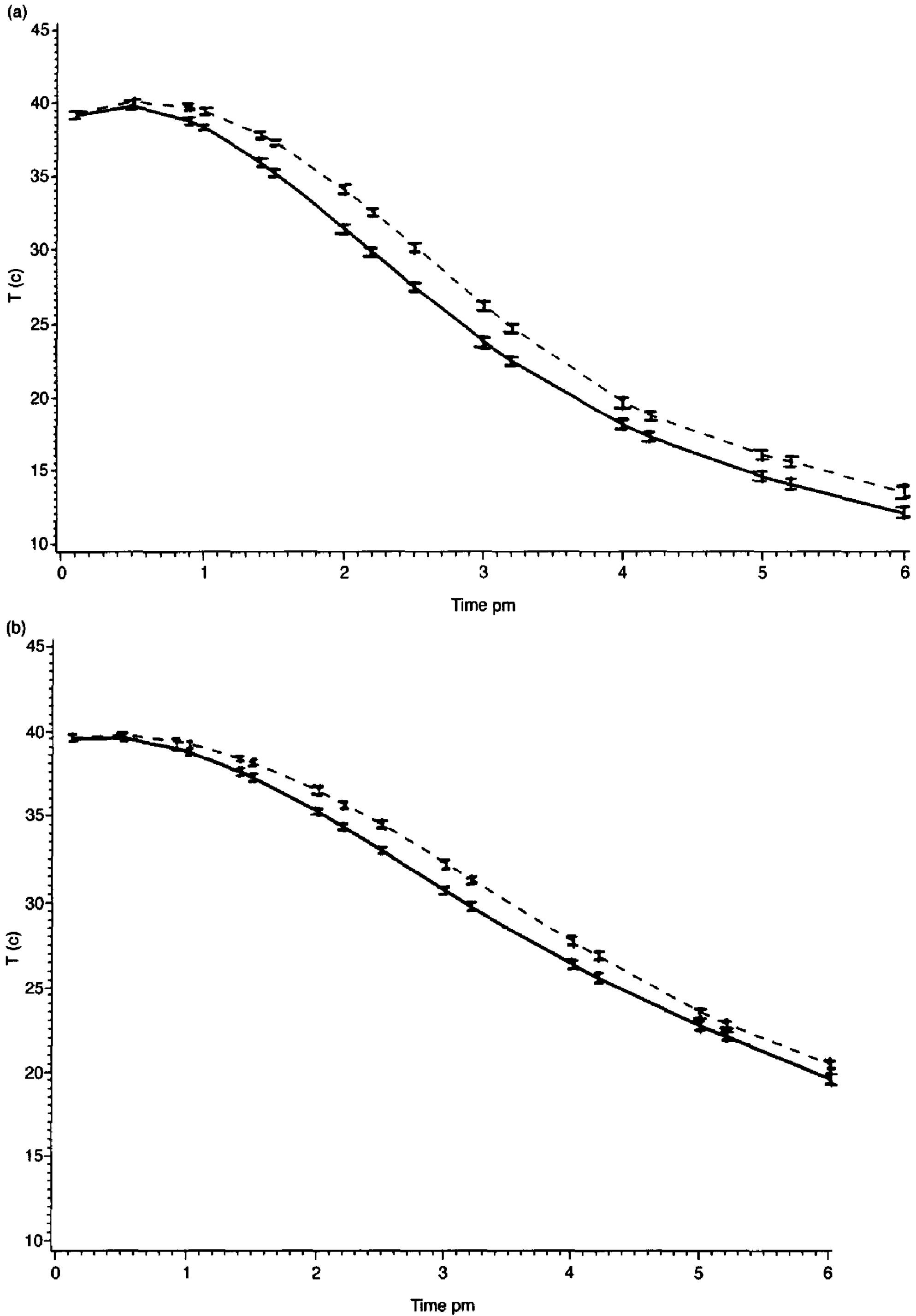

Fig. 1. (a) Temperature in LD from 5 min to 6 hr *post mortem* (pm) in control (-) and cooled (——) pigs. (b) Temperature in BF from 5 min to 6 hr *post mortem* (pm) in control (-) and cooled (——) pigs.

