CARCASS AND MEAT QUALITY CHARACTERISTICS OF THREE HALOTHANE GENOTYPES IN PIGS

by

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Declaration

I the undersigned hereby declare that the work contained in this thesis is my own original work and has not previously in its entirety or in part been submitted at any university for a degree

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Date: 29 / 8 / 1995

SUMMARY

The object of this study was to determine the effect of the halothane gene in pigs on certain production, carcass, meat and processed meat characteristics.

Fifty nine (gilts = 25, castrates = 34) Landrace x Large white pigs of three halothane genotypes (NN = 31, Nn = 17, nn = 11) were raised under commercial conditions from 27 kg to 86 kg live weight. Variables measured during this period were days to slaughter and ADG. Upon reaching slaughter weight (86 kg) the pigs were transported to a commercial abattoir and slaughtered and classified according to factory procedures. Variables measured at the classification point were pH_1, carcass length, warm carcass weight, fat thickness, meat depth and percentage predicted lean yield. After a 24 h cooling period the carcases were weighed, pH_{24} measured, cut to factory specifications and samples of the loin removed to determine drip loss. Certain portions (left hand side ham and back) of the carcass were removed, deboned and frozen for further processing.

The backs and hams were defrosted after all the pigs were slaughtered. The backs were used to manufacture back bacon according to commercial procedures followed in the factory. This consists of brine injection, immersion in brine for 24 h, smoking and tempering or cooling. Weights of the individual samples (n = 59) were recorded before and after each of the processes mentioned. The deboned hams were grouped according to genotype and minced using a 20 mm mincerplate. Spices, preservatives and ice water were added to the minced meat and tumbled for 30 min to enhance the water binding potential of the meat. The ham mixture of each genotype was canned (20 cans/genotype), weighed and sterilized. After a cooling period the hams were removed from the cans, residual water drained off and the weight of each sample determined.

Samples (n = 59) removed from the processed backs were used to determine chemical composition. Moisture content of the samples were determined by method of freeze drying. The dried samples were then analyzed for protein content (Kjeldahl), fat content (ether extraction) and sodium content (chromatography). The variables were analyzed in a two way classification model using the method of least squares to estimate differences between means. Correlations between variables and multiple linear regression equations for certain dependent variables were also calculated. Interaction between sex and genotype for certain variables (days to slaughter, ADG, carcass length, percentage bacon yield) were discounted due to unalterability of the castrate:gilt ratio (days to slaughter, ADG, carcass

length) or due to low significant values for sex in the model (percentage bacon yield).

The presence of the halothane gene in homozygous form (nn) caused enhanced growth rate (days to slaughter, ADG). The nn pigs grew significantly ($P < 0.05$) faster than the NN- and Nn pigs during the growth (days to slaughter) phase (NN = 81.2, Nn = 84.6 and nn = 74.1). No significant differences between the genotypes for ADG were observed (NN = 0.729, Nn = 0.710 and nn = 0.765).

Most of the carcass and meat quality characteristics did show statistically significant differences. No differences in the means for percentage chilling loss (NN = 4.4, Nn = 4.7 and nn = 5.2) and pH_{24} (NN = 5.87, Nn = 5.81 and nn = 5.80) were found. The correlations between pH_1 and pH_{24} ($r = 0.27$, $P < 0.05$) and drip loss ($r = -0.39$, $P < 0.05$) were significant. Percentage drip loss differed significantly ($P < 0.05$) between all three genotypes (NN = 1.69, Nn = 2.39 and nn = 1.06). Fat thickness (NN = 20.0, Nn = 18.8 and nn = 26.1), meat depth (NN = 50.9, Nn = 52.4 and nn = 46.8) and percentage predicted lean yield (NN = 66.1, Nn = 66.7 and nn = 63.0) differed significantly ($P < 0.05$ for meat depth, $P < 0.001$ for fat thickness and percentage predicted lean yield) between nn and NN as well as Nn, with no differences between NN and Nn, ruling out the incorporation of this gene and its proposed advantages. Correlations between drip loss and days to slaughter (growth phase) ($r = 0.47$, $P < 0.001$) and ADG ($r = -0.38$, $P < 0.05$) were significant.

The processed meat (back bacon) only showed significant ($P < 0.05$) differences between Nn and nn for percentage pumped yield (NN = 9.7, Nn = 8.1 and nn = 12.6). Percentage moisture loss differed ($P < 0.001$) significantly between nn and NN as well as Nn, with no differences between NN and Nn (NN = 2.5, Nn = 1.2 and nn = 7.7). There were no differences in means for percentage bacon yield (NN = 7.2, Nn = 6.9 and nn = 4.8). The chemical composition of the analyzed samples showed significant ($P < 0.05$) differences between NN and nn for percentage protein (NN = 72.4, Nn = 71.6 and nn = 69.1) and sodium concentration ($mg.kg^{-1}$ DM) (NN = 12096, Nn = 12477 and nn = 13446). Percentage moisture differed ($P < 0.05$) between Nn and NN as well as nn (NN = 50.2, Nn = 44.8 and nn = 49.4). Percentage fat in the samples did not differ significantly (NN = 5.9, Nn = 5.7 and nn = 7.6). None of the multiple regressions calculated were sufficiently accurate to be of any use as a predictive model.

The incorporation of the halothane gene in pig production under South African production and processing conditions seems to have no real benefits for the

producer, processor and consumer alike and the exclusion thereof in breeding programs is strongly recommended.

OPSOMMING

Die doel van hierdie studie was om die invloed van die halotaangeen in varke op sekere produksie-, karkas-, vleis- en geprosesseerde vleiseienskappe te bepaal.

Nege-en-vyftig (soggies = 25, burgies = 34) Landras x Grootwit varke van die drie halotaangenotipes (NN = 31, Nn = 17, nn = 11) is onder kommersiële produksietoestande gehuisves en grootgemaak vanaf 27 kg tot 86 kg lewendige gewig. Veranderlikes gemeet tydens die groeifase is dae tot slagting en gemiddelde daaglikse toename (GDT). Met voltooiing van die groeifase (86 kg) is die varke na die naaste slagpale geneem, geslag en geklassifiseer volgens fabrieksprosedures. Die volgende veranderlikes is by die klassifikasiepunt gemeet: pH_1, karkaslengte, warm karkasmassa, vetdikte, oogspierdeursnit en die persentasie voorspelde maervleis in die karkas. Na verloop van 'n 24 uur verkoelingsperiode is die karkasse geweeg, pH_{24} gemeet en opgesny volgens fabriekspesifikasies. Monsters is vanaf die lende verwyder om dripverlies te bepaal. Dele van die karkas (linkerkantse ham en lende) is verwyder, ontbeen en gevries vir verdere verwerking.

Nadat al die varke geslag is, is die hamme en lendes ontvries. Die lendes is gebruik vir kommersiële spekvervaardiging volgens fabriekspesifikasies. Hierdie proses sluit in pekelinspuiting, dompeling in 'n pekelbad vir 24 uur, beroking en tempering of afkoeling. Al die lendes (n = 59) is voor en na elke proses geweeg. Die ontbeende hamme is volgens genotipe gegroepeer en deur 'n 20 mm plaat gemaal. Speserye, preserveermiddels en yswater is by die gemaalde vleis gevoeg en vir 30 minute getuimel om die waterbindingsvermoë van die vleis te verbeter. Die hammengsels is volgens genotipe gegroepeer en geblik (20 blikke/genotipe), geweeg en gesteriliseer. Na 'n afkoelingsperiode is die hamme uit die blikke gehaal, oortollige water gedreineer en elke blik se inhoud geweeg om sodoende kookverliese te bepaal.

Monsters is van elke lende (n = 59) geneem om die chemiese samestelling daarvan te bepaal. Die monsters is gevriesdroog om voginhoud te bepaal. Ontleding op die gedroogde monsters is gedoen om proteïeninhoud (Kjeldahl), vetinhoud (eterekstraksie) en natriumkonsentrasie (chromatografie) te bepaal. Elke veranderlike is ontleed as 'n tweerigtingklassifikasie model en die metode van kleinste kwadrate is gebruik om verskille tussen gemiddeldes te beraam. Korrelasies tussen veranderlikes asook veelvuldige lineëre regressies vir sekere afhanklike veranderlikes is bereken. Interaksie tussen geslag en genotipe is vir sekere veranderlikes (dae tot slag, GDT, karkaslengte) geïgnoreer vanweë die

onveranderbaarheid van die burg:sog verhouding of vanweë die lae betekenisvolheid van geslag in die model (persentasie spekopbrengs).

Die teenwoordigheid van die halotaangeen in homosigotiese vorm (nn) het aanleiding gegee tot verbeterde produksieprestasies (dae tot slagting, GDT). Die nn varke het minder ($P < 0.05$) dae geneem om slagmassa te bereik as die NN- en Nn varke (NN = 81.2, Nn = 84.6 en nn = 74.1). Geen betekenisvolle verskille vir GDT is tussen genotipes waargeneem nie (NN = 0.729, Nn = 0.710 en nn = 0.765).

Meeste van die vleis- en karkaskwaliteitseienskappe het nie enige betekenisvolle verskille vir persentasie koelverlies (NN = 4.4, Nn = 4.7 en nn = 5.2) en pH_{24} (NN = 5.87, Nn = 5.81 en nn = 5.80) gehad nie. Die korrelasies tussen pH_1 en pH_{24} ($r = 0.27$, $P < 0.05$) en dripverlies ($r = -0.39$, $P < 0.05$) was statisties betekenisvol. Persentasie dripverlies het betekenisvol ($P < 0.05$) verskil tussen al drie genotipes (NN = 1.69, Nn = 2.39 en nn = 1.06). Vetdikte (NN = 20.0, Nn = 18.8 en nn = 26.1), oogspierdeursnit (NN = 50.9, Nn = 52.4 en nn = 46.8) en persentasie beraamde maervleis in die karkas (NN = 66.1, Nn = 66.7 en nn = 63.0) het betekenisvol verskil ($P < 0.05$ vir oogspierdeursnit, $P < 0.001$ vir vetdikte en persentasie beraamde maervleis in die karkas) tussen nn en NN sowel as Nn, met geen verskille tussen NN en Nn. Hierdie resultate wys daarop dat die insluiting van die geen en die voorgestelde voordele daaraan verbonde nie aanbeveel word nie. Korrelasies tussen dripverlies en dae tot slagting ($r = 0.47$, $P < 0.001$) asook GDT ($r = -0.38$, $P < 0.05$) was statisties betekenisvol.

Die verwerkde vleis (rugspek) het slegs betekenisvolle verskille ($P < 0.05$) tussen Nn en nn gehad vir persentasie gepompde opbrengs (NN = 9.7, Nn = 8.1 en nn = 12.6). Die persentasie vogverlies het betekenisvol verskil ($P < 0.001$) tussen nn en NN sowel as Nn, met geen verskille tussen NN en Nn (NN = 2.5, Nn = 1.2 en nn = 7.7). Daar was geen verskille tussen genotipes vir persentasie spekopbrengs (NN = 7.2, Nn = 6.9 en nn = 4.8). Die chemiese samestelling van die monsters het betekenisvol verskil ($P < 0.05$) tussen NN en nn vir persentasie proteïeninhoud (NN = 72.4, Nn = 71.6 en nn = 69.1) en vir natriumkonsentrasie (mg. kg^{-1}) (NN = 12096, Nn = 12477 en nn = 13446). Die persentasie vog het betekenisvol verskil ($P < 0.05$) tussen Nn en NN sowel as nn (NN = 50.2, Nn = 44.8 en nn = 49.4). Die persentasie vet in die monsters het nie verskil nie (NN = 5.9, Nn = 5.7 en nn = 7.6). Nie een van die berekende veelvuldige linieëre regressies was akkuraat genoeg om as voorspellingsmodel te gebruik nie.

Dit blyk dat die insluiting van die halotaangeen in varkproduksie onder Suid-Afrikaanse produksie- en verwerkingstoestande geen werklike voordele vir die

produsent, verwerker of verbruiker het nie en die uitsluiting daarvan in die teelprogram word sterk aanbeveel.

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LIST OF ABBREVIATIONS.

ADG	average daily gain ($g.d^{-1}$).
ADP	adenosine diphosphate.
AMP	adenosine monophosphate.
ATP	adenosine triphosphate.
%BY	percentage bacon yield.
Ca^{2+}	Calcium.
cDNA	carrier deoxiribonucleic acid.
CL	carcass length (cm).
%CHL	percentage chilling loss.
Cp	Mallow's coefficient
CV	coefficient of variation.
DAYS	days to slaughter.
DFD	dry firm dark.
%DL	percentage drip loss.
DM	dry mass.
%F	percentage fat in sample.
FCR	food conversion ratio.
FT	fat thickness over loin area (mm).
Hal+	halothane positive phenotype (nn genotype).
Hal-	halothane negative phenotype (NN + Nn genotypes).
HGP	Henessey Grading Probe.
LMP	lean meat percentage.

M	mole.
%M	persentage moisture in sample.
MD	depth of *M. longissimus thoracis* (mm).
MH	Malignant Hyperthermia.
Mg^{2+}	Magnesium.
%ML	percentage moisture loss after pumping.
MLT	*M. longissimus thoracis*.
MS	mean square.
Na^{+}	sodium content of sample ($mg.kg^{1}$).
NN	homozygous halothane negative genotype.
Nn	heterozygous halothane negative genotype.
nn	homozygous halothane positive genotype.
p	parameters used in regression analysis.
P	Probability.
%P	percentage protein in sample.
PCR	polymerase chain reaction.
pH_1	pH measured after 1 h.
pH_{24}	pH measured after 24 h.
pH_{45}	pH measured after 45 min.
pH_i	initial pH.
pH_u	ultimate pH.
PSE	pale, soft and exudative.
PSS	Porcine Stress Syndrome.

pST	porcine somatotropin.
%PY	percentage pumped yield.
SS	sum of squares.
WCM	warm carcass mass (kg).
WHC	water holding capacity.

CONTENTS

INTRODUCTION

A genetic mutation in pigs, known as Malignant Hyperthermia (MH) has certain beneficial and detrimental effects on growth, carcass, meat and processed meat quality characteristics. These effects are discussed in detail, and where appropriate, in context with the present study.

MH is a hypermetabolic myopathy which is triggered in genetically susceptible humans and animals when exposed to anesthetics such as halothane gas (2-bromo-2 chloro-1,1,1-trifluoroethane) (Gronert & Theye, 1976) or by depolarizing muscle relaxants such as succinylcholine (Harrison *et al.*, 1969). In pigs it is also referred to as Porcine Stress Syndrome (PSS) (Topel, 1968) and is characterized by death induced by natural stressors such as transport, high ambient temperature, exercise, fighting, service and parturition (Patterson & Allen, 1972), as well as anesthesia (Hall *et al.*, 1966).

In recent years the volume of literature published on MH has grown considerably. There are several reasons for this:

Firstly, MH affects production performance, carcass characteristics and meat quality of pork. Phenotypic comparisons of breeds provided strong evidence that the halothane gene (which, in a resessive homozygous form, may trigger MH) has certain beneficial effects on economically important carcass traits (Webb *et al.*, 1982). Webb & Simpson (1986) found that MH positive British Landrace pigs, as defined by halothane reaction, showed significant advantages, when compared to MH free pigs, regarding food conversion ratio (FCR), eye muscle area, proportion lean in sample joints and visual conformation. This was accompanied by disadvantages in meat colour, incidence of pale, soft and exudative (PSE) pork and post weaning mortality. A link between MH and PSE pork was established by Harrison *et al.* (1969) and Nelson *et al.* (1974). PSE pork, resulting from the combination of a low initial pH (pH_1) and high temperature within the early postmortem muscle (Bendall, 1973; Wismer-Pedersen & Briskey, 1961 and McLoughlin, 1963) is a well known condition in the pig meat industry. High losses suffered during cooking of PSE muscle makes this muscle type undesirable for meat processors (Briskey, 1964). Historically, PSE was the first of the porcine stress syndromes to be described in a substantial report by Ludvigsen (1957). However, the difficulties associated with PSE in the preparation of sausages in Germany has been experienced for many years before this date (Wismer-Pedersen, 1959a).

Secondly, pigs are used as a model system for research in human malignant hyperthermia syndrome studies. Reports of pigs receiving or subjected to halothane and sux-amethonium anesthesia (Hall *et al.*, 1966; Harrison *et al.*, 1968) showed similarities to descriptions of the human malignant hyperthermia syndrome (Stephen, 1967; Wilson *et al.*, 1967). Similarities such as these have led to the assumption that the human and porcine syndromes are identical (Britt & Kalow, 1970). However, this assumption has been questioned by Heffron & Mitchell (1975).

Thirdly, another stimulus for research with regard to the malignant hyperthermia gene, and the effect it has in various guises [homozygous (NN) and heterozygous (Nn) non reactors, and the homozygous (nn) reactors], was the development of a non invasive diagnostic test to determine the genotype of the pig with regard to MH (Fujii *et al.*, 1991). This test is based on the assumption that a single point mutation in the porcine gene for the skeletal ryanodyne receptor (*ryr 1*) is the causal factor for MH. A correlation between the single point mutation of the *ryr 1* gene and MH was found in five major breeds of lean, heavily muscled pigs (Pietrain, Yorkshire, Landrace, Poland China and Duroc). This suggests that the mutation either has a common source for all these breeds or that it has been a recurring mutation selected for in each of these breeds. On the basis of further tests done to determine if the mutation is associated with a specific haplotype in all the breeds, Fujii *et al.* (1991) reported that a common ancestry for all the MH animals in all five pig breeds is indicated.

The *ryr 1* gene has been localized to a specific area on the relevant human chromosome and linked to the mutation causing MH in humans (MacLennan *et al.*, 1990). The porcine MH locus (Davies *et al.*, 1988) and the *ryr 1* gene (Harbitz *et al.*, 1990) was localized to a region that relates to the abovementioned human chromosome, suggesting linkage between porcine MH and the mutated *ryr 1* gene. In humans, MH is usually inherited in an autosomal dominant fashion, but in pigs with a similar phenotype, inheritance of the disease is autosomal recessive or co-dominant. The syndrome which occurs in one out of 50 000 anesthetized adult humans (Fujii *et al.*, 1991), can cause neurological, liver or kidney damage and is frequently fatal.

1. THE GENERAL STRUCTURE AND COMPOSITION OF MAMMALIAN SKELETAL MUSCLE

Mammalian muscles are classified into smooth, cardiac and skeletal muscle. Meat is mainly derived from skeletal muscle and will as such be the main focus of this review.

Because of the demands placed on it, skeletal muscle show a great variation in shape and composition as well as colour differences, ranging from red through pink to white (Davey & Winger, 1988). The colour differences are largely due to the varying concentrations of myoglobin in the muscle. Muscles with a high proportion of red muscle fibres are classified as "red" muscles, and those with a high proportion of white muscle fibres as "white" muscles. The red muscles are usually slow-acting and can sustain activity for extended periods of time whereas white muscles tend to be fast acting and are easily fatigued (for more detail see chapter 2).

The muscle is a highly ordered and complex composition of various structures. The muscle consists of bundles of muscle cells, called fibre bundles, which in turn can be seperated into successively smaller bundles, each bundle being surrounded by a connective tissue sheath. Surrounding the muscle is the outer sheath or epimesium, the bundles of muscle cells (fibre bundles) are in turn surrounded by the perimesium and the individual muscle cells are enveloped by the endomesium. The endomesium coveres a triple layered cell membrane or sarcolemma of approximately 10 nm thickness. Approximately 80 % of the muscle cell volume is made up of threadlike contractile cylinders, called myofibrils. These myofibrils are surrounded by the sarcoplasm which contains cell components such as the nuclei, responsible for cell differentiation and morphology; the sarcoplasmic reticulum, responsible for nervous impulse transmission; the mitochondria, responsible for oxidative metabolism; the supporting cytoskeletal framework and the glycogen granules which serve as a source of metabolic energy reserve.

At low magnification cross striations are seen in longitudinal sections of the skeletal muscle. With the use of phase contrast microscopy these striations appear as a repeating sequence of light and dark bands, called I and A bands, respectively. The I bands are bisected by structures called Z discs whereas the A bands are bisected by lighter areas called H zones. The H zones are in turn also bisected by dark M lines and N_2 lines can be seen crossing the I bands. The contractile unit, called a sarcomere, is bound at each end by a Z disc.

Morphologically the sarcomere consists of two sets of filaments (thick and thin filaments) running parallel to the muscle (Squire, 1981). The central part of the sarcomere is occupied by the thick filaments, 12 nm in diameter and 1500 nm in length. The thick filaments consist of myosin molecules which are in a side by side formation connected at the tail end. These filaments have regularly spaced cross bridges along their total length. These cross bridges consisting of the heads of myosin molecules. This is also the site for the interaction with ATP and the actin molecules, which are the basic components of the thin filaments. The thin filaments are arranged in a structure resembling a four stranded rope. The two major strands are called F actin and consist of a double winding of G actin molecules. The remaining two strands are called tropomyosin and lie in the helical grooves of the F actin strands. The tropomyosin strands are beaded at intervals with a troponin complex. The thick and thin filaments form a three dimensional lattice which is hexagonal in shape. This shape consist of six thin filaments surrounding a thick filament and each thin filament is in turn surrounded by three thick filaments (Fig. 1.1).

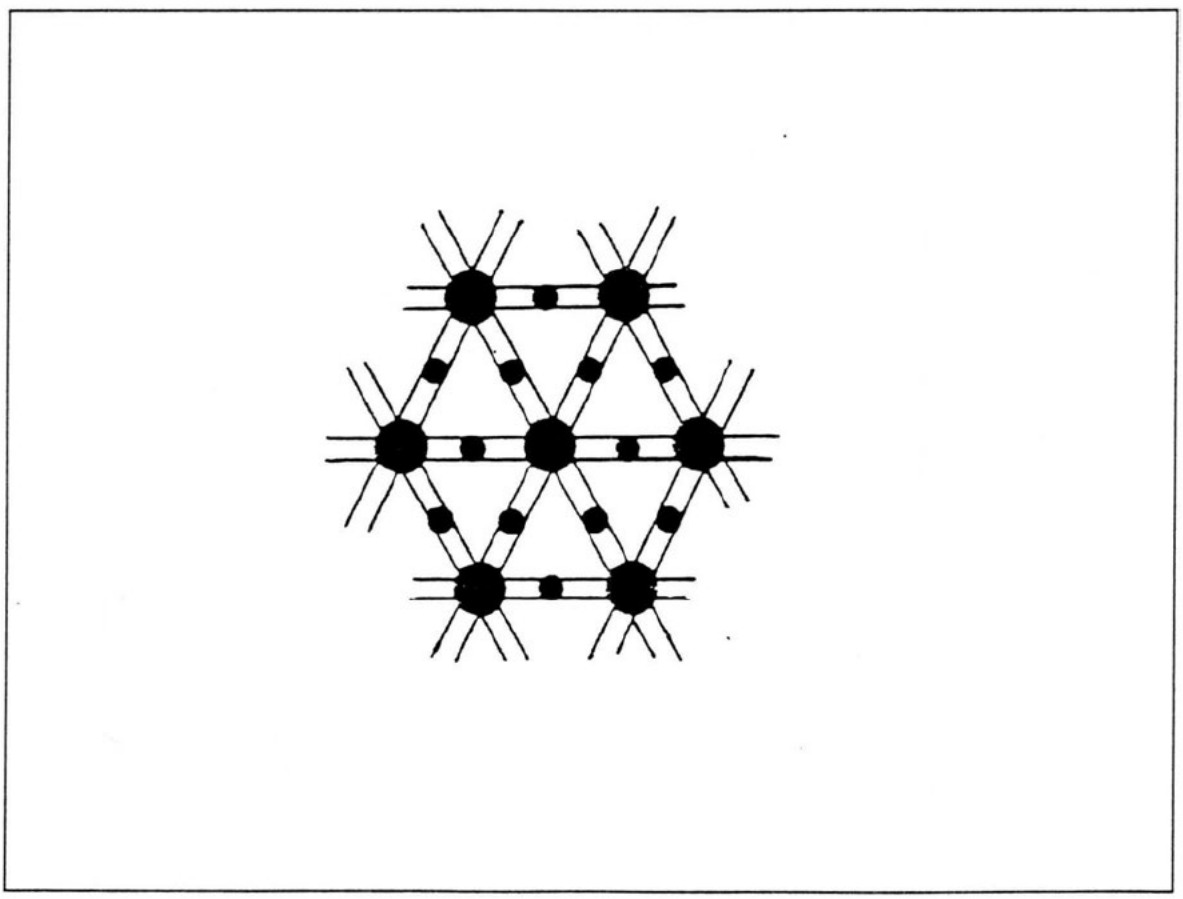

Figure 1.1 Diagrammatic cross-section of myofibril in region of M zone, showing myosin filaments within a network of M filaments and M bridges (Lawrie, 1984).

