

Effects of production systems on the muscle energy status post mortem and meat quality of beef cattle

By

ELSABE DU TOIT

B.Sc. (Agric) Animal Science, University of Pretoria

Submitted in partial fulfilment of the requirements for the degree

M.Sc. (Agric) Production Physiology

In the Department of Animal and Wildlife Sciences
University of Pretoria

Pretoria
2011

Supervisor: Prof EC Webb
Co-supervisor: Dr L. Frylinck



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I declare that this thesis for the degree M.Sc. (Agric) Production Physiology at the University of Pretoria has not been submitted by me for a degree at any other University.



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“Faith is a journey. The journey is not so much about a destination but a transformation.”
Jim and Rachel Britts

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ACKNOWLEDGEMENTS

I wish to express my sincere gratitude to the following people without whom this study would not have been possible:

Prof. E.C. Webb from the Department of Animal and Wildlife Sciences at the University of Pretoria, who acted as supervisor, for support, guidance and encouragement throughout the study.

Dr. L. Frylinck at the Meat Industry Centre of the Agricultural Research Council, who acted as co-supervisor, for support, advice and guidance throughout the study.

The personnel of the Agricultural Research Council – Animal Production Institute (ARC-API), Irene for assistance with the experiment and analyses of samples. (Dr. P.E. Strydom, H. Snyman and J. Anderson).

The Red Meat Research and Development South Africa (RMRD SA) for financial support.

Technology and Human Resources for Industry Programme (THRIP) for financial support.

My friends and family for moral support en encouragement throughout the study.

LIST OF ABBREVIATIONS

AF	- A age classification animals reared at a feedlot
AP	- A age classification animals reared on pasture
ABF	- AB age classification animals reared at a feedlot
ABP	- AB age classification animals reared on pasture
ADP	- Adenosine diphosphate
AMP	- Adenosine monophosphate
ATP	- Adenosine triphosphate
ARC	- Agricultural Research Council
API	- Animal Production Industry
BP	- B age classification animals reared on pasture
Br-X	- Brahman cross bred animals
CAI	- Calpain I
CAII	- Calpain II
CC	- Cold carcass weight
CIE	- Commission International De l' Eclairage
CP	- Creatine phosphate
DFD	- Dark, firm and dry
EDTA	- Ethylenediaminetetra-acetic acid
F	- Feedlot
Glu	- Glucose
G6P	- Glucose-6-phosphate
Glyc	- Glycogen
GlycP	- Glycolytic potential
Insol	- Insoluble collagen
LD	- Muscle <i>Longissimus dorsi</i>
Lakt	- Lactic acid
MFL	- Myofibril fragment length
Ng-X	- Nguni cross bred animals
P	- Pasture
pI	- Iso-electrical point
PML	- Percentage weight loss
PSE	- Pale, soft exudative
Sac	- Sarcomere
Si-X	- Simmental cross bred animals
Sol	- Soluble collagen
SolInsol	- Total collagen
SpesCAI	- Specific calpain I
SpesCAII	- Specific calpain II



SpesIN	- Specific calpastatin
SD	- Standard deviation
Temp	- Temperature
TotIN	- Total calpastatin
WC	- Warm carcass weight
WBSF	- Warner-Bratzler shear force
WBC	- Water holding capacity
\bar{X}	- Mean

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ABSTRACT

This study was part of a larger study to develop an animal production model to obtain the optimum beef tenderness. There is a variety of different beef production systems being used in South Africa. The production systems investigated in this study were; animals reared on pasture until A age classification group (AP), animals reared on pasture until AB age classification group (ABP), animals reared on pasture until B age classification group (BP), animals reared at a feedlot until A age classification group (AF) and animals reared at a feedlot until AB age classification group (ABF). These production systems affect a wide range of components in the muscle that contribute to meat quality. Meat quality refers mainly to tenderness and colour. Scientists and industry role players are uncertain of which production system is the best to produce high quality meat. The aim of the study was to determine the effects of different production systems on the post slaughter muscle energy metabolism and the related effects on meat quality (tenderness and colour).

In the experiment 180 steers of the following breed crosses were used: Nguni, Simmental and Brahman. These animals were reared until they reached the A, AB or B age classification, either on pasture or in a feedlot (B only on pasture). All the carcasses were electrically stimulated for approximately 15 sec, before analyses were done. The following properties were measured on the animals after slaughter: tenderness (Warner-Bratzler shear force), carcass pH, carcass temperature, sarcomere lengths, myofibrillar fragmentation length, calpain activity, muscle lactic acid concentration, muscle glycogen concentration, muscle ATP concentration, muscle glucose-6-phosphate concentration, muscle creatine phosphate concentration, muscle glucose concentration, water holding capacity, drip loss and meat colour. These measurements and determinations were done according to standard laboratory procedures at the ARC (Agricultural Research Council) at Irene.

Breed had no effect on the muscle energy status for muscle glycolytic potential, muscle lactic acid concentration, muscle glucose concentration, muscle glycogen concentration, muscle glucose-6-phosphate concentration, muscle ATP concentration and creatine phosphate concentration ($p>0.05$). Breed only had an influence on the tenderness at 7 days post mortem ($p<0.05$).

Older animals from the pasture had lower muscle energy levels than younger animals from the feedlot. Animals from the BP production system had the darkest colour meat with the highest hue angle. AF production system animals had the lightest colour meat with the lowest hue angle and the highest chroma. ABF production system animals had the lowest chroma. Animals from the ABF production system had the lowest shear force value at 1, 7 and 14 days post mortem and

animals from the AP production system had the highest shear force value at 1, 7 and 14 days post mortem.

This study showed that the energy status in the muscle post mortem does not influence the tenderness of the meat nor the colour ($p>0.05$). Shear force had a weak to medium positive correlation with muscle pH (between 0.186 and 0.410) and a weak to medium negative correlation with muscle temperature (between -0.157 and -0.268) ($p<0.05$). Muscle lactic acid concentrations (between -0.033 and -0.322), muscle glucose concentrations (between -0.066 and -0.155) and sarcomere length (between -0.276 and -0.326) had a weak to medium negative correlation with shear force ($p<0.05$). Muscle glycogen concentrations (between 0.026 and 0.166) and myofibrillar fragment length (between 0.248 and 0.447) had a weak to medium positive correlation with shear force ($p<0.05$). Shear force had a weak positive correlation with calpastatin activity (between 0.064 and 0.253) and a weak to medium negative correlation with calpain I activity (between -0.183 and -0.313) ($p<0.05$). The ratio of calpastatin: calpain I (between 0.323 and 0.348) and the ratio of calpastatin: calpain I + II (between 0.183 and 0.275) had a weak to medium positive correlation with shear force ($p<0.05$).

Breed had no effect on the muscle energy status for muscle glycolytic potential, muscle lactic acid concentration, muscle glucose concentration, muscle glycogen concentration, muscle glucose-6-phosphate concentration, muscle ATP concentration and creatine phosphate concentration ($p>0.05$).

If electrical stimulation was not used in this study the difference between the production systems in terms of muscle energy status and colour would have been more prominent. The conclusion is that if animals are slaughtered under “ideal” circumstances in terms of stress being kept to a minimum before slaughter and the carcasses are electrically stimulated in order to prevent cold shortening, the production system shows a small effect on the energy status of the animal and consequently also levels out the meat quality characteristics such as tenderness and colour. For more dramatic results and academic value it would have been more useful to include more variations of non-ideal slaughter conditions and non-electrical stimulation, as well as more breeds. A follow up study with no electrical stimulation can be helpful to explain some uncertainties. This follow up study will present its own challenges, for example higher frequency of DFD.

A follow up study on the effects of a larger variety of breeds can help to determine the exact effect of muscle energy metabolites in the different breeds, on the tenderness and colour of the meat.

CHAPTER 1

INTRODUCTION

This study was part of a larger study to develop an animal production model to obtain the optimum beef tenderness. There is a variety of different beef production systems being used in South Africa. The production systems used in this study were; animals reared on pasture until A age classification group (AP), animals reared on pasture until AB age classification group (ABP), animals reared on pasture until B age classification group (BP), animals reared at a feedlot until A age classification group (AF) and animals reared at a feedlot until AB age classification group (ABF) (see Appendix A for the RSA meat classification system). For commercial purposes the pasture animals are sold at AB or B age class according to the red meat classification system, whereas the feedlot animals are sold at A or AB age class according to the same classification system. The AP production system was included in this study to compare with the AF production system. There was no BF production system to compare with the BP production system in this study, because it is too expensive to keep the animals until they reach the appropriate age. These production systems affect a wide range of components in the muscle that contribute to meat quality. Meat quality refers to tenderness, colour, juiciness, flavour and odour. This study mainly focused on tenderness and colour. Scientists cannot decide which production system is the best to produce high quality meat. The aim of the present study was to determine the effects of different production systems on the post slaughter muscle energy metabolism and its effects on meat quality (tenderness and colour).

One of the properties the consumer expects of beef is tenderness. The producer must try and produce a meat product of consistent tenderness. In order to do this, the factors involved in tenderness must be known as well as how these factors can be manipulated.

The colour of the meat is the main characteristic meat can be judged on. Approximately 15% of retailed meats are discriminated against due to colour. The consumer links poor quality meat with a dark colour or a very light colour. The dark coloured meat is associated with a condition or phenomenon called dark, firm and dry (DFD) meat, where the pH of the meat does not drop below 5.8. DFD meat has variable tenderness and the shelf life is shorter than normal meat. Very light coloured meat is due to a pale, soft exudative (PSE) like condition. This condition usually occurs in beef when the meat is electrically over stimulated. PSE meats can either result in extremely tender meat or extremely tough meat (Mancini and Hunt 2005).

The main reason consumers do not like to buy dark coloured meat, is because dark coloured meat is associated with meat which is not fresh. As the meat gets older more of the dark (brown) metmyoglobin pigment accumulates in the meat. Metmyoglobin is produced when oxymyoglobin is

converted to metmyoglobin through the process of reduction (Mancini and Hunt, 2005, Muchenje, Dzama, Chimonyo, Raats and Strydom, 2009b; Abril, Campo, Önenç, Sañudo, Albertí, and Negueruela, 2001, Lawrie, 1974; Varnam and Sutherland, 1996).

The tenderness of meat is influenced by a variety of factors. All of these factors play a role in the end result (tenderness). They start on the farm, with feeding and handling. The next step is the transport of the animals to the abattoir. At the abattoir, they include the treatments before and after slaughter. The animal's age, gender and breed influence tenderness, as well as the weather on the day of slaughter. For optimum tenderness the amount of stress experienced by the animals should be minimized (Jelenikova, Pipek and Staruch, 2008; Lawrie, 1974; Hollung, Veiseth, Froystein, Aass, Langsrud, and Hildrum, 2007; Muchenje, Dzama, Chimonyo, Raats and Strydom, 2009a).

The conversion of muscle to meat involves a complex set of interrelated occurrences. Just after slaughter the blood circulation stops, therefore the oxygen and nutrient supply stops, as well as the removal of metabolic waste. But muscle metabolism does not stop directly after slaughter. The muscles are still trying to maintain tonicity, by utilizing energy. As there is no oxygen, this process takes place under anaerobic conditions, causing the production of lactic acid. Lactic acid is acidic and therefore the pH of the muscle falls, as the concentration of lactic acid increases. As the conversion continues calcium is released into the sarcoplasm. The drop in pH, as well as the increase in the calcium concentration in the muscle cytoplasm stimulates the activation of calpains.

Calpains are enzymes that breakdown myofibrils. There are two different calpains; calpain I and calpain II, also known as μ -calpain and m-calpain. Calpastatin another protein in the sarcoplasm, inhibits the action of calpains. Therefore the ratio between calpains and calpastatin is also important for the tenderisation of the meat.

ATP is required for muscle contraction and relaxation. After death the amount of ATP in the muscle starts to decrease. As the level of ATP decreases, more actomyosin forms, that cannot be released again. This is the onset of rigor mortis (Pulford, Fraga Vazquez, Frost and Fraser, 2008; Lawrie, 1974; Varnam and Sutherland; 1996, Forrest, Aberle, Hedrick, Judge and Merkel, 1996; Dransfield, 1994).

CHAPTER 2

LITERATURE REVIEW

A variety of different beef production systems are used in South Africa. These production systems are divided into animals reared on grass and those reared in a feedlot. They can be divided further according to the different ages that the cattle are sold at to the abattoir. For commercial purposes the grass fed animals are generally sold at AB or B age class, according to the red meat classification system (see Appendix A), whereas the feedlot animals are sold at A or AB age groups according to the same classification system. These production systems affect a variety of inherent muscle characteristics that contribute to the normal conversion of muscle to meat and thus meat quality. Meat quality refers to tenderness, colour, juiciness, flavour and odour. This study mainly focused on tenderness and colour.