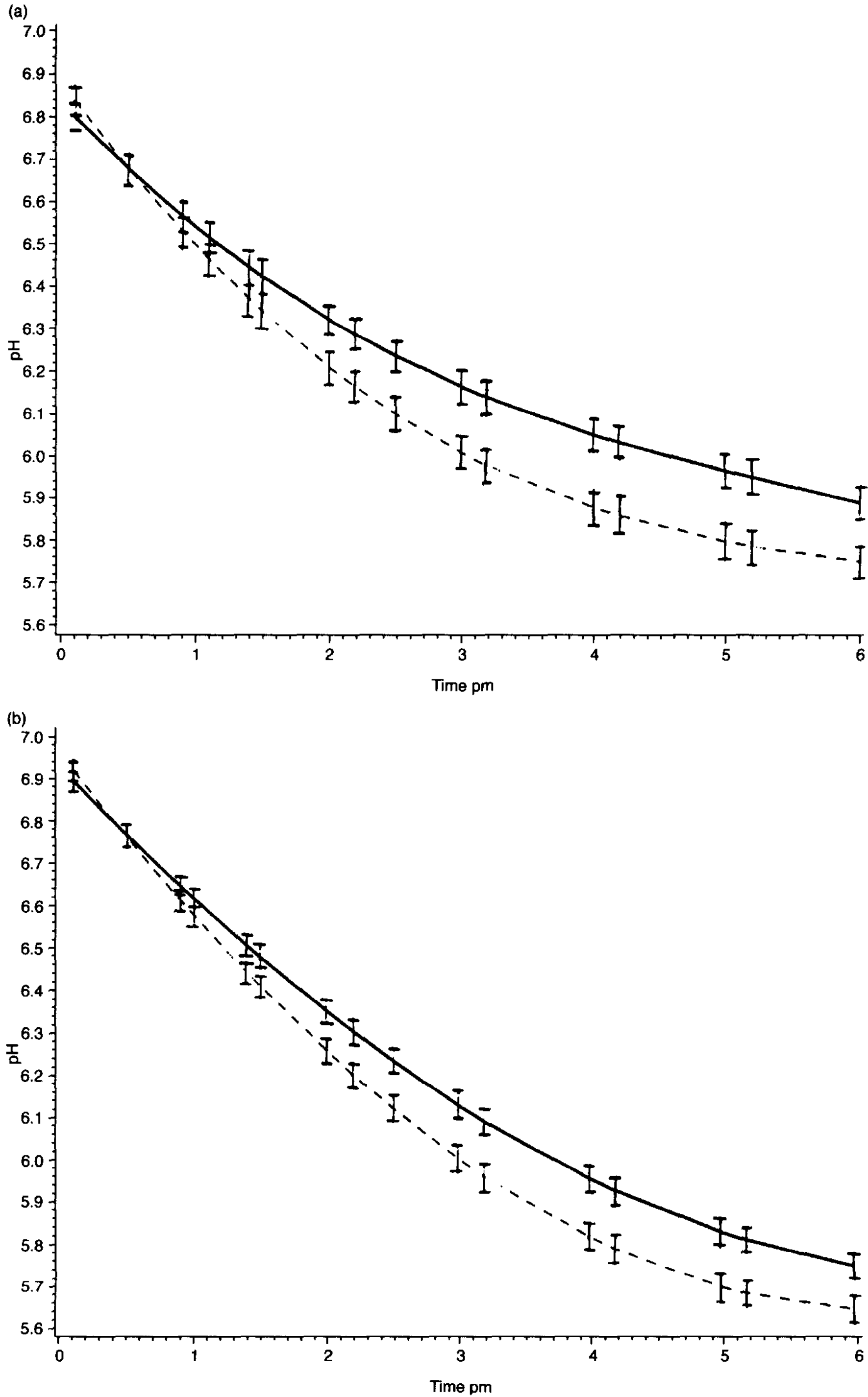

Fig. 2. (a) pH in LD from 5 min to 6 hr *post mortem* (pm) in control (-) and cooled (——) pigs. (b) pH in BF from 5 min to 6 hr *post mortem* (pm) in control (-) and cooled (——) pigs.

To investigate the effect of glycolytic potential in LD on the slope of the pH curve of LD and BF within treatment, the pigs were divided in four groups according to glycolytic potential in LD ≤ 150, 151–175, 176–225, $> 225\,\mu g\ g^{-1}$. The curves showed that the level of glycolytic potential in LD did not influence the early pH fall in LD and BF from exsanguination to 5 hr *post mortem*, but 5 to 6 hr *post mortem* the pigs with the highest glycolytic potential ($> 225\,\mu g\ g^{-1}$) had a significantly lower pH (0·1–0·2 units) than pigs with the lowest glycolytic potential ($\leq 150\,\mu g\ g^{-1}$). The other glycolytic potential groups were intermediate but not significantly different from the others.