2. CLASSIFICATION OF SKELETAL MUSCLE AND SKELETAL MUSCLE FIBRE TYPES

Lorenzini (1678) (as cited by Pearson & Young, 1989) is generally credited with recognizing the differences in the colour intensity of muscles. The association between colour intensity and speed of contraction was made by Ranvier (1874) who observed that the red muscles contracted slower but sustained contraction longer than white muscles. Needham (1926) and Denny-Brown (1929) pointed out that, when histologically examined, muscles are a mixture of red and white fibres. The apparent differences in colour were ascribed to a preponderance of the red or the white fibres present in the muscle. They also recognized that some fibres were intermediate in type, showing an approximately equal distribution of red and white fibre characteristics. Dubrowitz and Pearse (1960a, b) showed that the activity of certain enzymes in the muscle, such as phosphorylase and various dehydrogenase, can be used as a basis for classifying muscle fibre types.

2.1. Methods of classification and identification of skeletal muscle

Muscle types are generally identified on the fibre types present in the muscle that gives the muscle its characteristic appearance or physiological behaviour.

2.1.1. Appearance of the muscle

Differentiation between muscle types on the basis of colour enjoy widespread use because of the ease of of application (Peachy & Franzini-Armstrong, 1983). A widely used example of red muscle is the *M. masseter* of ruminants, responsible for the chewing action. The *M. pectoralis* of the chicken has been studied as an example of white muscle. Certain muscles are also characterized by the seperate white and red areas in the same muscle, such as the *M. semitendinosus* of the pig and the *M. lattisimus dorsi* of the chicken.

2.1.2. Physiological behaviour of the muscle

The difference in speed of contraction have been and is still used to differentiate between muscle types (Martonosi & Beeler, 1983). The difference in speed of contraction is used to classify muscles as fast twitch or slow twitch muscle types. Resistance to fatigue is also used to classify muscles and can be combined with speed of contraction. According to Goldspink (1983) muscles can be classified as tonic or phasic, with phasic muscles subdivided into (a) slow twitch oxidative (SO or type I), (b) fast twitch glycolytic (FG, FF or type IIB) and (c) fast twitch oxidative (FO, FR or type IIA). Tonic muscles contract very slowly and when activated by a single impulse, produce no significant response. Tonic fibres have a very low specific

activity for myosin ATPase, which is probably responsible for their ability to develop and maintain isometric tension with relatively little expenditure of energy. Goldspink (1983) cited the anterior *M. latissimus dorsi* of birds as a good example of a tonic skeletal muscle.

Phasic muscles are more widely distributed in the vertebrate skeletal muscle and are more diverse in their reactions and speed of contraction than tonic muscles. The abovementioned classification of phasic muscles is based on speed of contraction (fast or slow) and their predominant reaction (oxidative or glycolytic) which depends on relative blood supply, number of mitochondria and speed of hydrolyzing ATP.

2.1.2.a Slow twitch oxidative muscle

The slow twitch oxidative muscles maintains posture and carry out slow, repetitive movements. They are slow to respond to a contracting stimulus, and are efficient and economical in executing these functions. They contain large numbers of mitochondria and, because of a slow rate of ATP hydrolization, are resistant to fatigue (Goldspink, 1983). The *M. masseter* muscle of the ruminant is a good example of this muscle type.

2.1.2.b Fast twitch glycolytic muscle

Fast twitch glycolytic muscles have a rapid response to a single stimulus. These muscles are used for rapid movement and have a high speed of contracting with a high ATPase spesific activity. ATP levels are high and few mitochondria are present. The muscles have an inability to replenish energy stores (ATP and CP) during contraction and thus fatigue easily. ATP and CP are restored to normal levels upon cessation of activity. The *M. pectoralis* (breast muscle) of the chicken is an example of a fast twitch glycolytic muscle.

2.1.2.c Fast twitch oxidative muscle

Since the fast twitch oxidative muscles are adapted for rapid, repetitive movements these muscles are the first used, followed by the contraction of slow twitch oxidative muscles. Rate of contraction is similar to fast twitch glycolytic, but with a slower rate of contracting. It contains larger numbers of mitochondria than fast twitch glycolytic muscles and are thus less subject to fatigue and can recover faster from the effects of exercise. The red portion of the *M. vastus* muscle of the rat is classified as a fast twitch oxidative muscle (Saltin & Golnick, 1983).

2.2 Methods of classification and identification of skeletal muscle fibre types

Since all muscles contain a mixture of fibre types, classification based on fibre type is much more precise. However, fibre typing is not always clear cut and a certain amount of diversity does exist. Histochemical staining is one of the more frequently used methods to differentiate between fibre types, with several procedures needed to varify a specific fibre type. A review of the commonly used methods to classify fibre types was published by Cassens & Cooper (1971).

Table 2.1 Fibre types grouped according to physical, chemical or morphological characteristics.

Colour	Twitch	ATPase	Energy supp.	Mitochondria
Red	slow	I	oxidative	plentifull
	fast	IIA	oxidative	intermediate
White	fast	IIB	glycolytic	few

Colour is frequently used to identify different fibre types as described by Moody & Cassens (1968), in order to differentiate between muscle fibres with a preponderance for oxidative (red fibres), glycolytic (white fibres) or a combination of oxidative and glycolytic (intermediate fibres) metabolic activity.

Engel (1962) and Robbins *et al.* (1969) classified muscle fibres as type I or type II based on their reactivity to myofibrillar ATPase. Type I is high in mitochondrial enzymes (NADH-TR and SDH) and low in myofibrillar ATPase and phosphorylase (Cassens & Cooper, 1971). Type I fibres are thus similar or parallel to red fibres based on physical appearance and histochemical reactions. Stein & Padykula (1962) classified muscle fibres into three types (A, B & C) based on staining with SDH, ATPase and nonspesific esterase. Types B and C represents two types of red fibres. Using the hind limb of the rat, Yellin (1969) measured responses to SDH and phosphorylase staining. Type B fibres exibited moderate oxidative and low glycolytic activity, whereas type C showed high oxidative and moderate glycolytic activities. The system developed by Peter *et al.* (1972) classifies fibres into three types based on speed of contraction, glycolytic and oxidative capacity. On the basis of these criteria they classify fibres as fast twitch glycolytic, fast twitch oxidative and slow twitch oxidative.

2.2.1 Red fibres (slow twitch, high oxidative activity)

To identify red fibres oxidative stains such as NADH-tetrazolium reductase (NADH-TR) or succinate dehydrogenase (SDH) are used. Red fibres tend to be smaller in size, contain more myoglobin and lipid, and have more mitochondria than white fibres.There is a more abundant blood supply to red fibres and they contain more RNA, which explains their ability to synthesize more protein than white fibres. Red fibres have a higher calcium content than white fibres which is probably related to the larger number of mitochondria present therein.

Young & Bass (1984) examined the effects of castration on bovine muscle fibre types, using myofibrillar ATPase staining to classify the fibres as types I, IIA and IIB. In the MLT muscle of steers, type IIB fibres comprized 32 % of the total compared to 8% for bulls. No significant differences where observed between bulls and steers for type I fibres and no type IIB fibres were observed in the *M. splenius* muscle of bulls and rarely in steers. The effect of castration on muscle fibre types in bovine seem minor, with diversity between breeds being more evident (Young & Bass, 1984). Comparing sexes, Young & Foote (1984), showed that the relative areas of type I and IIA fibres in the *splenius* muscle of bovine females were more like steers than bulls, whereas type IIB in the *M. longissimus thoracis* (MLT) muscle was intermediate between steers and bulls. Young & Foote (1984) suggested that a link may exist between a high proportion of type IIB fibres and the incidence of dark cutters.

2.2.2 White fibres (fast twitch, high glycolytic activity)

Compared to red fibres, white fibres have narrower Z lines, contract and relax more rapidly and are less fatigue resistant (Gauthier, 1970; Saltin & Golnick, 1983; Wiles *et al.*, 1979; Burke *et al.*, 1971). White fibres have a smaller blood supply (Romanul, 1965) than red fibres, but have more abundant and better developed SR, which is apparently related to their faster rates of contraction and relaxation (Schiaffino *et al.*, 1970).

Based on mitochondrial activity (Engel, 1962; Robbins *et al.*, 1969) white fibres are roughly equivalent to type II fibres, which have a low mitochondrial enzyme activity and exhibit high myofibrillar ATPase and phosphorylase activity (Cassens & Cooper, 1971). Peter *et al.* (1972), using myofibrillar ATPase staining techniques, classified white muscle fibres as type IIB or fast twitch glycolytic fibres. Young & Foote (1984) suggested that a link may exist between a high proportion of type IIB fibres and the incidence of dark cutting beef. Solomon *et al.* (1985) compared fibre types of the MLT muscle of intact male water buffaloes and domestic beef bulls. All types of

muscle fibre were present in the beef bulls, with an absence of type IIB in the water buffalo. Solomon *et al.* (1985) suggested that artificial selection for beef traits have led to an increased incidence of glycolytic fibres, whereas the preponderance of type I and IIA fibres in water buffalo were attributed to natural selection.

2.2.3 Intermediate fibres(intermediate twitch, oxidative & glycolytic activity)

Intermediate fibres have a contraction rate intermediate between red (slow) and white (fast) fibres, and display both oxidative and glycolytic activity (Dubrowitz & Pearse, 1960a).

The classification system used by Engel (1962) and Robbins *et al.* (1969) failed to recognize intermediate fibres, reacting to both glycolytic and oxidative stains. Peter *et al.* (1972) classified intermediate fibres as fast twitch oxidative glycolytic or type IIA, based on speed of contraction, glycolytic and oxidative capacity.

Young & Bass (1984) noted that type IIA fibres occupied a significantly greater proportion of the total area than type I fibres in bulls, which was suggested as being associated with increased serum concentrations of androgens. Salviati *et al.* (1982) also identified type IIC fibres in rabbit muscle, using the peptide mapping procedure of Cleveland *et al.* (1977). Type IIC is an intermediate fibre showing the coexistence of fast twitch and slow twitch isoforms of myosin light chains and troponin subunits (myosin light chains are molecules that range in size from 14 000 to 20 000 daltons, compared to myosin heavy chains approximately 220 000 daltons in size). Type IIC is thus a variant of type IIA, differing in type of myosin light chains and troponin subunits.

2.2.4 Muscle fibre characteristics as influenced by physical activity

Frequently used muscles, or those used for maintaining posture, have a higher proportion of type I (slow twitch oxidative) fibres compared to muscles that are seldom used (Kiessling & Hansson, 1983). In comparison with other mammalian muscles, pig muscles in general have a lower percentage of type I fibres and is therefore naturally lighter in colour.

Hanson *et al.* (1991) and Essén-Gustavsson *et al.* (1988) showed that moderate exercise increased oxidative capacity in pigs, but with little improvement in meat colour or WHC. They conclude that much heavier exercise may be necessary to produce desirable effects on carcass lean:fat composition and meat quality, however, this was not a commercially realistic approach. Similarly, Warris *et al.* (1985) found no improvement in colour and WHC in pigs from free range versus intensive production systems.

The adaptation in muscle that takes place due to physical activity appears only in those muscles that are involved in the exercise. At low work intensities, type I and IIA fibres are recruited, while type IIB fibres are used for high work intensities. Thus, both physical activity and inactivity are important factors that influence adaptations in muscles and metabolic profiles (Essén-Gustavsson, 1992).

2.2.5 Effect of breed and different muscles on fibre characteristics

Within and between pig breeds the muscles shows marked differences in fibre type composition, fibre areas (size) and metabolic profiles (Rahelic & Puac, 1981; Monin *et al.*, 1987).

The MLT has a large proportion of type IIB fibres (80 - 90 %) while *M. vastus intermedius* has a large proportion of type I fibres (70 - 80 %). A characteristic readily observed in porcine muscles is the clustering of red (type I) and white (type IIA &IIB) fibres into groups. This is in contrast to the more commonly found checkerboard mode seen in other species (Cassens & Cooper, 1971). The oxidative capacity can vary markedly within all the fibre types in a specific muscle. Type IIB fibres in the vicinity of type I and type IIA fibres display a higher oxidative capacity compared with type IIB fibres close to the fascicular border. The metabolic profile can differ in *M. gluteus* and MLT of different breeds of pigs (Hampshire, Yorkshire, Swedish Landrace) which nevertheless have similar fibre type compositions (Essén-Gustavsson & Fjelkner-Modig, 1985).

Wild pigs have, in comparison with domestic pigs, a higher oxidative capacity as well as a higher fibre ratio of type IIA to type IIB (Solomon & West, 1985). Wild and cross bred pigs also have a higher oxidative capacity with regard to type I and IIA fibres, when compared to domestic pure bred pigs. Fibre type composition and cappilary:fibre ratio are similar in domesticated stress susceptible and stress resistant pig breeds, but on cross section fibre areas were shown to be larger and capillarization (cap.mm^{-2}) lower in the former (Cooper *et al.*, 1969; Essén-Gustavsson *et al.*, 1992).

2.2.6 Giant fibres

Giant fibres are particularly prevalent in the musculature of stress susceptible pigs and found predominantly in the border of fascicles among type II fibres (Cooper *et al.*, 1969). They have low amylophosphorylase and high ATPase activities and are thus biochemically intermediate between red and white fibres (Lawrie, 1984). Many giant fibres and glycogen depleted fibres were found in MLT of pigs that had been transported over a distance of 25 or 70 km before slaughter, when compared with

pigs that had been housed and fed at the abattoir for 5 days (Klosowska & Klosowski, 1985).

Administration of porcine somatotropin (pST) has been shown to result in an increased fibre area for all fibre types in MLT (Solomon *et al.*, 1990). Giant fibres were observed in some of the pST treated pigs.

2.2.7 Summary

From the foregoing review it is clear that although PSE is commonly related to a stressfull environment prior to slaughter, muscle fibre type may also play an important role in the colour of pork. Various factors have been implicated in fibre type, including breed, sex, adaptation and stress. PSE develops as a result of a rapid post mortem pH decline, to values below 6 in 45 min post mortem. To minimize these factors in the present study, all pigs were obtained from the same cross breed and kept under the same environmental conditions.

2.3 Muscular contraction

Muscular contraction in the living animal is characterized by the sliding action of the thick and thin filaments of the sarcomere (Squire, 1981). Contraction in the muscle is initiated by nerve stimulation of the muscle cell membrane which causes depolarization. The depolarization is dispersed through the transverse tubule (T-tubule) system of the muscle fibre and induces depolarization of the sarcoplasmic reticulum (SR). This causes the release of Ca^{2+} from the SR into the sarcoplasma and the free Ca^{2+} concentration rises from approximately 5×10^{8} M to 10^{-5} M. This triggers the cyclical cross bridging between thick and thin filaments in the region of the overlap of the thick and thin filaments (Smellie, 1974). The troponin complex on the tropomyosin strands receive the calcium ions and facilitate the movement of these strands toward the centre of the F-actin helix. This gives the swivelling myosin heads access to the actin. ATP, bound at the sites on the myosin, is then dephosphorylated and the released energy used for the relative sliding of the thick and thin filaments. This process will continue as long as sufficient ATP is supplied, or until the SR retrieves the released Ca^{2+}.

3 DEVELOPMENT OF RIGOR MORTIS AND THE ASSOCIATED CHEMICAL AND PHYSICAL CHANGES WITHIN POSTMORTEM MUSCLE

Upon slaughtering the musculature of the animal is still biochemically active and remain so, often for many hours. This activity will remain until rigor mortis is finally and irreversibly established. The physical and biochemical processes and changes have been extensively reviewed by Bendall (1979).

3.1 Chemical changes in the living muscle

Pyruvic acid is oxidized to CO_2 and H_2O by way of acetyl-CoA entering the citric acid cycle and its associated phosphorylation. In living muscle a total of 12 mol ATP is formed for every mole acetate utilized in the cycle. However, after death the residual oxygen becomes depleted and this reaction, which is aerobic, ceases. Thus, glycogen and high energy compounds are utilized in an attempt to maintain normal muscle cell activity. These compounds are discussed on the basis of their levels in the muscle at the time of death and the consequences of their breakdown following death.

1 ATP - ADP + P_i

2 creatine-P + ADP - creatine + ATP

3 **Under aerobic conditions:**

$(glycogen)_n$ + 9ADP + 9P_i - $(glycogen)_{n-1}$ + 9ATP + 2 pyruvate --->

2 pyruvate + 30ADP + 3P_i - 6CO_2 + 30ATP

4 **Under anaerobic conditions:**

$(glycogen)_n$ + 3ADP + 3P_i - $(glycogen)_{n-1}$ + 3ATP + 2 pyruvate --->

2 pyruvate - 2 lactate

5 2ADP - ATP + AMP

6 AMP - IMP + NH_3

Figure 3.1 Chemical changes in muscle during aerobic and anaerobic metabolism (Barton-Gade *et al.*, 1988).

3.1.1 High-energy phosphate compounds and their intermediates

The concentration of ATP in the resting, living muscle ranges from 5.7-8.1 mol/g (Bendall, 1973), depending on the species or the muscle involved. In resting muscle, ATP is slowly dephosphorylated to ADP, producing free energy for various metabolic requirements. This process of dephosphorylation is continued after slaughter and ATP levels drops. This drop is caused by the less efficient system of ATP resynthesis via anaerobic metabolism, which cannot keep pace with the rate of

ATP depletion. For example, ATP levels in beef muscle drop to about 17 % of its initial (live) value at 48 h postmortem (Bodwell *et al.*, 1965). Thus, after an initial delay, the ATP concentration level is lowered and the muscle enters a rapid phase of rigor development, which continues until all the ATP is depleted. Following the depletion of the ATP, actin-myosin cross bridges are completely formed and the muscle thus enters a state of inextensibility or rigor mortis.

During the early delayed phase of rigor development, ATP is resynthesized through the transfer of phosphate from creatine phosphate and, to a lesser degree, through glycolysis. During this phase the thick and thin filaments slide past each other with the resultant extensibility and contraction upon stimulation. This situation remains as long as the creatine phosphate stores last and ATP can be resynthesized. If the creatine phosphate stores are however depleted, resynthesis of ATP depends largely on glycolysis, which signals the second or rapid stage of rigor development.

This phase is characterized by a drop in ATP concentration in the muscle which cannot be sufficiently replenished by glycolysis. Cross bridges between actin and myosin form in the region of the overlap between the thick and thin filaments. The ADP, which is the product of the ATP dephosphorylation, is converted to AMP and ATP by the phosphotransferase-enzyme myokinase. For every 2 molecules of ADP used, one molecule AMP and ATP each are formed. The AMP is then irreversibly deaminated to inosine monophosphate and ammonia.

3.1.2 Glycogen

Glycogen is used in the skeletal muscle as the substrate for glycolysis. Glycogen normally comprizes about 1 % of the muscle weight according to Lawrie (1979). Pig muscle shows a rapid depletion of glycogen, reaching low levels within 3 - 5 hours after death (Sayre *et al.*, 1963a, b; Beecher *et al.*, 1965a, b; Bodwell *et al.*, 1966). The amount of glycogen remaining when the muscle goes into rigor mortis falls in the range of 2 - 30 % of resting levels and depends on the original resting concentration and the rate at which phosphorylase and the other glycolytic enzymes become inactive and are no longer capable of catabolizing glycogen to glucose and lactic acid. The development of a low pH as a result of lactic acid production during anaerobic glycolysis is a major factor in the inactivation of phosphorylase and the other glycolytic enzymes. Thus muscles that undergo rapid glycolysis may have as high a level of residual glycogen as those that hydrolyze it more slowly, since the enzyme systems may be inactivated earlier due to the rapid drop in pH associated with the early production of lactic acid.

3.1.3 pH changes in the postmortem muscle

Resting pH values vary between muscles and species of farm animals from between 7.08 to 7.30 (Bendall, 1973). Bate-Smith (1948) reported a normal pH value of 7.4 for living muscle. He also indicated that bicarbonate gives a buffering effect on CO_2 release and breakdown of CP with P*i* production, resulting in a nett pH of about 7.6 for living muscle.

Stress associated with slaughter may cause a marked drop in the pH of unanesthetized muscle. Beef muscle pH falls to the range of 6.9 - 7.0 within 10 min postmortem, but decline to about 5.5 - 5.6 at 48 h postmortem (Bodwell *et al.*, 1965). Cassens & Newbold (1966) reported similar results. Pearson *et al.* (1973a, b) reported mutton muscle pH values of about 6.9 - 7.0 in less than 30 min postmortem. Marsh & Thompson (1958) reported similar results for lamb muscle 30 - 40 min postmortem. Hallund & Bendall (1965) reported pork muscle pH at 10 - 15 min postmortem to be 6.6 - 6.8. These results suggest that pH for the MLT muscle declines curvilinearly from an initial value until its final 48 h pH of about 5.4 - 5.7 (Greaser, 1986), with pork muscles showing an inherent faster pH decline than that of beef and mutton.

Various factors may influence and alter the usual pattern of change in pH, such as exhaustion, stress causing situations and prerigor muscle stimulation. The distance of sampling from the muscle surface may influence pH readings, as Tarrant (1981) indicated that beef semimembranosus muscle requires 24 - 48 h to reach an ultimate pH at 1.5 cm from the surface, but needs only 12 and 6 h at 6 and 8 cm, respectively.

Species and type of muscle also have an effect on postmortem pH. Lawrie (1979) reported that the drop in muscle pH is faster in pork MLT when compared to beef MLT, which is intermediate, and slowest in horsemeat MLT. The most notable differences occur during the early postmortem period (first 3 h). Different muscles from the same species also have different postmortem curves (Lawrie, 1979). For example, The *M. psoas*, *M. semimembranosus* and MLT have distinctly different curves. The MLT and *M. semimembranosus* reached pH 5.8 at 3.7 and 5.4 h, respectively, with the *M. psoas* reaching pH 5.8 at 2.5 h postmortem.

The pH drop is accelerated by mincing or grinding (Newbold & Scopes, 1971). Grinded lamb muscle resulted in a decrease from pH 6.82 to 5.63 within 6 h, compared to a pH of 6.46 for the unground controls (Pearson *et al.*, 1973b). The addition of calcium and epinephrine accelerated the pH drop over that of mincing alone, although the decline appeared to be associated with some mechanism other than the conversion of phosphorylase *b* (inactive) to phosphorylase *a* (active).

Newbold & Small (1985), in accordance with these results, found that conversion of phosphorylase from the *b* to *a* form is only transitory in nature and probably does not significantly contribute to the rapid pH decline that results from electrical stimulation.

3.1.4 The effect of stress on postmortem glycolysis

Preslaughter stress greatly affects the levels of high-energy phosphates in the muscle. The condition of the pig with regard to PSE, PSS and DFD are related to preslaughter stress and is a consequence of the alteration in the levels of high energy phosphates and their resultant metabolites (Briskey, 1964; Sybesma & Eikelenboom, 1978). Fast glycolyzing muscles are present in pig and bovine muscles, although less frequent in bovine than in pig muscles (Fischer & Hamm, 1980).

Results by Fischer & Hamm (1980) demonstrated that ATP and CP levels in fast glycolyzing muscles arise from a combination of rapid glycolysis and glycolysis occuring earlier postmortem than in normal muscle. Both these compounds were present in the fast glycolyzing muscles at lower initial concentrations but after 1 h postmortem, CP was almost absent and ATP levels were very low, indicating a rapid utilization of the high energy phosphates. The lower initial pH values were associated with lower muscle glycogen levels, lower WHC, paler colour and higher concentrations of lactic acid when compared to normal muscles. The effects on beef muscles are however less severe than in the case of pork muscles (Fischer & Hamm, 1980).

A clear link between stress, muscle quality and the rate of glycolysis has been shown. Stress greatly accelerates the rate of glycolysis and is responsible for PSE muscle (Briskey *et al.*, 1959a; Briskey, 1964; Sair *et al.*, 1970). Callow (1936) reported that the depletion of glycogen stores, caused by stress, was a contributing factor to the development of spoilage of bacon, due to the high ultimate pH and reduced shelf life.

3.1.5 The effect of temperature on postmortem glycolysis

Postmortem glycolysis is significantly affected by muscle temperature (Marsh, 1954). High postmortem temperatures cause an accelerated drop in pH (used as an indicator of glycolysis) whereas lower temperatures retards the rate of glycolysis. Results from Bodwell *et al.* (1966) showed that pork MLT held at -29°C for 3 h resulted in a pH decline from 6.54 to 6.24, whereas MLT held at 38°C for 3 h rapidly declined to pH 5.48. Thus, high postmortem temperatures caused an accelerated glycolysis, whereas low temperatures delayed the glycolytic effect. Full rigor, at both temperatures (23°C and 38°C), were reached after 48 h postmortem.

3.2 Physical changes in postmortem muscle

At the time of death skeletal muscles are flaccid and highly extensible. However, within a few hours it becomes inextensible and relatively rigid resulting in the condition known as rigor mortis. The time of onset of rigor mortis can be influenced by a number of factors such as temperature or struggling at the time of death. Higher temperatures and struggling speeds up glycolysis and hasten the onset of rigor mortis. Stimulation of the respiratory system accelerates aerobic metabolism and delays rigor development (Lawrie, 1979). Lawrie (1979) reported that thin strips of muscle exposed to the atmosphere produces ATP so efficiently that rigor mortis can be delayed and CP resynthesized to levels above its original concentration.