There is an extensive discussion among scientists and industry role players, about which one of the production systems produces the best quality meat. It is said that meat from grass fed animals is healthier, because the meat contains more antioxidants than that of feedlot animals. It is, however, also said that meat from grass fed animals is tougher than that of feedlot animals, because animals that are grass fed get more exercise (Muchenje, Dzama, *et al.*, 2009b) and are usually older when ready for the market. Which of these opinions are correct? The purpose of this study was to evaluate the effects of beef production system on meat quality.

According to Mancini and Hunt (2005) nearly 15 % of retail beef is discounted in price due to surface discolouration. Surface discolouration is associated with poor meat quality and has to be prevented. The discolouration is mainly due to the very high ultimate pH values of the meat (pH of more than 5.8). Carcass pH is affected by the energy available in the muscle at the time of slaughter.

Meat tenderness is receiving attention, because consumers want consistent meat tenderness. There are many physiological factors that influence meat tenderness. This study will therefore also focus on muscle energy metabolism and the related effects on meat tenderness.

Different beef production systems influence the tenderness, juiciness, flavour and colour of beef. Flavour is only of importance to the consumer if there are off flavours (Viljoen, De Kock and Webb, 2002; Lawrie, 1974; Varnam and Sutherland, 1996). Water holding capacity is one of the most important factors that influence juiciness of meat upon mastication. This is defined as the ability of meat to retain its water during the application of external forces, such as cutting, heating, grading, or pressing (Muchenje, Dzama, *et al.*, 2009b; Lawrie, 1974). Higher water holding capacity correlates positively with the juiciness of meat. The water in the muscle is mainly held by capillary forces between thick and thin filaments. In these filaments the proteins, actin and myosin

are important in the formation of a protein network, necessary for binding the water and fat in further processing (Muchenje, Dzama, *et al.*, 2009b).

The pH of the meat influences the water holding capacity of the meat (Muchenje, Dzama, *et al.*, 2009b; Lawrie, 1974). This is important for meat processing because, as proteins are able to hold more water, they become more soluble (Muchenje, Dzama, *et al.*, 2009b). Enzymes are included in the protein fraction and require water to function.

When the iso-electrical point (pI) of the proteins is reached the water holding capacity is at a minimum (Muchenje, Dzama, *et al.*, 2009b; Lawrie, 1974). At the iso-electrical point there are equal positive and negative charges on the amino acid side chains, resulting in a maximum number of salt bridges between peptide chains and the net charge is zero (Muchenje, Dzama, *et al.*, 2009b). The iso-electrical point of meat proteins is at the pH between 5.0 and 5.5; this is at the pH of normal meat (after 24 hours) (Muchenje, Dzama, *et al.*, 2009b; Lawrie, 1974; Varnam and Sutherland, 1996). An increase or a decrease of the pH away from the pI will result in a higher water holding capacity. This is caused by an increase in available negative or positive charges on the protein structure (Muchenje, Dzama, *et al.*, 2009b). Meat with a high pH has a higher water holding capacity than normal meat (Muchenje, Dzama, *et al.*, 2009b; Lawrie, 1974).

Drip loss is the loss of fluid from the shrinkage of muscle protein in the form of drip. The exposure of proteins to low pH at high temperatures causes less water to be retained between the actin and myosin filaments, therefore increasing drip loss. This typically occurs during the PSE condition. Temperature also influences the pI of the meat. A high carcass temperature and low pH post mortem is associated with a higher metabolism and breakdown of proteins, therefore the higher the breakdown the higher the drip loss. Some other factors influencing drip loss are glycolytic metabolism, difference in collagen structure and intermuscular fat content (Muchenje, Dzama, *et al.*, 2009b).

2.1 Colour of meat

Colour is the first meat characteristic through which the consumer can judge if the meat quality is acceptable. The colour of the meat plays an important role in the meat industry (Mancini and Hunt, 2005; Muchenje, Dzama, *et al.*, 2009b; Abril, Campo, *et al.*, 2001; Lawrie, 1974; Varnam and Sutherland, 1996). According to Mancini and Hunt (2005) nearly 15 % of retail beef is discounted in price due to surface discolouration. Colour is an indication of freshness (Mancini and Hunt, 2005; Muchenje, Dzama, *et al.*, 2009b, Abril, Campo, *et al.*, 2001).

Myoglobin is the pigment in meat that is mainly responsible for the colour of meat (Mancini and Hunt, 2005; Muchenje, Dzama, *et al.*, 2009b; Lawrie, 1974; Varnam and Sutherland, 1996). But light reflection also plays a role in the colour. Meat with a low light reflection appears darker

than a meat sample with a higher light reflection (Abril, Campo, *et al.*, 2001; Lawrie, 1974; Varnam and Sutherland, 1996; Forrest, Aberle, *et al.*, 1973). Diet has an effect on muscle colour, by altering the glycogen store, chilling rate and antioxidant accumulation. All of the above factors influence intrinsic colour traits, pH, oxygen consumption and metmyoglobin reducing activity (Mancini and Hunt, 2005). There is a small amount of hemoglobin in the muscle that can provide colour (Muchenje, Dzama, *et al.*, 2009b; Abril, Campo, *et al.*, 2001).

Myoglobin is fixed to cell tissues of the muscle and does not circulate in the blood (Muchenje, Dzama, *et al.*, 2009b; Abril, Campo, *et al.*, 2001; Lawrie, 1974). Different states of myoglobin occur in the muscle, each with a different colour. They include deoxymyoglobin, oxymyoglobin, metmyoglobin and carboxymyoglobin. Deoxymyoglobin occurs when there is a very low oxygen level present, like in vacuum sealed meat. The colour associated with deoxymyoglobin is purplish-red or purplish-pink. Oxymyoglobin occurs in the presence of oxygen (Mancini and Hunt, 2005; Lawrie, 1974). The colour associated with oxymyoglobin is cherry red (Mancini and Hunt, 2005; Muchenje, Dzama, *et al.*, 2009b; Lawrie, 1974). Metmyoglobin in the meat is just below the oxymyoglobin layer and just above the deoxymyoglobin layer. The colour associated with metmyoglobin is brown (Mancini and Hunt, 2005; Lawrie, 1974). Not a lot is known about carboxymyoglobin, all that is known are that carbon dioxide plays a role in its formation (Mancini and Hunt, 2005).

The different amounts and proportion of myoglobin will determine the exact colour of the meat (Lawrie, 1974). As meat age, the proportion of myoglobins changes to form a more brown coloured meat (Mancini and Hunt, 2005). Bloom time will also influence the proportion of myoglobins (Wulf and Wise, 1999).

The pH of the meat, meat structure and physical state of the muscle will mainly influence the light reflection of the meat (Abril, Campo, *et al.*, 2001; Lawrie, 1974; Varnam and Sutherland, 1996; Forrest, Aberle, *et al.*, 1973). High water holding capacity results in a high light absorption, resulting in a dark coloured meat. High water holding capacity is associated with a high pH (therefore DFD meat). Low water holding capacity is associated with a PSE meat. This low water holding capacity results in high light reflection, therefore a light coloured meat (Abril, Campo, *et al.*, 2001; Varnam and Sutherland, 1996). Bloom can be defined as the process of exposing the meat to air after removing the meat sample from its packaging, to restore its natural colour.

Colour measurements are done using the Commission International De l' Eclairage (CIE) colour system. The three fundamental colour coordinates are L*, a* and b*. The L* measures the lightness and is a measure of the light reflection (100 = white, 0 = black), a* measures positive red and negative green and b* measures positive yellow and negative blue. Spectral colour (hue angle) and colour saturation (chroma) can be calculated from the a* and b* coordinates (Young,

Priolo, Simmons and West, 1999; Muchenje, Dzama, *et al.*, 2009b). Hue angle and Chroma is calculated as follows: hue angle = $\tan^{-1}(b^*/a^*)$ and Chroma = Square root of $(a^{*2} + b^{*2})$.

2.2 Tenderness of meat

Tenderness has been identified as the most important factor determining the consumer - eating satisfaction of beef (Jelenikova, Pipek, *et al.*, 2008; Lawrie, 1974). Tenderness is determined by a variety of factors. All these factors are related to each other. Factors influencing tenderness starts at the farm level. If the animals are treated correctly, then there is a greater possibility that the meat will be tender. The next place where tenderness is influenced is when the animals are transported to the abattoir, followed by the abattoir and then during and after slaughter. The animal's age, gender, breed and genetics play an important role in tenderness as well as the weather conditions before slaughter (Hollung, Veiseth, *et al.*, 2007; Muchenje, Dzama, *et al.*, 2009a; Muchenje, Dzama, *et al.*, 2009b, Jelenikova, Pipek, *et al.*, 2008). Generally, females and castrates have more tender meat than bulls (Jelenikova, Pipek, *et al.*, 2008).

On the farm the animals must be handled frequently so that they are used to handling. If they are not handled frequently, then they will be wild and use more energy (due to stress) than animals that are used to handling, during transport and handling at the abattoir (Kadim, Mahgoub, Al-Ajmi, Al-Maqbaly, Al-Mugheiry and Bartolome, 2004). The nutrition on the farm also affects the tenderness to some degree (Muchenje, Dzama, *et al.*, 2009b). All of the above components differ amongst the different production systems.

During transport the animals must not be mixed, they must have enough space to move around in, but not too much. Mixing the animals will decrease carcass quality, due to stress. The distance and time that the animals travel, may influence the tenderness of the meat. The longer the distance and time the animals travel the more stress is experienced by the animal. Stress will decrease the meat tenderness.

At the abattoir the animals should not be mixed, because mixing causes stress in the animals (Jelenikova, Pipek, *et al.*, 2008). The amount of time that animals are without food will influence the tenderness of the meat. The animals should always have some water to drink, or they will dehydrate severely (Hollung, Veiseth, *et al.*, 2007). Dehydration causes stress. The animals must be handled gently to prevent as much stress as possible (Silva, Patarata and Martins, 1999). Another important factor at the abattoir is the weather conditions. If the weather is hot more meat defects occurs, such as DFD meat (Kadim, Mahgoub, *et al.*, 2004).

The animals must be slaughtered in such a way to minimize stress. Most abattoirs use a captive bolt to stun the animals before the jugular vein is cut.

There are a large number of factors that influence meat tenderness after slaughter. These include the use of electrical stimulation (Janz, Aalhus and Price, 2001; Hwang, Devine and Hopkins, 2003; Pearce, Hopkins, Williams, Jacob, Pethick and Phillips, 2009; Hollung, Veiseth, *et al.*, 2007; Rosenveld, North, Devine, Micklander, Hansen, Dobbie and Wells, 2008; Muchenje, Dzama, *et al.*, 2009b), the temperature of storage and conditioning, humidity during storage and conditioning, whether hot boning is used, whether wrapping is used and the time the meat is matured/conditioned (Marsh, Ringkob, Russell, Swartz and Pagel, 1987; Rosenveld and Andersen, 2003; Rosenveld, North, *et al.*, 2008; Jelenikova, Pipek, *et al.*, 2008; Simmons, Daly, Mudford, Richards, Jamison Pleiter, 2006, Hwang and Thompson, 2001; Devine, Payne, Peachey, Lowe, Ingram and Cook, 2002). The cooking process also plays an important role in the ultimate tenderness of the piece of meat.

Stress has a large effect on tenderness (Hwang, Devine, *et al.*, 2003; Tornberg, 1996; Scanga, Belk, Tatum, Grandin and Smith, 1998; Forrest, Aberle, *et al.*, 1973). To obtain a tender piece of meat stress must be minimized.

There are two types of stress that play a role in tenderness. These are long term and short term stress. If long term stress occurs, the meat from such cattle will be darker than that the meat normal cattle. This condition is termed dark, firm and dry (DFD) meat. But when short term stress occurs, the meat from such animals appears pale and wet. This is called pale, soft and exudative (PSE) meat. PSE usually occurs in pigs and poultry. It seldom occurs in beef and lamb. Short term stress anti mortem will seldom course PSE in beef. These two conditions have different effects on the tenderness of the meat; these types of meat are not preferred by the consumer (Tornberg, 1996; Scanga, Belk, *et al.*, 1998; Forrest, Aberle, *et al.*, 1973).