A model of the rate of the pH fall ($\Delta pH\ hr^{-1}$) can be estimated from the pH model by differentiating the polynomials of the pH fall and are described by the following polynomials where t is any selected time from 5 min to 6 hr *post mortem*:

$$\text{Control} - \text{LD} : \Delta\text{pH hr}^{-1} = 0{\cdot}4442 - 0{\cdot}1182t + 0{\cdot}0083t^2$$

$$\text{Cooled} - \text{LD} : \Delta\text{pH hr}^{-1} = 0{\cdot}3390 - 0{\cdot}0936t + 0{\cdot}0083t^2$$

$$\text{Control} - \text{BF} : \Delta\text{pH hr}^{-1} = 0{\cdot}4193 - 0{\cdot}0664t$$

$$\text{Cooled} - \text{BF} : \Delta\text{pH hr}^{-1} = 0{\cdot}3366 - 0{\cdot}0464t$$

In LD and BF the rate of the pH fall was reduced by time *post mortem*, and was lowest in cooled carcasses from exsanguination to 4 hr *post mortem*. From 4 to 5 hr *post mortem* the rate of the pH fall was equal for the control and the cooled carcasses. From 5 to 6 hr the rate of the pH fall is highest in cooled carcasses [Figs 3(a) and (b)]. The difference in rate of pH fall between treatments was highest when the temperature difference was greatest i.e. at 3–4 hr *post mortem*, but the difference in pH level was greatest 5–6 hr *post mortem* [see Figs 1(a) to 3(b)].

The relationship between rate of pH fall and temperature is illustrated in Figs 4(a) and (b). From these figures a linear relationship between temperature and rate of pH fall is seen when the temperature is below approximately 37°C. At temperatures above approximately 37°C the rate of the pH fall is on average higher, although very variable (0·20 to 0·45 pH units per hour). The figures show the measured temperature and estimated rate of pH fall for all pigs at each measuring point between exsanguination and 6 hr *post mortem*. The relationship between rate of pH fall and temperature was independent of treatment, but the relationship varied between muscles.

The rate of the pH fall can be described by pH and temperature level when muscle temperature is below 37°C by using a general model $\Delta pH\ hr^{-1} = \text{constant} + k_1T + k_2pH + w$, and the estimated models for the two muscles are:

$$\text{LD} : \Delta\text{pH hr}^{-1} = -0{\cdot}1192 + 0{\cdot}0078T + 0{\cdot}013\ \text{pH}$$

$$\text{BF} : \Delta\text{pH hr}^{-1} = -0{\cdot}2854 + 0{\cdot}0130T + 0{\cdot}0137\ \text{pH}$$

The above models of temperature and pH fall as well as rate of pH fall make it possible to calculate these values at any time from 5 min to 6 hr *post mortem*. Temperature, pH level