3.2.1 Effect of rigor mortis on meat tenderness

Early work done on the effect of rigor mortis on meat tenderness resulted in conflicting conclusions. Paul *et al.* (1944) showed that delayed cooking after slaughter resulted in more tender meat than cooking immediately after slaughter. Ramsbottom *et al.* (1945) reported that beef steaks fried in deep fat were maximally tender if cooked immediately after slaughter and increased in toughness on delaying cooking for 24 - 48 h. Paul *et al.* (1952) reported that steaks fried in deep fat within 1 h of death had the lowest shear force values and reached maximum shear force readings 24 - 48 h postmortem, after which tenderness increased on subsequent holding. Large roasts cooked at 63°C (internal temperature) less than 1 hour after death were the toughest when cooked immediately, with decreasing shear force values as cooking was delayed. These results suggest that prerigor meat is tender, and, if cooked in thin pieces at high heat so that rapid heat penetration is achieved, it is even more tender, as suggested by Ramsbottom *et al.* (1945). With large roasts, heat penetration is delayed and the muscles go into rigor mortis before cooking is completed, resulting in tough meat.

4 PORCINE STRESS SYNDROME (PSS) AND MALIGNANT HYPERTHERMIA (MH)

Sudden stress induced death; pale, soft and exudative (PSE) meat and halothane induced malignant hyperthermia (MH) are manifestations of the porcine stress syndrome (Mitchell & Heffron, 1982; Louis *et al.*, 1990). The search for the basic biochemical lesion in PSS had an early breakthrough in 1966 when it was discovered that PSS was very similar to MH (Hall *et al.*, 1966).

Deaths due to MH arise from uncontrolled skeletal muscle contractions with attendant hypermetabolic and hyperthermic reactions. It occurs frequently at market weight (± 100 kg) and after severe physical exertion (Vögeli *et al.*, 1992). MH is also triggered by anesthetics such as halothane (2-bromo-2-chloro, 1,1,1-trifluoroethane) and by depolarizing muscle relaxants such as succinylcholine (Mitchell & Heffron, 1982). An impending episode of MH is usually preceded by the onset of muscle rigidity or muscle stiffness, which is indistinguishable from rigor. This occurs within 1 - 4 min. of initiation of halothane anesthesia (Harrison *et al.*, 1968) and is followed by a depletion of muscle ATP and a drop in muscle pH. Both are established causes of rigor (Szent-Györgi, 1944). Since the muscle rigidity could not be ascribed to rigor, Britt & Kalow (1970) put forward the theory that the rigidity is caused by sustained high levels of cytoplasmic Ca^{2+}.

4.1 Aetiology of MH

Pearson & Young (1989) reviewed hormonal changes associated with MH and the subsequent effect on certain metabolic pathways. Epinephrine and norepinephrine are released from the adrenal medulla and stimulates glycogen breakdown in both the liver and muscles to release glucose and lactate, respectively. This is brought about by cyclic AMP binding to protein kinase receptors to yield the active phosphorylase kinase, that in the presence of Ca^{2+} catalyzes phosphorylation of phosphorylase *b* to the active *a* form. This speeds up the transformation of glycogen to glucose or lactate, as the case may be. In stressfull situations anaerobic metabolism is used to help meet ATP demands by the muscles. This results in the formation of lactic acid, which is favoured by the action of epinephrine, causing a buildup of tissue acidity. White muscles are unable to metabolize lactic acid, and buildup will continue unless it is transported to the liver, where it can be reconverted into glycogen. High tissue temperatures and failure of the muscles to reduce acidity are contributing factors to the MH condition and, if left uncontrolled, can lead to death due to generalized acidosis and/or abnormally high body temperatures.

The catecholamines also stimulate fat mobilization, yielding free fatty acids in the blood. Wood *et al.* (1977) showed that fat mobilization can be stimulated by noradrenaline more readily in Pietrains (stress prone) than in Large White pigs. The catecholamines also accelerates heart rate and increases smooth muscle tone, thus increasing blood pressure. Hall *et al.* (1977) reported that blocking the α- but not the ß-adrenergic pathways of the sympathetic nervous system (SNS) can prevent the development of MH in Pietrain pigs, and on occasion, α-adrenergic stimulation can induce it. Moss (1987) showed that a further link between the SNS, catecholamines and metabolism was established in the PSE condition by the raised rate of utilization of thyroxin, released to increase metabolic rate and releasing more energy during periods of stress.

4.2 PSS, MH and Ca^{2+} regulation in the muscle

The main sources of Ca^{2+} in the muscle are the extracellular fluids, sarcolemma, mitochondria and the sarcoplasmic reticulum (SR). During MH the Ca^{2+} levels initially increase (Berman *et al.*, 1970) and the fact that the sarcolemma is normally impermeable to extracellular Ca^{2+} suggest an outward movement of Ca^{2+} into the extracellular space rather than the reverse (Britt, 1979). As the sarcolemma and mitochondria contain 10 % each of the cellular Ca^{2+}, their contribution in raising the myoplasmic Ca^{2+} concentration must be small. Since 80 % of all the Ca^{2+} in skeletal muscle is found in the SR (Sulakhe *et al.*, 1973), by implication the SR should be the site of the basic lesion.

The Ca^{2+} release channel (ryanodine receptor) is a large protein that spans the gap between the T-tubules and the SR. This channel is activated by ATP, Ca^{2+}, caffeine, halothane and ryanodine (a plant alkaloid). Inhibition is facilitated by ruthenium red, tetracaine calmodulin, and high magnesium levels (Lai *et al.*, 1988). The Ca^{2+} release channel of the SR from MH positive pigs has a higher affinity for ryanodine binding than normal SR and requires a higher concentration of Ca^{2+} to inhibit ryanodyne binding (Mickelson *et al.*, 1988; Fill *et al.*, 1990).

Contraction of the skeletal muscle in vivo is triggered by the release of Ca^{2+} from the SR after depolarization of the T-tubules (Endo, 1977). Franzini-Armstrong (1970) suggested that the communication between the T-tubules and the SR occurs at the triad junction where arrays of electron dense projections, called 'foot' structures, are interposed. The use of ryanodine, that binds to the Ca^{2+} release channel in junctional SR and modulates its activity, has allowed the purification of the ryanodine receptor (Inui *et al.*, 1987; Lai *et al.*, 1988). The purified receptor protein has been morphologically identified with the "foot" structure (Inui *et al.*,

1987; Saito *et al.*, 1988) and has also been shown to function as a Ca^{2+} release channel when reconstituted into planar lipid bilayers (Imagawa *et al.*, 1987; Lai *et al.*, 1988). The alteration of the MH Ca^{2+} release channel was also observed in these planar lipid bilayers (Fill *et al.*, 1990).

The studies by Mickelson *et al.* (1988) and Fill *et al.* (1990) agrees on the alteration of the Ca^{2+} regulated gating of the MH Ca^{2+} release channel. It is possible that this Ca^{2+} dependent inhibition of Ca^{2+} release may serve *in situ* as a means to close the channel after the signal for Ca^{2+} release is completed. Thus, the abnormality could explain both the increased rate of Ca^{2+} release from the SR as well as the increased muscle twitch force, which could be the result of a greater SR Ca^{2+} release (Gallant *et al.*, 1989; Mickelson *et al.*, 1988).

Comparison of the cDNA of the ryanodine receptor of a Yorkshire and Pietrain pig by MacLennan *et al.* (1990) demonstrated a nucleotide base change (C1843 to T1843) that resulted in the mutation of Arg 615 (normal) to Cys 615 (MH) (Fujii *et al.*, 1991). This mutation is highly correlated with MH in five major breeds of heavily muscled pigs (Fujii *et al.*, 1991; Otsu *et al.*, 1991). Haplotyping suggests that the mutation in all five breeds has a common origin (Fujii *et al.*, 1991). The skeletal muscle ryanodine (*ryr 1*) gene and the porcine MH locus have been localized to pig chromosome 6p11-q21 suggesting linkage between porcine MH and *ryr 1* (Davies *et al.*, 1988; Harbitz *et al.*, 1990). In contrast to pigs, MH muscles in humans may contain two populations of SR Ca^{2+} release channels and therefore MH in humans does not arise from a single defect (Fill *et al.*, 1991).

5 METHODS USED FOR IDENTIFYING THE MH GENOTYPE IN PIGS

In the past, progeny testing schemes and certain other experiments have had various suggestions as to the genetic basis of porcine MH. These tests suggested a major gene with complete penetrance (Smith & Bampton, 1977), high penetrance (Andresen, 1979a), variable penetrance (Cheah & Cheah, 1979) or incomplete penetrance (Ollivier *et al.*, 1975). A single autosomal gene with complete penetrance (Jones *et al.*, 1972) as well as two different dominant genes have also been suggested as a causative defect. These experiments however indicated that there was a phenotypic variety which varied from three to five different phenotypes. This phenotypic variety was based on results from tests such as postmortem glycolysis rates, response to halothane anesthesia, hormone studies and the activity of different blood enzymes.

5.1 Serological tests

Several enzymes have been measured in an attempt to identify MH susceptible pigs. Certain enzymes, such as lactate dehydrogenase, serum glutamate oxalate transferase and aldolase have been found to show increased levels in pigs which have been under physical stress, as well as during a period of MH (Sybesma & Eikelenboom, 1969; Eikelenboom & Minkema, 1974).

Since all of these enzymes are found in most tissues of the body, an increase in activity in the plasma is not necessarily indicative of a muscle defect. The metabolical changes associated with MH occurs principally in skeletal muscle and there is thus a need for a more specific enzyme marker. Serum creatine phosphokinase (SCPK) is found mainly in the cytoplasm of cardiac and skeletal muscle and is therefore an ideal marker for detecting a muscle defect such as MH (Baskin & Deaner, 1970). The usefullness of SCPK levels as a means for detecting the presence of a muscular disease was considered after it was used to indicate muscle injury (Tammisto & Airaksinen, 1966) and muscle disease (Hess *et al.*, 1964). The use of SCPK levels in determining MH susceptibility in humans seems to have fallen in disrepute because of a low correlation between SCPK levels and MH susceptibility, nonspecific activity and variability (Britt *et al.*, 1976; Ellis *et al.*, 1975).

Virtually all reports indicate that in stress susceptible pigs or pigs in the state of MH, SCPK levels alone as a method for determining a predisposition to MH is not very accurate because of variability (Nelson *et al.*, 1974), overlap between normal and susceptible values and a lack of appreciation of the causes of variation and limitations of the test. Three factors were identified which cause most of the

variability in SCPK activity in the pig (Mitchell & Heffron, 1975a; Heffron & Mitchell, 1975) of which animal age is the most important. Between 11 and 28 weeks of age protein anabolism reaches a peak and is well correlated with maximum SCPK activity. No significant differences between normal and stress susceptible pigs regarding SCPK levels occur and therefore it does not have any diagnostic value. However, before 11 and after 28 weeks clear differences in SCPK levels between the two phenotypes do exist and can be used to differentiate between them. The other two factors are diurnal variations in SCPK activity which can increase by as much as 50 %, as well as muscle activity which can increase enzyme activity fourfold within one hour.

The results from these studies show that the absolute SCPK activity values can only be properly interpreted in conjuntion with knowledge of familial or genetic predisposition to MH.

5.2 Hematological tests

Analysis of blood groups, platelet function and erythrocyte fragility have been implicated in MH susceptibility in pigs (Mitchell & Heffron, 1982). Because of certain similarities between muscles and platelets, tests have been based on certain platelet disfunctions in an effort to link it with muscle disease.

Solomons *et al.* (1978, as cited by Mitchell & Heffron, 1982), found that humans susceptible to MH have abnormal platelet metabolism. Jensen *et al.* (1976) and Andresen (1979b, 1980) found a correlation between the H blood group system and halothane sensitivity, as well as an association between MLT colour and halothane susceptibility (Jensen *et al.*, 1976). Rasmussen & Christian (1976) found that two of the H blood groups (a\a and -\-) do correlate with PSS susceptibility. During MH hemolysis occur with potassium, SCPK and myoglobin escaping into the plasma (Berman *et al.*, 1970). It has been suggested that because of the intracellular fluids escaping into the plasma, MH may be characterized by a membrane defect which is identifiable by increased erythrocyte fragility (Britt & Kalow, 1970).

On the premise that MH can be identified by a defect in the cell membrane, Harrison & Verburg (1973) tested the osmotic fragility of the membranes in sodium chloride solutions and found significant differences between normal and stress susceptible pigs. Tests with sodium chloride ranging between 0.077 - 0.137 M

(King *et al.*, 1976; Cheah & Cheah, 1978) caused a fourfold increase in hemoglobin precipitation from the erythrocytes of susceptible pigs when compared to normal pigs. However, it is not a very specific test as hemolysis does not always occur during MH in pigs (Berman *et al.*, 1970; Nelson *et al.*, 1974). The test for osmotic fragility is not entirely reliable and should be done in conjunction with other diagnostic tests.

5.3 Halothane gas challenge

The use of halothane gas to identify stress susceptible pigs has, until a few years ago, been widely used (Dimarco *et al.*, 1976; King *et al.*, 1976; Eikelenboom & Minkema, 1974; Sybesma & Eikelenboom, 1978).

In a typical test, two to three month old pigs are subjected to the halothane challenge test (3 - 5 % halothane with oxygen through a face mask) for several minutes. Those that develop extensor muscle rigidity during the test are diagnosed as MH susceptible. This method of testing was also popular because it was cheap, quick and the results were immediately available (Lister *et al.*, 1981). However, some problems associated with this method of testing were encountered. Some of the major limitations of this test is its low sensitivity (Webb *et al.*, 1986) and the risk that reactors may die unless proper therapy is applied immediately. This has been an accepted consequence since the first halothane testing has precipitated MH in pigs (Harrison *et al.*, 1968).

Ahern *et al.* (1980) have suggested that if a recovery is made after the halothane challenge, the pigs were not susceptible to MH or were undergoing the initial responses to halothane. The variation in responses to the halothane challenge could be explained by the variation in the concentration of the gas used. However, Berman *et al.* (1970) found that MH, once initiated, will continue independent of the halothane concentration in the tissue. Levels of halothane in oxygen ranging between 1 and 10 % (Mitchell & Heffron, 1975b) have been used and had varying responses. Mitchell & Heffron (1975b) used a 10% halothane concentration with oxygen which gave an 8 min time duration of MH as opposed to a 50 - 70 min time duration for a 1 % halothane concentration (Gronert & Theye, 1976).

However, the correlation between phenotypical and genotypic diagnosis was not very clear due to the lack of a test identifying the heterozygous (Nn) pig, as well as the fact that a low percentage of the Nn pigs do react to the halothane challenge. Webb & Jordan (1978) found that the probability of wrongly predicting genotype with the halothane challenge was 5 ± 1 %. Cheah & Cheah (1979) found this to be higher at

8 - 12 %. On young pigs (3 - 5 weeks) the test appears to be highly inaccurate (Carden & Webb, 1984).

5.4 Halotyping

With the discovery of the mutation responsible for the physiological defect associated with MH (Fujii *et al.*, 1991), a simple diagnostic tool became available for testing on a large scale and clearly identifying all three genotypes associated with MH.

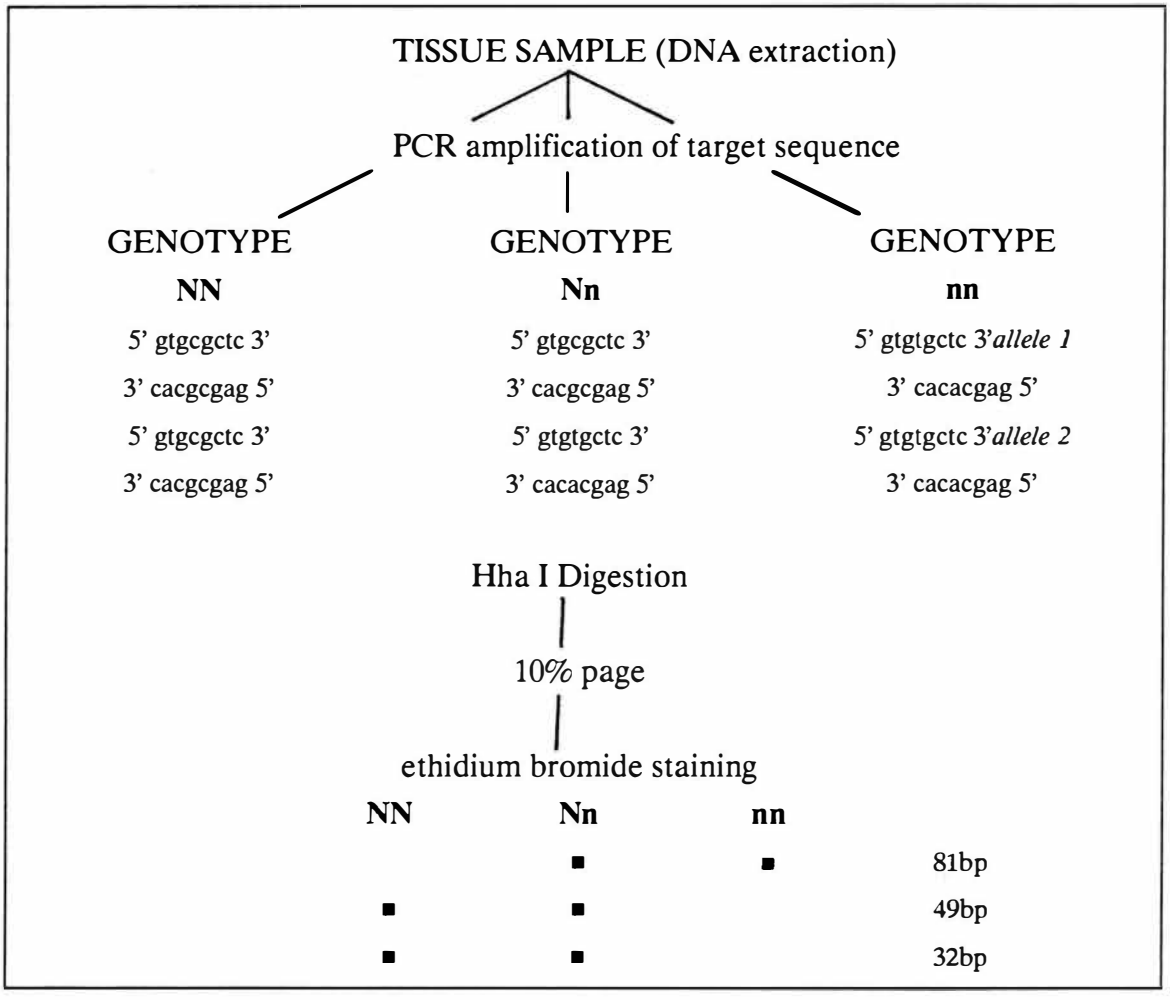

Figure 5.1 Diagram showing the procedure to detect the c/t transition associated with porcine MH. DNA is extracted from the tissue sample and the target region in the ryanodyne receptor gene is amplified by polimerase chain reaction producing an 81 bp fragment. Digestion of this product by Hha I (GCG/C) yields two fragments of 49 and 32 bp for normal animals (NN), three fragments of 49, 32 and 81 bp for heterozygotes (Nn) and only the 81 bp DNA fragment for mutant homozygous individuals (nn) (Houde & Pommier, 1993).

Linkage studies have traced the defect to chromosome 6 (Harbitz *et al.*, 1990) and molecular probes for a gene that codes for a calcium channel in the muscle (ryanodine receptor) has been shown to cosegregate with the halothane gene (MacLennan *et al.*, 1990). The point mutation identified by Fujii *et al.* (1991) consists of a substitution of thymidine for cytosine at position 1843 on the cDNA which leads to the substitution of arginine for cysteine at position 615 on the ryanodine receptor of MH carrier pigs. This transition in the DNA deletes a Hha I restriction site and creates a Hgi AI site, which forms the basis of the test, schematically represented in Fig 5.1.

Using polymerase chain reactions (PCR) to amplify the region where the defect occurs, it is possible to demonstrate the presence of the mutation by a restriction endonuclease digestion assay.

6 WATER HOLDING CAPACITY, DARK, FIRM AND DRY AND PALE, SOFT AND EXUDATIVE PORK

6.1 Water Holding Capacity of meat

Skeletal muscle contains about 75 % water, with the myofibrillar proteins playing a dominant role in water binding. Most of the "unbound" water is located in the spaces between the thick filaments of myosin and the thin filaments of actin and tropomyosin. Factors such as pH, sarcomere length, ionic strength, osmotic pressure and pre or post rigor condition of the muscle has an influence on the interfilament space, which can vary between 320 Å and 570 Å (Offer & Trinick, 1983). This difference in interfilament space corresponds to a threefold change in volume and has a much larger influence on the variation of interfilament water content than the approximately 5 % of the total water in the muscle directly bound to hydrophilic groups on the proteins.

The tenacity with which muscle binds water differs from the immobilized or bound water to the free or unbound water. Offer & Trinick (1983) presented evidence that most of the water in the muscle is held by cappilary forces between the thick and thin filaments. Offer & Trinick (1983) explained the water holding capacity (WHC) of myofibrils as being mainly determined by interfilament spacing, which in turn is mainly determined by long range electrostatic forces. Myofibrils exposed to salt (as in curing) swell due to increased negative charges on the filaments as well as the effect of salt on the restraining links. These links consist of attachments of actin filaments to the Z line, attachment of myosin filaments to M line proteins and cross links between actin and myosin filaments. Swelling is thus an association between expansion of the filament lattice and the disassociation of the cross links.

6.1.1 Effect of pH on water holding capacity

WHC is at a minimum around pH 5.0, which corresponds to the isoelectric point of the principal muscle proteins (Lawrie, 1984). At pH 5.0 the nett charge of the myosin and the actomyosin are at a minimum so that WHC is at its lowest.

In the pH range of 5.0 - 6.5 any alteration in pH has a great influence on WHC. This change is related to the ionization states of histidine and, to a lesser extent, glutamic acid (Hamm, 1986). The changes in WHC between 5.0 - 6.5 are reversible but become irreversible below pH 4.5 or above pH 10.0 (Hamm, 1986). Muscle swelling and pH are closely related. Hamm (1986) show that swelling and WHC of beef muscle are minimal near pH 5.0. Addition of acid or base shifts the pH away from the isoelectric point of the muscle proteins, thereby increasing WHC and

interfilament spacing, thus swelling occurs. Differences in WHC between species do occur, for instance the WHC of pork is higher than that of beef. Intramuscular fat content tends to have a higher WHC (Saffle & Bratzler, 1959). This could possibly be due to the intramuscular fat loosening up the microstucture, allowing more water to be retained.

6.1.2 Effect of salt on water binding

Divalent cations such as Ca^{2+} and Mg^{2+} lower the WHC of meat by reducing the electrostatic repulsion between negatively charged groups. This tightens the muscle structure, causing shrinkage (Pearson & Young, 1989). Magnesium salts have a much stronger effect on WHC than sodium salts of the same ionic strength because of the stronger bonding between magnesium and the myofibrillar proteins.

With the use of NaCl and $MgCl^2$, shrinkage does not occur because the chlorine ions superimpose the effect of cations on WHC. Removal of the divalent cations by exchange resins or other methods increase WHC (Pearson & Young, 1989).

Salted pre rigor meat maintains a high WHC for a period of time (a few days) during refrigeration and has superior WHC and fat binding characteristics compared to conventional processes. Pre rigor salting results in accelerated ATP breakdown (Hamm, 1977) and an increased solubilization of the myofibrillar proteins, but does not appear to protect the proteins against loss of solubility. However, WHC remains unchanged although the loss of solubility is similar to that of post rigor salted meat. Pre rigor salted meat also maintains a high WHC during freezing and thawing.

6.2 Dark, firm and dry pork

Dry, firm and dark (DFD) pork is dark in colour, firm in texture and dry to the touch. It does not absorb curing salts readily and is thus more susceptible to spoilage (Newton & Gill, 1981). It is essentially the same condition as dark cutting in beef, being associated with low muscle glycogen reserves at the time of slaughter as well as a high ultimate pH (Pearson & Young, 1989).

The high ultimate pH causes a high rate of surviving activity of the cytochrome enzymes. This, together with the muscle proteins being above their isoelectric point and thus with a higher WHC, causes the layer of bright red oxymyoglobin to disappear and the purple red myoglobin will predominate to such an extent that the meat appears dark (Lawrie, 1984). The incidence of high pH or DFD pork has had a major impact on the production of Wiltshire sides, where salt concentrations may be high enough to prevent microbial spiolage, but produces a fiery colour, sticky

consistency and spoiled flavour, called "glazy" bacon (Lawrie, 1979). Monin *et al.* (1981) has concluded that DFD and PSE pork may be triggered by different mechanisms and that genetic selection may not neccesarily reduce its incidence.

6.3 Pale, soft and exudative pork

Pale, soft and exudative (PSE) pork is characterized by an unusual pale colour, and having soft and watery lean meat along with an open structure (Briskey, 1964). Occasionally PSE muscle also appears in beef, although the incidence is much lower and not considered a serious problem (Hunt & Hedrick, 1977). The mechanism of the development of PSE muscle is closely related to the PSS and DFD mechanisms (Gronert, 1980).