Stress as well as the feeding ration shortly before slaughter will effect the amount of energy in the muscle before slaughter. The energy is important in the tenderisation process (O'Halloran, Troy and Buckley, 1997; Rosenveld and Andersen, 2003; Hollung, Veiseth, *et al.*, 2007, Muchenje, Dzama, *et al.*, 2009a; Hwang and Thompson, 2001; Lawrie, 1974, Forrest, Aberle, *et al.*, 1973). If the energy levels are not correct, the ultimate pH will not be correct for optimum tenderness (Thompson, 2002; Kadim, Mahgoub, *et al.*, 2004; Jelenikova, Pipek, *et al.*, 2008; Bekhit, Farouk, Cassidy and Gilbert, 2007; Lawrie, 1974).

The following are indicators of tender meat; long sarcomere lengths, large number of myofibril fragments, short myofibril fragment length, an ultimate pH (pHu) of below 5.5, an intermediate pH drop, low levels of connective tissue, rapid glycolysis, high water holding capacity and low drip loss and high calpain activity (Marsh, Ringkob, *et al.*, 1999; Purchas and Aungsupakorn, 1993; O'Halloran, Troy, *et al.*, 1997; Rosenveld and Andersen, 2003; Gratacos-Cubarsi and Lametsh, 2008; Pearce, Hopkins, *et al.*, 2009; Hollung, Veiseth, *et al.*, 2007; Rosenveld, North, *et al.*, 2008; Thompson, 2002; Hannula and Puolanne, 2004; Muchenje,

Dzama, *et al.*, 2009a; Muchenje, Dzama, *et al.*, 2009b; Kadim, Mahgoub, *et al.*, 2004; Jelenikova, Pipek, *et al.*, 2008; Pulford, Fraga Vazquez, *et al.*, 2008; Hwang and Thompson, 2001; Devine, Payne and Wells, 2002b; Lawrie, 1974; Varnam and Sutherland, 1996). There is a long standing debate on whether water holding capacity and fat content have an effect on tenderness.

Electrical stimulation is used to increase tenderness by preventing cold shortening through the depletion of energy in the muscles. The energy decrease causes the pH to reach its ultimate pH sooner, preventing the occurrence of cold shortening (Hwang and Thompson, 2001; Devine, Payne, Peachey, *et al.*, 2002a).

Cold shortening occurs when muscle temperature drops below 10 °C while the pH is still above 6.1 (Muchenje, Dzama, *et al.*, 2009b). The sarcomere length is short, because of a higher proportion of actomyosin. This condition is irreversible and the meat will be tough (Muchenje, Dzama, *et al.*, 2009b; Devine, Payne, *et al.*, 2002a; Forrest, Aberle, *et al.*, 1973).

The type of electrical stimulation influences the effect on the meat. There is a difference between high, medium and low voltage electrical stimulation. The duration of electrical stimulation also plays a role in the effect of electrical stimulation on the meat tenderness. According to the literature medium voltage to low voltage electrical stimulation, with medium duration stimulation produces the optimum result (Hwang, Devine, *et al.*, 2003; Hollung, Veiseth, *et al.*, 2007).

Tenderness can be measured in two ways, objectively or subjectively. Objectively by using a Warner-Bratzler shear force (WBSF) test (Tornberg, 1996; Thompson, 2002; Muchenje, Dzama, *et al.*, 2009b) and subjectively through a sensory taste panel. The sensory panel will normally evaluate meat attributes (like example tenderness) on a scale of 1 to 10 (with 1 very poor and 10 very good) for the following; softness on tongue and cheek, resistance to tooth pressure, ease of fragmentation, mealiness, adhesion and residue after chewing (Forrest, Aberle, *et al.*, 1973). The following are normal ranges of a sensory score of some meat quality characteristics (Table 2.1) (Muchenje, Dzama, *et al.*, 2009b).

Table 2.1 Normal ranges of sensory scores of some meat quality characteristics (Muchenje, Dzama, *et al.*, 2009b).

Meat quality characteristics	Sensory score (0= lowest, 10= highest)
Taste at 2 days	4.70 – 5.50
Taste at 14 days	5.80
Aroma at 2 days	5.21 – 5.70
Aroma at 21 days	5.02 – 5.70
Juiciness at 2 days	3.30 – 6.60
Juiciness at 21 days	4.38 – 5.60
Flavour at 2 days	3.10 – 5.89
Flavour at 21 days	5.39 – 5.93
Tenderness at 2 days	2.10 – 6.40
Tenderness at 21 days	5.50 – 6.47
Residual at 2 days	4.19 – 4.98
Residual at 21 days	4.21 – 4.76
Overall acceptability at 2 days	1.80 – 5.65
Overall acceptability at 21 days	4.26 – 4.96

2.3 Muscle structure

Figure 2.1 illustrates the structure of a muscle cell. Muscles are surrounded by a connective tissue sheath, known as the epimysium (Varnam and Sutherland, 1996; Forrest, Aberle, *et al.*, 1996). From the inner surface of the epimysium, connective tissue penetrates in the muscle, separating the muscle fibre into bundles (Lawrie, 1974; Varnam and Sutherland, 1996). This penetrating connective tissue constitutes the perimysium that contains the blood vessels and nerves. From the perimysium, a fine connective tissue framework passes further inwards to surround each individual muscle fibre (Lawrie, 1974). This is called the endomysium (Lawrie, 1974; Varnam and Sutherland, 1996).

The size of the muscle fibre bundles determines the texture of the muscle. The muscle fibres do not attach directly to the bone, which they move or in relation to which their force is exerted. The endomysium, perimysium and epimysium blend with massive aggregates of connective tissue (or tendons) and these attach to the skeleton. This connective tissue holds and supports the muscle. It is probable that all substances passing into or out of muscle cells diffuse through some type of connective tissue, resulting in a highly intimate relationship between muscle cells and connective tissue (Lawrie, 1974; Varnam and Sutherland, 1996).

The connective tissue includes formed elements and an amorphous ground substance in which the formed elements are embedded. The amorphous ground substance is rich in proteoglycans and glycoproteins. Various types of cells are present in the amorphous substance, fibroblasts, macrophages, mast cells and fat cells. From the fibroblasts the fibrous proteins collagen and elastin are synthesized. The formed elements consist of the fibres of collagen, elastin and reticulin. Collagen fibres are straight, inextensible and non-branching. These fibres are the major determinant of texture of cooked meat, the quality of the collagen rather than the quantity being important. Collagen, a protein of connective tissue, is converted into the water soluble fraction, when it is heated above 80°C. Elastin fibres are elastic, branching and yellow in colour. The quantity of elastin in muscles is very low. Reticulin fibres resemble collagen, but are associated with substantial quantities of a lipid containing myristic acid. Reticulin is stained black by ammoniacal silver solution in contrast to collagen, which stains brown (Lawrie, 1974; Varnam and Sutherland, 1996).

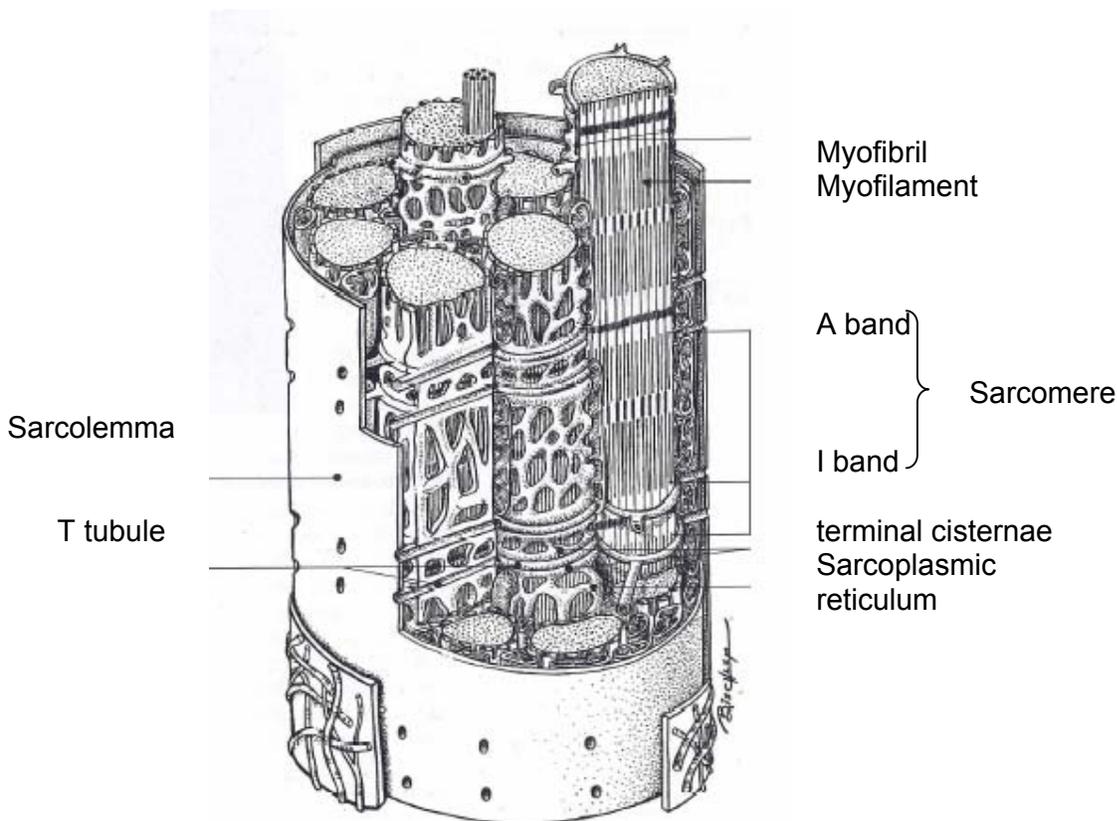


Figure 2.1: The structure of a muscle cell as edited from Frandson, Wilke and Fails (2003)

The properties of collagen fibres, and to a lesser extent elastin fibres, have a large impact on the structural stability in muscles (Varnam and Sutherland, 1996). The hierarchy of the

structural elements of collagen is as follows; primary fibre, fibril, filament and then protofibril. The protofibrils are composed of three helically interwoven polypeptide chains (Lawrie, 1974).

The essential structural unit of all muscles is the fibres. The fibres are long, narrow, multinucleated cells that stretch from one end of the muscle to the other and have a diameter of between 10 and 100 μm . There are many factors influencing the diameter of the muscle fibre, including species, genetics, age, breed, gender and plane of nutrition. A given muscle contains fibres of different diameters, with the smaller ones more on the outside and the larger ones more in the middle of the muscle (Lawrie, 1974).

Underneath the endomysium and surrounding the muscle fibre, is a sheath, the sarcolemma. The sarcolemma is a double membrane. The sarcolemma transmits the forces of the myofibrils to connective tissue structure in the contraction process of the muscle. The myofibrils are situated in the sarcolemma, surrounded by a fluid phase, the sarcoplasm (Lawrie, 1974).

Figure 2.2 illustrate a light micrograph and electron micrograph of a skeletal muscle, as well as a schematic representation of a sarcomere. The myofibrils have dark and light striations. The dark A-band has a central lighter area called the H-zone and the light I-band has a central dark area called the Z-line. A sarcomere is the difference between two adjacent Z-lines. It is the functional unit of the myofibril. The myofibrils are composed of numerous parallel filaments. These filaments are divided into thick and thin filaments (Lawrie, 1974; Varnam and Sutherland, 1996).

The thick filaments consist mainly of a protein named myosin, which consist of a head and a tail. The heads of the myosin are towards the Z-line and the tails are towards each other. The thin filaments consist mainly of the protein actin (Lawrie, 1974; Varnam and Sutherland, 1996). Actin filaments continue through the Z-line, but do not transverse the H-zone, which bound each sarcomere. The myosin filaments on the other hand transverse only through the A-band (Lawrie, 1974). Actin and myosin are the primary proteins responsible for the contraction of muscles.

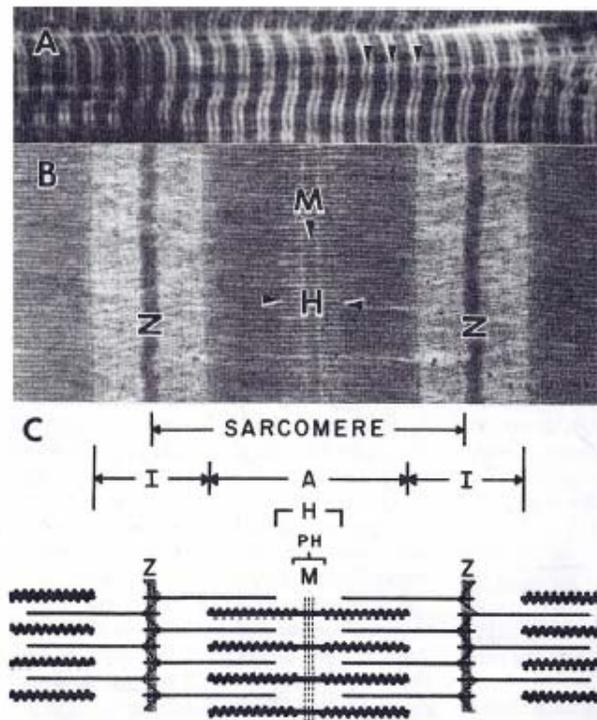


Figure 2.2: A light micrograph (A) and electron micrograph (B) of a skeletal muscle, as well as a schematic representation (C) of a sarcomere (Frandsen, Wilke, et al., 2003).