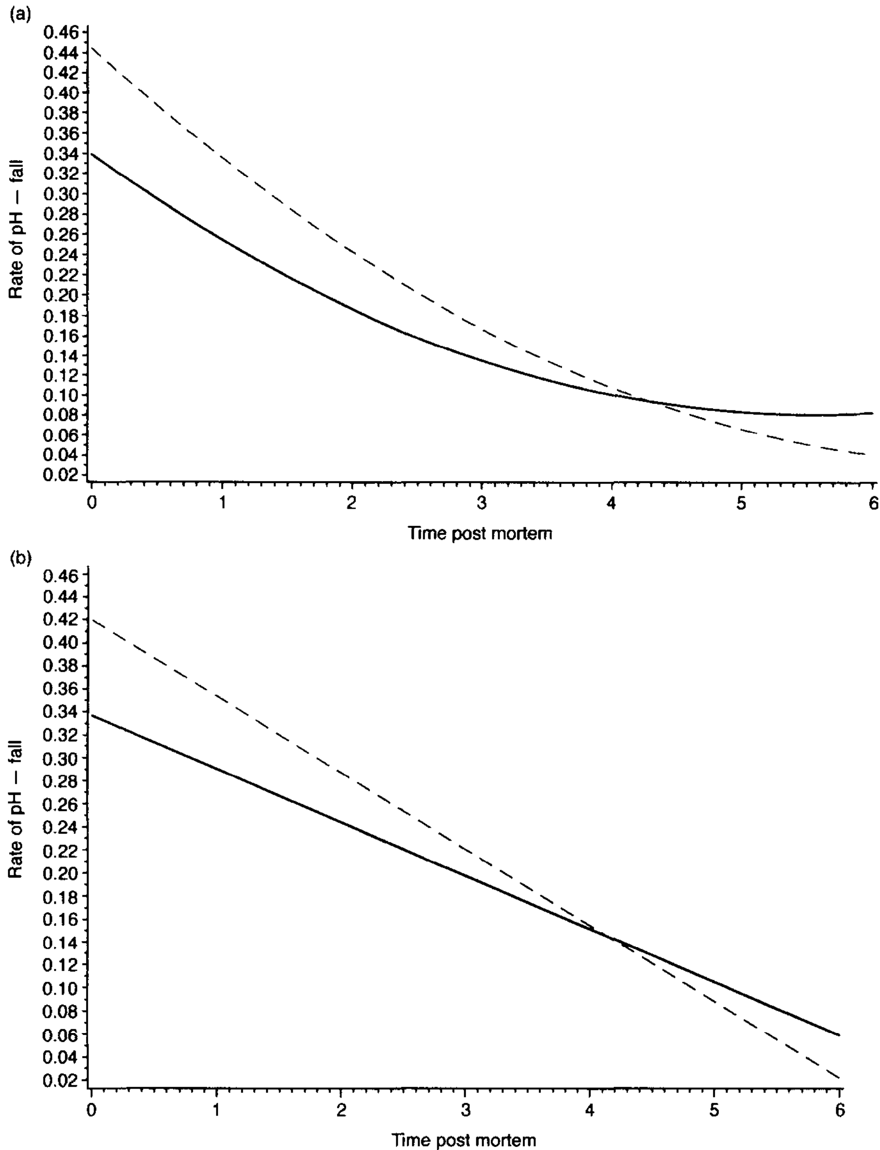

Fig. 3. (a) Rate of pH fall in LD from 5 min to 6 hr *post mortem* in control (-) or cooled (——) pigs. (b) Rate of pH fall in BF from 5 min to 6 hr *post mortem* in control (-) and cooled (——) pigs.

and rate of pH fall are calculated at selected times *post mortem*: (1) at the time the temperature starts to fall (i.e. has reached its maximum); (2) 45 min *post mortem*; (3) at the time the temperature is below 37°C and (4) at the time rigor is complete (see Table 3). The definition used for estimating the completion of rigor is when pH reaches 6·00 (Bendall, 1973).

The temperature in LD and BF begins to fall after 20 and 30 min *post mortem* respectively, which corresponds to the period just after gut removal, and before the experimental cooling process takes place. The maximum temperature for both treatments was 40°C in