Ludvigsen (1954) first described PSE, however he described it as a dystrophic condition (such as with vitamin E / selenium deficiency) in the muscles of the pig, which is superficially characterized by a pale, exudative appearance of the flesh. Wismer-Pedersen (1959a) reported that this condition was recorded in Germany many years before, although the aetiology was unknown. In Denmark the dystrophy, known as "muskeldegeneration" appeared to be prevalent in pigs of the Landrace breed (Lawrie, 1960). A striking biochemical characteristic of this condition was the low pH measured immediately postmortem; values of 5.4 being attained 30 min after death. It appeared that the MLT was particularly susceptible to this condition (Ludvigsen, 1954).

Blaxter & Wood (1952, as cited by Lawrie, 1960), reported that low potassium and high sodium concentrations were common to white exudative muscles in both pigs and calves. Concomittantly, a higher level of potassium and a lower level of sodium was found in the blood (Ludvigsen, 1954). In contrast, Topel *et al.* (1967) and Briskey *et al.* (1959b) found no consistent differences for either sodium or potassium in several porcine muscles from hams and MLT muscles grouped according to colour morphology. Another feature was the absence of myoglobin in the muscle. However, Blaxter & Wood (1952, as cited by Lawrie, 1960), only mentioned myoglobinuria in the white muscle condition in calves. Biörck (1949, as cited by Lawrie, 1960), suggested that the condition in calves was suggestive of an accute onset which is associated with a vitamin E deficiency. Ludvigsen (1957) concluded from his observations of PSE muscle that hypofunction of the thyroid gland and adrenal cortex appeared to be involved in the development of muscle changes associated with PSE meat.

6.3.1 Causes of pale, soft and exudative pork

Pale, soft and exudative (PSE) pork is the result of very rapid glycolysis postmortem, which causes a decline in pH levels while muscle temperatures are still high (Briskey & Wismer-Pedersen, 1961). The rapid decline in pH results in sarcoplasmic proteins precipitating on myofibrils and thus impedes the uptake of muscle exudate (Bendall *et al.*, 1963). Although pH appears to be the primary factor that determines the severity of the PSE condition, there are many subtleties in this relationship. The most obvious is the temperature x pH interaction, it being normally thought of that a low pH at a high body temperature is more damaging than the same pH attained at a low temperature, e.g. when carcases are chilled rapidly.

Bendall *et al.* (1963) reported that the early postmortem rate of pH decline is about twofold faster for PSE muscle than for normal muscle, about 1.04 pH units.hour ^{1} decline compared to 0.65 units.hour ^{1} for normal muscle at 37°C. Comparison of postmortem concentrations of metabolites show that soon after death the glycogen concentration is much lower in PSE muscle compared to normal muscle (Greaser, 1986). Lactate levels are close to double in PSE muscle, even as early as 15 min postmortem. ATP and CP concentrations are lower in PSE muscle (early postmortem) and are virtually depleted after one hour postmortem. The hexose monophosphate levels tend to be higher in normal muscle during the first three hours postmortem (Fischer & Augustine, 1977).

The completion of rigor requires much less time in PSE muscle when compared to normal muscle and is closely related to the pattern of depletion of ATP (Bendall *et al.*, 1963). Myosin-ATPase is also reduced twofold in PSE muscle, and PSE myofibrils fail to contract on addition of MgATP (Sung *et al.*, 1977). The PSE muscles also have a higher salt soluble and heat lable collagen content than normal muscle at 48 h postmortem (McClain *et al.*, 1967), but this increase is probably due to the rapid pH drop.

Normal muscle has an increased SR ATPase activity postmortem but decreases in PSE muscle (Greaser *et al.*, 1969a). No significant differences between normal and PSE muscles exist with regard to calcium binding activity immediately postmortem, although calcium uptake by the SR decreases much more rapidly in PSE muscle (Greaser *et al.*, 1969a, b). The possibility exists that a reduced calcium binding activity by the SR may trigger a rapid ATP turnover in PSE muscle.

Protein solubility in PSE muscle declines more rapidly than that of normal muscle, and show a rapid pH drop while muscle temperatures are still high. It also has only 50 - 75 % as much soluble sarcoplasmic and myofibrillar protein as normal pig

muscle (Bendall & Wismer-Pedersen, 1962). The sarcoplasmic protein creatine kinase suffers most from denaturation (Scopes & Lawrie, 1963). There is no apparent change in myoglobin solubility of the PSE muscle, indicating that the pale colour results from the light coloured precipitates of the sarcoplasmic proteins masking the usual red colour (Goldspink & McLoughlin, 1964). Bendall (1973) concluded that the increased light scattering originated from denaturation of the sarcoplasmic proteins in PSE muscle.

The softness associated with PSE pork is the least understood aspect of pork quality. It may involve microstructural changes, in addition to changes in fluid distribution, because Swatland (1992) reported that laser diffraction measurements of sarcomere lengths may be correlated with X ray diffraction measurements of filament spacing. In excised pre rigor samples longer sarcomere lengths may be recorded than in samples left intact untill rigor is completed. In such cases there is evidence that PSE muscles may have longer sarcomeres than normal muscles (Eikelenboom & Nanni Costa, 1988). However, this relationship is lost when care is taken in handling the muscle samples and no elongation of the sarcomeres take place. Thus, some of the softness of PSE pork may originate from the lengthening of the sarcomeres when the meat is handled.

7 PRODUCTION PARAMETERS

7.1 Average daily gain, food conversion ratio and days to slaughter

Although it is generally perceived that the halothane gene reduces growth rate (Sather *et al.*, 1991a), there is considerable variation in the results found in the literature (Webb *et al.*, 1982). Some of the variation has been attributed to breed effects at the halothane locus in the populations from the various studies reviewed (Sather *et al.*, 1991a).

Webb & Simpson (1986) found little indication of a halothane phenotype effect on growth rate when halothane positive (Hal+) and halothane negative (Hal-) pigs were compared in growth performance trails. Averaged over different feeding levels (scale and ad lib.) Hal+ pigs showed significant improvement over Hal- pigs with regard to FCR. However, any economic advantages in growth from the H+ pigs were overshadowed by losses due to mortality and PSE. In a comparison of NN and Nn pigs, Pommier *et al.* (1992) reported that days to slaughter were similar for both genotypes and barrows reached market weight significantly ($P < 0.001$) earlier than gilts.

This observation is also reflected in the greater ADG of the barrows. Similar results were achieved by Sather *et al.* (1991a) comparing NN with Nn pigs. ADG during the grower/finisher period were similar for both genotypes with castrates growing faster ($P < 0.007$) and attaining market weight earlier than gilts ($P < 0.045$).

7.2 Transportation and preslaughter environment

7.2.1 Time of last feeding

The type of management that pigs are subjected to during the 24 h period prior to slaughter is known to have a considerable effect on the ultimate quality of their meat (Warris, 1987). Advantages of feed withdrawal prior to slaughter include lower mortality rates during transportation (Guise, 1987), better meat colour and WHC (Warris, 1982; Jones *et al.*, 1985; Eikelenboom *et al.*, 1991), lower carcass weight losses during chilling and cutting (Warris & Brown, 1983; Lopez-Bote & Warris, 1988) and a lower risk of bacterial contamination from the gut during evisceration. Certain disadvantageous conditions associated with feed withdrawal are lower yield of dressed carcases (Jones *et al.*, 1985; Fischer *et al.*, 1988) and a higher incidence of DFD meat (Fischer *et al.*, 1988; Eikelenboom *et al.*, 1991).

The main reason for feed withdrawal prior to slaughtering is to lower the risk of death during transit, especially with adverse weather conditions (hot, humid) and

stress susceptible pig breeds. Loss of carcass yield due to fasting prior to slaughter amounts to about 1 % in the first 24 h without food and can increase to 2 % or more if dehydration occurs during transportation. Jones *et al.* (1988) showed that weight loss due to feed restriction occurred non preferentially from fat and lean portions of the carcass and that these losses were similar for the three genotypes. Murray *et al.* (1989) indicated that within the lean tissue, these weight losses occurred through metabolic effects which had little influence on the proportions of fat, protein and moisture involved in the weight loss.

Evidence suggest that the length of time from the last feeding to time of slaughter should not exceed 18 hours (Warris, 1982) so that the maximal benefits, as outlined above, can be realized, without the resultant loss of economically important traits such as carcass yield which occurs when longer periods of feed withdrawal are implemented.

7.2.2 Environment and transportation

Preslaughter handling has a profound effect on meat quality aspects of pigs, especially those related to PSE and DFD syndromes (Barton-Gade *et al.*, 1988).

Changes in the preslaughter environment can cause PSE or DFD (or both) in stress susceptible pigs, which are relatively insensitive to changes in the environment. Stress resistant pigs will develop PSE meat only after severe short term stress or DFD meat after prolonged stress (Barton-Gade *et al.*, 1988). Nielsen (1981) proposed a simple scheme to illustrate the relationship between genotype, energy reserves and meat quality. The meat quality can only be improved by regulating energy reserves since the genotype of the pig is fixed. Short transportation time and lairage, together with feeding until the day of slaughter, will result in high energy reserves at slaughter, thus increasing PSE frequency and lowering DFD frequency. Longer lairage and transportation, together with feed withdrawal will result in lower energy reserves, thus less PSE and more DFD meat.

Barton (1974) showed that the effect of prolonged preslaughter treatment can be quite dramatic. Transportation in well equipped vehicles with adequate ventilation, partitions and non slip floors, decreased the PSE frequency by 25 % when transportation time was increased from 15 min to 3 h. The DFD frequency increased correspondingly. A very long period of transportation and/or lairage can result in an increase in both PSE and DFD meat in stress susceptible pigs, as some of them can relax and recoup energy losses (Augustini & Fischer, 1981).

Guise & Penny (1989) reported that recommended stocking densities are rarely adhered to and overcrowding is frequently encountered. This can cause fighting with resultant skin damage and a higher incidence of prolapse. Floor area allowed should be 0.5 - 0.6m^2 for pigs of 90 kg live weight and slightly more for heavier pigs (Barton-Gade *et al.*, 1988).

7.2.3 Temperature

Factors affecting body temperature at slaughter are of great interest because of the role of muscle temperature in PSE development and the possibility of measures to control it.

Long & Tarrant (1990), using pigs from a single source, found no differences in body temperatures or MLT temperatures between pigs slaughtered in summer (mean temp. = 19.5°C) or winter (mean temp. = 11°C). Using a wider range of temperatures (12°C to 24°C), Warris (1991) found a correlation between ambient temperature, MLT muscle temperature at 1 h postmortem and drip loss. However, it is unclear whether the observed effects were due to the influence of high ambient temperatures or the cooling rate of the carcass on the killing line. Long & Tarrant (1990) found that cold water showering prior to slaughtering brought about a drop in loin muscle temperature which was sufficient to reduce paleness and drip in loin chops.

8 MEAT QUALITY

8.1 Drip loss

There is evidence that the high drip loss and softness of PSE meat is caused by myosin denaturation, while the paleness and high light scattering properties could be attributed to the denaturation of sarcoplasmic proteins (Staborsvik *et al.*, 1984; Honikel & Kim, 1986; Offer, 1991).

Myofibrils occupy in excess of 80 % of the volume of muscle fibres and *in vivo* they hold, via capillary forces, most of the cellular water in the spaces between the thick and thin filaments of the myofibrillar lattice (Offer & Trinick, 1983). Lateral shrinkage at the onset of rigor causes some drip from normal meat. Offer (1991) suggested the shrinkage is the result of two events associated with rigor in normal meat; (a) the decline in pH and (b) the irreversible attachment of myosin heads to actin at rigor.

Postmortem glycolysis in a typical skeletal muscle will normally proceed to an ultimate pH of about 5.5 and, as this is the isoelectrical point of the principal proteins in muscle, some loss in WHC is an inevitable consequence of death of the animal (Lawrie, 1984). The extent of postmortem pH decline will, therefore, affect the WHC, and the higher the ultimate pH, the less will be the diminution in WHC (Cook *et al.*, 1926; as cited by Lawrie, 1984). An increase in the rate of pH decline postmortem worstens the extent of sarcoplasmic protein denaturation (Bendall & Wismer-Pedersen, 1962). When a fast rate of pH fall postmortem is due to elevated temperatures, the enhanced loss of WHC observed is partly due to increased denaturation of the muscle proteins, and partly to enhanced movement of water into extracellular spaces.

The irreversible attachment of the myosin heads to actin at rigor causes a diminished filament lattice spacing: at the onset of rigor there is 4.4 % decrease in space corresponding to a 9 % volume decrease (Offer, 1991). The denaturation of the doublet heads of the myosin molecules may cause additional shrinkage in PSE meat (a 12 % reduction in the myofibrillar lattice space), resulting in more fluid being expelled from the myofibrillar lattice and thus increasing drip loss (Tarrant, 1992). However, it is not known what proportion of myosin molecules would have to be denatured to allow this additional shrinkage to occur and since between 20 - 50 % has been observed in PSE muscles (Staborsvik *et al.*, 1984; Honikel & Kim, 1986) this theory seems appropriate.

Muscle fibre type also has an influence on meat quality aspects such as pH, reflectance values and drip loss. Karlsson *et al.* (1992) found that the proportion of glycogen depleted type IIB fibres in the MLT muscles of NN pigs were positively correlated to pH at exsanguination and ultimate pH, and negatively correlated to drip loss and reflectance values. Lundström *et al.* (1989) reported that nn pigs had poorer meat quality and a greater content of PSE meat compared to NN pigs, given that all the pigs had a similar fibre type composition and enzyme activity in the muscle. The nn pigs had a larger proportion of glycogen depleted type IIB fibres at slaughter, which was negatively correlated with pH at exsanguination, and positively correlated with drip loss and reflectance values.

Drip loss can be minimized if carcass chilling is quick and efficient, but within the limits necessary to avoid cold shortening (Honikel, 1987; as cited by Tarrant, 1992). Honikel (1987; as cited by Tarrant, 1992) noted that fast chilling reduced drip loss and paleness of PSE muscles when using hot boned pork muscles. He concluded that fast chilling can diminish, but not solve the PSE problem. Offer (1991) pointed out that the benefits of fast chilling are greatest for carcases with slow or intermediate rates of pH decline, where appreciable cooling can take place before the carcases set in rigor, thereby reducing the amount of denatured myosin. To be effective in lessening drip loss, cooling must occur pre rigor, when myosin heads are unattached and susceptible to denaturation.

The results from various studies (Murray *et al.*, 1989; De Smet *et al.*, 1992; Sather *et al.*, 1991a, b; Lundström *et al.*, 1989) seem to indicate that the presence of the halothane gene is accompanied by an increase in drip loss, compared to pigs that do not have the halothane gene. Murray *et al.* (1989) reported that comparison of the three genotypes (NN, Nn and nn) with regard to drip loss from the MLT muscle showed the nn genotype to have a higher percentage drip loss than the NN genotype. Sather *et al.* (1991a) reported that a greater drip loss was observed at both the anterior end ($P = 0.009$) and at the centre ($P = 0.002$) of the loins from the Nn pigs when compared to NN pigs. However, this was one quarter to one half less than the differences previously reported by Murray *et al.* (1989). Lundström *et al.* (1989) reported that drip loss from Nn pigs are intermediate to the homozygous (NN,nn) genotypes, but Murray *et al.* (1989) found that the drip loss from the Nn and nn pigs were similar. In contrast, Barton-Gade (1985) (as cited by Sather *et al.*, 1991a) reported WHC values for Nn and NN pigs that did not differ. Fisher *et al.* (1994) reported no significant differences in chilling loss (expressed as percentage difference between warm and cold carcass mass) between the three genotypes.

8.2 Initial pH

The development of PSE is usually attributed to an increased glycolysis rate postmortem (Wismer-Pedersen, 1959a). In muscles which develop DFD, muscle glycogen is already depleted before slaughter. This leaves less substrate available for postmortem glycolysis and the ultimate pH is higher than normal (Bendall & Swatland, 1988).

When PSE develops in a muscle, pH drops to values lower than 5.8 at 45 min postmortem (Wismer-Pedersen, 1959a). In comparison, normal muscles decrease from a pH of approximately 7.0 in the living state (6.9-7.3) (Bendall, 1973) to pH 5.3 - 5.8 within 24 h postmortem (Wismer-Pedersen, 1959a; Briskey & Wismer-Pedersen, 1961). The combination of a high carcass temperature within one hour postmortem and low pH contributes significantly to protein denaturation (Wismer-Pedersen, 1959a; Honikel & Kim, 1986; Offer, 1991). This contributes to the pale colour and reduced WHC of PSE meat (Martin *et al.*, 1975; Honikel & Kim, 1986; Wismer-Pedersen, 1959a).

Enfält *et al.* (1993) reported that the postmortem pH decline rate is probably somewhat higher in PSE muscles than in normal or DFD muscles. Comparison of PSE ($pH_{45} < 5.8$), normal and DFD MLT (pH ultimate > 5.87), with regard to the rate of pH decline 50 min postmortem, Enfält *et al.* (1993) showed significant differences ($P < 0.05$) between PSE and DFD at pH_i (initial pH at exsanguination) and between PSE and normal, DFD at pH_{45} (45 minutes after exanguination). The development of PSE characteristics in the muscles thus seems to be initiated by a combination of a lower muscle pH already at exanguination due to lactate accumulation before slaughter, and a faster pH decline postmortem.

The rate of pH decline seems to change with time and actual pH level (Bendall *et al.*, 1963). Under a constant temperature of 37°C, Bendall *et al.* (1963) reported that normal muscles had a biphasic decline in pH_1 with a lower rate when $pH \geq 6.5$ (90 min postmortem). PSE muscles however, had a constant rate of pH decline during the first 150 min postmortem. Enfält *et al.* (1993) also reported a linear decline in pH for PSE muscles.

Murray *et al.* (1989) reported a significant difference ($P \leq 0.05$) in pH_{45} between all three genotypes, with NN the highes, nn the lowest and Nn intermediate. Fisher *et al.* (1994) reported similar results with significant differences ($P < 0.001$). Comparison of Hal- and Hal+ pigs (Oliver *et al.*, 1993) showed that pH_{45} in the MLT and the

semimembranosus of the Hal+ pigs were lower ($P < 0.001$) than that of the Hal- pigs.

Cheah *et al.* (1995), using biopsy samples of crossbred (Landrace x Large white) Nn pigs, showed that innate variations in meat quality , as defined by pH_1, do exist, ranging from 43 % normal to 57 % PSE. However, Kauffman *et al.* (1993) concluded that ultimate pork quality can only be reliably assessed after the development of full rigor. They emphasize that pH_{45} can be used successfully when groups of carcases, rather than single carcases, are screened for quality variations when conducting pork quality research.

8.3 Ultimate pH

Preslaughter handling of pigs, which may affect muscle glycogen content and therefore ultimate pH, was shown to be an important factor influencing the incidence of PSE meat (Monin *et al.*, 1981). However, when ante mortem treatments that lower muscle glycogen stores are applied to pigs with a tendency to produce PSE meat, the muscles remain firm in texture, dark in colour and do not develop the expected PSE characteristics (Briskey *et al.*, 1959a; Sayre *et al.*, 1961). Bendall & Wismer-Pedersen (1962) showed that muscles going into rigor mortis at a constant temperature of 37°C always develop PSE meat. Since then it became evident that temperature plays an important role in PSE development, even though muscles exhibit normal rates of pH decline. This also implies that PSE may occur in normal glycolysing muscles under bad chilling conditions.

It has been observed that the adverse effect of halothane sensitivity, which increases the rate of pH decline and therefore PSE incidence, was counterbalanced in the case of Belgian Landrace pigs by frequent high ultimate pH_u (24 hours postmortem) (Sellier *et al.*, 1988). This interaction between muscle glycogen content and PSE incidence is probably due to the fact that a minimum pH must be reached at a given temperature to induce significant protein denaturation. This in turn results in detectable PSE characteristics. Thus, the rapid glycolysis that occurs in PSE muscle must reach a sufficiently low pH before the typical PSE characteristics appear (Fernandez *et al.*, 1994).

Fisher *et al.* (1994) and Murray *et al.* (1989) reported no significant differences in pH_{24} between the three genotypes. Sather *et al.* (1991a) reported no significant diference between NN and Nn in pH_u measured in the MLT. Oliver *et al.* (1993) suggest that to increase fresh pork quality and quantity of manufactured products,

such as dry cured ham, it will suffice to use stress resistant pigs that, in addition, show relatively high levels of intramuscular fat.

9 FAT THICKNESS, MUSCLE DEPTH AND PREDICTED LEAN

Hal+ (nn) pigs are generally assumed to yield more lean meat when compared with Hal- (Nn+NN) pigs, but with an adverse effect on meat quality (rate of pH decline, colour, WHC) and a higher incidence of PSE. Whereas the halothane gene is recessive for stress susceptibility, it appears additive for lean content and certain meat quality criteria (Simpson & Webb, 1989).

9.1 Fat thickness

Comparison of back fat thickness between the three halothane genotypes have resulted in various and often conflicting results. Jones *et al.* (1988) compared back fat depths of the three genotypes (NN, Nn and nn) measured at different locations (C, K, shoulder, mid back, back, loin, lumbar and 3/4th last rib). For the majority of the comparisons the back fat thickness between nn and NN pigs were not significant. Of interest is that the fat depth, of the nn pigs, at the loin area was significantly ($P < 0.05$) more than that of the NN pigs. The Nn pigs tended to have more back fat than either the NN or nn pigs, although few of the comparisons were statistically significant. Sather *et al.* (1991a) compared NN and Nn pigs, with no significant differences in back fat thickness, although the Nn pigs tended to have a lower value. Pommier *et al.* (1992) reported similar results.

Various studies (Eikelenboom *et al.*, 1980a, b; De Smet *et al.*, 1992; Fisher *et al.*, 1994) reported that the Nn genotype had a fat thickness intermediate to the homozygotes (NN, nn), with NN having the highest and nn the lowest values. Comparison of gender (barrows vs. gilts) in studies (Jones *et al.*, 1988; Sather *et al.*, 1991a; Pommier *et al.*, 1992) consistently show that the gilts have significantly lower back fat thickness values at the grading site than the barrows, and thus a greater predicted lean meat yield at similar carcass weights.

Jones *et al.* (1988) reported that although the nn and NN carcases had similar fat thickness measurements, the nn pigs had a lower proportion of carcass fat. Researchers (Jones *et al.*, 1988; Sather *et al.*, 1991a; Pommier *et al.*, 1992) emphasize that a single measurement of fat thickness may not necessarily identify the greater lean meat content of pigs with the halothane gene in homo or heterozygous form.

9.2 Muscle depth and predicted lean meat yield

The percentage predicted lean meat in a pig carcass in South Africa is based on a single measurement with a Hennessy Grading Probe (HGP) or an Intrascope, on a position between the 2nd and 3rd last ribs, 45 mm from the midline while the carcass

is in a hanging position (Government Notice No. R. 1748, 26 June 1992). The percentage meat is calculated, depending on the technique, by means of the following formulae:

HGP: **LMP** = 72.5114-(0.4618 x **FT**) + (0.0547 x **MD**)

Intrascope: **LMP** = 74.4367-(0.4023 x **FT**)

Examining the formula for predicting percentage lean meat (with HGP) in the carcass, it is clear that the fat thickness on the measurement position makes a much larger contribution (approx. 10 times) to predicting lean meat yield than the muscle depth on the same position. Results from Nel *et al.* (1993), comparing NN pigs with Nn and nn (Nn+nn) pigs, indicate that MLT muscle depth at the point of measurement differed significantly ($P < 0.001$) between groups with the Nn+nn group having the higher value. Percentage predicted lean meat was similar for both groups. In concurrence, Fisher *et al.* (1994) reported similar results with no differences observed between the three genotypes. Nel *et al.* (1993) reported that no significant correlation between pH_1 (as predictor of meat quality) and MLT muscle depth or percentage lean meat. The study did however show an interaction between muscle depth and meat colour, and between percentage lean meat and colour.

Results from Jones *et al.* (1988) show that nn carcases had a significantly higher ($P < 0.05$) proportion of lean and a lower proportion of bone, skin and fat than NN carcases. The Nn carcases had a higher value than the NN carcases, but not intermediate to nn and NN carcases. This is not in accordance with other results (Sather & Murray, 1989; Sather *et al.*, 1989; Andresen *et al.*, 1981) that reported expected carcass lean yield from Nn pigs to be approximately equal to the mid value of their parental lines (NN and nn) at final farm weights (90 - 100 kg). Sather *et al.* (1989) reported that differences in predicted lean yield did not differ among genotypes. Sather *et al.* (1991a) and Pommier *et al.* (1992) found no significant differences in lean meat content comparing NN with Nn genotype.

Comparison of sexes show that gilts, compared to barrows, have significantly higher lean depth and less fat thickness at similar carcass weights and thus result in a greater predicted lean yield (Sather *et al.*, 1991a; Jones *et al.*, 1988; Sather *et al.*, 1989; Pommier *et al.*, 1992). Results from various studies (Simpson & Webb, 1989; Andresen *et al.*, 1981; Eikelenboom *et al.*, 1980a; Jones *et al.*, 1988) show that the nn genotype have shorter carcases than NN genotypes, with Nn genotypes showing an

intermediate position. Results from Pommier *et al.* (1992) however show no significant differences in length between NN and Nn genotypes.

10 INFLUENCE OF MEAT QUALITY AND GENOTYPE ON PROCESSED PORK PRODUCTS

10.1 Cured pork products

The overall composition of bacon is one of the most variable pork products with the lean:fat ratio one of its most important quality aspects (Möller *et al.*, 1992).