The proteins in the muscle can be divided into water soluble, those that are soluble in salt solution and insoluble proteins that are not soluble in salt solution. The water soluble proteins are mainly those from the sarcoplasm. The proteins that are soluble in salt are mainly the myofibrillar proteins and the insoluble proteins are mainly proteins of connective tissue and other formed structures. The sarcoplasmic proteins (myogen and globulins) are known to represent a complex mixture of about 50 components, many of which are enzymes in the glycolytic cycle. Almost all the sarcoplasmic proteins have been crystallized (Lawrie, 1974).

Myosin is the most abundant of the myofibrillar proteins. It has a strong affinity for magnesium and calcium ions and has ATPase and actin-binding properties. It is suggested that the actin filaments are attached to the Z-line by a meshwork of tropomyosin and tropomyosin extending along the helical groove in the actin filament. The interaction of actin, myosin and ATP is extremely complex (Lawrie, 1974).

Troponin promotes the aggregation of tropomyosin and prevents the formation of actomyosin. It is composed of two factors that are involved in the contraction of muscles, a calcium-sensitizing factor and inhibiting factor. The protein α -actinin promotes the lateral association of F-actin and β -actinin inhibits polymerization of F-actin (Lawrie, 1974).

2.4 Muscle contraction

For a muscle in rest in the living animal, the beads composing the actin filaments are prevented from binding on the corresponding projections of the myosin by the magnesium complex of ATP. ATP is very important for muscle functioning and therefore the supply of ATP is important. To supply the ATP, both soluble and insoluble proteins play a role. The most immediate source to regenerate ATP is from the reaction with creatine phosphate and ADP. This reaction is controlled by the enzyme creatine kinase, which is soluble in the sarcoplasm (ADP + creatine phosphate \leftrightarrow creatine + ATP). The main source of ATP regeneration in the living animal occurs when ADP is converted to ATP, through the oxidation of glucose to carbon dioxides and water. But when ATP is required in larger amounts that cannot be maintained by the normal respiration system or when oxygen levels are low, ATP is regenerated by means of the conversion of glycogen to lactic acid. The conversion of glycogen to lactic acid is a very inefficient way of generating ATP (Lawrie, 1974).

The energy required for contraction of muscles is derived from hydrolysis of ATP, catalyzed by ATPase in the myosin heads (Varnam and Sutherland, 1996). There is also ATPase in the sarcoplasm; it functions at a slower rate than that of ATPase in the myosin head. Its function is mainly to maintain the muscle tone and body temperature (Lawrie, 1974).

Muscle contraction is initiated by the release of calcium ions from the sarcoplasmic reticulum in response to a nerve impulse (Varnam and Sutherland, 1996). The nerve impulse arrives at the motor end plate and cause a depolarising of the sarcolemma. This causes a release of calcium ions from the sarcolemma and sarcotubular system, mainly at the region of the Z-line. A highly acidic protein is responsible for the storage of calcium in the sarcoplasmic reticulum. The calcium diffuses and forms firm chelate links between the bound ADP of the F-actin filament and the ATP which is positioned to be bound at the end of an asymmetrical extended, rapidly snaking polypeptide, which is part of the myosin filament (Lawrie, 1974).

Formation of the calcium link neutralizes the residual electrical charge of the bound ATP so that the polypeptide can now spontaneously twist and contract to form an α -helix. This drags the actin along the myosin filament and brings the ATP into the region of activity of the ATPase, which will split off the terminal phosphate of ATP. To form the next link with actin, the bound ADP must become bound ATP either by direct exchange with cytoplasmic ATP or by the activity of ATP: creatine phosphotransferase or ATP: AMP phosphotransferase. When ATP is rephosphorylated, the α -helix becomes more stretched and the hydrogen bonds are broken to form a more extended polypeptide chain. The movement is caused by the repulsion of the negative charge on the ionised ATP and fixed negative charges on the myosin. The ATP will now compete with inorganic

phosphate for the bounds further down the actin, this whole cycle will repeat several times during the active period (Lawrie, 1974).

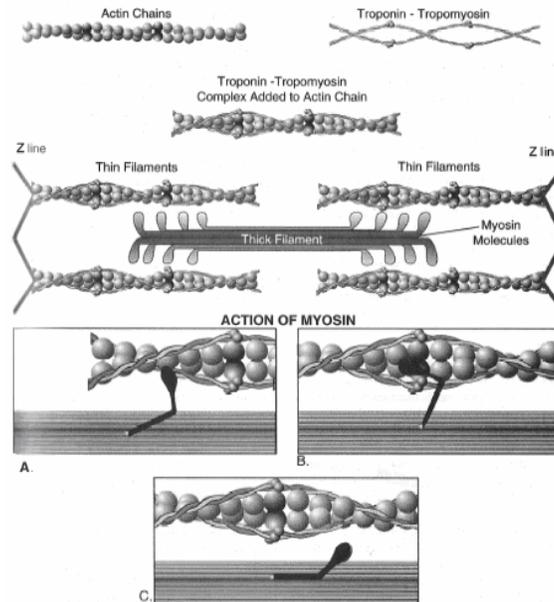


Figure 2.3 Formation and activation of actomyosin. A, B and C illustrate the sequence of myosin as it binds to actin and rotate the thin filament past the thick filament (Frandsen, Wilke, *et al.*, 2003).

Figure 2.3 illustrate the formation and activation of actomyosin. During contraction of the muscle the thin and thick filaments overlap more than during rest, in a cyclic fashion, causing the muscle to shorten in the same cyclic fashion. As the muscle shortens, the width of the I-band decreases as the thin filaments are drawn into the space between the thick filaments in the centre of each sarcomere. On the other hand the thickness of the A-band stays constant throughout contraction. The sarcomere length is dependent on the extent of overlap between the thick and thin filament. The length of the thick and thin filaments remains constant at all times (Varnam and Sutherland, 1996).

The maximum reversible contraction varies between 20 – 50 per cent of the length of the sarcomere at rest (approximately 3.6 μm). Relaxation results from removal of calcium and the myofibrils losing their ability to hydrolyse ATP (Varnam and Sutherland, 1996). Relaxing-factor in the sarcotubular membrane pumps out the calcium ions from the sarcoplasm (Lawrie, 1974).

Hydrolysis of ATP occurs at a concentration below 0.5 μM (Varnam and Sutherland, 1996). The ATP acts as a plasticiser, allowing separation of the actin and myosin (Lawrie, 1974,

Varnam and Sutherland, 1996). Now ATP levels can be re-established for the upcoming contraction (Lawrie, 1974).

2.5 The conversion of muscle to meat

The conversion of muscle to meat involves a complex set of interrelated occurrences (Pulford, Fraga Vazquez, *et al.*, 2008). The conversion starts directly after death, when the circulation stops. Circulation in the live animal provides nutrients and oxygen to organs, including muscles. Therefore, when the circulation stops, neither oxygen nor nutrients can be delivered to the muscles. Just because the circulation stopped, does not mean the metabolism in the muscle stopped. The muscles still require energy to maintain its tone as well as to maintain body temperature (Lawrie, 1974; Varnam and Sutherland, 1996; Forrest, Aberle, *et al.*, 1973). Because of the lack of oxygen in the muscles the muscle will use other metabolic pathways to regenerate ATP. These include the regeneration of ATP through the conversion of creatine phosphate to creatine, resulting in the conversion ADP to ATP, the conversion of two ADPs to one ATP and one AMP and through anaerobic respiration that results in lactic acid. These methods are less efficient than aerobic respiration and the levels of ATP decreases (Lawrie, 1974; Varnam and Sutherland, 1996).

The pH of the muscle decreases from the normal of 7.2 to between 5.5 and 5.8 during the first 24 hours post slaughter (Brewer, Zhu, Cassidy and Gilbert, 2001; Varnam and Sutherland, 1996; Forrest, Aberle, *et al.*, 1973). This decrease is because of the production of lactic acid (Muchenje, Dzama, *et al.*, 2009b; Lawrie, 1974; Varnam and Sutherland, 1996; Forrest, Aberle, *et al.*, 1996). The rate of pH decline differs between animals, even when compared within the same muscle (Simmons, Daly, *et al.*, 2006; Varnam and Sutherland, 1996). If the pH is too acidic the energy metabolism stops, because the enzymes involved in the system cannot function at the low pH. ATP is required for muscle contraction and relaxation (Varnam and Sutherland, 1996). As the levels of ATP decrease, more actomyosin forms that cannot be released again (Lawrie, 1974; Varnam and Sutherland, 1996). This is when rigor mortis sets in. Rigor mortis also occurs when the energy supply is depleted (Hwang, Devine, *et al.*, 2003; Rosenveld and Andersen, 2003). There is a relationship between glycolytic rate and the muscle contraction process (Rosenveld and Andersen, 2003). Structural changes of meat occurring during the rigor mortis development are both longitudinal and lateral contraction of myofibrillar weight (Tornberg, 1996).

The onset of rigor mortis is not the end of the conversion from muscle to meat. After the onset or even before the onset of rigor mortis enzymes start breaking down the proteins in the muscle (Lawrie, 1974). This proteolytic action is, loosening up the myofibrillar held together laterally, weakening of the myofibrillar length and myofibril fragmentation (Tornberg, 1996). The

enzymes involved are mainly calpains and cathepsins. The following table illustrates their activity: (Table 2.2) (Varnam and Sutherland, 1996)

Table 2.2 Function and activity of calpains and cathepsins (Varnam and Sutherland, 1996).

Location	Protease	Activity
Sarcoplasmic	Calpain I (μ - calpain)	Releases α -actinin, Z-nin
	Calpain II (m-calpain)	Degrades desmin, filamin, connectin, nebulin
		Degrades troponins, tropomyosin
		Degrades C- and M-proteins
Lysosomal	Cathepsin B	Degrades myosin, actin, troponin T Degrades collagen
	Cathepsin L	Degrades myosin, actin, troponins Degrades tropomyosin, α -actinin Degrades collagen
	Cathepsin D	Degrades myosin, actin, α -actinin Degrades troponins, tropomyosin Degrades collagen

These enzymes break down different parts of the muscle. But the actomyosin stays intact, no enzyme break this bond.

The post mortem tenderisation process is influenced by the pH and calcium concentration of the meat, which may involve activation or inactivation of the proteolytic enzymes. Cathepsins are more reactive in low pH conditions, while calpains are more reactive in higher pH conditions. The level of calpains in the muscle increases during the growth of the animal (Dransfield, 1994). They are calcium dependent proteases and are responsible for the myofibrillar degradation observed during post mortem ageing (Livisay, Xiong and Moody, 1996). Calpain I is activated first (at pH 6.3), when the calcium levels are still low. As the calcium levels increase calpain II starts to activate (Dransfield, 1994; Livisay, Xiong, *et al*, 1996; Feidt and Brun-Bellut, 1999). Both of these calpains are unstable and their activity decreases over time (Dransfield, 1994). Calpastatin is an inhibitor of the calpains, therefore the higher the concentration, the lower the calpain activity, and the lower the tenderness.

The proteins in the meat are degraded at different rates (Pulford, Dobbie, Fraga Vazquez, Frazer, Frost and Morris, 2009). The conversion of muscle to meat has a significant effect on meat

quality (Livisay, Xiong, *et al.*, 1996; Simmons, Daly, *et al.*, 2006). The following are normal ranges of meat: (Table 2.3) (Muchenje, Dzama, *et al.*, 2009b):

Table 2.3 Normal ranges of meat characteristics (Muchenje, Dzama, *et al.*, 2009b).

