

## GENETICS OF MEAT QUALITY IN PIGS

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### INTRODUCTION

Recent increased industry interest in the genetic aspects of pigmeat quality in the UK began with the results of a trial coordinated by the Meat and Livestock Commission (MLC) and published in 1986 (Kempster *et al.*, 1986; Wood *et al.*, 1986). Groups of 100 pig carcasses representing either end of the commercial range of fat thickness (8 and 16 mm P<sub>2</sub>) exhibited small but important differences in eating quality, with loin chops from the leaner group being ranked less tender and significantly less juicy by trained taste panellists and consumers. The implication was that continuing emphasis on fatness in grading schemes was having some detrimental effects on meat quality.

Since 1986, options for improving quality especially at low overall levels of carcass fatness have been sought, including genetic options. There has also been work in other countries to identify the genetic factors controlling meat quality.

It is accepted that the consumer's definition of meat quality is now very wide and includes aspects of safety, animal ethics, nutrition etc. In this presentation we are interested mainly in the visual appearance of meat (colour, drip loss) and its eating quality i.e. the intensities of tenderness, juiciness, pork flavour and abnormal flavour when the meat is cooked.

### THE PSE CONDITION

Pigmeat exhibiting the PSE (pale soft exudative meat) condition is visually unattractive, has a lower yield throughout the butchery and retail chain because of increased drip loss and is relatively dry and tough when eaten (Bejerholm, 1984). The condition is part of the porcine stress syndrome and its genetic cause is a mutation of the ryanodine receptor gene which controls calcium movement in the muscle cell. The mutant gene is recessive so when inherited from both parents the PSE condition is expressed, especially if the pig is stressed in the preslaughter period. Dissemination of the defective gene has been encouraged because of the close association with genes controlling leanness and muscularity, traits which are very much in demand all over the world.

Up until recently, attempts to control the PSE condition depended on the use of the halothane test in which nn individuals react by strong muscle contraction to the anaesthetic halothane. The aim was to separate nn and NN breeding animals into separate lines so that Nn progeny having advantages in lean yield but not exhibiting PSE were slaughtered and not used for breeding. However some Nn individuals, undetected in the test, were often wrongly placed in the NN breeding group and this probably explains why studies have shown a high and variable incidence of PSE in the slaughter generation (Murray *et al.*, 1989).

Recently, the availability of the Toronto DNA test has made specific identification of nn and NN breeding lines possible. When the Nn offspring then have a significantly higher PSE incidence than NN such as in the Canadian study of Pommier and Houde (1993) (Table 1) the truly recessive nature of the gene is called into question. Danish research suggests that Nn are more

susceptible to pre-slaughter stressors than NN (Barton-Gade, 1984). Because of results like these, some breeding companies have attempted to remove the gene altogether from pig stocks, relying on additive genetic selection alone to raise levels of leanness and conformation.

Table 1. Effect of halothane genotype on *longissimus* muscle colour and PSE incidence.

	NN	Nn	nn
n	693	198	22
L <sup>c</sup>	50.9 <sup>a</sup>	54.1 <sup>b</sup>	56.6 <sup>b</sup>
a <sup>c</sup>	1.87	1.97	1.99
b <sup>c</sup>	10.04 <sup>a</sup>	10.83 <sup>a</sup>	11.06 <sup>b</sup>
Free water	41.4 <sup>a</sup>	43.8 <sup>b</sup>	44.6 <sup>b</sup>
% PSE	53.7	79.8	90.9

913 loins visually exhibiting the PSE condition were used. Genotype was determined using the restriction endonuclease assay. The high PSE incidence in NN shows either a high environmental component or that other genetic factors than the halothane gene are involved.

<sup>ab</sup> Means with different superscripts are significantly different ( $p < 0.01$ ) <sup>c</sup> CIELAB colour coordinates.