When curing PSE meat, which has a reduced WHC, the nett gain during curing is less than that of normal meat. However, curing whole sides with intact membranes reduces the amount of fluid loss (Wismer-Pedersen, 1968). Smith & Lesser (1982) reported that PSE carcases yielded 1.6 % less bacon and increased drip loss (1 %) compared to normal carcases. The relationship between PSE and the appearance of cured bacon is not so clear. During curing the added nitrite reacts with the pigments in the meat and converts it to nitrosomyoglobin. Taylor *et al.* (1973) reported that PSE bacon was paler than normal bacon when measured with a reflectometer, but visible colour differences were only seen in two toned *semitendinosus* muscles. However, Warris & Akers (1980) found an inverse relationship between pH_1 and the appearance of Wiltshire bacon; improved bacon quality being associated with lower pH_1 values in the carcass.

Comparison of conventional processing with hot processing in terms of yields or major quality traits of bacon showed no significant advantages or disadvantages. Neel *et al.* (1988) observed no significant change in postmortem treatments such as smokehouse yields or slicing yields. Sensory attributes appear unaffected by time of processing, exept for crispness ratings, which is lower for hot processed bacon (Abu-Baker *et al.*, 1983; Neel *et al.*, 1988).

Results from Carpenter *et al.* (1969) showed a significant positive correlation ($r = 0.60$, $P < 0.05$) between percentage cooling loss with bacon weight and a negative correlation ($r = -0.51$, $P < 0.01$) with percentage seperable lean. Carcass weight had a positive correlation ($r = 0.54$, $P < 0.01$) with percentage cooking loss and age ($r = 0.42$, $P < 0.01$). Cooking loss had a negative correlation ($r = -0.51$, $P < 0.01$) with percentage seperable lean. Saffle & Bratzler (1959) also reported increased percentage cooking losses with increased fatness. Correlation between percentage cooking loss and fatness ($r = -0.49$, $P < 0.01$) (Carpenter *et al.*, 1963).

Nusbaum & Rust (1978) found positive correlations ($r = 0.63$) between percentage pumped yield ($r = 0.63$), percentage final yield ($r = 0.66$) and percentage total residual salt ($r = 0.59$) when correlated with saltiness, as determined by a taste

panel. Motycka *et al.* (1981) reported on various methods of tumbling and injecting brine into bacons. No treatment was consistently superior for brine incorporation, yield or quality. Results showed that the use of only multineedle injection or injection combined with tumbling had significantly higher ($P < 0.01$) percentage final yields, compared to other combinations of injection and tumbling.

10.2 Cooked cured pork products

The condition of the raw material used for ham manufacture is of particular importance since large pieces of muscle is used and the PSE condition has an adverse effect on the process. The reduced WHC of PSE meat leads to an increased percentage gelatinous cookout (% jelly) in the cans due to a higher than normal degree of aggregation of meat proteins, especially for pasteurized, canned hams (Wismer-Pedersen, 1968). PSE hams processed with polyphosphates showed significant improvement in WHC and technological yield (Davis *et al.*, 1975). Honkavaara (1988) compared PSE pork ($pH_1 \leq 5.8$) with non-PSE pork ($5.8 \leq pH_1 \leq 6.4$) in cooked cured ham production. The PSE pork resulted in acceptable yet decreased sensory scores and technological yield at 94.0 % compared to 105.9 % for non-PSE ham. Similar relationships between pH and ham quality was reported by Müller (1991). Increased pH values resulted in higher cooked ham yields, with a concomittant decrease in the amount of juice exudation. The hams with the higher pH levels were also more tender.

In a review by Fernandez & Tornberg (1991) the effect of various glycogen levels in the muscle prior to slaughtering on ultimate pH and processing characteristics were examined. Earlier studies indicated that pigs fed a high sugar diet before slaughtering had a higher weight gain during tank curing and a lower weight loss during maturation, due to the accumulated reducing sugars in the meat. Heat processing of the canned hams caused increased shrinkage in the sugar fed group due to the lower pH. Wismer-Pedersen (1959b) suggested that the effect of sugar on weight loss was associated with the raw meat.

Production of ham from pre rigor meat showed, when compared with conventional processes, similar or increased cooking yield (CY) (Mandigo & Hendrickson, 1966; Van Laack *et al.*, 1989), increased tenderness in cooked hams (Arganosa & Henrickson, 1969; Mullins *et al.*, 1958) and increased cured pigment stability (Parr & Henrickson, 1970). However, at pH values below 5.9 the benefit of pre rigor meat in ham production is greatly reduced (Troeger & Waltersdorf, 1987).

Tumbling and massaging is a method by which mechanical agitation is employed to aid in the rapid difusion of brine (Krause *et al.*, 1978), which promotes the formation of a protein exudate which enhances muscle cohesion (Fukazawa *et al.*, 1961a, b), makes the muscle more pliable, produces fewer voids, provides more efficient retention of brine and improves yield (Torr, 1965). Most researchers attribute the complex binding phenomenon in processed meat products to myofibrillar proteins in general, but Nakayama & Sato (1971) and Samejima *et al.* (1969) reported that myosin and actomyosin contribute to the development of binding. MacFarlane *et al.* (1977) measured binding strength of muscle proteins and their results suggest that myosin exhibits superior binding capabilities to actomyosin in the presence or absence of salt. The heat induced binding (during cooking) between meat pieces is a complex phenomenon, affected by the composition of the exudate and the extent of cellular disruption and breakage which takes place during massaging or tumbling (Siegel *et al.*, 1978).

Gillet *et al.* (1981) showed that increased massaging resulted in an increased distribution of cure ingredients with a resultant more uniform colour. Krause *et al.* (1978) and Ockerman *et al.* (1978) reported similar results for tumbling. Ockerman *et al.* (1978) reported that short term tumbling (30 min) improved muscle cohesion but did not significantly affect yield. Apparently a longer time is required to increase yield when compared to cohesion. Motycka & Bechtel (1983) reported no significant differences in yield when comparing continuous tumbling with intermittent tumbling. Results from Krause *et al.* (1978) suggest that the shorter tumbling periods may result in brine movement and protein extraction sufficient to result in acceptable ham quality. The improvement in yield caused by the development of a surface moisture barrier of exudate requires a longer period of tumbling interrupted with sufficient rest periods to permit the movement of the salt soluble proteins. Chow *et al.* (1986) studied the main effects (electrical stimulation, rigor condition and tumbling) and their interactions on certain quality characteristics of canned pork shoulder. The cooking yield was not influenced by rigor condition, but it increased due to tumbling of pre rigor meat when compared to post rigor meat.

11 MATERIALS AND METHODS

Landrace x Large White crosses (n = 59) of the three genotypes (genotype and sex specified in Table 11.1) were sourced from a stud in the Western Cape, minimizing any possible breed effects. The pigs originated from a population of known genotype, with regard to the halothane gene, which were tested by ADSRI at Irene using the protocol adapted from the method described by Fujii *et al.* (1991).

Table 11.1 Number and sex of pigs in each genotype

	Genotype		
Sex	NN	Nn	nn
Castrates	19	10	5
Gilts	12	7	6
Total	31	17	11

The pigs were housed at the University of Stellenbosch piggery during the growth phase of the experiment. Upon reaching 27 kg live weight the pigs were individually weighed and started the growth phase of the experiment. The growth phase was from 27 kg to 86 kg live weight, similar to the specifications of the National Pig Testing Centre. The pigs were housed in 10 m^2 pens equipped with water nipples and partially slatted floors. Each pen contained no more than 10 pigs at any time. Pigs of similar genotype were penned together. Sexes (castrates and gilts) were mixed. Sexes were not housed seperately as split sex feeding is not commercially practiced in the Western Cape. Feeding was ad lib and the maize based ration contained 16 % crude protein (CP), balanced for amino acid availability. Upon reaching an individual live weight of 86 kg the pigs were weighed and feed withheld for approximately 12 hours prior to slaughtering. This was done to minimize any possible risk of death during transport (see chapter 7.2.1).

The pigs were then transported by road to the local abattoir ± 25 km away. Transportation was done under conditions of minimal stress with no overcrowding or mixing of unfamiliar pigs and during the early morning thus avoiding excessive temperatures. No electrical prodders were used to handle the pigs. Upon arrival at the abattoir the pigs were put in lairage for approximately 2 h prior to slaughtering. Commercial slaughter procedures were followed. This consists of electrical stunning (90 V AC, ear to ear for 3 - 5 sec), sticking within 30 sec, shower scalding for 10 min at 60°C and mechanical removal of the hair.

The initial measurements (pH_1, warm carcass mass, carcass length, percentage lean meat in the carcass) were taken 60 min after sticking and exsanguination. The carcases were then chilled for 24 h at 2°C whereafter additional measurements (pH_{24}, cold carcass mass) were recorded. The carcases were then cut into commercial joints according to factory specifications. The left-hand side hams (without shank) and backs needed for further experimental work were then removed from the processing line.

The ham was removed from the carcass side by sawing in a line perpendicular between the last lumbar and first sacral vertebra. The back was removed from the shoulder at a line perpendicular between the 6th and 7th thoracic vertebra. The belly was removed by sawing along an imaginary line (approximately 18 cm) parallel to the midline. All the carcass joints were then individually wrapped, tagged, fast frozen and kept at -40°C till further processing.

11.1 Production performance values

11.1.1 Days to slaughter

Days to slaughter (DAYS) was calculated as time (in days) to complete the growth phase (27 kg to 86 kg live weight) and recorded for every individual pig in the trail.

11.1.2 Average daily gain

Average daily gain (ADG) was calculated as live weight gained during the growth phase (27 to 86kg live weight), divided by DAYS. The results are expressed as kg live weight gained per day ($kg.d^{-1}$).

11.2 Carcass and meat quality measurements

11.2.1 Percentage predicted lean, fat thickness and muscle depth

For each carcass LMP was estimated with a Hennessy Grading Probe (HGP) on a position between the 2nd and 3rd last rib, 45 mm from the midline (Goverment Notice No. R. 1748, 26 June 1992). Fat thickness at the point of measurement was displayed by the HGP and the muscle depth (of the MLT) was calculated using the appropriate formula since it was not indicated on the monitor during the classification process. Muscle and fat thickness values are expressed in mm.

11.2.2 Carcass length

Individual carcass length (CL) was determined at the classification point after the carcases were split along the midline. The measurements were taken, with the carcases suspended from the hind legs, as the distance (in cm) from the 1st thoracic vertebra to the nearest visible point of the pelvic bone.

11.2.3 pH

The pH values were measured with a glass probe in the MLT on a point horizontal to the HGP incision, 45 mm from the midline. Two measurements per carcass were taken. pH_1 was measured 60 min postmortem at the classification site. pH_{24} was measured 24 h postmortem after the carcases were cooled at 2°C for 24 h. The pH measurements were taken according to the procedure described by Hofmann (1988). The glass probe was calibrated using standard buffers (pH = 7 and pH = 4). The meter was calibrated after every fifth reading and rinsed with distilled water between measurements.

11.2.4 Drip loss

After chilling for 24 h the carcases were separated and the left hand side ham and back removed. A slice, 25 mm thick, was removed from the anterior end of each back at the position of the 6th thoracic vertebra. This sampling position was used because the backs are separated from the shoulder at the 6th/7th thoracic vertebra, according to factory specification. The samples were weighed individually and placed, under atmospheric pressure, in a net enclosed in a plastic bag in such a way that the exudate did not come into contact with the meat sample. The samples were stored for 48 h at 2°C. Thereafter the meat was removed, gently dried with absorbent tissue and weighed. Drip loss is reported as the weight loss as a percentage of the original weight of the sample.

11.2.5 Chilling loss

Chilling loss is expressed as the percentage loss in mass during the 24 h chilling period prior to processing of the carcass. This is calculated as the difference between warm carcass mass (60 min postmortem) and cold carcass mass (24 h postmortem), expressed as a percentage of warm carcass mass.

11.3 Processing procedures

11.3.1 Preparation of cured meat (back bacon)

The subcutaneous fat, skin and bone were removed from the backs, leaving a fat layer of approximately 1 cm. After storage and defrosting individual weights were recorded before further processing. The backs were injected with an 11.6 % bacon brine solution (see Table 11.2) using a Belan multi-needle brine injector. The brine injector was calibrated to pump 20 % by weight at an operating pressure of 2 bar. Individual weights were recorded after brine injection.

The backs were then submersed in a similar brine solution for 24 h, before being hung on smoke racks and dry-smoked (with Meranti wood chips) in a Maurer

smoker for 2 h at 50°C. After smoking the backs were chilled (tempered) for 12 h at 2°C and weighed. The backs were then cut in half perpendicular to the median (spinal column) and a sample was removed for determining moisture, protein, Na^+ and fat content. Three variables were calculated for each back, percentage pumped yield (%PY), percentage moisture loss (%ML) and percentage bacon yield (%BY). The %PY was calculated as the percentage gain in mass after brine injection, %ML as the percentage moisture loss after brine injection and %BY as the percentage nett gain after completion of the process.

Table 11.2 Bacon brine ingredients added as a percentage of back mass

Ingredients	(%)
Phosphate	0.4
NaCl	2.0
$NaNO_2$	0.016
Dextrose	1.0
Ascorbic acid	0.048

Table 11.3 Ham brine ingredients added as a percentage of the meat

Ingredients	(%)
Phosphate	0.9
NaCl	2.5
$NaNO_2$	0.016
Dextrose	0.9
Ascorbic acid	0.048
Water	26.0

11.3.2 Preparation of cooked cured meat (canned ham)

The hams were deboned and all visible skin and subcutaneous fat removed. The top and silver side of each ham was then removed according to factory specifications. The cuts from each genotype (NN, Nn and nn) were then grouped together and minced through a 20 mm mincing plate.

An 8 kg sample of each of the three genotypes was then collected for ham preparation. The minced meat from each sample was put into a tumbler together with the ingredients listed in Table 11.3. No gelatin was added so that any water not bound with the meat after sterilization could easily be separated to accurately

determine actual moisture binding. The meat was then tumbled for 10 min followed by a 10 min rest period. This cycle was repeated three times for each of the three samples (see chapter 10.2).

The tumbled meat of each of the three genotypes was then canned (300g per can, 20 cans per genotype), sealed and sterilized at 124°C in a Rotomat autoclave. Total cycle time in the autoclave was 21 min. After 24 h the cans were opened and the ham removed. The meat of each can was carefully dried with absorbent tissue and weighed. Cooking loss is expressed as the difference in weight after sterilization as a percentage of the weight prior to sterilization.

11.3.3 Chemical analysis

Moisture content was determined by method of freeze drying. The samples removed from the backs were weighed and put into the freeze drier for 72 h at 40°C whereafter the weight of the samples were again determined. Moisture content is expressed as the weight difference after drying as a percentage of the initial (before drying) weight.

Nitrogen content was determined with use of the Kjeldahl system and expressed as the percentage protein in the sample on a dry matter (DM) basis. Fat content was determined with ether extraction and expressed as a percentage of the sample on a DM basis. Na^+ content was spectroscopically determined and expressed as mg Na^+ per kg of the sample on DM basis.

11.4 Statistical analysis

11.4.1 Null hypothesis and test of significance

Analyses of variance were performed on all the variables measured using the General Linear Models (GLM) procedure of SAS (Statistical Analysis System Institute, Inc., 1988). A full model was used to determine the presence of two way interaction. The model was

$$Y_{ij} = \mu + G_i + S_j + GS_{ij} + e_{ij}$$

Y_{ij} = Dependent variable

μ = Overall mean

G_i = Genotype effect

S_j = Sex effect

GS_{ij} = Interaction between genotype and sex

e_{ij} = Residual

Variables that showed sex x genotype interaction are indicated in Table 1, Annex C (bold printed values). Discussion of the results will show the appropriate ANOVA's which were calculated using the mean square value of the sex x genotype interaction as error term to determine F values for the main effects (sex and genotype), thus eliminating or confirming the relative importance of the main effects in the presence of interaction.

The differences between genotypes, phenotypes and sexes were, where appropriate, tested seperately by means of the null hypothesis (H_o), with H_o:$\mu = \mu_o$ and the alternate hypothesis (H_a) being H_a:$\mu \neq \mu_o$. This was done by means of contrast analyses and estimated means (± standard error) are reported in the tables contained in the text, while the significance level for differences between genotypes, phenotypes and sexes are represented in Annex A. Differences between the variables were reported significant if the probability of rejection of H_o < 5%.

11.4.2 Correlation

The correlation coeficient (r) measures the degree of closeness of the linear relationship between two variables (Snedecor & Cochran, 1980). If one variable (e.g. X_1) can be expressed as an exact linear function of another variable (e.g. X_2), then the correlation is +1 or -1, depending on the nature of the relationship, being direct (+1) or inverse (-1). Another property of r is that it is without unit or dimension since the units of its numerator and denominator are both the products of the units in which X_1 and X_2 are measured. SAS determines the true product correlation P (Pearson) and is defined as:

$$P_{xy} = \text{cov}(x,y)/\sqrt{[\text{var}(x)\ \text{var}(y)]}$$

The true correlation is estimated by the sample correlation r (Pearson product moment) and is calculated as:

$$r_{xy} = \Sigma(x_i-x)\ (y_i-y)/\sqrt{[\Sigma x_i-x)^2\ \Sigma(y_i-y)^2]}$$

where x and y are the sample means of X and Y.

The significance level of the correlation is indicated as PROB>|R| (SAS: users guide: Basics, 1988). It is important to note that even if convincing evidence of a association does exist, it does not prove that x is the cause of the variation in y and that evidence of the causality must come from other sources.

11 4 3 Multiple linear regression

Regression (R^2) values were calculated using the SAS RSQUARE procedure. R^2 is the square of the multiple correlation coefficient and, if expressed as a percentage, gives an indication of the percentage variation in the dependent variable as explained by the independent variables. If a model with p parameters is fitted, then

R^2 = 1 - [Residual Sum of Squares/Total Sum of Squares]

It is used as a summary measure of the quality of the fitted model, a higher R^2 indicating a closer regression and thus a more accurate predictive model.

The Cp-statistic (Mallows, 1973) was used for selecting suitable models for predicting the dependent variables. This method uses the full model with all the variables as a base of reference and it makes adjustments for the number of variables included. A possible drawback is the lack of a significance test to compare different subsets. Mallows defines

$Cp = RSS_p/\delta^2 + 2p\text{-}n$

RSS_p = Residual Sum of Squares where only p variables are included in the model.

δ^2 = Residual Mean Square for the full model.

n = number of observations.

If there is no bias in the chosen model then Cp should be on average equal to p. Thus, choosing a suitable model would be to plot Cp against p and choose the simplest model with Cp near to but preferably less than p.

Table 12.1a Pooled statistics of the 59 carcases

Variable	Mean	Std. dev.	Min.	Max.
Days to slaughter	80	9.57	63	106
ADG	0.73	0.075	0.533	0.928
Chilling loss	4.6	1.91	1.43	11.51
Drip loss	1.8	0.931	0.513	4.89
Pumped yield	9.8	5.74	-0.439	31.6
Moisture loss	3.1	4.46	-4.40	22.3
Bacon yield	6.7	6.83	-13.6	27.4
Moisture	48.5	5.15	32.01	58.1
Fat thickness	20.8	4.49	14.4	33.6
Meat depth	50.6	4.82	38.5	62.0
LMP	65.7	2.23	59.1	69.2
Carcass length	76.3	2.04	72.5	80.5
pH_1	5.8	0.328	5.04	6.51
pH_{24}	5.8	0.256	5.41	6.50
Protein content	71.6	4.67	59.3	82.2
Fat content	6.1	2.78	2.19	13.9
Sodium content	12458	1904	5510	14700

Table 12.1b Average values for food convertion ratio (FCR) for each genotype

Characteristic	Genotype NN (n = 31)	Nn (n = 17)	nn (n = 11)
[1]FCR	2.66	2.64	2.80

[1]Since the pigs were housed in groups the mean (without std. error) FCR was only calculated for each genotype and is not discussed in the text.

12 RESULTS AND DISCUSSION

Four (DAYS, ADG, CL, %BY) of the seventeen variables measured showed interaction between sex and genotype. Interaction for the first three variables (DAYS, ADG, CL) were expected. The results of the four interactions will be discussed first before the main effects of the other thirteen variables are discussed. Included in the results and discussion are phenotypic comparisons (Hal- vs Hal+) for all the variables. This was done to compare the results of this study with results from studies done before the advent of PCR technology and subsequent accurate genotype identification.

12.1 Interaction

12.1.1 Days to slaughter

Table 12.2 ANOVA table for days to slaughter (DAYS)

Source	Df	SS	MS	F	Pr>F
Sex	1	1015.69	1015.69	18.31	0.0001
Genotype	2	948.79	474.40	8.55	0.0006
Interaction	2	408.24	204.12	3.68	0.0319
Error	53	2939.17			
Total	58	5311.89			

The results in Table 12.2 indicate significant (P = 0.0319) genotype x sex interaction. Therefore the interaction results are given in Table 12.3, which shows that in both sexes the nn genotype had the least DAYS and the Nn genotype the highest DAYS.

Table 12.3 Days to slaughter (DAYS) means for genotype and sex

	Genotype			
Sex	NN	Nn	nn	WA[2]
C	77.4	78.6	74.6	77.3
G	87.3	93.3	73.7	85.7
WA[1]	81.2	84.6	74.1	

[1] Weighed averages for genotypes
[2] Weighed averages for sex

The presence of the halothane gene caused both faster and slower growth, which is supported by Webb *et al.* (1982), who reported on the variation in results found in literature. In this study the halothane gene only expressed approximately 10 % faster growth in the resessive (nn) form. However, the expected progeny of males and females is 50 % each and currently not alterable for production purposes. The main effect of genotype is thus still an important factor in pork production. The ANOVA for DAYS (Table 12.2) clearly shows that sex is the most important factor (MS = 1015.69), followed by genotype (MS = 474.40) and that the interaction is relatively small (MS = 204.12) in comparison to the main effects. Castrates finished the test approximately 10% faster than the gilts (77.3 vs 85.7 DAYS respectively, Table 12.3), which is supported by results from Pommier *et al.* (1992). Tests for differences in means between sexes were thus significant, but sex cannot be manipulated. Although not statistically justifiable, sex was thus ignored and different tests performed on genotype only, mainly to compare with existing literature.

Table 12.4 Average values (with standard errors) for days to slaughter (DAYS) for different genotypes

Characteristic	Genotype NN (n = 31)	Nn (n = 17)	nn (n = 11)
Days to slaughter (DAYS)	81.2^{a} (±1.62)	84.6^{a} (±2.19)	74.1^{b} (±2.72)

$^{a-b}$ Values in the same row with different superscripts differ ($P < 0.05$)

The results from Table 12.4 show that the nn pigs had a significant ($P < 0.05$) advantage in days to slaughter when compared to both NN and Nn pigs. No significant differences where observed between NN and Nn pigs, although the NN pigs were slaughtered approximately 10 % earlier. This is similar to results from Pommier *et al.* (1992) and Sather *et al.* (1991a). Comparison of the two phenotypes (Hal+ and Hal-) (Table 12.5) indicate that the Hal+ pigs were slaughtered significantly earlier than the Hal- pigs.

Table 12.5 Average values (with standard errors) for days to slaughter (DAYS) for different phenotypes

Characteristic	Phenotype	
	Hal- (n = 48)	Hal+ (n = 11)
Days to slaughter (DAYS)	82.4[a] (±9.83)	74.1[b] (±2.72)

[a-b] Values in the same row with different superscripts differ ($P < 0.05$)

12.1.2 Average daily gain

Table 12.6 ANOVA table for ADG

Source	Df	SS	MS	F	Pr>F
Sex	1	0.056	0.056	13.99	0.0005
Genotype	2	0.028	0.014	3.47	0.0385
Interaction	2	0.033	0.017	4.16	0.0209
Error	53	0.213	0.004		
Total	58	0.330			

A similar trend was observed for ADG, therefore similar procedures were followed as in the case of DAYS. Average daily gain showed significant genotype x sex interaction (Table 12.6).

Table 12.7 ADG means for genotype and sex

Sex	Genotype NN	Nn	nn	WA[2]
C	0.764	0.746	0.747	0.752
G	0.672	0.658	0.780	0.703
WA[1]	0.729	0.710	0.765	

[1] Weighed averages for genotypes
[2] Weighed averages for sex

Results from Table 12.7 show clearly that the presence of the halothane gene only had a positive effect on the nn gilts, which can also be deduced from the Table 12.3

since ADG is calculated as the total mass gained.DAYS ^{1}. The total mass gained was not a constant value and was thus responsible for the difference in probability values (Annex A, Table 1) comparing ADG with DAYS. Since sex is unalterable, the genotype x sex interaction was discounted and the data analyzed with genotype (Table 12.8) as a main effect, similar to DAYS. The results for phenotypic comparison of ADG are presented in Table 12.9.