Meat characteristic	Range
Lightness (L*)	33.2 – 41
Redness (a*)	11.1 – 23.6
Yellowness (b*)	6.1 – 11.3
Colour saturation	16.1 – 20.9
Sarcomere length (µm)	1.75 – 2.31
WBSF at 2 days post mortem (N)	38.1 – 143.6
WBSF at 21 days post mortem (N)	16.9 – 59.9
Myofibril fragment length at 2 days post mortem (µm)	26.2 – 34.2
Myofibril fragment length at 14 days post mortem (µm)	19.2 – 24.7
pH _u	5.5 – 6.7
Drip loss (%)	0.14 – 3.89
Water holding capacity (%)	37.0 – 72.7
Cooking loss (%)	13.1 – 34.5
Moisture (%)	73.8 – 77.9
Protein content (%)	20.0 – 22.8
Fat content (%)	0.7 – 3.0

2.6 Dark, firm and dry meat (DFD) and pale, soft and exudative (PSE)

Long and short term stress in animals give rise to meat quality problems, like dark, firm and dry meat (DFD) and pale, soft and exudative (PSE) respectively (Tornberg, 1996; Scanga, Belk, *et al.*, 1998; Kadim, Mahgoub, *et al.*, 2004; Forrest, Aberle, *et al.*, 1996). DFD occurs when stress, just before slaughter, causes a depletion of glycogen concentration in the muscles and therefore a reduction in glycolysis that is needed to reduce the pH through lactic acid production (Scanga, Belk, *et al.*, 1998; Viljoen, De Kock, *et al.*, 2002; Thompson, 2002; Muchenje, Dzama, *et al.*, 2009a; Muchenje, Dzama, *et al.*, 2009b; Silva, Patarata, *et al.*, 1999). The following may play a role in the occurrence of DFD meat; handling practices, weather, growth promoters, genetics, disposition (Scanga, Belk, *et al.*, 1998), nutritional states and feeding management before slaughter (Muchenje, Dzama, *et al.*, 2009a) and the animal's gender (Scanga, Belk, *et al.*, 1998, Muchenje, Dzama, *et al.*, 2009a, Muchenje, Dzama, *et al.*, 2009b).

The DFD condition occurs most in cattle and goats exposed to stressful conditions (Viljoen, De Kock, *et al.*, 2002). In beef cattle, this condition is also referred to as "dry cutting beef". In goats the condition is also associated with ante mortem stress and a high glycolytic potential in muscle tissue (Webb, Casey and Simela, 2005).

The ultimate pH of DFD meat is higher than 5.8 - 6.2 (Tornberg, 1996; Viljoen, De Kock, *et al.*, 2002; Muchenje, Dzama, *et al.*, 2009a; Livisay, Xiong, *et al.*, 1996; Brewer, Zhu, *et al.*, 2001; Pulford, Fraga Vazquez, *et al.*, 2008, Varnam and Sutherland, 1996).

Thompson (2002) suggested that even a pH above 5.5 is an indicator of DFD meat. DFD has a more variable tenderness than normal meat, because of relative short sarcomere lengths that are swollen laterally and have a small extracellular space, the changes in the activity of the calpain system (Livisay, Xiong, *et al.*, 1996), high sarcoplasmic protein solubility and high water holding capacity (Purchas, Yan and Hartley, 1999; Purchas and Aungsupakorn, 1993; Purchas, 2004; Tornberg, 1996; Thompson, 2002; Livisay, Xiong, *et al.*, 1996; Silva, Patarata, *et al.*, 1999). With DFD there is no proteolysis of actin and myosin filaments when you look at SDS-PAGE analysis (Livisay, Xiong, *et al.*, 1996) therefore lower myofibrillar protein solubility than normal pH meat (Zhang, Farouk, *et al.*, 2005, Silva, Patarata, *et al.*, 1999).

Meat with a high ultimate pH has a dark colour as measured by the L* coordinate of the CIE colour system (Scanga, Belk, *et al.*, 1998; Muchenje, Dzama, *et al.*, 2009a; Muchenje, Dzama, *et al.*, 2009b; Kadim, Mahgoub, *et al.*, 2004; Livisay, Xiong, *et al.*, 1996). The dark colour is because the meat surface does not scatter light and has a higher light absorption than normal pH meat (Scanga, Belk, *et al.*, 1998; Kadim, Mahgoub, *et al.*, 2004; Livisay, Xiong, *et al.*, 1996). High pH meats are more brown in colour than normal pH meats (Muchenje, Dzama, *et al.*, 2009b; Zhang, Farouk, *et al.*, 2005).

High pH meat is considered a quality defect, particularly in beef on retail displays that is destined for table cuts (Zhang, Farouk, *et al.*, 2005). Consumers prefer the colour and general appearance of normal meat (cherry red) more than that of DFD meat (Viljoen, De Kock, *et al.*, 2002; Muchenje, Dzama, *et al.*, 2009b; Livisay, Xiong, *et al.*, 1996); although according to Viljoen, De Kock, *et al.* (2002) there is no difference in hedonic rating between fried normal and DFD meat.

DFD meat have an increased charge repulsion between proteins that leads to excessive water entrapment in muscle fibre networks, thus making the meat sticky and lacking in desirable texture (Livisay, Xiong, *et al.*, 1996; Brewer, Zhu, *et al.*, 2001). High pH also promotes growth of micro organisms which lead to development of off-odours and often slime formation that results in a shorter shelf life (Muchenje, Dzama, *et al.*, 2009a; Muchenje, Dzama, *et al.*, 2009b; Livisay, Xiong, *et al.*, 1996, Silva, Patarata and Martins, 1999). Heifers and bulls yield higher incidences of DFD meat than steers (Scanga, Belk, *et al.*, 1998, Zhang, Farouk, *et al.*, 2005).

It will seem that a threshold value (approximately 100 $\mu\text{mol/g}$) exists at slaughter for glycolytic potential. A high ultimate pH (DFD meat) is associated with a glycolytic potential below this threshold value and above this value no effect on pH can be observed (Wulf, Emmett, Leheska and Moeller, 2002). According to Wulf, Emmett, *et al.* (2002) higher glycolytic potential is associated with increased tenderness, and low glycolytic potential is associated with DFD meat and results in substantially less palatable cooked steaks.

PSE meat occurs when a low pH (below 5.5) is reached at a high muscle temperature soon after slaughter causing denaturation of muscle proteins and a decrease in electrostatic repulsion between myofilaments (Klont, Brocks and Eikelenboom, 1998).

PSE meat has a large variation in sarcomere length. The long sarcomeres in PSE meat is apparently caused by reduced shortening, due to the denaturation of the sarcoplasmic proteins during rigor (Tornberg, 1996). The short sarcomere can be caused by a high percentage of rigor development in the warm-shortening region and that the denaturation of the myosin heads caused longitudinal and lateral contraction of the myofibrillar weight (Tornberg, 1996). PSE pork has reduced myofibrillar fragmentation and protein degradation compared to normal meat (Livisay, Xiong, *et al.*, 1996).

It is possible that variations in proteolytic activity in DFD and PSE muscles are responsible, in part, for changes in protein functionality and meat quality (Gratacos-Cubarsi and Lametsh, 2008; Livisay, Xiong, *et al.*, 1996). The rapid production and accumulation of lactic acid in PSE meat causes decreases in water holding capacity and protein solubility, thereby reducing the textural quality of meat (Livisay, Xiong, *et al.*, 1996, Simmons, Daly, *et al.*, 2006). Post mortem glycolysis decreases muscle pH, making the meat appear brighter (high light reflection) and superficially more wet (Abril, Campo, *et al.*, 2001).

PSE is typically associated with pig and poultry, due to a high rate of glycolysis in some breeds of pigs. This condition is only sporadically seen in beef and lamb which have been subjected to high levels of electrical stimulation (over stimulation) combined with slow chilling rates (Simmons, Daly, *et al.*, 2006).

Figure 2.4 illustrates the effect of DFD, electrically over stimulation and electrical stimulation on the pH value decline post mortem. DFD meat's pH never goes below 6.6. The pH of the electrical stimulated and electrically over stimulated meat's pH initially falls quicker than that of normal meat (Frylinck, 2001).

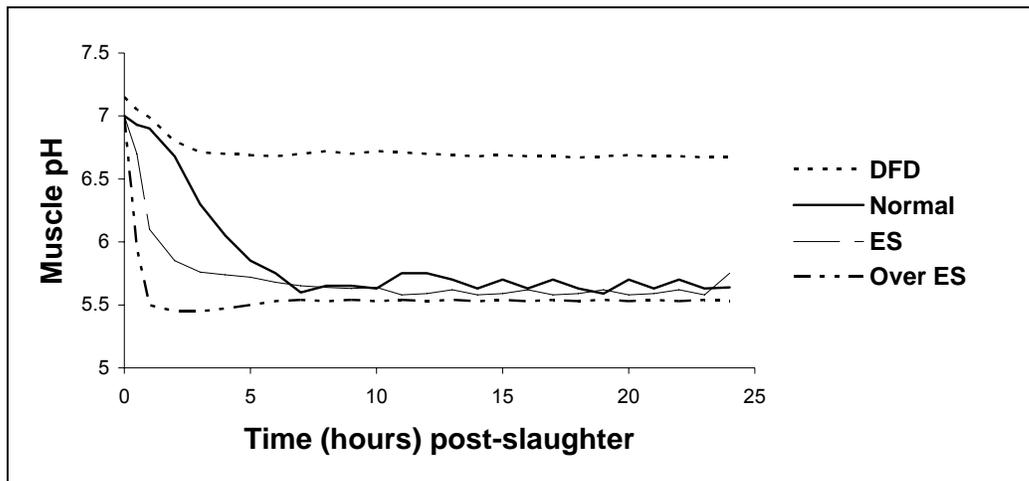


Figure 2.4 Examples of the rate and extent of decline in muscle pH for normal muscles and muscles with the DFD (dark, firm and dry), ES (electrical stimulation) and Over ES (electrical over stimulation) (Frylinck, 2001).

CHAPTER 3

METHODS AND MATERIALS

3.1 Animals and the treatment of the animals

One hundred and eighty (180) steers of three breed types (Nguni, Brahman and Simmental cross bred animals) were raised at a feedlot or on pasture, until reaching A, AB or B age (only pasture) classification and a 2 or 3 fat classification, according to the South African red meat classification system. The cattle were slaughtered under optimal slaughter conditions. All the animals were castrated shortly after weaning. The experiment was carried out in the feedlot, pasture, abattoir and meat science laboratories of the ARC-ANPI (Agricultural Research Council - Animal Nutrition and Production Industry), ARC-AII's (Agricultural Research Council - Animal Institution Irene) Groblersdal farm and the meat industry centre of ARC-ANPI. Normal animal management was applied to the animals (weighing, vaccinations, etc.). No special feeding regime was used, only the normal feeding regime used at the farm. These animals were slaughtered in the period between September and March of the next year, A and AB age animals were slaughtered in the beginning of the period and B age animals were slaughtered nearer to the end of this period.

The animals underwent the following pre and post slaughter practices. The animals were given the same non-aggressive growth promoter (Zeraplix-Intervet) at the specified finishing periods, they were transported the same number of kilometres to the abattoir (approximately 50 km) and feed was withdrawn 3 hours before slaughter. Electrical stimulation for approximately 15 seconds was used.

The following was measured in samples of the *m. longdissimus dorsi* (LD); the carcass pH value decline at 1, 2, 3, 4 and ultimate pH at 24 hours after slaughter, the temperature 1, 2, 3, 4 and 24 hours post slaughter, water holding capacity and drip loss at 1 day post slaughter, muscle lactate, glycogen, ATP, glucose-6-phosphate, creatine phosphate and glucose-concentration at 1, 2, 4 and 24 hours post slaughter, collagen solubility in 1 day post slaughter samples, activity of the calpain enzymes and its inhibitor calpastatin in meat samples 1 and 24 hours post mortem samples, myofibril fragment lengths on day 1, 7 and 14, sarcomere lengths at 1 and 3 days post mortem, the colour of the meat on day 1, tenderness of the meat on day 1, 7 and 14 (maximum shear force values were used). All the analyses were done according to standard laboratory methods at the ARC-API meat industry centre.

3.2 The measurement of pH and temperature

Carcass pH and temperature were measured in the LD with a digital handheld meat pH meter (Sentron, model 1001) fitted with a polypropylene spear type gel electrode between the ribs.

3.3 The measurement of water holding capacity and drip loss

Water holding capacity was determined by placing a 0.5 cm³ sample of the LD on a filter paper. The filter paper with the meat sample on was placed in a press at a constant 35 kg/cm² for 30 seconds. Directly after removing the filter paper, the borders of the meat and the fluid ring were marked. Water binding capacity was calculated from the area of water pressed out (area of meat/ area of moisture). The areas were measured by means of a video Image Analyser (Kentron, Germany) (Strydom, Frylinck, Van der Westhuizen and Burrow, 2008; Irie, Izumo and Mohri, 1996).

Drip loss was measured on the samples from the LD as follows; a sample of 5 g (cut into a cube on 10 x 10 x 20 mm) was taken and suspended in a bottle (200ml), for 3 days at 4±2 °C. The amount of drip was expressed as a percentage of initial weight (Strydom, Frylinck, *et al.*, 2008).