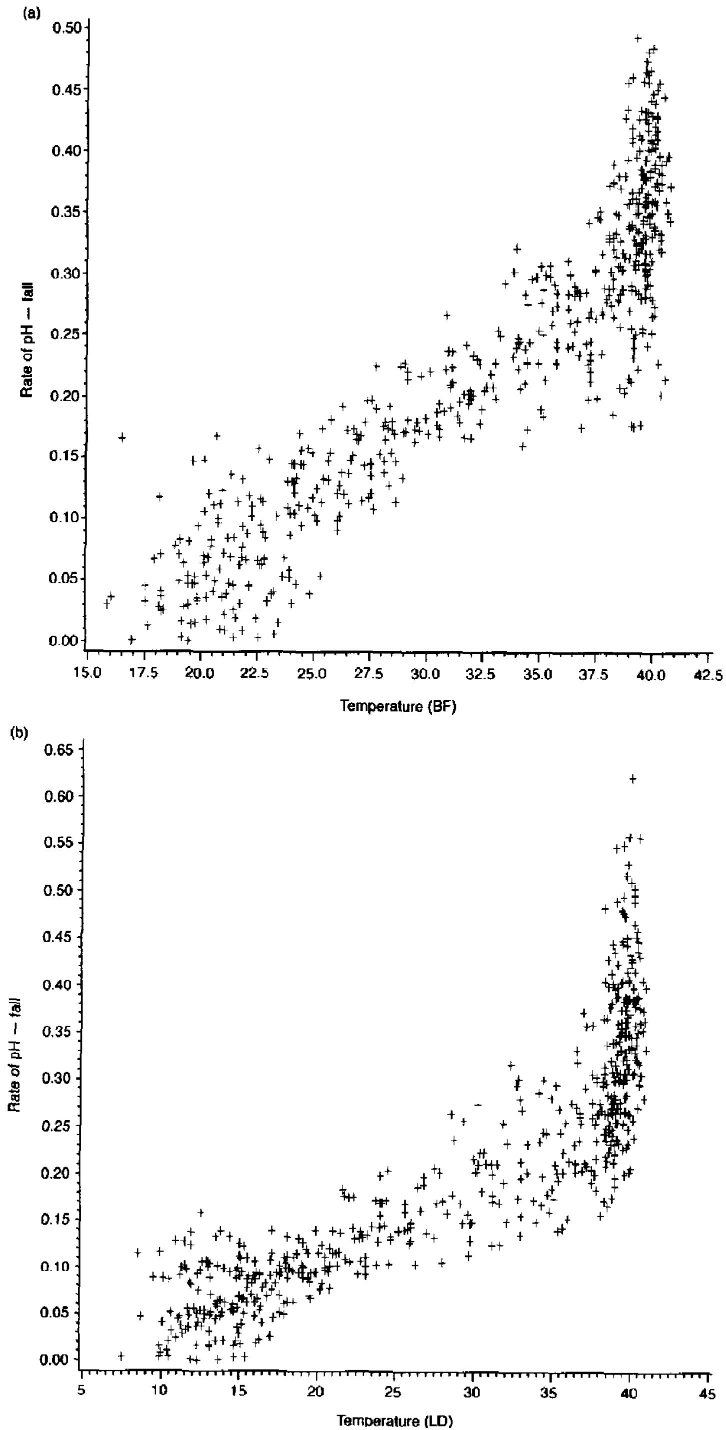

Fig. 4. (a) Rate of pH fall in relation to temperature in BF (all pigs). (b) Rate of pH fall in relation to temperature in LD (all pigs).

TABLE 3
Temperature (T), pH and Rate of pH Fall Calculated from the Models at Various Times *Post mortem* in Control and Cooled pigs. The Selected Control Points Were (1) When the Temperature Had Reached the Maximal Level; (2) 45 mins *Post mortem* (Pm); (3) When the Temperature Was Below 37°C and (4) When Rigor Was Complete (pH < 6·0)

	Time pm (min)		*Temperature (°C)*		*pH*		*Rate of pH fall (units h^{-1})*	
	Contr.	*Cool.*	*Contr.*	*Cool.*	*Contr.*	*Cool.*	*Contr.*	*Cool.*
m. l. dorsi								
1. Max temp.	35	26	40·1	39·8	6·64	6·70	0·38	0·30
2. 45 min pm	—	—	40·0	39·2	6·58	6·60	0·36	0·27
3. Temp. < 37°C	97	74	—	—	6·30	6·48	0·27	0·24
4. Rigor	182	272	27·6	16·0	—	—	0·13	0·09
m. b. femoris								
1. Max temp.	25	19	39·8	39·7	6·79	6·82	0·39	0·32
2. 45 min pm	—	—	39·6	39·3	6·66	6·69	0·37	0·30
3. Temp. < 37°C	112	94	—	—	6·30	6·46	0·22	0·16
4. Rigor	186	223	32·2	27·6	—	—	0·22	0·16

both LD and BF, and the pH levels were 6·6–6·7 and 6·8 respectively. From exsanguination to maximum temperature was reached, the temperature rose by 1°C and pH fell by 0·3 units in LD. In BF, the temperature rose 0·3°C and pH fell 0·2 units. As expected there was no difference in pH level and temperature 45 min *post mortem*, but the rate of the pH fall was lowest in the cooled carcasses, which indicates that cooling has had an effect at this time that later results in differences in pH level. The temperature was below 37°C in both LD and BF approximately 20 min earlier in the cooled carcasses than in the control group, and at this temperature pH levels were higher and the rate of pH fall lower in the cooled carcasses. Rigor was complete 1½ and ½ hr later in cooled than in control carcasses for LD an BF respectively. At the time of rigor onset the temperature was lowest and the rate of pH fall slowest in the cooled carcasses.

Meat quality

There was no significant effect of the cooling process on the meat quality parameters measured (Table 4), but there was a tendency towards a lower drip loss and higher ultimate pH in LD and BF in the cooled carcasses. Ultimate pH in LD, BF and SM was affected by the glycolytic potential in LD, higher glycolytic potential in LD resulting in a lower ultimate pH in all muscles.

Halothane carriers

From 50 min to 6 hr *post mortem* the eight halothane carriers had lower pH's than the 64 halothane free pigs. The day after slaughter the halothane carriers had a lower content of soluble proteins and higher drip loss than halothane free pigs in LD, but there was an interaction between treatment and halothane gene in content of soluble proteins and drip loss for BF. Control halothane carriers (six pigs) had a lower content of soluble proteins and higher drip loss of in BF compared with non-carriers, whereas there was no difference between genotypes for the cooled carcasses (Table 5).