Table 12.8 Average values (with standard errors) for ADG for different genotypes

Characteristic	Genotype NN (n = 31)	Nn (n = 17)	nn (n = 11)
ADG	0.729 (±0.0123)	0.710 (±0.0180)	0.765 (±0.0224)

The results in Table 12.8 show that the nn pigs had the highest ADG, followed by the NN- and Nn pigs. These differences were not statistically significant at the 5 % level although the differences between the lowest (Nn) and the highest (nn) values approached significance (P = 0.0617). Although the deviation from the null hypothesis was not significant, the small sample size gives only a weak confirmation of the null hypothesis Snedecor & Cochran, 1980). This supported by Runtgren *et al.* (1990), whose results indicate that the nn pigs had the highest ADG compared to the other two genotypes, although none of the differences reached significance (P > 0.05). In accordance with previous studies (Pommier *et al.*, 1992; Sather *et al.*, 1991a; Webb *et al.*, 1982) the results from this study do not indicate any significant differences in ADG between NN and Nn pigs.

Comparison of the two phenotypes (Table 12.9) show that the Hal+ pigs have a higher ADG than the Hal- pigs, although not significant (P = 0.0753) at the 5 % level.

Table 12.9 Average values (with standard errors) for ADG for different phenotypes

Characteristic	Phenotype Hal (n = 48)	Hal+ (n = 11)
ADG	0.723 (±0.080)	0.765 (±0.022)

These results indicate that there is reason to believe that the presence of the halothane gene in the recessive form (nn) has certain limited beneficial effects on growth rate. However, phenotypic studies show that although the Hal+ pigs have an advantage in FCR (Webb *et al.*, 1982; Webb & Simpson, 1986), this advantage does not appear when comparing ADG between the two phenotypes (Webb & Simpson, 1986; Lampo *et al.*, 1985). Lampo *et al.* (1985) also stated that daily growth and food conversion seem to be relatively independent of the halothane gene.

12.1.3 Carcass length

Table 12.10 ANOVA table for carcass length (CL)

Source	Df	SS	MS	F	Pr>F
Sex	1	2.819	2.819	0.76	0.3859
Genotype	2	17.909	8.995	2.43	0.0979
Interaction	2	26.333	13.167	3.57	0.0351
Error	53	195.447	3.688		
Total	58	242.508			

The results in Table 12.10 show significant genotype x sex interaction with no significant main effects for carcass length (CL).

Table 12.11 Carcass length (CL) means for genotype and sex

	Genotype			
Sex	NN	Nn	nn	WA^2
C	76.8	75.8	74.4	76.1
G	76.0	78.0	75.9	76.6
WA^1	76.5	76.7	75.2	

[1] Weighed averages for genotypes
[2] Weighed averages for sex

The averages in Table 12.11 indicate clearly the effect of the halothane gene in homozygous (nn) form, causing a decrease in the average length of the carcases for both gilts and castrates. As the result of the effect that the interaction had on both sexes, it was discounted in calculating averages for genotypes and phenotypes. The same reasons regarding the sex ratios (as for DAYS and ADG) is maintained which propose that, although not significant as a main effect, genotype plays an important

role in pork production and producers may be interested to know what the effect of the gene on CL is. Although deemed not relevant in this study, differences between sexes were not significant.

Carcass length did not differ significantly between genotypes (Table 12.12) although NN vs nn approached significance at the 5 % level with P = 0.0804 and Nn vs nn approached significance with P = 0.0678. Results for phenotypic comparison approached significance (P = 0.0508) with the Hal pigs having the longest carcases (Table 12.13).

Table 12.12 Average values (with standard errors) for carcass length (CL) for different genotypes

Characteristic	Genotype NN (n = 31)	Nn (n = 17)	nn (n = 11)
Carcass length (CL)	76.5 (±0.361)	76.7 (±0.488)	75.2 (±0.606)

Jones *et al.* (1988) reported significant differences in length between all genotypes, with NN- having the longest carcases, nn- the shortest and Nn pigs intermediate length carcases. The results from this study seem consistent with results reported by Webb & Simpson (1986) who reported that Hal- pigs had longer carcases than the Hal+ pigs. Pommier *et al.* (1992) reported no significant difference comparing NN with Nn pigs, although the Nn pigs tended to have longer carcases. Consistent with this study, Pommier *et al.* (1992) reported no significant differences between sexes.

Table 12.13 Average values (with standard errors) for carcass length (CL) for different phenotypes

Characteristic	Phenotype Hal- (n = 48)	Hal+ (n = 11)
Carcass length (CL)	76.6 (±0.98)	75.2 (±0.606)

12.1.4 Percentage bacon yield

Table 12.14 ANOVA table for percentage bacon yield (%BY)

Source	Df	SS	MS	F	Pr>F
Sex	1	45.36	45.36	1.03	0.3159
Genotype	2	35.24	17.62	0.40	0.6735
Interaction	2	280.74	140.37	3.17	0.0500
Error	53	2345.22	44.25		
Total	58	2706.56			

Table 12.14 indicate that %BY variation was caused by genotype x sex interaction (P = 0.0500) and eliminates sex as a factor (P = 0.3159). Furthermore, during the manufacturing of bacon the sex of the pig is disregarded and is generally not traceable.

Table 12.15 Percentage bacon yield (%BY) means for genotype and sex

	Genotype			
Sex	NN	Nn	nn	WA^2
C	6.4	10.3	5.5	7.1
G	8.4	2.0	4.3	4.8
WA^1	7.2	6.9	4.8	

[1] Weighed averages for genotypes
[2] Weighed averages for sex

The genotype x sex interaction is seen in Table 12.15. Due to the fact that the interaction caused both the highest (castrates) and lowest (gilts) %BY value with the halothane gene present, made it impossible to interpret the result. The resultant %BY trend is ascribed to chance, however it is discussed as an observation (including phenotype and sex) in chapter 12.3.1

12.2 Carcass characteristics and meat quality

12.2.1 Chilling and drip loss

The inclusion of the halothane gene seems to cause an increase in percentage chilling loss (%CHL) (Table 12.16), although none of the differences between the genotypes were significant, similar to results from Fisher *et al.* (1994).

Table 12.16 Average values (with standard errors) of carcass characteristics of different genotypes

Characteristic	Genotype NN (n = 31)	Nn (n = 17)	nn (n = 11)
Chilling loss (%CHL)	4.4 (±0.345)	4.7 (±0.466)	5.2 (±0.580)
Drip loss (%DL)	1.69^{a} (±0.148)	2.39^{b} (±0.199)	1.06^{c} (±0.248)
Fat thickness (FT)	20.0^{d} (±0.665)	18.8^{d} (±0.899)	26.1^{e} (±1.12)
Meat depth (MD)	50.9^{a} (±0.807)	52.4^{a} (±1.09)	46.8^{b} (±1.35)
LMP	66.1^{d} (±0.329)	66.7^{d} (±0.445)	63.0^{e} (±0.554)
pH_1	6.05^{a} (±0.051)	5.65^{b} (±0.069)	5.82^{b} (±0.085)
pH_{24}	5.87 (±0.046)	5.81 (±0.063)	5.80 (±0.078)

$^{a-c}$ Values in the same row with different superscripts differ ($P < 0.05$)

$^{d-f}$ Values in the same row with different supescripts differ ($P < 0.001$)

The NN pigs had the lowest value (4.4 %) with nn the highest (5.2 %) and Nn intermediate (4.7 %). However, if a comparison of the coefficient of variation (CV) is made, both NN and Nn pigs had a CV < 10 % (7.8 % and 9.9 % respectively) while the nn pigs had a CV > 10 % (11.1 %). This indicates a small but more varied distribution of %CHL values for the nn pigs.

Table 12.17 Average values (with standard errors) of carcass characteristics of different phenotypes

Characteristic	Phenotype	
	Hal- (n = 48)	Hal+ (n = 11)
Chilling loss (%CHL)	4.5 (±1.79)	5.2 (±0.580)
Drip loss (%DL)	1.93[c] (±0.951)	1.06[d] (±0.248)
Fat thickness (FT)	19.6[c] (±3.19)	26.1[d] (±1.12)
Meat depth (MD)	51.5[a] (±4.43)	46.8[b] (±1.35)
LMP	66.3[c] (±1.60)	63.0[d] (±0.554)
pH_1	5.91 (±0.345)	5.82 (±0.085)
pH_{24}	5.85 (±0.267)	5.80 (±0.078)

[a-b] Values in the same row with different superscripts differ ($P < 0.05$)

[c-d] Values in the same row with different superscripts differ ($P < 0.001$)

The Hal-pigs had a lower %CHL than the Hal+ pigs but not significant. Differences between sexes were not significant (Table 12.18) with no genotype x sex interaction present (Annex A, Table 4).

Table 12.18 Average values (with standard errors) of carcass characteristics of castrates (C) and gilts (G)

Characteristic	Sexes C (n = 34)	G (n = 25)
Chilling loss (%CHL)	4.4 (±0.327)	4.9 (±0.381)
Drip loss (%DL)	1.43[c] (±0.146)	2.23[d] (±0.169)
Fat thickness (FT)	22.4[a] (±0.709)	18.7[b] (±0.826)
Meat depth (MD)	49.8 (±0.818)	51.7 (±0.954)
LMP	64.9[a] (±0.354)	66.7[b] (±0.412)
pH_1	5.94 (±0.056)	5.83 (±0.063)
pH_{24}	5.87 (±0.044)	5.80 (±0.051)

[a-b] Values in the same row with different superscripts differ ($P < 0.05$)

[c-d] Values in the same row with different superscripts differ ($P < 0.001$)

Drip loss (%DL) did show considerable differences ($P < 0.05$) between all the genotypes with Nn the highest (2.39 %), nn the lowest (1.06 %) and NN intermediate (1.69 %). The nn pigs do however, in comparison with the other two genotypes, show a much wider distribution of %DL values with a CV of 23.4 % , compared to a CV < 10% for NN and Nn pigs (8.8 % and 8.3 % respectively).

Phenotypic differences (Table 12.17) show a similar trend with the Hal- pigs having a significant higher ($P < 0.001$) %DL than the Hal+ pigs. A significant difference between sexes was observed (Table 12.18) with the gilts having a significantly higher ($P = 0.0007$) %DL than the castrates.

These results are contrary to most studies (Murray *et al.*, 1989; De Smet *et al.*, 1992; Sather *et al.*, 1991a,b; Lündstrom *et al.*, 1989) which seem to indicate that the presence of the halothane gene causes an increase in %DL. However, Murray *et al.* (1989) reported no significant differences in %DL between Nn and nn pigs, and

Barton-Gade (1985) (as cited by Sather *et al.*, 1991a) reported that WHC values for Nn and NN pigs were not significantly different. In a breed comparison Moss (1985) reported that some carcases classified as non-PSE (pH > 5.9) had a %DL as high as PSE (pH < 5.9) carcases.

If the condition of the carcass is such that it has a high glycolytic potential (GP) (GP = sum of the concentration of glycogen, glucose-6-phosphate, glucose and lactic acid; Monin *et al.*, 1981), a high internal temperature with a slow decrease thereof and a fast rate of glycolysis, it will lead to membrane leakage and protein denaturation, the direct cause of a high %DL (Honikel & Kim, 1986; Honikel, 1985). However, as the differences between pH_1 and pH_{24} for all the genotypes are relatively small compared to other studies (Fisher *et al.*, 1994; Pommier *et al.*, 1992; Murray *et al.*, 1989; De Smet *et al.*, 1992), there is reason to believe that the GP at the time of slaughter may have been low due to the length of time from last feed or glycogen depletion due to transport, insufficient rest period pre-slaughter and pre-slaughter handling. The low GP would thus cause a much higher ultimate pH, with a concomitant lessened loss in exudate. A further explanation for the lower %DL observed in the nn pigs would be that PSE muscles release their drip within the first day post mortem (hence the higher %CHL), leveling off afterwards, whereas normal meat releases drip with a lag phase after the second day post mortem (Honikel *et al.*, 1986).

12.2.2 pH_1

Genotypic comparison (Table 12.16) shows that the NN pigs had a significantly higher ($P < 0.05$) pH_1 value compared with Nn- and nn pigs, with no significant differences between Nn- and nn pigs. Values for the two phenotypes (Table 12.17) do not differ significantly although the Hal- pigs had the highest pH_1 values. Genotype x sex interaction was absent and no significant differences between the sexes (Table 12.18) were found.

Results from other studies (Fisher *et al.*, 1994; Murray *et al.*, 1989; Oliver *et al.*, 1993) show that NN pigs have the highest pH_1 values, with nn the lowest and Nn intermediate. However, Cheah *et al.* (1995) and Sellier (1987) reported that crossbred Nn pigs (Landrace x Large White) show a large variation in meat quality, as defined by pH_1, ranging from 43 % normal to 57 % PSE. The results from Table 12.16 seem to confirm these findings. The large proportion of PSE carcases, especially among the NN pigs, (Annex B, Table 1) do suggest that poor pre- and/or post slaughter management cannot be excluded in producing these results, as proposed by Barton-Gade *et al.* (1988) (see chapter 7.2.2). Poor preslaughter

conditions can lead to a lower muscle GP (Monin *et al.*, 1981) and thus result in a lower pH_1 value. Kaufmann *et al.* (1993) concluded that reliable assesment of meat quality can only be done after full rigor development and that the initial pH measurements for determining meat quality should be used when groups of carcases, rather than single carcases, are measured. Moss (1987) suggests that an accurate assesment of PSE condition can only be made if a combination of pH_1 and meat colour measurements are used.

12.2.3 pH_{24}

The pH_{24} values for the three genotypes (Table 12.16) indicate no significant differences, ranging from 5.80 (nn) to the highest value of 5.87 (NN). Results for the two phenotypes (Table 12.17) and sexes (Table 12.18) show no significant differences with no genotype x sex interaction present.

The post mortem drop in pH (from pH_1 to pH_{24}) is relatively small with a high pH_{24}, compared to results from other studies (Fisher *et al.*, 1994; Murray *et al.*, 1989; Schmitten *et al.*, 1987). As pH_{24} did not reach 5.5, which is the isoelectric point of the principal muscle proteins (Lawrie, 1984), the subsequent denaturation of these proteins were not as acute, thus lessening the characteristic features, such as high %DL, of PSE meat. pH_{24} is determined by muscle GP prior to slaughtering (Monin *et al.*, 1981), whereas the rate of pH decline post mortem is determined by, among others, the halothane status (Monin & Sellier, 1985). It is thus probable that a low GP at slaughter could, to a certain degree, mask the rapid decline in pH characteristic of nn pigs, since the amount of substrate would only allow limited glycolysis. This could then lead to a diminished difference in meat quality between genotypes as defined by pH_1, pH_{24}, colour and %DL.

12.2.4 Fat thickness

The results from Table 12.16 indicate that the nn pigs had a significantly higher ($P < 0.001$) fat thickness (FT) than NN as well as Nn pigs, which in turn did not differ from one another. Phenotypic comparison (Table 12.17) resulted in a similar pattern with the Hal+ pigs having a significantly higher ($P < 0.001$) FT compared to Hal- pigs. The castrates (Table 12.18) had a significantly higher ($P < 0.05$) FT than the gilts, with no genotype x sex interaction (Annex A, Table 4).

These results are not consistent with reported FT values (Eikelenboom *et al.*, 1980a; De Smet *et al.*, 1992; Fisher *et al.*, 1994). Their results indicate that the Nn pigs have a value intermediate to the homozygotes (NN, nn), with NN having the highest and nn the lowest values. Comparing of Hal+ with Hal- pigs, Webb & Simpson (1986) found no significant differences in FT measured over a number of points along the

back. However, it seems that the results of the present study is partially in accordance with Jones *et al.* (1988) who reported that the FT of nn pigs, over the loin area, were significantly higher than that of the NN pigs. Also consistent with the results from the present study are the reports from Pommier *et al.* (1992) and Sather *et al.* (1991a) who compared NN- and Nn pigs, showing no significant differences, although Nn pigs tended to have a lower FT.

Comparison of gender (Table 12.18) shows values consistent with other studies (Jones *et al.*, 1988; Sather *et al.*, 1991a; Pommier *et al.*, 1992) which report that gilts have significantly lower FT than barrows. However, it must be emphasized that various reports (Jones *et al.*, 1988; Pommier *et al.*, 1992; Sather *et al.*, 1991a) indicate that a single FT measurement may not necessarily identify the greater lean content of carcases from pigs with the halothane gene in homo- (nn) or heterozygous (Nn) form.

12.2.5 Meat depth and predicted percentage lean meat yield

The nn pigs had a significantly lower ($P<0.05$) meat depth (MD) (as measured with the HGP) compared to both the NN- and nn pigs (Table 12.16). The phenotypic comparison (Table 12.17) showed a similar trend with the Hal- pigs having a significantly higher ($P < 0.05$) value. The gilts had a higher MD than the barrows (Table 12.18), though not significant. Genotype x sex interaction was absent, with genotype mainly responsible for the observed effect (Annex A, Table 4). Comparison of genotype, phenotype and gender indicate that percentage lean meat yield (LMP) has an inverse trend to that of the measured FT. This is largely due to the formulae used to calculate LMP. The contribution of FT is almost 10 times larger (see formula, chapter 9.2) to LMP than the MD on the same measurement position.

The results from various studies (Jones *et al.*, 1988; Nel *et al.*, 1993, Sather *et al.*, 1991a; Pommier *et al.*, 1992) indicate that the presence of the halothane gene in hetero- or homozygous form (Nn, nn) seem to cause an increase in MD over the loin area, although not always statistically significant. The results of the comparison between NN- and Nn pigs are similar to studies (Pommier *et al.*, 1992; Jones *et al.*, 1988; Sather *et al.*, 1991a) which indicate that the Nn pigs have a higher MD, although not statistically significant. Similar to the present study's results, these studies also report that gilts have a higher measured MD over the loin compared to barrows of the same weight.

The percentage LMP from the Nn pigs is expected to be approximately equal to the mid value of the NN- and the nn pigs, with nn the highest and NN the lowest (Andresen *et al.*, 1981; Sather & Murray, 1989; Fisher *et al.*, 1994). However, as the

results from this study indicate, the LMP of the nn genotype was the lowest with no significant difference between NN- and Nn pigs, casting doubt that the producer can consistently exploit the potential advantages in carcass yield attributable to the halothane gene under the South African classification system.

12.3 Manufactured products

12.3.1 Cured pork products (back bacon)

The results from Table 12.19 seem to indicate that the nn pigs, in spite of initially absorbing the most brine (%PY) during needle injection, had the highest moisture loss (%ML) ($P < 0.001$) during further processing compared to the other two genotypes. This resulted in the nn pigs having the lowest nett gain (%BY), though not statistically significant.

Table 12.19 Average values (with standard errors) of the processing characteristics of the three genotypes

Characteristic	Genotype NN (n = 31)	Nn n = 17)	nn (n = 11)
Pumped yield (%PY)	9.7 (±1.01)	8.1[a] (±1.37)	12.6[b] (±1.69)
Moisture loss (%ML)	2.5[d] (±0.696)	1.2[d] (±0.940)	7.7[e] (±1.16)
Bacon yield (%BY)	7.2 (±1.24)	6.9 (±1.67)	4.8 (±2.08)

[a-b] Values in the same row with different superscripts differ ($P < 0.05$)

[d-e] Values in the same row with different superscripts differ ($P < 0.001$)

This is consistent with results from studies (Wismer-Pedersen, 1968; Smith & Leser, 1982) which report a higher moisture loss and a reduced nett gain for cured meat from PSE carcases. Due to the design of the present study to investigate the effect of genotype and not meat type (normal, PSE and DFD) and processing characteristics, statistical analysis could not be performed on meat type. Since no literature is available on processing characteristics of genotypes, comparison to meat types is considered appropriate.

Table 12.20 Average values (with standard errors) of the processing characteristics of the different phenotypes

Characteristic	Phenotype	
	Hal (n = 48)	Hal+ (n = 11)
Pumped yield (%PY)	9.1 (±6.06)	12.6 (±1.69)
Moisture loss (%ML)	2.0[c] (±3.44)	7.7[d] (±1.16)
Bacon yield (%BY)	7.1 (±6.72)	4.8 (±2.08)

[c-d] Values in the same row with different superscripts differ ($P < 0.001$)

Comparison of the two phenotypes (Table 12.20) show that Hal+ pigs have a significantly higher percentage moisture loss with a higher percentage brine pumped into the backs approaching significance at the 5 % level (Annex A, Table 1). None of the variables show significant differences between the sexes.

A possible explanation for the initial higher percentage moisture gain (%PY) of the nn pigs could be the more open structure of the myofibrils of the PSE meat (Briskey, 1964), thus allowing more moisture into the fibrillar network. The subsequent higher loss (%ML) and resultant lower nett gain (%BY) is probably due to a higher state of protein denaturation in the nn pigs and thus resulted in an impeded moisture uptake.

Table 12.21 Average values (with standard errors) of the processing characteristics of castrates (C) and gilts (G)

Characteristic	Sex	
	C (n = 34)	G (n = 25)
Pumped yield (%PY)	10.2 (±0.989)	9.2 (±1.15)
Moisture loss (%ML)	2.8 (±0.768)	3.6 (±0.895)
Bacon yield (%BY)	7.4 (±1.17)	5.6 (±1.38)

Table 12.22 Average values (with standard errors) of the chemical analysis of cured meat from the three genotypes

Characteristic	Genotype		
	NN (n = 31)	Nn (n = 17)	nn (n = 11)
Moisture content (%M)	50.2[a] (±0.831)	44.8[b] (±1.12)	49.4[a] (±1.39)
Protein content (%P)	72.4[a] (±0.823)	71.6 (±1.11)	69.1[b] (±1.38)
Fat content (%F)	5.9 (±0.489)	5.7 (±0.661)	7.6 (±0.822)
Sodium content (Na^+)	12096[a] (±335)	12477 (±453)	13446[b] (±563)

[a] [b] Values in the same row with different superscripts differ ($P < 0.05$)

The high percentage of PSE carcases in the Nn group seem to produce anomalous results in this regard, which could call into question the choice of pH values used to discriminate between PSE, DFD and normal carcases.

Results from the chemical analysis of the lean muscle in the back bacon appears in Table 12.22. The moisture content (%M) was the highest for the NN pigs, differing significantly ($P < 0.05$) from the Nn pigs, but not from the nn pigs. The nn pigs had a higher ($P < 0.05$) %M than the heterozygous Nn pigs. Phenotypic comparison

(Table 12.23) indicate that no significant differences in %M exists between Hal- and Hal+ pigs.

Table 12.23 Average values (with standard errors) of the chemical analysis of cured meat of different phenotypes

Characteristic	Phenotype	
	Hal- (n = 48)	Hal+ (n = 11)
Moisture content (%M)	48.3 (±5.42)	49.4 (±1.39)
Protein content (%P)	72.1 (±4.67)	69.1 (±1.38)
Fat content (%F)	5.8^{a} (±2.49)	7.6^{b} (±0.82)
Sodium content (Na^{+})	12231 (±2009)	13446 (±563)

$^{a-b}$ Values in the same row with different superscripts differ ($P < 0.05$)

The results in Annex A Table 4 strongly suggest that the causative factor for %M is genotypic, and not sex or genotype x sex interaction.

Percentage protein (%P) (expressed on a dry matter basis) only differs significantly between NN and nn pigs, though the phenotypic comparison (Table 12.23) approached significance at the 5 % level ($P = 0.063$) with the Hal- pigs having a higher value. No differences between sexes or genotype x sex interaction was observed. Percentage fat (%F) (expressed on dry matter basis) did not differ significantly between genotypes but approached significance ($P = 0.068$) comparing NN with nn and Nn with nn ($P = 0.066$). The Hal+ pigs had a significantly higher ($P < 0.05$) F (Table 12.23) compared to the Hal- pigs. Comparison of the sexes showed no significant differences with no genotype x sex interaction present.

Table 12.24 Average values (with standard errors) of the chemical analysis of cured meat of castrates (C) and gilts (G)

Characteristic	Sex	
	C (n = 34)	G (n = 25)
Moisture content (%M)	48.8 (±0.888)	48.1 (±1.04)
Protein content (%P)	71.5 (±0.808)	71.6 (±0.943)
Fat content (%F)	6.3 (±0.479)	5.9 (±0.559)
Sodium content (Na^+)	12552 (±328)	12328 (±383)

Sodium content (Na^+) (expressed on dry matter basis) (Table 12.22) was the highest for the nn pigs, differing only significantly ($P < 0.05$) from the NN pigs, with the heterozygous (Nn) pigs intermediate. The difference between Hal- and Hal+ pigs approached significance ($P = 0.071$), with the Hal+ pigs having the highest Na^+ content. No difference between sexes or interaction was observed.

In contrast to the results of this study other reports indicate that, comparing fresh lean meat, nn pigs have the lowest protein and the highest fat content, with Nn intermediate and the NN pigs having the highest protein and lowest fat content (Murray *et al.*, 1989; Sather *et al.*, 1991a). This could indicate that the nn pigs, which grow faster, mature faster and thus deposit fat in different depots. The correlation between subcutaneous fat and marbling fat (in the MLT) was not significant as will be described in chapter 12.4. However, studies (Murray *et al.*, 1989; Sather *et al.*, 1991a) show a trend similar to the results in Table 12.22 in that an increase in fat content is accompanied by a corresponding lower moisture content. The higher sodium content of the lean meat from the nn pigs is consistent with the higher percentage moisture loss as this causes a higher concentration of sodium in the muscle. From the results it becomes apparent that the presence of the halothane gene, especially in homozygous (nn) form will result in deminished economic returns for the meat processor.