Pommier and Houde (1993)

#### EFFECTS OF SELECTION FOR LEAN CONTENT IN THE ABSENCE OF THE HALOTHANE GENE.

Most studies of the genetics of meat quality have been conducted in pig populations carrying the halothane gene. Medium to high values for the heritability of muscle quality traits such as pH and drip loss and similarly sized correlations between these traits and carcass lean content would be expected in such populations. There have been far fewer studies in halothane gene-free pig stocks but results of from 2 such studies (Hovenier *et al*, 1992; Cameron, 1990; Cameron and Enser, 1991) have still shown significant heritabilities for muscle quality traits (0.2 to 0.4) and generally unfavourable correlations with carcass lean content. Both studies also found that the heritability of marbling fat was high (0.5 to 0.6) and the work of Cameron and Enser (1991) found significant negative genetic correlations between eating quality traits and carcass lean content (Table 2). This was also observed by Lo *et al* (1992b). All this suggests that separate attention should be paid to meat quality in selection schemes even when the halothane gene has been eliminated.

Table 2. Heritabilities and genetic correlations with carcass lean weight in 160 Duroc and halothane gene-free Landrace pigs.

	$h^2$	genetic correlation
<u>Cameron and Enser (1991)</u>		
Marbling fat (%)	0.53	-0.41
Muscle pH <sup>a</sup>	0.20	-0.50
Muscle moisture	0.26	0.47
Tenderness <sup>b</sup>	0.23	-0.29
Juiciness <sup>b</sup>	0.18	-0.47
Pork flavour <sup>b</sup>	0.16	-0.16
<u>Cameron (1990)</u>		
Fat firmness	0.43	-0.38
Muscle colour <sup>c</sup> lightness	0.20	0.12
hue	0.19	-0.06
saturation	0.42	0.41
Fatty acids backfat		
C16:0	0.71	-0.38
C18:0	0.53	-0.30
C18:1	0.69	-0.22
C18:2	0.67	0.47

<sup>a</sup> ultimate pH    <sup>b</sup> taste panel scores    <sup>c</sup> CIELAB system

### MARBLING FAT: THE DUROC EFFECT

Although some studies, usually involving different breeds, have shown high correlations between marbling fat and total fat or lean content others have shown that the within-breed correlation is moderate (e.g. 0.11 in Polish Large Whites in the study of Duniec *et al.*, 1961). A breed with unusually high values for marbling fat in relation to carcass fat is the Duroc and it is possible to conclude across many published studies that this breed has improved juiciness and tenderness in comparison with 'white skinned' breeds.

The possibility that increasing tenderness in Duroc pigs is due to increasing marbling fat comes from results such as those of Bejerholm and Barton-Gade (1986) which showed wide variations in both characteristics. However in an equally wide ranging study, Goransson *et al* (1992) showed no association.

The results of a British study (Meat and Livestock Commission, 1992) showed that the concentration of haem pigments and the redness of muscle increased as the proportion of Duroc genes increased (Table 3). This suggested that Durocs had a higher concentration of red oxidative (as opposed to white glycolytic) muscle fibres which in some studies, reviewed by Wood (1994) has been linked with higher eating quality scores. Individual muscles with more red fibres also often contain higher lipid deposits. However, Karlsson *et al* (1993) found no difference in the proportions of muscle fibre types between Swedish Yorkshire pigs selected for 2 or 4 generations for lean tissue growth rate or any clear association between fibre type and lipid content.

Table 3. Effect of the Duroc breed on meat quality.

	Duroc genes (%)			
	0	25	50	75
<u>Measurements on <i>m. longissimus</i></u>				
Haem pigments (mg/g)	0.61	0.64	0.67	0.67
L* brightness	54.0	53.8	53.3	53.6
a* redness	2.2	2.7	2.9	3.1
Saturation	4.5	5.2	5.4	5.7
Marbling fat (%)	0.70	0.86	1.08	1.27
<u>Scores of trained taste panel (1-8)</u>				
Tenderness	4.96	5.03	5.32	5.38
Juiciness	4.09	4.11	4.18	4.38
Flavour	3.88	3.99	3.96	3.98

Meat and Livestock Commission (1992)

A review of current marbling fat levels (defined as ether-extractable lipid as a percentage of the weight of fresh *m. longissimus*) in European and American pigs showed much lower levels in the former (0.5 to 2.5%) than the latter (2.0 - 5.0%) (Wood, 1993). Recent work at Bristol has found significant numbers of pigs below 0.5%, some as low as 0.2%. It is difficult to believe that levels as low as this do not adversely affect eating quality.