TABLE 4
Means and Standard Deviation for Content of Soluble Proteins and Drip Loss in LD and BF and Ultimate pH in LD, BF, SM, and SC in Control or Cooled Pigs

	Control (n = *32*)	*Cooled* (n = *32*)	p-*value*
Soluble protein LD	0·181 (0·009)	0·178 (0·011)	0·330
Soluble protein BF	0·172 (0·008)	0·171 (0·008)	0·890
Drip loss LD (%)	4·81 (1·84)	4·23 (1·86)	0·215
Drip loss BF (%)	3·89 (1·28)	3·47 (1·63)	0·252
Ultimate pH LD	5·50 (0·12)	5·53 (0·15)	0·389
Ultimate pH BF	5·58 (0·14)	5·60 (0·18)	0·721
Ultimate pH SM	5·55 (0·12)	5·57 (0·15)	0·567
Ultimate pH SC	5·98 (0·23)	5·99 (0·21)	0·826

TABLE 5
Means for Content of Soluble Proteins and Drip Loss in LD for Pigs With and Without the Halothane Gene and in BF for Control or Cooled Pigs With and Without the Halothane Gene

	Halothane gene free (n = *64*)		*Halothane carrier* (n = *8*)		p-*value*
Soluble proteins LD	0·18[a]		0·16[b]		0·0001
Drip loss LD	4·4[a]		10·3[b]		0·0001
	Control (n = *32*)	*Cooled* (n = *32*)	*Control* (n = *6*)	*Cooled* (n = *2*)	p-*value*
Soluble proteins BF	0·17[a]	0·17[a]	0·15[b]	0·17[a]	0·0011
Drip loss BF	3·8[a]	3·6[a]	6·9[b]	3·0[a]	0·0004

[ab]Values marked with different superscript within row, are significantly different ($p < 0{\cdot}05$)

DISCUSSION

pH and temperature conditions are important *post mortem* factors known to affect meat quality, particularly drip loss. The benefits of rapid chilling was acknowledged by the Danish pig industry leading to the introduction of tunnel chilling operating at a temperature of approximately −20°C. In practice chilling is usually commenced at about 45 min *post mortem*. Most work dealing with the effect of chilling rate on pork has not included the very early *post mortem* period, i.e. cooling during the slaughter process.

In the present work an early *post mortem* cooling procedure was used. The cooling took place at approximately 30 min *post mortem* by showering for 12 min with cold water (10–12°C). The cooling process reduced the temperature in LD and BF and the reduction was greater in LD than in BF. The explanation for this is that the loins were showered on both the outside and the inside of the carcass, while the hams (BF) were only showered on the outside. Furthermore the BF is close to other muscles in the ham and they continue to supply heat to the cooled muscle surface. Surprisingly neither carcass weight nor lean meat content had any effect on temperature, perhaps as a result of the small variation between carcasses. The reduction of temperature in the cooled carcasses by 1–2°C resulted in a lower rate of the pH fall and a pH 0·1 to 0·2 units higher throughout the measuring period (from exsanguination to 6 hr *post mortem*). The cooling affected the rate of the pH fall immediately, but the effect on pH is delayed by 2–3 hr.

The rate of the pH fall in the muscles is reduced by time from exsanguination until ultimate pH is reached, due to a reduction in the glycogen content in the muscle, a falling temperature and falling pH reduce the metabolic/enzymatic activity (Bendall, 1973). Klont *et al.* (1994), found that the content of glycogen decreased and lactate increased with increasing temperature between 38 and 42°C in halothane free pigs. Klont *et al.* (1994), based on results found by Bendall (1973), calculated that a reduction in temperature of 1°C reduces the metabolic rate by 8% in the interval from 38–30°C. The results of this study also show that the rate of the pH fall is reduced when the temperature declines. The correlation between temperature and rate of pH fall [Fig. 4(a) and (b)] led to the hypotheses that the rate of the pH fall was independent of temperature above approximately 37°C, but decreased with decreasing temperature below approximately 37°C. There could be two explanations for this. Firstly, the effect could be purely enzymatic. Early *post mortem*, when the glycogen reserves are higher, activity of the metabolic enzymes are likely to be at their optimal level at physiological temperatures 39 ± 2°C. As temperature falls below a certain level, about 37°C, the enzyme activity and hence metabolic rate are progressively reduced. Secondly, the effect could be controlled by the glycogen levels. A high glycogen content early *post mortem* will keep the metabolic rate high which will keep the temperature high. When the content of glycogen is limited the metabolic rate falls as well as the heat production and the muscle temperature is reduced.

If the first hypothesis is correct it is possible to reduce the rate of the pH fall by decreasing the temperature. If the second hypothesis is correct it is not possible to reduce the metabolic rate by changes in temperature early *post mortem* as the rate of the pH fall will only depend on the amount of glycogen. The results of this study support the first hypothesis. Furthermore this study showed that the pH fall in LD early *post mortem* did not depend on the glycolytic potential in LD. However, pigs with the highest level of glycolytic potential in LD ($>225\,\mu g\ g^{-1}$) had a lower pH in LD and BF than pigs with the lowest glycolytic potential in LD ($\leq 150\,\mu g\ g^{-1}$) 5 to 6 and 24 hr *post mortem* irrespective of treatment. At 5 to 6 hr *post mortem* the level of glycogen in the muscles of pigs with a low glycolytic potential in LD had obviously reached a level that affected the metabolic rate. If the pigs had been exhausted at slaughter the level of glycolytic potential would have been even lower and the pH-fall would probably have been affected earlier *post mortem*. This study therefore shows that in halothane free pigs the metabolic rate early *post mortem* was not affected by the glycolytic potential, but was mainly affected by the temperature in the muscles, thus confirming earlier work (Klont *et al.*, 1994 and Monin *et al.*, 1995).