3.4 The measurement of the energy status in the muscle post mortem

Lactic acid concentration in the muscle samples from the LD was determined by using the method as described by Gutmann and Wahlefield (1974) and modified by Dalrymple and Hamm (1973). A 3 g muscle sample was homogenised with 15 ml of 0.6 N perchloric acid at full speed for 100 seconds in an ice bath, and then centrifuged for 20 minutes at 40000 g at 0°C. The resulting supernatant was decanted through filter paper to remove any floating material and then neutralized with 5.4 N potassium hydroxide until a methyl orange colour. The resulting potassium perchlorate was allowed to settle out for 20 minutes at 0°C and the supernatant decanted through filter paper to eliminate the precipitate. 0.02 ml of the neutralized extract was used to determine the absorbance at 340 nm.

Glucose and glycogen concentration in the LD muscle sample were determined by using the method as described by Keppler and Decker (1974) and modified by Dalrymple and Hamm (1973). A 3 g muscle sample was homogenised with 15 ml of 0.6 N perchloric acid at full speed for 100 seconds in an ice bath. 0.02 ml of the homogenate was pipetted into a 5 ml test tube, and then 1.0 ml of amyloglucosidase solution was added. The amyloglucosidase solution contains 1 mg of enzyme protein per 1 ml of 0.2 M acetate buffer (pH 4.8). 0.02 ml 5.4 N potassium hydroxide was added to neutralize the perchloric acid. The solution was incubated for 2 hours in a 40°C water

bath with frequent mixing. The incubated solution was allowed to cool before 0.10 ml of 3N perchloric acid was added to precipitate the proteinous material. The precipitate was allowed to settle out for 10 minutes at 0°C. The tube was then centrifuged at 1000 g for 10 minutes, the resulting supernatant was decanted through filter paper. 0.5 -1.0 ml of supernatant was used to determine the absorbance at 340 nm.

Glucose-6-phosphate, ATP and creatine phosphate concentration in the muscle was determined by the method described by Bernt, Bergmeyer and Möllering (1974) and Lamprecht, Stein, Heinz and Weisser (1974) as modified by Dalrymple and Hamm (1973). A 3 g muscle sample was homogenised with 15 ml of 0.6 N perchloric acid at full speed for 100 seconds in an ice bath and then centrifuged for 15 minutes at 300 rpm. 1 ml of tri-ethanolamine/potassium carbonate solution was added to 3 ml of supernatant fluid, and then allowed to stand for 15 minutes in an ice bath. The supernatant was decanted through filter paper to remove the perchlorate precipitate. The supernatant was allowed to reach 25°C, and then 2.00 ml of the buffered solution was used to determine the absorbance at 340 nm.

Glycolytic potential was calculated as follow: Glycolytic potential = 2 * (glucose-6-phosphate + glucose + glycogen) + lactic acid (Wulf, Emmett, *et al.*, 2002).

3.5 The measurement of collagen content and solubility

To determine collagen content and collagen solubility, the following procedure was performed in triplicate. Total collagen was estimated by determining the hydroxy proline content in a hydrolysed sample. To hydrolyse the sample, 0.5 g of a pulverised freeze-dried meat sample of the LD was added to 30 ml of 6 N hydrochloric acid and then hydrolysed at 110°C for 16 hours. After hydrolysis, active carbon was added; the solution was filtered and diluted with 100 ml of distilled water.

The intramuscular collagen was determined by modifying the method described by Hill (1966). 12 ml of a 1 per cent sodium chloride solution was added to 1 g of a pulverized freeze-dried LD sample and then heated in a water bath for 60 minutes at 78°C. This sample was centrifuged at 1000 rpm for 30 minute then hydrolysed for 16 hour at 110°C. Active carbon was added to the hydrolysed sample, filtrated and diluted by 100 ml of distilled water. The nitrogen content had been determined after the sample had been digested in a micro Kjeldahl system.

Hydroxy-proline was calorimetrically determined after neutralising the acid in LD sample by adding 10 per cent potassium hydroxide and oxidised with Chloramine-T for 20 minutes. Ehrlich's reagent was added and the sample was placed in a water bath for 15 minutes at 60 °C. The absorbance was measured at 558 nm in a 1cm³ cuvette.

Collagen solubility was calculated by dividing total hydroxy-proline by the total hydroxy proline plus the residue in the filtrate (Marais, 2007).

3.6 The measurement of calpains and calpastatins

To determine calpains and calpastatin, 5 g of the frozen meat sample of the LD was used. The calpains and calpastatins were separated by using a two-step gradient ion exchange chromatography method, as described by Dransfield (1996).

The muscle sample was homogenised in 100ml of 50 mM Tris(2-amino-2-[hydroxymethyl]-propane-1,3-diol)-hydrochloric acid, 2 mM ethylenediaminetetra-acetic acid (EDTA), 0.01 % sodium azide, 10 mM phenolmethanesulphonyl fluoride (buffer A) and 150 nM pepstatin. Hydrochloric acid was added to the extract until the pH reached 7.5 and then stirred for 1 hour at 4°C. The extract was then centrifuged at 25000 g for 20 minutes, and then the supernatant was filtrated through glass wool. The filtrate was made up to 500 mM by adding crystalline sodium chloride. Calpains were separated from calpastatin using hydrophobic chromatography on a phenol-separose column (25 mm diameter x 150mm, 1.5 ml/minute). A calpain containing fraction was eluted with buffer A with added 10 % (v/v) ethylene glycol. The fraction was chromatographed on a DEAE-sephacel column (25mm diameter x 90 mm, 1 ml/minute, 2.00 ml fraction) and eluted with a linear gradient for 0 to 450 mM NaCl in buffer A (Dransfield, 1996).

The calpain assay was determined by using azo-casein as substrate (Dransfield, 1996). The azo-casein eliminates the problem of background absorbance of non-specific proteins in the extracts.

One unit of calpain is defined as an increase in absorbance at 366 nm of 1.0 per hour at 25°C. One unit of calpastatin is defined as the amount that inhibits one unit of m-calpain activity (Frylinck, Van Wyk, Smith, Strydom, Van Marle-Köster, Webb, Koohmaraie, Smith, 2009).

3.7 The measurement of sarcomere lengths and myofibrillar fragment lengths

Sarcomere lengths were measured by means of a Video Image Analyzer (Kentron, Germany). The average of five different measurements on the same LD meat sample was used. The microscope slides were immersed in distilled water. 250 mg muscle sample was homogenised at half maximum speed for 12 to 30 seconds in 19.5 ml of a chilled solution of 0.1 M potassium chloride, 0.039 M sodium tetraborate and 6 mM EDTA. Three drops of the homogenate was transferred to a slide and covered with a cover slip (Heinze and Bruggenmann, 1994).

The myofibril fragment length was measured by using a Video Image Analyzer (Kentron, Germany). The extractions were done by using the methods of Culler, Parrish, Smith and Cross

(1978), as modified by Heinze and Bruggenmann (1994). The sample was thinly sliced and any visible fat and connective tissue was removed, and then scissor minced. 3 g of the minced sample was homogenised in 50 ml 0.02 M potassium phosphate buffer (pH 7.00) for 30 seconds at 20000 rpm. The buffer contains 100 mM potassium chloride 1 mM magnesium chloride, 1 mM EDTA and 1 mM sodium nitrogen. The homogenised sample was centrifuged at 1000 g for 15 minutes at 4 °C. The supernatant was discarded and the pellet resuspended in 50 ml of the same buffer and centrifuged as described above. The supernatant was discarded again and the pellet resuspended in 10 ml of the buffer. The suspension was filtrated through a 1000 µm polyethylene strainer under a light vacuum, an additional 5 ml of buffer was use to lubricate the passage through the strainer. The filtrate was than filtered through a 250 µm polyethylene strainer under a light vacuum. The myofibril fragment length was measured by using a Video Image Analyzer (Kentron, Germany).

3.8 The measurement of tenderness (Warner-Bratzler shear force)

The tenderness was determined by using a Warner-Bratzler shear force device mounted on an Instron Universal testing machine, model 4301, Series IX Automated materials testing system. Frozen meat samples were cut into 30 mm steaks from the LD with a band saw, before being thawed at 2-4°C overnight. The next morning the meat was prepared by using an oven-boiling method using direct radiant heat. The steaks were cooked at 200°C until the internal temperature reached 70°C. Then the steaks were cooled until they reached room temperature. The cooled steaks were cored to produce six cylindrical samples per steak. The sample of 12.5 mm diameter was removed parallel to the grain. The samples were sheared perpendicular to the fiber direction and shear force was measured in Newton (N).

3.9 The measurement of meat colour

The colour was measured in triplicate by means of a Minolta Chromameter, model CR200 (Minolta, Japan). Before the measurements were taken, the Minolta were calibrated on a standard white calibration tile (calibration values; L*: 97.81, a*: -5.56, b*: +7.38) (Simela, 2005). Measurements werS taken at three different spots on the LD sample. The following calculations were done; hue angle = $\tan^{-1} (b^*/a^*)$ and Chroma = Square root of $(a^{*2} + b^{*2})$ (Young, Priolo, *et al.*, 1999)

3.10 Statistical evaluation of results

The effect of the 5 production systems, the 3 different breeds as well as the combined effect of the production system and the breed on the meat quality was evaluated by means of MANOVA on GCM (SAS, 1996).

The Bonferroni method multiple range test was used for the multiple comparisons of means, between production systems as well as between breeds. A partial correlation was done between the variables, with the two tailed student t test to determine significance at a level of $p < 0.05$.

CHAPTER 4

RESULTS AND DISCUSSION

Production system is defined by a specific feeding system combined with the age of the animal. There were two feeding systems in this experiment namely feedlot (F) and pasture (P). There were three age groups namely A, AB and B according to the red meat classification system of South Africa (see Appendix A). Production systems used in this study were; AF, ABF, AP, ABP and BP.

4.1 Effect of production system and breed on carcass characteristics

The effect of the interaction between breed and production system on the warm and cold carcass weight as well as percentage weight loss are summarised in Table 4.1. The interactions between breed and production system had an effect on the warm and cold carcass weight as well as the percentage weight loss ($p < 0.05$). Nguni animals from the AP production system (148 kg and 143 kg respectively) had the lightest warm and cold carcass weight and Simmental animals from the BP production system (266 kg and 285 kg respectively) had the tendency for heaviest warm and cold carcass weight. Brahman animals from the AF production system (1.73 %) had the lowest percentage weight loss and Nguni animals from the AP production system (3.02 %) had the highest percentage weight loss.

Average warm and cold carcass weights for this study were; 207 ± 45.21 kg and 202 ± 44.54 kg respectively. There were differences between production systems for warm and cold carcass weight ($p < 0.05$). The mean percentage weight loss of carcasses for this study was 2.50 ± 0.66 %. There were no differences for percentage weight loss between production systems ($p > 0.05$).

According to Strydom, Frylinck, Montgomery and Smith (2009), the mean warm carcass weight for 18 month old Bonsmara steers from feedlots is 339 kg. Strydom (2008) noted that animals from South African feedlots have an average warm carcass weight of 250 kg (these animals are mainly A age animals). Average warm carcass weight of animals from the ABP production systems reported in literature range from 107 kg, for Nguni, to 149 kg, for Brahman (Mapiye, Chimonyo, Dzama, Strydom, Muchenje and Marufu, 2009; Muchenje, Dzama, Chimonyo, Raats and Strydom, 2008; Muchenje, Dzama, *et al.*, 2009a). Warren, Scollan, Enser, Hughes, Richardson and Wood (2008) noted that Aberdeen Angus cattle from the BP production systems had an average warm carcass weight of 399 kg and Holstein Friesland of the same production system had a warm carcass weight of 363 kg.