Effective selection for marbling fat depends on developing an *in vivo* test, probably based on ultrasound. Some success has been achieved in cattle (e.g. Park *et al.*, 1994) but low levels of marbling fat in pigs makes individual identification much more difficult. Progress towards higher marbling fat levels is also constrained by the finding in 2 studies that mean values for the crossbred progeny are less than the average value of the parental breeds (Wood *et al.* 1988; Lo *et al.* 1992a).

Early work with the Meishan and other Chinese pig breeds suggested some advantages for meat quality over European breeds, perhaps associated with marbling fat. However, Ellis *et al.* (1990) found similar overall acceptability of eating quality between purebred Meishan and Large White pigs, slightly higher tenderness and juiciness in the Meishan being balanced by more abnormal odour and flavour. Young (1992) found that although Meishan, Fengjing and Minzhu crossbred pigs were fatter than Durocs, they had lower marbling scores.

In an general sense it can be concluded that variations in muscle pH/colour and marbling fat explain an important part of the genetic variation in eating quality seen in commercial pig stocks. This was illustrated in the MLCs study of genotypes from different breeding companies (Meat

and Livestock Commission, 1989). The extreme groups in terms of tenderness and juiciness were also extreme in drip loss and marbling fat (Table 4) However, more information is required on the relationships between eating quality and muscle characteristics to identify the important components of eating quality that can be included in a breeding objective.

### HANDLING CHARACTERISTICS

When early studies were done in the UK to identify the meat quality problems associated with genetically lean pigs (Wood *et al.*, 1986), much emphasis was given by retailers to the problems of fat separation and soft fat. These were identified as being directly due to the low level of fat tissue in the carcass and to relative immaturity in the development of fat as measured by a high concentration of water, a low concentration of lipid and small fat cells. Genetic studies show that the heritabilities of tissue firmness and the fatty acids which control it are moderate to high and that genetic progress could be made independently of total fat content (Cameron and Enser, 1991).

Table 4. Carcass leanness and meat quality in the crossbred progeny of 'white' sires from two commercial breeding companies.

	Company K	Company M	
P <sub>2</sub> fat thickness (mm)	14.1	11.0	*
lean in carcass (%)	53.7	58.0	*
Fat in carcass (%)	21.6	16.0	*
Drip loss (%) <sup>a</sup>	3.8	4.7	*
Marbling fat (%) <sup>b</sup>	0.89	0.74	*
Tenderness <sup>c</sup>	5.4	4.4	*
Juiciness <sup>c</sup>	4.5	3.9	NS
Pork flavour <sup>c</sup>	4.3	4.7	NS

<sup>a</sup> In *m. longissimus* <sup>b</sup> Ether extract of *m. longissimus* <sup>c</sup> Taste panel scale 1-8 for roast pork

\* Differences significant P<0.05)

Meat and Livestock Commission (1989)

### PROTEOLYSIS: THE CONDITIONING EFFECT

Until recently in the UK it was believed that the process of conditioning in pigmeat was accomplished extremely rapidly after slaughter so very little ageing was done. More recent studies, incorporated in the MLC's *Blueprint for Lean and Tender Pork*, show important tenderising effects occurring for at least 7 or 14 days postmortem following storage of loins at 1°C. These changes are due to the action of proteolytic enzymes on the myofibrillar proteins gradually breaking down the fibrous structure of muscle.

Work by Koohmaraie's group at Clay Center Nebraska over the last few years has shown that the calpain enzyme system is particularly significant in meat tenderisation (Koohmaraie, 1992). The same enzymes (calpain-1, calpain -2 and calpastatin) which are responsible for myofibrillar protein degradation *in vivo* also control the process post mortem (Bardsley *et al.*, 1992). Results of a US beef breeding project showed that substantial genetic improvement in tenderness could

be made by selecting on the basis of calpastatin activity (Shackelford *et al*, 1992). It is possible that the same would apply in pigs in which faster rates of muscle growth (and turnover) are associated with more tender meat (Warkup and Kempster, 1991).

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