Despite the effect of early cooling on pH fall the cooling process did not result in significant changes in protein denaturation or drip loss. During the time interval from 5 min *post mortem* to rigor, pH fell by 0·29 and 0·19 units per hour for the control and cooled carcasses respectively. This is much lower than previously assumed for normal carcasses (0·6 units per hour or 0·01 unit per minute) by Offer (1991), but this could be a result of classifying halothane carriers as normal pigs. Presumably the temperature reduction at the slaughter line in this work was not great or early enough to reduce protein denaturation and drip loss significantly. For practical reasons the batch chilling process began 20 min later than normal. Had the chilling process started earlier or had it been more efficient, then the protein denaturation in the cooled carcasses may have been reduced further either by an earlier reduction in rate of pH fall or a reduction in pH fall over a longer period. Earlier work has shown that the earlier and the faster the temperature is reduced, the larger the reduction in drip loss, due to a reduction in myosin denaturation in fast glycolytic muscles (Honikel, 1987).

The cooled halothane carriers had protein denaturation and drip loss in BF at the same level as the halothane free pigs, whereas non-cooled halothane carriers showed greater

protein denaturation and higher drip loss in BF than halothane free pigs. These results confirm earlier work which showed that cooling of hot boned parts from halothane carriers resulted in an improvement in water holding capacity (Honikel, 1987; Woltersdorf and Troeger, 1986). The meat quality from halothane carriers has been shown to be very variable (Jensen and Barton-Gade, 1985; Lundström *et al.*, 1989; Sather *et al.*, 1991; Leach *et al.*, 1996; Pommier and Pomar, 1996) and the results from this study indicates that variation in the cooling of the carcasses early *post mortem* may be one factor leading to this situation.

The experimental cooling procedure used in this trial is not realistic for use at commercial abattoirs, on hygienic and water usage grounds. Under commercial slaughter conditions there are other possible methods for reducing the muscle temperature early *post mortem* such as using chilled water in processes that use water, optimization of the scalding procedure, use of skinning or reducing time from exsanguination to chilling. The extent of meat quality improvement will however, depend on the pig population concerned and is likely to be greatest when the halothane gene is present, since the cooling process did seem to reduce protein denaturation and drip loss in BF in the few halothane carriers in this work. If the treatment of the pigs prior to slaughter had been more stressful (lairage for a longer time, fighting during lairage, forcing pigs to the stunning pen in an abattoir with higher killing rates etc.) the effect of cooling on the slaughter line might have resulted in significant differences in protein denaturation and drip loss, as a pig stressed before slaughter shows higher muscle temperature, faster pH fall, higher protein denaturation and higher drip loss (Warris *et al.*, 1995; van der Wal *et al.*, 1997).

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REFERENCES

Barton-Gade, P., Warriss, P., Brown, S. N. and Lambooij, B. (1996) Methods of assessing meat quality. *Proc. EU Seminar 'New information on welfare and meat quality of pigs as related to handling. transport and lairage conditions'*. Mariensee, Germany June 1995, 23–34

Bendall, J. R. (1973) In *Structure and function of Muscle*. ed. G. H. Bourne. Vol. II, 2nd edn. part 2, pp. 243–309. Academic Press, New York.

Fujii, J., Otsu, K., Zoranto, F., De Leon, S., Kahnna, V. K., Weiler, J. E., O'Brien, P. J. and Mc Lennan, D. H. (1991) Identification of a mutation in porcine ryanodine receptor associated with malignant hypertamia. *Science* **253**, 448–451.

Honikel, K. O. (1987). Influence of chilling on meat quality attributes of fast glycolysing pork muscles. In *Evaluation and Control of Meat Quality of Pigs*. eds. P. V. Tarrant, G. Eikelenboom, G., Monin. Martinus Nijhof Publishers, pp. 273–283.

Honikel, K. O. and Kim, C. J. (1986) Causes of the development of PSE pork. *Fleischwirtschaft* **66**, 349–353.

Honikel, K. O., Kim, C. J., Hamm, R. and Ronchales, P. (1986) Sarcomere shortening of pre-rigor muscles and its influence on drip loss. *Meat Science* **16**, 267–282.