Table 4.1 Effect of the interaction between production system and breed on carcass weight and percentage weight loss

Production system (n=180)	Breed	Warm weight (kg) ($\bar{X} \pm SD$)	Cold weight (kg) ($\bar{X} \pm SD$)	Percentage weight loss (kg) ($\bar{X} \pm SD$)
AF	Br-X	222±6.61 ^{cde}	218.±6.16 ^c	1.73±0.29 ^a
	Ng-X	205.±18.16 ^{bc}	201±17.88 ^{abc}	1.86±0.22 ^a
	Si-X	239±6.05 ^{def}	234±6.11 ^c	1.98±0.22 ^{ab}
ABF	Br-X	246±40.66 ^{ef}	240±39.86 ^c	2.40±1.22 ^{bc}
	Ng-X	176±13.15 ^{ab}	172±12.85 ^{ab}	2.13±0.27 ^{ab}
	Si-X	236±22.67 ^{def}	231±26.01 ^c	2.04±0.33 ^{ab}
AP	Br-X	181±21.98 ^{ab}	175±21.51 ^{ab}	3.01±0.28 ^c
	Ng-X	148±27.71 ^a	143±27.24 ^a	3.02±0.49 ^c
	Si-X	170±15.29 ^{ab}	165±14.79 ^{ab}	2.93±0.40 ^c
ABP	Br-X	207±33.97 ^{bcd}	201±32.91 ^{bc}	2.16±0.27 ^{bc}
	Ng-X	162±21.58 ^a	157±21.17 ^a	2.84±0.20 ^c
	Si-X	182±19.91 ^{ab}	177±19.56 ^{ab}	2.98±0.29 ^c
BP	Br-X	267±42.10 ^f	260±41.61 ^c	2.39±0.31 ^{bc}
	Ng-X	186±26.32 ^{abc}	182±23.01 ^{abc}	2.60±0.37 ^{bc}
	Si-X	266±36.84 ^f	285±37.52 ^c	2.97±1.16 ^c

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

Warm weight ~ average warm carcass weight, Cold weight ~ average cold carcass weight, Percentage weight loss ~ average percentage of weight loss during carcass cooling.

Br-X ~ breed average for Brahman cross bred animals, Ng-x ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmentaler cross bred animals.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

Carcass weights in this study were less than that reported in literature. The differences in weight between this study and those in literature can be due to breed differences which agree with the findings of Warren, Scollan, *et al.* (2008). Cattle carcasses produced in South Africa have approximately 18 % carcass fat which is considerably lower than those in the United States of America. Carcasses from the United States of America weigh approximately 400 kg while those from South Africa weigh approximately 220 kg (Webb and O'Neill, 2008).

4.2 Effect of production system on carcass pH and carcass temperature at 1, 2, 3, 4 and 24 hours post mortem.

Effect of production system on pH at 1, 2, 3, 4 and 24 hours post mortem is summarised in Table 4.2. According to previous studies carcass pH values post mortem, at 1, 2, 3, 4 and 24 hours are approximately; 6.0, 5.9, 5.8, 5.7 and 5.6 (these values are mainly from AB age animals fed a feedlot diet and AB aged animals from pasture) (Marsh, Ringkob, *et al.*, 1987; Muchenje, Dzama, *et al.*, 2009b). Immonen, Ruusunen, Hissa and Puolanne (2000) found a difference in carcass pH between production systems with carcass pH of cattle from the AP production system being higher than that of animals from the AF production system.

Table 4.2 Effect of production system on carcass pH at 1, 2, 3, 4 and 24 hours post mortem.

Production system (n=180)	pH ₁ ($\bar{X} \pm SD$)	pH ₂ ($\bar{X} \pm SD$)	pH ₃ ($\bar{X} \pm SD$)	pH ₄ ($\bar{X} \pm SD$)	pH ₂₄ ($\bar{X} \pm SD$)
AF	6.10±0.10	5.86±0.13	5.69±0.16 ^a	5.57±0.19 ^a	5.48±0.11 ^a
ABF	6.07±0.12	5.81±0.52	5.77±0.18 ^{ab}	5.66±0.18 ^{ab}	5.64±0.08 ^b
AP	6.10±0.25	5.96±0.28	5.90±0.25 ^b	5.86±0.26 ^c	5.68±0.12 ^b
ABP	6.06±0.24	5.90±0.28	5.82±0.27 ^{ab}	5.75±0.27 ^b	5.64±0.12 ^b
BP	6.12±0.23	5.93±0.26	5.86±0.33 ^b	5.80±0.33 ^b	5.66±0.18 ^b
Total	6.09±0.20	5.89±0.32	5.81±0.26	5.73±0.27	5.62±0.14

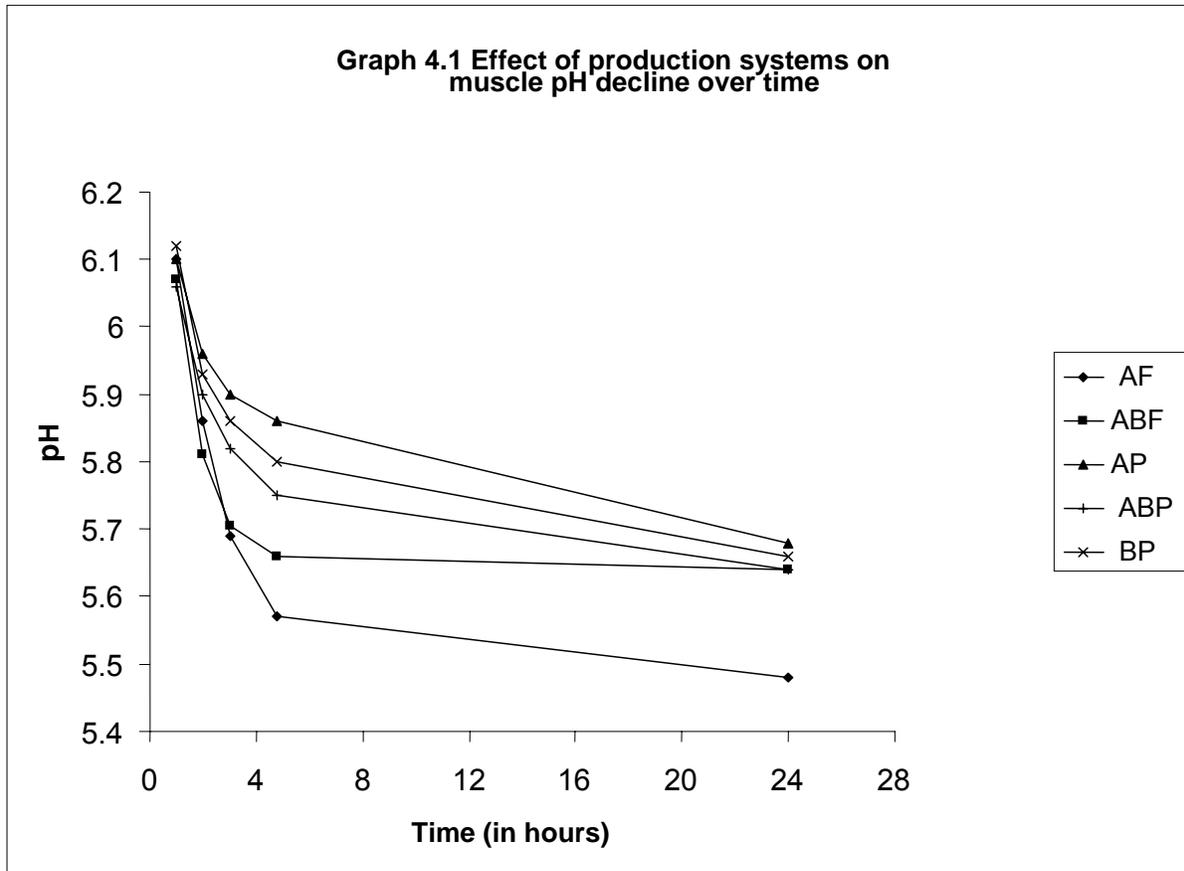
^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

pH₁ ~ average pH 1 hour after slaughter, pH₂ ~ average pH 2 hours after slaughter, pH₃ ~ average pH 3 hours after slaughter, pH₄ ~ average pH 4 hours after slaughter, pH₂₄ ~ average pH 24 hours after slaughter.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

There were differences between carcass pH values of cattle from the different production systems. Specifically carcass pH values at 3, 4 and 24 hours post mortem ($p < 0.05$) differed between production systems. At 1 and 2 hours post mortem ($p > 0.05$) there were no differences. Carcasses from the AP production system and BP production system showed higher carcass pH at 3, 4 and 24 hours post mortem than those from the AF production system (Table 4.2).



Effect of production system on the carcass pH as time progresses is illustrated in Graph 4.1. Carcass pH values decreased as time progressed, the rate of this decrease differed between production systems; carcasses from the AF production system had the highest rate of pH decrease followed by the ABF production system ($p < 0.05$). AP production system had the lowest rate of pH decline ($p < 0.05$). Carcass pH values of carcasses from the ABF production system were the lowest from 1 to 3 hours post mortem, while carcass pH of carcasses from the AF production system were the lowest from 3 to 24 hours post mortem. The pH decreases over time as the concentrations of lactate concentrations in the carcass increases (Marsh, Ringkob, *et al.*, 1987; Muchenje, Dzama, *et al.*, 2009b). The increase in lactate concentrations occurs, because anaerobic glycolysis occurred after death (Muchenje, Dzama, *et al.*, 2009b; Lawrie, 1974; Varnam and Sutherland, 1996). The ultimate carcass pH (pH_{24}) from the AF production was lower ($p < 0.05$) than that of the other production systems (ABF, AP, ABP and BP). As discussed in more detail later animals from the AF production system had higher carcass lactic acid concentrations ($p < 0.05$) than the animals from the other production systems (ABF, AP, ABP and BP).

The carcass pH from the ABF production system increases between 4 and 24 hours post mortem. Vetharanim, Thompson, Devine and Daly (2010) explain this occurrence as caused by a

greater alkalising effect of adensine monophosphate deamination at low pH than the acidifying effect of the inosine monophosphate dephosphorylation. Between pH 4 and 24 the conditions are perfect for this occurrence.

Feeding system per se had an influence on carcass pH at 2, 3, 4 and 24 hours post mortem ($p < 0.05$). Carcasses from the feedlot (pH 1 hour: 6.09 ± 0.11 , pH 2 hours: 5.84 ± 0.38 , pH 3 hours: 5.73 ± 0.17 , pH 4 hours: 5.62 ± 0.19 and pH 24 hours: 5.56 ± 0.12) constantly had a lower carcass pH than the carcasses from cattle on pastures (pH 1 hour: 6.10 ± 0.24 , pH 2 hours: 5.93 ± 0.27 , pH 3 hours: 5.86 ± 0.29 , pH 4 hours: 5.80 ± 0.29 and pH 24 hours: 5.66 ± 0.14). According to Sami, Augustini, *et al* (2004) there are differences in carcass pH values between feeding systems and their findings suggest that animals on pasture had higher carcass pH values than animals from the feedlot. Ultimate pH range of the feedlot animals and the pasture animals reported in literature are; 5.47 to 5.61 and 5.47 to 5.75 respectively (Serra, Gil, Gispert, Guerrero, Olivier, Sanudo, Campo, Panea, Olleta, Quotanilla and Piedrafita, 2004; Serra, Guerrero, Guardis, Gil, Sanudo, Panea, Campo, Olleta, Garcia-Cachan, Piedrafita and Oliver, 2008; Blanco, Villalba, Bidner, Meisinger and McKeith, 2009; Boles and Suran, 2002; Insausti, Beriain, Purroy, Alnerti, Gorraiz and Alzueta, 2001; Immonen, Ruusunen, *et al.*, 2000; Immonen, Schaefer, Puolanne, Kauffman and Nordhein, 2000). The pH values differ between the production system due to the conditioning of the animals and the glycolytic potential (discussed later in dissertation).

Age per se only had an influence on carcass pH at 24 hours post mortem ($p < 0.05$). The youngest animals showed lower pH values than the older animals. Carcass pH of cattle in the A and AB age classification groups did not differ ($p > 0.05$). Thompson, Perry, *et al.* (2006) states that the older the animals the highest the ultimate pH will be. Ultimate pH values for the different age classification groups (based on similar chronological age as in SA system) reported in literature are approximately: A: 5.53, AB: 5.61 and B: 5.65 (Serra, Gil, *et al.*, 2004; Insausti, Beriain, *et al.*, 1999; Serra, Guerrero, *et al.*, 2008; Revilla and Vivar-Quintana, 2006; Blanco, Villalba, *et al.*, 2009; Boles and Swan, 2002). These differences can be explained by differences in conditioning and glycolytic potential (discussed later in dissertation).

The effect of breed on carcass pH post mortem is summarised in Table 4.3. Breed per se had no effect on carcass pH values post mortem ($p > 0.05$). Until 3 hours post mortem carcass from Brahman cross bred animals had the tendency for the highest carcass pH values. From 3 hours post mortem Simmental cross bred animals had the tendency for the highest pH values. Nguni cross bred animals constantly had the tendency for the lowest carcass pH values (Table 4.3).