12.3.2 Cooked cured pork products (canned hams)

Highly significant differences ($P < 0.001$) in percentage moisture loss (exudate) between all three genotypes were observed (Table 12.25). The NN pigs had the lowest percentage exudate, with Nn the highest and nn intermediate. These results give a clear indication of the effect of the halothane gene on WHC in both the heterozygous (Nn) and homozygous (nn) form. Both the Nn- and nn pigs had moisture losses in excess of 15 % (16.7 % and 15.5 % respectively) compared to the NN pigs (13.3 %)

Table 12.25 Average values (with standard errors) of percentage cooking loss in canned hams of different genotypes (n = 20 cans.genotype^{-1})

Characteristic	Genotype NN (n = 20)	Nn (n = 20)	nn (n = 20)
% Cooking loss	13.33^{d} (±0.107)	16.73^{e} (±0.107)	15.45^{f} (±0.107)

$^{d\text{-}f}$ Values in the same row with different superscripts differ ($P < 0.001$)

Wismer-Pedersen (1968) reported that the reduced WHC of PSE meat leads to an increased percentage gelatinous cook out in cans due to a higher degree of aggregation of meat proteins, especially for pasteurized, canned hams.

This could probably explain the high moisture loss or cook-out of the hams from the Nn- and nn pigs, as both genotypes had a high proportion (70.6 % and 63.6 %) of PSE carcases (Table 12.13) Ockerman *et al.* (1978) reported that short term tumbling (30 min.) improved muscle cohesion but did not significantly affect ham yield, and that longer tumbling periods are required to enhance yield compared to cohesion.

The reduced moisture binding capability of the high proportion PSE meat present in the Nn and nn genotype samples was thus probably enhanced by the short term tumbling period (30 min. total) used in this study, thus leading to a lower yield in the finished product. The results of this study supports results by Honkavaara (1988), who reported reduced TY for cooked, cured ham from PSE pork, compared to non-PSE pork. Müller (1991) reported similar relationships between pH and ham quality, higher pH values resulting in higher cooked ham yield with a concomittant decrease in the amount of juice exudation.

12.4 Correlations

The correlations are given in Annex C Table 1. The production characteristics show significant correlations with carcass quality characteristics. The positive correlation between ADG and fat thickness ($r = 0.41$, $P < 0.05$) agrees with results in literature (Sönnichsen *et al.*, 1984; Schwörer *et al.*, 1980; Hovenier *et al.*, 1992). These authors contribute the higher backfat thickness to ad lib. feeding, thus enabling pigs with a higher appetite to grow faster and attain a higher fat thickness.

The growth rate (ADG) is also negatively correlated to LMP ($r = -0.40$, $P < 0.05$), thus reflecting the large influence that the backfat thickness has on the calculation of the LMP in the carcass. The correlations between production and meat quality characteristics are low exept for days to slaughter ($r = 0.47$, $P < 0.001$) and ADG ($r = -0.38$, $P < 0.05$) with drip loss, the results indicating that the faster growing pigs have a lower drip loss. These correlations suggest that the advantages of the faster growing pigs is offset by a higher backfat thickness, lower LMP and thus lower financial returns.

The strong correlation between pH_1 and pH_{24} ($r = 0.27$, $P < 0.05$) and drip loss ($r = -0.39$, $P < 0.05$) suggest that pH_1 could be a good indicator of meat quality as a high pH_1 value would indicate lower drip loss and a higher ultimate pH value, resulting in non-PSE meat. Similarly, Hovenier *et al.* (1992) reported high genetic correlations (between breeds) between pH values, drip loss and meat colour, arguing that the mechanism of pH, WHC and meat colour still exist in the absence of the halothane gene.

Drip loss has a significant correlation with fat thickness ($r = -0.46$, $P < 0.001$), meat depth ($r = 0.30$, $P < 0.05$) and LMP ($r = 0.46$, $P < 0.001$). The results indicate that the pigs with a high drip loss had less backfat, a higher meat depth and a subsequent higher LMP. Results in literature (Murray *et al.*, 1989: De Smet *et al.*, 1992) show that the presence of the halothane gene results in high drip losses and lower backfat values, however the results from the present study indicate the opposite effect.

The cured processed meat (bacon) show a strong correlation between percentage pumped yield and percentage bacon yield ($r = 0.76$, $P < 0.001$), which is in accordance with the results from Nusbaum & Rust (1978). A significant correlation also exists between pumped yield and percentage moisture loss ($r = -0.55$, $P < 0.001$). This suggests that meat with a high pump yield sufferes less moisture loss and results in a higher bacon yield, thus enhanced economic returns. The correlation between percentage moisture loss and backfat thickness ($r = 0.43$,

$P < 0.001$), meat depth ($r = -0.29$, $P < 0.05$) and LMP ($r = -0.44$, $P < 0.001$) suggest that fatter pigs have higher moisture losses and thus have a lower nett gain in bacon yield, although the correlations for bacon yield were not significant. It must be stressed however that in this study the nn pigs, which are prone to PSE where incidently of higher backfat thickness.

The high correlation between LMP and fat thickness ($r = -0.99$, $P < 0.001$) and meat depth ($r = 0.65$, $P < 0.001$) was due to the fact that LMP is a dependent variable that is calculated using fat thickness and meat depth as independent variables (see chapter 9.2). The correlation between backfat thickness and meat depth is also significant ($r = -0.57$, $P < 0.001$) indicating that the higher backfat was accompanied by a reduction in meat depth and a subsequent lower percentage lean in the carcass. The results in Table 12.14 comparing genotypes clearly illustrates this relationship.

The results from the chemical analysis only show the moisture content having any significant correlation with meat quality characteristics. The positive correlation between moisture content and pH_1 ($r = 0.35$, $P < 0.05$) indicate that processed meat with a higher pH_1 has better moisture retention, also supported by the negative correlation between pH_1 and drip loss ($r = -0.39$, $P < 0.05$).

The sodium content showed a significant correlation with backfat thickness ($r = 0.29$, $P < 0.05$), meat depth ($r = -0.31$, $P < 0.05$), LMP ($r = -0.31$, $P < 0.05$) and protein content ($r = -0.55$, $P < 0.05$). Protein and fat content showed a negative correlation ($r = -0.37$, $P < 0.001$). These results suggest that processed meat from pigs with a high backfat thickness and low LMP have higher sodium levels compared to leaner pigs. Table 12.10 show that the bacon from the nn pigs had the lowest protein and highest fat and sodium content, thus supporting the correlation values. This correlation has however no practical implications for processing purposes.

The results of the present study, although not all statistically significant, seem to suggest that the presence of the halothane gene causes a tendency in meat and processed products that is characterized by increased backfat, lower lean yield, higher sodium content and lower WHC, eventually resulting in a product of inferior quality and subsequent deminished economic returns for both the producer and processor.

12.5 Multiple linear regressions

Multiple linear regression values (R^2) were calculated for drip loss (%DL), chilling loss (%CHL), percentage moisture (%M), percentage pumped yield (%PY),

(MD), sodium content (Na^+), fat content (%F), protein content (%P), pH_1 and pH_{24} in an attempt to find a prediction equation of value.

12.5.1 Drip loss

The results from Annex D Table 1 show that FT alone does not give a good predictive model to determine %DL. However, if combined with WCM and pH, R^2 increases by 0.07 units with R^2 = 0.416 and Cp = 2.496, indicating little bias in the equation. Inclusion of more variables do not greatly improve R^2 ,R^2 only improving 0.04 units (R^2 = 0.454) with all eight variables in the model. These results suggest that the prediction of %DL from the above mentioned variables would not result in an accurate value and thus of no or limited use.

12.5.2 Chilling loss

The R^2 values for %CHL (Annex D, Table 2) are low and shows minor improvements with the inclusion of more than one variable in the regression equation. The R^2 for pH_{24} is 0.054, improving only 0.04 units to R^2 = 0.094 with all the variables included. These results suggest that predicting %CHL is not accurate enough to be of much use to the processor.

12.5.3 Percentage pumped yield, moisture loss and bacon yield

The %PY and %BY show similar trends in so far that both have comparatively low R^2 values, using either one or multiple variables in the regression equations. %PY has a R^2 = 0.084 (Annex D, Table 3) with Cp = 0.1274 using FT, %P and Na^+ as independend variables. The R^2 value only increases with 0.02 units to R^2 = 0.104 if all the variables are used. R^2 for %BY (Annex D, Table 5) differs only with 0.034 units between the three variable (pH_1, MD, Na^+) regression equation (R^2 = 0.089) and eight variable equation (R^2 = 0.123). This suggests that the use of more than three variables does not greatly improve R^2 for %BY or %PY.

The single variable regression equations predicting %ML (Annex D, Table 4) indicate that FT is the variable that has the best predictive value, more than twice that of MD. The use of more than four variables (pH_1, FT, MD, Na^+) to predict %ML (R^2 = 0.249) does not greatly improve R^2, as the best R^2 with least bias, using all the variables, is only R^2 = 0.258, an improvement of 0.01 units. The low R^2 values predicting back bacon quality (%BY, %PY, %ML) in this study would not justify the recording of the additional variables and the calculation of the appropriate R^2 values.

12.5.4 Percentage moisture in sample

Calculation of the regression equations for %M (Annex D, Table 6) shows that single variables do not give high R^2 values. Using WCM, pH_1, pH_{24}, and Na^+ improves R^2 to 0.224, with the inclusion of all the variables only improving R^2 with 0.024 units to R^2 = 0.248, thus not giving the calculated regression equations any good predictive value.

13 CONCLUSIONS

The conclusions for this study can be devided into three main catagories namely growth, carcass and meat quality characteristics and processed meat quality characteristics.

13.1 Growth characteristics

According to the present results the values for growth characteristics (Days to slaughter, ADG) were the result of genotype x sex interaction. However sex as a main effect was disregarded since it is a fixed effect and as such cannot be manipulated. There was a significant decrease in days to slaughter for nn pigs compared to NN and Nn pigs (NN = 81.2, Nn = 84.6 and nn = 74.1). No statistically significant differences appeared for ADG (NN = 0.729, Nn = 0.710 and nn = 0.765). The inclusion of the halothane gene in terminal crossings to produce Nn progeny thus holds no compelling advantages for the producer. The advantages of increased growth rate of the nn pigs is more than offset by the risk of possible death during any of the stress causing activities such as mating or transport. Correlation values between production and meat quality characteristics are low except for days to slaughter (r = 0.47) and ADG (r = -0.38) with drip loss. Growth rate (as described by ADG) is negatively correlated with LMP (r = -0.40), suggesting that faster growth is accompanied by increased fat thickness, lower LMP and subsequently lower returns for the producer.

13.2 Carcass and meat quality characteristics

The presence of the halothane gene failed to produce any financially advantageous carcass characteristics such as less backfat (NN = 20.0, Nn = 18.8 and nn = 26.1), higher meat depth (NN = 50.9, Nn = 52.4 and nn = 46.8) or a higher LMP (NN = 66.1, Nn = 66.7 and nn = 63.0). Carcass length, the result of sex x genotype interaction, deminished with the halothane gene in homozygous form (NN = 76.5, Nn = 76.7 and nn = 75.2). The interaction was discounted since the effect of the gene was observed in both sexes. These results suggest that inclusion of the halothane gene could lead to deminished returns for the producer, especially if the current considerations to slaughter heavier carcases are implemented.

Chilling loss (the percentage difference between warm and cold carcass mass determined within the first 24 h post mortem) did not differ significantly (NN = 4.4, Nn = 4.7 and nn = 5.2) between any of the genotypes but drip loss (the percentage exudate lost from the loin portion in the plastic bag 48 h post mortem) showed

nn = 1.06). These results could call in to question the method currently used to determine drip loss since PSE muscles released exudate, measurable as chilling loss, within the first 24 hours post mortem, whereas normal muscles release drip after a lag phase of approximately 48 hours, measurable as drip loss.

The pH_1 values were NN = 6.05, Nn = 5.65 and nn = 5.82, with the NN pigs having a significantly higher ($P < 0.05$) pH_1 value than both Nn- and nn pigs, which in turn did not differ from each other. The majority of Nn- and nn carcases had a $pH_1 < 5.9$ (70.6 % and 63.6 % respectively), compared to only 29 % for the NN carcases. The strong correlation between pH_1 and pH_{24} (r = 0.27) as well as drip loss (r = -0.39) strongly suggest that pH_1 could be implemented as an indicator of meat quality. The pH_{24} values did not differ significantly (NN = 5.87, Nn = 5.81 and nn = 5.80). The pH_1 values can be used to discriminate between PSE and non-PSE carcases, thus if the pH_1 values are included in the current classification system, it could lead to the eventual eradication of the halothane gene.

13.3 Processed meat characteristics

The results for back bacon clearly shows the disadvantages associated with the halothane gene in homozygous form, resulting in a higher moisture loss (NN = 2.5, Nn = 1.2 and nn = 7.7) and a lower nett gain (NN = 7.2, Nn = 6.9 and nn = 4.8). The percentage bacon yield was the result of sex x genotype interaction. The percentage pumped yield was the highest for nn pigs (NN = 9.7, Nn = 8.1 and nn = 12.6) but this characteristic did not cause an increase in the percentage bacon yield for this genotype. These losses are currently only suffered by the meat processor and are recovered by increased prices to the consumer. A strong correlation between percentage pumped yield and percentage bacon yield (r = 0.76) and percentage moisture loss (r = -0.55) suggest that processed meat with high pump yields suffer less from moisture loss and thus result in higher nett bacon yields.

Comparison of the chemical composition of the samples show that the NN pigs had the highest percentage protein content (NN = 72.4, Nn = 71.6 and nn = 69.1), highest percentage moisture content (NN = 50.2, Nn = 44.8 and nn = 49.4) and lowest sodium content (expressed as $mg.kg^{-1}$) (NN = 12096, Nn = 12477 and nn = 13446). None of the correlation values for chemical composition with meat quality characteristics such as Na^+ with fat thickness (r = 0.29), meat depth (r = -0.31) and LMP (r = -0.31) seem to have much practical implication for current production or processing methods. Prediction models, using multiple linear regression equations, show that none of the dependent variables (percentage drip loss, percentage chilling loss, percentage pumped yield, percentage moisture loss,

loss, percentage chilling loss, percentage pumped yield, percentage moisture loss, percentage bacon yield, percentage moisture content of the sample) could be accurately predicted with the independent variables used.

Comparison of the canned ham products indicate that the presence of the halothane gene results in significantly higher percentage cooking losses for both the Nn- and nn genotypes (NN = 13.33, Nn = 16.73 and nn = 15.45). Since the experimental work was carried out for canned products this cooking loss is in a gelatinous form and thus not lost during the sterilization/cooking process, the processor does not suffer any direct financial losses. However, the products manufactured from Nn- and nn carcases could visually appear inferior and so doing dissuade the consumer from purchasing the product again. This meat type, when used for pasteurized non sealed hams, will result in a lower yield.

According to the present results the inclusion of the halothane gene in either the hetero- or homozygous form (Nn or nn) holds no financial advantages for both the producer and the meat processor. On the contrary, it could lead to losses for the producer since this study suggests that any advantages in production performance could be undone by the lower carcass classification due to a higher back fat value, thus deminishing finiancial return. The meat processor, in turn, has meat that , when processed, results in products of inferior quality and higher production losses. A possible solution would be to incorporate pH_1 measurements in the current classification system so that PSE and non-PSE carcases can be identified, especially if the processor is manufacturing products that is targeted at the more affluent or discerning consumer. Based on this criteria, premiums could then be paid to producers that deliver better quality carcases and so doing help expedite the eradication of the halothane gene from the national herd.

Annex A

Table 1 Probability of values > F for comparison between genotypes, as well as between phenotypes

Characteristic	Genotype			Phenotype
	NN vs Nn	NN vs nn	Nn vs nn	NN + Nn vs nn
DAYS	0.2124	0.0282	0.0038	0.0052
ADG	0.4050	0.1730	0.0617	0.0753
%CHL	0.6182	0.2267	0.4759	0.2991
%DL	0.0064	0.0347	0.0001	0.0007
%PY	0.3422	0.1485	0.0425	0.0557
%ML	0.2539	0.0003	0.0001	0.0001
%BY	0.8939	0.3424	0.4481	0.3529
%M	0.0003	0.6243	0.0121	0.2232
FT	0.3016	0.0001	0.0001	0.0001
MD	0.2661	0.0124	0.0021	0.0023
LMP	0.2666	0.0001	0.0001	0.0001
CL	0.7521	0.0804	0.0678	0.0508
pH_1	0.0001	0.0272	0.1282	0.7696
pH_{24}	0.4705	0.4841	0.9427	0.6848
%P	0.5494	0.0420	0.1617	0.0630
%F	0.8106	0.0681	0.0660	0.0456
$\%Na^+$	0.5014	0.0441	0.1856	0.0709

DAYS = days to slaughter (growth phase), ADG = average daily gain, %CHL = chilling loss, %DL = drip loss, %PY = percentage pumped yield, %ML = percentage moisture loss, %BY = percentage bacon yield, %M = moisture content, FT = fat thickness (mm), MD = meat depth (mm), LMP = percentage predicted lean yield, CL = carcass length (cm), pH_1 = initial pH, pH_{24} = ultimate pH, %P = percentage protein, %F = percentage fat, Na^+ = sodium content ($mg.kg^{-1}$)

Table 2. Probability of values > F for comparison between genotypes, as well as phenotypes

Characteristic	**Genotype**			**Phenotype**
	NN vs Nn	NN vs nn	Nn vs nn	NN + Nn vs nn
% Cooking loss	0.0001	0.0001	0.0001	0.0024

Table 3 Probabilities of values > F for comparison between castrates (C) and gilts (G)

Characteristic	C vs G
Days to slaughter (DAYS)	0.0005
ADG	0.0012
Chilling loss (%CHL)	0.2576
Drip loss (%DL)	0.0007
Percentage pumped yield (%PY)	0.5267
Percentage moisture loss (%ML)	0.4972
Percentage bacon yield (%BY)	0.3284
Percentage moisture (%M)	0.5716
Fat thickness (FT)	0.0013
Meat depth (MD)	0.1468
LMP	0.0016
Carcass length (CL)	0.4163
pH_1	0.2149
pH_{24}	0.3398
Percentage protein (%P)	0.9260
Percentage fat (%F)	0.6337
Sodium content (Na^+)	0.6593

DAYS = days to slaughter (growth phase), ADG = average daily gain, %CHL = chilling loss, %DL = drip loss, %PY = percentage pumped yield, %ML = percentage moisture loss, %BY = percentage bacon yield, %M = moisture content, FT = fat thickness (mm), MD = meat depth (mm), LMP = percentage predicted lean yield, CL = carcass length (cm), pH_1 = initial pH, pH_{24} = ultimate pH, %P = percentage protein, %F = percentage fat, Na^+ = sodium content ($mg.kg^{-1}$)

Table 4 Probabilities of values > F for genotype, sex and genotype x sex interaction

Characteristic	Sex	Genotype	Gen x Sex
DAYS	0.0001	0.0001	0.0319
ADG	0.0005	0.0385	0.0209
%CHL	0.2588	0.5432	0.2852
%DL	0.0001	0.0001	0.1577
%PY	0.5082	0.0998	0.1136
%ML	0.4385	0.0002	0.4226
%BY	0.3159	0.6735	0.0500
%M	0.5368	0.0014	0.9846
FT	0.0001	0.0001	0.1047
MD	0.1070	0.0028	0.1056
LMP	0.0001	0.0001	0.0773
CL	0.3859	0.0979	0.0351
pH_1	0.1548	0.0001	0.5989
pH_{24}	0.3538	0.7297	0.9095
%P	0.9249	0.1247	0.4859
%F	0.6290	0.1222	0.5339
Na^+	0.6583	0.1312	0.9574

DAYS = days to slaughter (growth phase), ADG = average daily gain, %CHL = chilling loss, %DL = drip loss, %PY = percentage pumped yield, %ML = percentage moisture loss, %BY = percentage bacon yield, %M = moisture content, FT = fat thickness (mm), MD = meat depth (mm), LMP = percentage predicted lean yield, CL = carcass length (cm), pH_1 = initial pH, pH_{24} = ultimate pH, %P = percentage protein, %F = percentage fat, Na^+ = sodium content (mg.kg 1)

Annex B

Table 1 Classification of meat quality within each genotype, upper values indicate number of pigs in each class with bracketed values indicating proportion within each genotype

	Genotype		
Meat quality	NN	Nn	nn
Normal	20	4	4
($pH_1 \geq 5.9$, $pH_{24} < 6.2$)	(64.5%)	(23.5%)	(36.4%)
DFD	2	1	0
($pH_{24} \geq 6.2$)	(6.5%)	(5.9%)	0
PSE	9	12	7
($pH_1 \leq 5.9$)	(29.0%)	(70.6%)	(63.6%)

Annex C

Table 1 Correlation (r) values for the production, carcass, meat quality, processed meat quality and chemical characteristics

	ADG	CL	pH_1	pH_{24}	%CHL	%DL	%PY	%ML	%BY	FT	MD	LMP	%P	%F	Na+	%M
DAYS	**-0.88**	0.17	-0.14	0.03	0.16	**0.47**	-0.05	-0.21	0.10	**-0.48**	**0.30**	**0.48**	0.18	-0.15	-0.12	-0.01
ADG		0.09	0.23	-0.01	-0.17	**-0.38**	-0.01	0.13	-0.09	**0.41**	-0.18	**-0.40**	-0.09	0.13	0.01	0.03
CL			0.25	0.06	0.14	**0.36**	-0.14	0.06	-0.16	-0.17	-0.06	0.15	0.19	-0.12	-0.24	-0.09
pH_1				**0.27**	-0.05	**-0.39**	0.03	-0.11	0.10	0.01	-0.20	-0.03	0.02	0.01	0.04	-0.12
pH_{24}					-0.23	-0.22	-0.08	-0.03	-0.05	0.01	-0.20	-0.03	0.02	0.01	0.04	-0.12
%CHL						0.03	0.00	0.20	-0.13	+0.06	-0.09	-0.06	0.02	-0.01	0.05	0.12
%DL							-0.11	0.04	-0.12	**-0.46**	**0.30**	**0.46**	0.09	-0.17	-0.15	-0.23
%PY								0.12	**0.76**	0.22	-0.04	-0.21	0.06	0.01	0.15	0.20
%ML									**-0.55**	**0.43**	**-0.29**	**-0.44**	-0.05	0.14	0.01	-0.07
%BY										-0.10	0.15	0.11	0.09	-0.08	0.12	0.21
FT											**-0.57**	**-0.99**	-0.17	0.24	**0.29**	-0.02
MD												**0.65**	**0.28**	-0.21	**-0.31**	-0.05
LMP													0.19	-0.24	**-0.31**	0.02
%P														**-0.37**	**-0.55**	0.05
%F															0.08	0.03
Na+																0.05

DAYS = days to slaughter, ADG = average daily gain, CL = carcass length (cm), pH_1 = initial pH, pH_{24} = ultimate pH, %CHL = percentage chilling loss, %DL = percentage drip loss, %PY = percentage pumped yield, %ML = percentage moisture loss, %BY = percentage bacon yield, FT = fat thickness (mm), MD = meat depth (mm), LMP = percentage predicted lean yield, %P = percentage protein, %F = percentage fat, Na+ = sodium content ($mg.kg^{-1}$), %M = percentage moisture.