Honikel, K. O., Kim, C. J. and Hamm, R. (1984) The influence of temperature on post mortem changes in porcine muscles. In *Proceedings of the 30th European Meeting of Meat Research Workers*, pp. 95–96.

Jensen, P. and Barton-Gade, P. A. (1985) Performance and carcass characteristics of pigs with known genotypes for halothane susceptibility. In *Stress Susceptibility and Meat Quality in Pigs*, ed. *J. B. Ludvigsen, EAAP. Publication No* **33**, 80–87.

Klont, R. E., Talmant, A. and Monin, G. (1994) Effect of temperature on porcine-muscle metabolism studied in isolated muscle-fibre strips. *Meat Science* **38**, 179–191.

Leach, L. M., Ellis, M., Sutton, D. S., McKeith, F. K. and Wilson, E. R. (1996) The growth performance, carcass characteristics, and meat quality of halothane carrier and negative pigs. *Journal of Animal Science* **74**, 934–943.

Lundström, K., Essen Gustavsson, B., Rundgren, M., Edfors-Lilja, I. and Malmfors, G. (1989) Effect of halothane genotype on muscle metabolism at slaughter and its relationship with meat quality: A within litter comparison. *Meat Science* **25**, 251–263.

Maribo, H. (1996) Temperature development in carcasses with different slaughter processes. *Poster Proceedings 42nd ICoMST, Norway*, pp. 448–449.

Monin, G. and Sellier, P. (1985) Pork of low technolological quality with a normal rate of muscle pH fall in the immediate post-mortem period: The case of the Hampshire breed. *Meat Science* **13**, 49–63.

Monin, G., Talmant, A., Aillery, P. and Collas, G. (1995) Effects on carcass weight and meat quality of pigs dehaired by scalding or singeing *post mortem*. *Meat Science* **39**, 247–254.

Offer, G. (1991) Modelling of the formation of pale, soft and exudative meat: Effects of chilling regime and rate and extent of glycolysis. *Meat Science* **30**, 157–184.

Offer, G. and Knight, P. (1988) The structural basis of water-holding in meat part 2: drip losses. In *Developments in Meat Science–4*, ed. R. Lawrie, pp. 173–243. Elsevier Applied Science, London.

Pearson, A. M. and Young, R. B. (1989) In *Muscle and Meat Biochemistry*. Academic Press, New York, 457 pp.

Pommier, S. A. and Pomar, C. (1996) Effect of halothane and preslaughter handling on animal performance and carcass and meat quality of crossbred pigs. *Journal of Animal Science* **74** suppl 1, 161.

Rasmussen, A. and Anderson, M. (1996) New method for determination of drip loss in pork muscles. *Poster Proceedings. 42nd ICoMST, Norway*, pp. 286–287.

Rutter, C. M. and Elashof, R. M. (1994) Analysis of longitudinal data: Random coefficient regression modelling. *Statistics in Medicine* **13**, 1211–1231.

SAS (1990). SAS/STAT User's Guide, Version 6, Fourth Edition, Volume 1 and 2. SAS Institute Inc., Cary, NC, 1686 pp.

Sather, A. P., Murray, A. C., Zawadski, S. M. and Johnson, P. (1991) The effect of the halothane gene on pork production and meat quality of pigs reared under commercial conditions. *Canadian Journal of Animal Science* **71**, 959–967.

Taylor, A. A. and Dant, S. J. (1971) Influence of carcass cooling rate on drip loss in pigmeat. *Journal of Food Technology* **6**, 131–139.

Troeger, K. and Woltersdorf, W. (1987) Influence of scalding and dehairing during pig slaughtering on meat quality. *Fleischwirtschaft* **67**, 66–70.

van der Wal, P. G., Engel, B. and Hulsegge, B. (1997) Causes for variation in pork quality. *Meat Science* **46**, 319–327.

van der Wal, P. G., Veerkamp, C. H., van Beek G. and Engel, B. (1994) The temperatures of hams, measured subcutanous and in the biceps femoris during slaughter and chilling I. Changes in temperature from exsanguination till the end of scalding. *Poster proceedings. 40th INCOMST, Haag, Holland*, S-III.02.

Warriss, P. D., Brown, S. N., Nute, G. R., Knowles, T. G., Edwards, J. E., Perry, A. M. and Johnson, S. P. (1995) Potential interactions between the effects of preslaughter stress and *post mortem* electrical stimulation of the carcasses on meat quality in pigs. *Meat Science* **41**, 55–68.

Woltersdorf, W. and Troeger, K. (1986) Warmzerlegung und warmfleischgewinnung beim schwein. I. *Einfluss der warmzerlegung auf die fleischbeschaffenheit*. *Fleischwirtschaft* **66**, 37–41.