Annex D

Table 1 The regression (R^2) model for drip loss (%DL)

p	R^2	Cp	FT	MD	%F	Na^+	%P	pH_1	pH_{24}	WCM
1	0.2117	17.253	+							
1	0.1539	22.545						+		
1	0.0911	28.306		+						
1	0.0466	32.379							+	
1	0.0426	32.751								+
1	0.0299	33.913			+					
1	0.0211	34.719				+				
1	0.0089	35.839					+			
2	0.3472	6.830	+					+		
2	0.2627	14.577	+							+
2	0.2572	15.083	+						+	
2	0.2160	18.853	+		+					
2	0.2153	18.917				+		+		
2	0.2147	18.978	+					+		
2	0.2142	19.017		+				+		
2	0.2140	19.035	+	+						
3	0.4163	2.496	+					+		+
3	0.3612	7.552	+					+	+	
3	0.3605	7.614	+			+		+		
3	0.3556	8.064	+				+	+		
3	0.3537	8.234	+		+			+		
3	0.3472	8.828	+	+				+		
3	0.3066	12.556	+						+	+
3	0.2890	14.162				+		+		+

%CHL = chilling loss, %DL = drip loss, %PY = percentage pumped yield, %ML = percentage moisture loss, %BY = percentage bacon yield, %M = moisture content, FT = fat thickness (mm), MD = meat depth (mm), LMP = percentage predicted lean yield, pH_1 = initial pH, pH_{24} = ultimate pH, %P = percentage protein, %F = percentage fat, Na^+ = sodium content ($mg.kg^{-1}$)

Table 1 (continued) The regression (R^2) model for drip loss (%DL)

p	R^2	Cp	FT	MD	%F	Na^+	%P	pH_1	pH_{24}	WCM
4	0.4357	2.722	+			+		+		+
4	0.4276	3.461	+					+	+	+
4	0.4241	3.785	+				+	+		+
4	0.4167	4.461	+		+			+		+
4	0.3714	8.612	+			+		+	+	
4	0.3689	8.841	+				+	+	+	
4	0.3673	8.992	+		+			+	+	
5	0.4436	3.996	+			+		+	+	+
5	0.4418	4.165	+	+		+		+		+
5	0.4361	4.687	+			+	+	+		+
5	0.4359	4.697	+		+	+		+		+
5	0.4348	4.797	+				+	+	+	+
5	0.4316	5.092	+	+				+	+	+
5	0.4285	5.379	+	+			+	+		+
5	0.4278	5.430	+		+			+	+	+
6	0.4527	5.161	+	+		+		+	+	+
6	0.4441	5.946	+			+	+	+	+	+
6	0.4438	5.974	+		+	+		+	+	+
6	0.4429	6.059	+	+			+	+	+	+
6	0.4428	6.065	+	+		+	+	+		+
6	0.4423	6.119	+	+	+	+		+		+
6	0.4361	6.679	+		+	+	+	+		+
6	0.4350	6.783	+			+	+	+	+	+

%CHL = chilling loss, %DL = drip loss, %PY = percentage pumped yield, %ML = percentage moisture loss, %BY = percentage bacon yield, %M = moisture content, FT = fat thickness (mm), MD = meat depth (mm), LMP = percentage predicted lean yield, pH_1 = initial pH, pH_{24} = ultimate pH, %P = percentage protein, %F = percentage fat, Na^+ = sodium content (mg.kg-1)

Table 1 (continued) The regression (R^2) model for drip loss (%DL)

p	R^2	Cp	FT	MD	%F	Na^+	%P	pH_1	pH_{24}	WCM
7	0.4544	7.005	+	+		+	+	+	+	+
7	0.4532	7.113	+	+	+	+		+	+	+
7	0.4442	7.941	+		+	+	+	+	+	+
7	0.4431	8.042	+	+	+		+	+	+	+
7	0.4430	8.055	+	+	+	+	+	+		+
7	0.3819	13.649	+	+	+	+	+	+	+	
7	0.3170	19.600		+	+	+	+	+	+	+
7	0.3081	20.416	+	+	+	+	+	+		+
8	0.4545	9.000	+	+	+	+	+	+	+	+

%CHL = chilling loss, %DL = drip loss, %PY = percentage pumped yield, %ML = percentage moisture loss, %BY = percentage bacon yield, %M = moisture content, FT = fat thickness (mm), MD = meat depth (mm), LMP = percentage predicted lean yield, pH_1 = initial pH, pH_{24} = ultimate pH, %P = percentage protein, %F = percentage fat, Na^+ = sodium content (mg.kg 1)

Table 2 The regression (R^2) model for chilling loss (%CHL)

p	R^2	Cp	FT	MD	%F	Na^+	%P	pH_1	pH_{24}	WCM
1	0.0539	-2.815							+	
1	0.0087	-0.314		+						
1	0.0081	-0.285								+
1	0.0033	-0.019	+							
1	0.0028	0.006				+				
1	0.0026	0.019						+		
1	0.0003	0.149					+			
1	0.0001	0.157			+					
2	0.0741	-1.929		+					+	
2	0.0614	-1.225							+	+
2	0.0580	-1.035				+			+	
2	0.0574	-1.005	+						+	
2	0.0544	-0.841					+		+	
2	0.0541	-0.823						+	+	
2	0.0540	-0.818			+				+	
2	0.0181	1.165		+						+
3	0.0835	-0.442		+					+	+
3	0.0786	-0.171		+			+		+	
3	0.0757	-0.014		+	+				+	
3	0.0750	0.026	+	+					+	
3	0.0746	0.049		+		+			+	
3	0.0742	0.071		+				+	+	
3	0.0647	0.593				+			+	+
3	0.0645	0.607	+						+	+

%CHL = chilling loss, %DL = drip loss, %PY = percentage pumped yield, %ML = percentage moisture loss, %BY = percentage bacon yield, %M = moisture content, FT = fat thickness (mm), MD = meat depth (mm), LMP = percentage predicted lean yield, pH_1 = initial pH, pH_{24} = ultimate pH, %P = percentage protein, %F = percentage fat, Na^+ = sodium content (mg.kg 1)

Table 2 (continued) The regression (R^2) model for chilling loss (%CHL)

p	R^2	Cp	FT	MD	%F	Na^+	%P	pH_1	pH_{24}	WCM
4	0.0878	1.318		+			+		+	+
4	0.0850	1.474	+	+					+	+
4	0.0839	1.537		+	+				+	+
4	0.0839	1.548		+				+	+	+
4	0.0836	1.551		+		+			+	+
4	0.0825	1.613		+		+	+		+	
4	0.0794	1.782	+	+			+		+	
4	0.0789	1.810		+	+		+		+	
5	0.0905	3.169		+		+	+		+	+
5	0.0894	3.233	+	+			+		+	+
5	0.0887	3.271		+			+	+	+	+
5	0.0878	3.318		+	+		+		+	+
5	0.0853	3.459	+	+		+			+	+
5	0.0852	3.463	+	+				+	+	+
5	0.0852	3.465	+	+	+				+	+
5	0.0841	3.523	+	+		+	+		+	
6	0.0928	5.041	+	+		+	+		+	+
6	0.0908	5.154		+		+	+	+	+	+
6	0.0907	5.161		+	+	+	+		+	+
6	0.0903	5.184	+	+			+	+	+	+
6	0.0895	5.228	+	+	+		+		+	+
6	0.0887	5.270		+	+		+	+	+	+
6	0.0854	5.450	+	+	+	+			+	+
6	0.0854	5.453	+	+	+			+	+	+

%CHL = chilling loss, %DL = drip loss, %PY = percentage pumped yield, %ML = percentage moisture loss, %BY = percentage bacon yield, %M = moisture content, FT = fat thickness (mm), MD = meat depth (mm), LMP = percentage predicted lean yield, pH_1 = initial pH, pH_{24} = ultimate pH, %P = percentage protein, %F = percentage fat, Na^+ = sodium content (mg.kg 1)

Table 2 (continued) The regression (R^2) model for chilling loss (%CHL)

p	R^2	Cp	FT	MD	%F	Na^+	%P	pH_1	pH_{24}	WCM
7	0.0934	7.014	+	+	+	+	+		+	+
7	0.0931	7.028	+	+		+	+	+	+	+
7	0.0909	7.146		+	+	+	+	+	+	+
7	0.0904	7.176	+	+	+		+	+	+	+
7	0.0855	7.444	+	+	+	+		+	+	+
7	0.0841	7.523	+	+	+	+	+	+	+	
7	0.0709	8.253	+		+	+	+	+	+	+
7	0.0291	10.557	+	+	+	+	+	+		+
8	0.0936	9.000	+	+	+	+	+	+	+	+

%CHL = chilling loss, %DL = drip loss, %PY = percentage pumped yield, %ML = percentage moisture loss, %BY = percentage bacon yield, %M = moisture content, FT = fat thickness (mm), MD = meat depth (mm), LMP = percentage predicted lean yield, pH_1 = initial pH, pH_{24} = ultimate pH, %P = percentage protein, %F = percentage fat, Na^+ = sodium content (mg.kg 1)

Table 3 The regression (R^2) model for percentage pumped yield (%PY)

p	R^2	Cp	FT	MD	%F	Na^+	%P	pH_1	pH_{24}	WCM
1	0.0468	-1.788	+							
1	0.0210	-0.348				+				
1	0.0061	0.483							+	
1	0.0040	0.6031					+			
1	0.0019	0.720		+						
1	0.0009	0.776						+		
1	0.0006	0.792								+
1	0.0001	0.822			+					
2	0.0570	-0.355	+				+			
2	0.0562	-0.310	+	+						
2	0.0542	-0.197	+			+				
2	0.0532	-0.142	+						+	
2	0.0506	0.001				+	+			
2	0.0487	0.107	+		+					
2	0.0472	0.193	+					+		
2	0.0471	0.198	+							+
3	0.0842	0.127	+			+	+			
3	0.0673	1.070	+	+		+				
3	0.0638	1.267	+				+		+	
3	0.0627	1.328	+	+			+			
3	0.0611	1.415	+			+			+	
3	0.0596	1.500				+	+		+	
3	0.0596	1.500	+	+					+	
3	0.0573	1.626	+	+	+					

%CHL = chilling loss, %DL = drip loss, %PY = percentage pumped yield, %ML = percentage moisture loss, %BY = percentage bacon yield, %M = moisture content, FT = fat thickness (mm), MD = meat depth (mm), LMP = percentage predicted lean yield, pH_1 = initial pH, pH_{24} = ultimate pH, %P = percentage protein, %F = percentage fat, Na^+ = sodium content (mg.kg 1)

Table 3 (continued) The regression (R^2) model for percentage pumped yield (%PY)

p	R^2	Cp	FT	MD	%F	Na^+	%P	pH_1	pH_{24}	WCM
4	0.0929	1.639	+			+	+		+	
4	0.0919	1.694	+	+		+	+			
4	0.0852	2.069	+			+	+	+		
4	0.0849	2.086	+		+	+	+			
4	0.0842	2.127	+			+	+			+
4	0.0719	2.810	+	+		+		+		
4	0.0707	2.881	+	+		+			+	
4	0.0684	3.009	+	+	+	+				
5	0.0974	3.388	+	+	+	+			+	
5	0.0968	3.424	+			+	+	+	+	
5	0.0947	3.538	+	+		+	+	+		
5	0.0939	3.587	+		+	+	+		+	
5	0.0929	3.638	+			+	+		+	+
5	0.0929	3.641	+	+	+	+	+			
5	0.0921	3.685	+	+		+	+			+
5	0.0860	4.026	+		+	+	+	+		
6	0.1029	5.084	+	+		+	+	+	+	
6	0.0985	5.328	+	+	+	+	+		+	
6	0.0978	5.366	+		+	+	+	+	+	
6	0.0976	5.379	+	+		+	+		+	+
6	0.0969	5.415	+			+	+	+	+	+
6	0.0958	5.480	+	+	+	+	+	+		+
6	0.0952	5.513	+	+		+	+	+		+
6	0.0939	5.586	+		+	+	+		+	+

%CHL = chilling loss, %DL = drip loss, %PY = percentage pumped yield, %ML = percentage moisture loss, %BY = percentage bacon yield, %M = moisture content, FT = fat thickness (mm), MD = meat depth (mm), LMP = percentage predicted lean yield, pH_1 = initial pH, pH_{24} = ultimate pH, %P = percentage protein, %F = percentage fat, Na^+ = sodium content (mg.kg 1)

Table 3 (continued) The regression (R^2) model for percentage pumped yield (%PY)

p	R^2	Cp	FT	MD	%F	Na^+	%P	pH_1	pH_{24}	WCM
7	0.1041	7.015	+	+	+	+	+	+	+	
7	0.1034	7.052	+	+		+	+	+	+	+
7	0.0985	7.326	+	+	+	+	+		+	+
7	0.0978	7.364	+		+	+	+	+	+	+
7	0.0960	7.469	+	+	+	+	+	+		+
7	0.0795	8.388	+	+	+	+		+	+	+
7	0.0728	8.764		+	+	+	+	+	+	+
7	0.0679	9.036	+	+	+		+	+	+	+
8	0.1044	9.000	+	+	+	+	+	+	+	+

%CHL = chilling loss, %DL = drip loss, %PY = percentage pumped yield, %ML = percentage moisture loss, %BY = percentage bacon yield, %M = moisture content, FT = fat thickness (mm), MD = meat depth (mm), LMP = percentage predicted lean yield, pH_1 = initial pH, pH_{24} = ultimate pH, %P = percentage protein, %F = percentage fat, Na^+ = sodium content (mg.kg-1)

Table 4 The regression (R^2) model for percentage moisture loss (%ML)

p	R^2	Cp	FT	MD	%F	Na^+	%P	pH_1	pH_{24}	WCM
1	0.1884	-0.304	+							
1	0.0854	6.639		+						
1	0.0192	11.098			+					
1	0.0120	11.588						+		
1	0.0029	12.200								+
1	0.0027	12.213					+			
1	0.0010	12.331							+	
1	0.0000	12.395				+				
2	0.2061	0.503	+					+		
2	0.2048	0.597	+			+				
2	0.1915	1.490	+	+						
2	0.1898	1.602	+		+					
2	0.1897	1.610	+							+
2	0.1896	1.619	+						+	
2	0.1889	1.668	+				+			
2	0.1095	7.015		+				+		
3	0.2351	0.555	+			+		+		
3	0.2118	2.120	+	+				+		
3	0.2112	2.161	+	+		+				
3	0.2085	2.342	+					+		+
3	0.2084	2.352	+				+	+		+
3	0.2080	2.381	+			+	+			
3	0.2072	2.434	+		+				+	
3	0.2066	2.470	+			+				+

%CHL = chilling loss, %DL = drip loss, %PY = percentage pumped yield, %ML = percentage moisture loss, %BY = percentage bacon yield, %M = moisture content, FT = fat thickness (mm), MD = meat depth (mm), LMP = percentage predicted lean yield, pH_1 = initial pH, pH_{24} = ultimate pH, %P = percentage protein, %F = percentage fat, Na^+ = sodium content ($mg.kg^{-1}$)

Table 4 (continued) The regression (R^2) model for percentage moisture loss (%ML)

p	R^2	Cp	FT	MD	%F	Na^+	%P	pH_1	pH_{24}	WCM
4	0.2492	1.598	+	+		+		+		
4	0.2392	2.276	+			+		+		+
4	0.2371	2.414	+			+	+	+		
4	0.2361	2.485	+		+	+		+		
4	0.2356	2.517	+			+		+	+	
4	0.2168	3.787	+	+			+	+		
4	0.2154	3.881	+	+				+		+
4	0.2142	3.961	+	+		+				+
5	0.2564	3.118	+	+		+		+		+
5	0.2499	3.556	+	+		+	+	+		
5	0.2496	3.574	+	+	+	+		+		
5	0.2492	3.598	+	+		+		+	+	
5	0.2417	4.107	+			+	+	+		+
5	0.2416	4.116	+		+	+		+		+
5	0.2400	4.225	+			+		+	+	+
5	0.2378	4.370	+			+	+	+	+	
6	0.2579	5.016	+	+	+	+		+		+
6	0.2572	5.064	+	+		+	+	+		+
6	0.2564	5.117	+	+		+		+	+	+
6	0.2500	5.549	+	+	+	+	+	+		
6	0.2499	5.556	+	+		+	+	+	+	
6	0.2496	5.574	+	+	+	+		+	+	
6	0.2430	6.040	+		+	+	+	+		+
6	0.2426	6.047	+			+	+	+	+	+

%CHL = chilling loss, %DL = drip loss, %PY = percentage pumped yield, %ML = percentage moisture loss, %BY = percentage bacon yield, %M = moisture content, FT = fat thickness (mm), MD = meat depth (mm), LMP = percentage predicted lean yield, pH_1 = initial pH, pH_{24} = ultimate pH, %P = percentage protein, %F = percentage fat, Na^+ = sodium content ($mg.kg^{-1}$)

Table 4 (continued) The regression (R^2) model for percentage moisture loss (%ML)

p	R^2	Cp	FT	MD	%F	Na^+	%P	pH_1	pH_{24}	WCM
7	0.2581	7.002	+	+	+	+	+	+		+
7	0.2579	7.015	+	+	+	+		+	+	+
7	0.2572	7.061	+	+		+	+	+	+	+
7	0.2500	7.549	+	+	+	+	+	+	+	
7	0.2435	7.984	+		+	+	+	+	+	+
7	0.2253	9.215	+	+	+		+	+	+	+
7	0.2193	9.617	+	+	+	+	+		+	+
7	0.1556	13.907		+	+	+	+	+	+	+
8	0.2581	9.000	+	+	+	+	+	+	+	+

%CHL = chilling loss, %DL = drip loss, %PY = percentage pumped yield, %ML = percentage moisture loss, %BY = percentage bacon yield, %M = moisture content, FT = fat thickness (mm), MD = meat depth (mm), LMP = percentage predicted lean yield, pH_1 = initial pH, pH_{24} = ultimate pH, %P = percentage protein, %F = percentage fat, Na^+ = sodium content (mg.kg 1)

Table 5 The regression (R^2) model for percentage bacon yield (%BY)

p	R^2	Cp	FT	MD	%F	Na^+	%P	pH_1	pH_{24}	WCM
1	0.0237	0.658		+						
1	0.0141	1.205				+				
1	0.0103	1.427	+							
1	0.0093	1.479						+		
1	0.0076	1.577					+			
1	0.0069	1.618			+					
1	0.0021	1.893							+	
1	0.0002	1.999								+
2	0.0547	0.894		+		+				
2	0.0478	1.285				+	+			
2	0.0384	1.821		+				+		
2	0.0343	2.052	+			+				
2	0.0309	2.252				+		+		
2	0.0264	2.509		+	+					
2	0.0258	2.540		+			+			
2	0.0244	2.620		+						+
3	0.0886	0.957		+		+		+		
3	0.0793	1.490		+		+	+			
3	0.0677	2.153	+			+	+			
3	0.0618	2.488				+	+	+		
3	0.0575	2.731		+	+	+				
3	0.0568	2.774	+			+		+		
3	0.0565	2.790	+	+		+				
3	0.0565	2.790		+		+				+

%CHL = chilling loss, %DL = drip loss, %PY = percentage pumped yield, %ML = percentage moisture loss, %BY = percentage bacon yield, %M = moisture content, FT = fat thickness (mm), MD = meat depth (mm), LMP = percentage predicted lean yield, pH_1 = initial pH, pH_{24} = ultimate pH, %P = percentage protein, %F = percentage fat, Na^+ = sodium content (mg.kg 1)

Table 5 (continued) The regression (R^2) model for percentage bacon yield (%BY)

p	R^2	Cp	FT	MD	%F	Na^+	%P	pH_1	pH_{24}	WCM
4	0.1080	1.851		+		+	+	+		
4	0.0937	2.669		+		+		+		+
4	0.0923	2.751		+		+		+	+	
4	0.0906	2.847	+	+		+		+		
4	0.0905	2.853		+	+	+		+		
4	0.0869	3.059	+			+	+	+		
4	0.0824	3.315	+	+		+	+			
4	0.0821	3.332		+		+	+			+
5	0.1141	3.506		+		+	+	+		+
5	0.1129	3.577		+		+	+	+	+	
5	0.1111	3.678	+	+		+	+	+		
5	0.1081	3.846		+	+	+	+	+		
5	0.0983	4.405	+			+	+	+	+	
5	0.0977	4.442		+		+		+	+	+
5	0.0971	4.473		+	+	+		+		+
5	0.0951	4.588	+	+		+		+		+
6	0.1194	5.204		+		+	+	+	+	+
6	0.1172	5.330	+	+		+	+	+	+	
6	0.1165	5.371	+	+		+	+	+		+
6	0.1141	5.504		+	+	+	+			+
6	0.1130	5.571		+	+	+	+	+	+	
6	0.1115	5.655	+	+	+	+	+	+		
6	0.1014	6.229	+		+	+	+	+		+
6	0.1013	6.234		+	+	+		+	+	+

%CHL = chilling loss, %DL = drip loss, %PY = percentage pumped yield, %ML = percentage moisture loss, %BY = percentage bacon yield, %M = moisture content, FT = fat thickness (mm), MD = meat depth (mm), LMP = percentage predicted lean yield, pH_1 = initial pH, pH_{24} = ultimate pH, %P = percentage protein, %F = percentage fat, Na^+ = sodium content ($mg.kg^{-1}$)

Table 5 (continued) The regression (R^2) model for percentage bacon yield (%BY)

p	R^2	Cp	FT	MD	%F	Na^+	%P	pH_1	pH_{24}	WCM
7	0.1229	7.002	+	+		+	+	+	+	+
7	0.1195	7.201		+	+	+	+	+	+	+
7	0.1177	7.300	+	+	+	+	+	+	+	
7	0.1165	7.371	+	+	+	+	+	+		+
7	0.1027	8.154	+	+	+	+		+	+	+
7	0.1015	8.227	+		+	+	+	+	+	+
7	0.0858	9.119	+	+	+	+	+		+	+
7	0.0453	11.431	+	+	+		+	+	+	+
8	0.1230	9.000	+	+	+	+	+	+		+

%CHL = chilling loss, %DL = drip loss, %PY = percentage pumped yield, %ML = percentage moisture loss, %BY = percentage bacon yield, %M = moisture content, FT = fat thickness (mm), MD = meat depth (mm), LMP = percentage predicted lean yield, pH_1 = initial pH, pH_{24} = ultimate pH, %P = percentage protein, %F = percentage fat, Na^+ = sodium content ($mg.kg^{-1}$)

Table 6 The regression (R^2) model for moisture content (%M)

p	R^2	Cp	FT	MD	%F	Na^+	%P	pH_1	pH_{24}	WCM
1	0.1250	3.190						+		
1	0.0143	10.553							+	
1	0.0076	11.001								+
1	0.0030	11.307					+			
1	0.0022	11.358				+				
1	0.0021	11.368		+						
1	0.0010	11.436			+					
1	0.0005	11.468	+							
2	0.1750	1.865						+	+	
2	0.1437	3.945				+		+		
2	0.1401	4.190						+		+
2	0.1268	5.070	+					+		
2	0.1267	5.077			+			+		
2	0.1251	5.182					+	+		
2	0.1251	5.185		+				+		
2	0.0222	12.026							+	+
3	0.2018	2.081				+		+	+	
3	0.1927	2.689						+	+	+
3	0.1771	3.728			+			+	+	
3	0.1770	3.733	+					+	+	
3	0.1759	3.806		+				+	+	
3	0.1753	3.847					+	+	+	
3	0.1623	4.708				+		+		+
3	0.1519	5.405	+			+		+		

%CHL = chilling loss, %DL = drip loss, %PY = percentage pumped yield, %ML = percentage moisture loss, %BY = percentage bacon yield, %M = moisture content, FT = fat thickness (mm), MD = meat depth (mm), LMP = percentage predicted lean yield, pH_1 = initial pH, pH_{24} = ultimate pH, %P = percentage protein, %F = percentage fat, Na^+ = sodium content (mg.kg-1)

Table 6 (continued) The regression (R^2) model for moisture content (%M)

p	R^2	Cp	FT	MD	%F	Na^+	%P	pH_1	pH_{24}	WCM
4	0.2245	2.575				+		+	+	+
4	0.2122	3.391	+			+		+	+	
4	0.2088	3.615				+	+	+	+	
4	0.2029	4.008			+	+		+	+	
4	0.2027	4.022		+		+		+	+	
4	0.1943	4.584	+					+	+	+
4	0.1931	4.665		+				+	+	+
4	0.1930	4.670			+			+	+	+
5	0.2347	3.894	+			+		+	+	+
5	0.2334	3.981				+	+	+	+	+
5	0.2269	4.413		+		+		+	+	+
5	0.2245	4.575			+	+		+	+	+
5	0.2190	4.941	+			+	+	+	+	
5	0.2156	5.168	+		+	+		+	+	
5	0.2140	5.271			+	+	+	+	+	
5	0.2130	5.340	+	+		+	+	+		
6	0.2434	5.317	+			+	+	+	+	+
6	0.2353	5.858	+		+	+		+	+	+
6	0.2349	5.882			+	+	+	+	+	+
6	0.2347	5.893	+	+		+		+	+	+
6	0.2346	5.901		+		+	+	+	+	+
6	0.2288	6.286	+		+	+	+	+	+	
6	0.2270	6.408		+	+	+		+	+	+
6	0.2209	6.812	+	+		+	+	+	+	

%CHL = chilling loss, %DL = drip loss, %PY = percentage pumped yield, %ML = percentage moisture loss, %BY = percentage bacon yield, %M = moisture content, FT = fat thickness (mm), MD = meat depth (mm), LMP = percentage predicted lean yield, pH_1 = initial pH, pH_{24} = ultimate pH, %P = percentage protein, %F = percentage fat, Na^+ = sodium content (mg.kg 1)

Table 6 (continued) The regression (R^2) model for moisture content (%M)

p	R^2	Cp	FT	MD	%F	Na^+	%P	pH_1	pH_{24}	WCM
7	0.2477	7.031	+		+	+	+	+	+	+
7	0.2439	7.285	+	+		+	+	+	+	+
7	0.2365	7.773		+	+	+	+	+	+	+
7	0.2353	7.857	+	+	+	+		+	+	+
7	0.2305	8.177	+	+	+	+	+	+	+	
7	0.1975	10.370	+	+	+		+	+	+	+
7	0.1811	11.461	+	+	+	+	+	+		+
7	0.0542	19.902	+	+	+	+	+		+	+
8	0.2482	9.000	+	+	+	+	+	+	+	+

%CHL = chilling loss, %DL = drip loss, %PY = percentage pumped yield, %ML = percentage moisture loss, %BY = percentage bacon yield, %M = moisture content, FT = fat thickness (mm), MD = meat depth (mm), LMP = percentage predicted lean yield, pH_1 = initial pH, pH_{24} = ultimate pH, %P = percentage protein, %F = percentage fat, Na^+ = sodium content (mg.kg 1)

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