Table 4.3 Effect of breed on the carcass pH at 1, 2, 3, 4 and 24 hours post mortem

Breed (n=180)	pH ₁ ($\bar{X} \pm SD$)	pH ₂ ($\bar{X} \pm SD$)	pH ₃ ($\bar{X} \pm SD$)	pH ₄ ($\bar{X} \pm SD$)	pH ₂₄ ($\bar{X} \pm SD$)
Br-X	6.13±0.16	5.94±0.21	5.83±0.23	5.74±0.23	5.62±0.12
Ng-X	6.10±0.08	5.87±0.25	5.76±0.25	5.70±0.28	5.63±0.18
Si-X	6.10±0.20	5.87±0.45	5.84±0.28	5.75±0.29	5.62±0.13

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

pH₁ ~ average pH 1 hour after slaughter, pH₂ ~ average pH 2 hours after slaughter, pH₃ ~ average pH 3 hours after slaughter, pH₄ ~ average pH 4 hours after slaughter, pH₂₄ ~ average pH 24 hours after slaughter.

Br-X ~ Breed average for Brahman cross bred animals, Ng-X ~ Breed average for Nguni cross bred animals, Si-X ~ Breed average for Simmentaler cross bred animals.

The effect of the interaction between breed and production system on carcass pH decline is summarised in Table 4.4. The interactions between breed and production system had an effect on the carcass pH value at 4 and 24 hours post mortem ($p < 0.05$) but not for the pH at 1, 2 and 3 hours post mortem ($p > 0.05$). The carcass pH ranges from 6.00 to 6.25 at 1 hour post mortem, 5.58 to 6.13 at 2 hours post mortem, 5.68 to 6.01 at 3 hours post mortem, 5.54 to 6.03 at 4 hours post mortem and 5.48 to 5.73 at 24 hours post mortem. The pH ranges for this study is in the same ranges as those reported in the literature (Serra, Gil, *et al.*, 2004; Insausti, Beriain, *et al.*, 1999; Serra, Guerrero, *et al.*, 2008; Revilla and Vivar-Quintana, 2006; Blanco, Villalba, *et al.*, 2009; Boles and Swan, 2002). The carcasses for the Nguni animals from the AP production system constantly the highest carcass pH values.

Carcass temperature will decrease over time, because the carcasses are stored in a cold room after slaughter. As the metabolism after death decreases, the carcass temperature decreases as well. Metabolism slows down after death because there is a decrease in availability of nutrients and oxygen post mortem. According to Thompson, Perry, *et al.* (2006) body weight influences temperature fall and therefore temperature at a certain time. If animal's carcass weights differ, there will be a difference in temperature as well. A significant difference was expected, because there was a significant difference between the different weights in production systems. Differences can also be explained by differences in metabolic rate and enzyme activity (discussed later in dissertation).

Table 4.4 Effect of the interaction between breed and production system on carcass pH decline.

Production system (n=180)	Breed	pH ₁ ($\bar{X} \pm SD$)	pH ₂ ($\bar{X} \pm SD$)	pH ₃ ($\bar{X} \pm SD$)	pH ₄ ($\bar{X} \pm SD$)	pH ₂₄ ($\bar{X} \pm SD$)
AF	Br-X	6.11±0.09	5.85±0.16	5.71±0.17	5.58±0.16 ^a	5.48±0.07 ^{ab}
	Ng-X	6.08±0.15	5.85±0.13	5.68±0.19	5.60±0.28 ^a	5.50±0.18 ^{abc}
	Si-X	6.11±0.06	5.88±0.10	5.69±0.12	5.54±0.12 ^a	5.46±0.05 ^a
ABF	Br-X	6.13±0.08	5.93±0.17	5.81±0.16	5.70±0.16 ^{ab}	5.66±0.09 ^{abcd}
	Ng-X	6.00±0.13	5.58±0.17	5.71±0.16	5.60±0.18 ^a	5.60±0.06 ^{abcd}
	Si-X	6.11±0.10	5.64±0.96	5.81±0.20	5.70±0.21 ^{ab}	5.66±0.11 ^{abcd}
AP	Br-X	6.09±0.16	5.93±0.14	5.88±0.24	5.80±0.18 ^{ab}	5.68±0.08 ^{cd}
	Ng-X	6.25±0.34	6.13±0.41	6.01±0.28	6.03±0.34 ^b	5.73±0.17 ^d
	Si-X	6.00±0.18	5.85±0.20	5.83±0.22	5.77±0.19 ^{ab}	5.63±0.07 ^{abcd}
ABP	Br-X	6.09±0.23	5.96±0.27	5.82±0.23	5.74±0.23 ^{ab}	5.62±0.07 ^{abcd}
	Ng-X	6.03±0.21	5.82±0.26	5.74±0.34	5.65±0.18 ^a	5.63±0.12 ^{abcd}
	Si-X	6.07±0.28	5.95±0.32	5.91±0.33	5.87±0.36 ^{ab}	5.67±0.14 ^{bcd}
BP	Br-X	6.20±0.17	6.03±0.24	5.90±0.28	5.84±0.30 ^{ab}	5.64±0.14 ^{abcd}
	Ng-X	6.00±0.20	5.76±0.17	5.71±0.29	5.67±0.28 ^a	5.67±0.25 ^{bcd}
	Si-X	6.19±0.24	6.00±0.29	6.00±0.39	5.86±0.39 ^{ab}	5.69±0.14 ^{bcd}

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) with the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

pH₁ ~ average pH 1 hour after slaughter, pH₂ ~ average pH 2 hours after slaughter, pH₃ ~ average pH 3 hours after slaughter, pH₄ ~ average pH 4 hours after slaughter, pH₂₄ ~ average pH 24 hours after slaughter.

Br-X ~ breed average for Brahman cross bred animals, Ng-x ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmentaler cross bred animals.

AF~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

Effect of production system on the carcass temperature at 1, 2, 3, 4 and 24 hours post mortem is summarised in Table 4.5. Differences for production systems on carcass temperatures at 1, 2, 3, 4 and 24 hours post mortem were observed ($p < 0.05$). At 2 hours post mortem, the carcasses from the BP production system (28.2 °C) differed significantly from all other production systems (AF, ABF, AP and ABP). There were differences ($p < 0.05$) for the carcass temperature at 3 and 4 hours post mortem between all the production systems (AF: 28.2 °C and 24.3 °C, ABF: 23.4 °C and 19.2 °C, AP: 16.6 °C and 13.4 °C, ABP: 19.9 °C and 16.9 °C, and BP: 23.0 °C and 19.7

°C). These results were expected due to the difference that occurred between the production systems carcass weights.

Carcasses from the AP production system (33.3 °C, 16.6 °C and 13.4 °C) had the tendency to have the lowest carcass temperature at 1, 3 and 4 hours post mortem, while the carcasses from the ABP production system (25.0 °C) had the tendency for the lowest carcass temperature at 2 hours post mortem and the carcasses from the ABF production system (3.5 °C) had the tendency for the lowest carcass temperature at 24 hours post mortem. Carcasses from the AF production system (39.0 °C, 32.9 °C, 28.2 °C and 24.3 °C) had the tendency for the highest temperature at 1, 2, 3 and 4 hours post mortem whereas carcasses from the ABP production system (7.5 °C) had the highest temperature at 24 hours post mortem.

Table 4.5 Effect of production system on the carcass temperature at 1, 2, 3, 4 and 24 hours post mortem

Production system (n=180)	Temp ₁ (°C) ($\bar{X} \pm SD$)	Temp ₂ (°C) ($\bar{X} \pm SD$)	Temp ₃ (°C) ($\bar{X} \pm SD$)	Temp ₄ (°C) ($\bar{X} \pm SD$)	Temp ₂₄ (°C) ($\bar{X} \pm SD$)
AF	39.0±0.80 ^d	32.9±2.29 ^c	28.2±2.48 ^d	24.3±1.85 ^d	7.2±1.06 ^c
ABF	37.3±6.25 ^{cd}	30.6±3.08 ^c	23.4±2.79 ^c	19.2±2.36 ^c	3.5±2.78 ^a
AP	33.3±3.10 ^a	27.3±3.22 ^a	16.6±2.78 ^a	13.4±2.50 ^a	5.8±0.90 ^b
ABP	34.8±2.28 ^{ab}	25.0±3.72 ^a	19.9±3.01 ^b	16.9±3.06 ^b	7.5±2.31 ^c
BP	36.2±1.99 ^{bc}	28.2±3.66 ^b	23.1±3.99 ^c	19.7±3.50 ^c	6.7±2.02 ^{bc}
Total	36.1±3.86	27.9±5.59	22.3±4.89	18.8±4.43	6.2±2.40

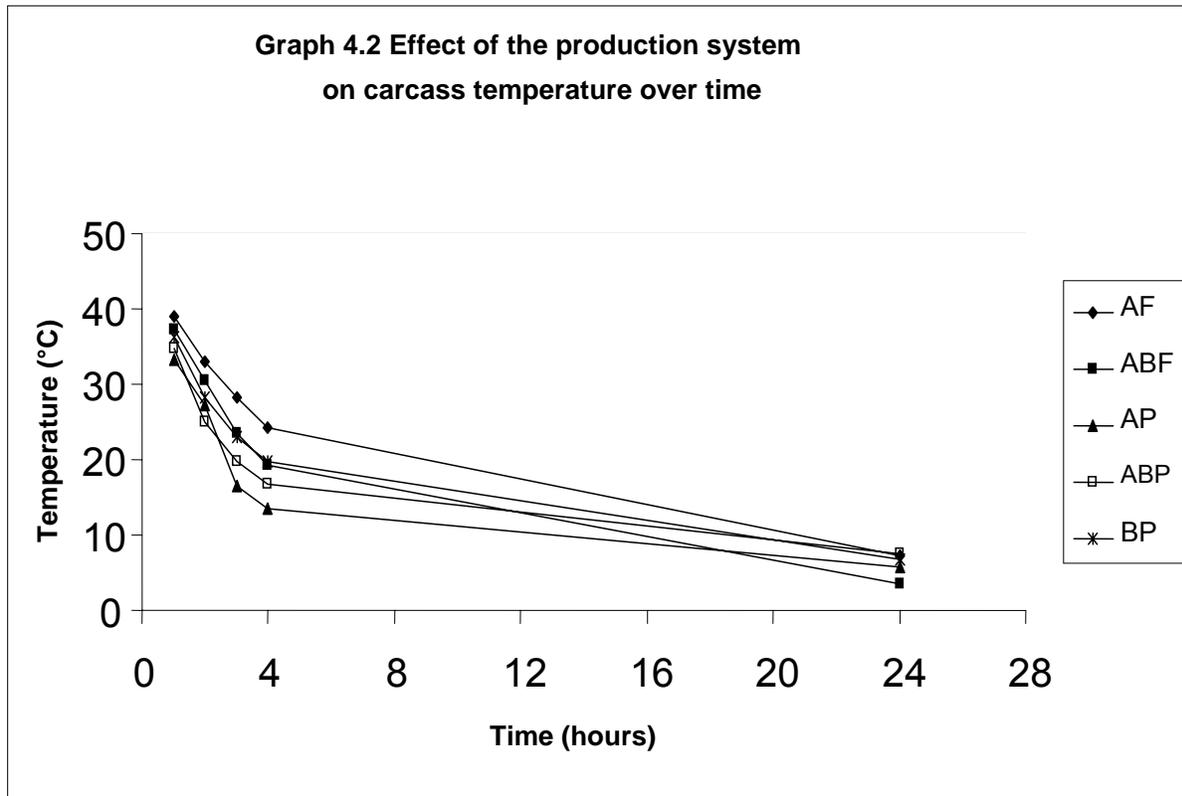
^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean ± standard deviation.

Temp₁ ~ average temperature 1 hour after slaughter, Temp₂ ~ average temperature 2 hours after slaughter, Temp₃ ~ average temperature 3 hours after slaughter, Temp₄ ~ average temperature 4 hours after slaughter, Temp₂₄ ~ average temperature 24 hours after slaughter.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

Graph 4.2 illustrates the effect of the different production systems on carcass temperature as time progresses. Temperature decreased as time progressed due to the cooling of the carcasses as well as a reduction in metabolism after death. The rate of temperature decline differed between the production systems. Carcasses from the AF production system seems to have a constantly higher temperature and carcasses from the AP production system seems to have a lower temperature up to approximately 18 hours post mortem, from where carcasses from ABF production system seems to have a lower temperature.



When only the effect of feeding system is considered; there were differences between the feeding systems for the carcass temperature ($p < 0.05$). Animals from the pasture (34.9 ± 2.71 °C, 25.4 ± 4.27 °C, 20.3 ± 4.27 °C and 16.9 ± 4.00 °C respectively) had a lower resulting temperature at 1, 2, 3 and 4 hours post mortem than animals from the feedlot (38.1 ± 4.51 °C, 31.8 ± 2.93 °C, 25.8 ± 3.57 °C and 21.7 ± 3.33 °C respectively). Animals from the feedlot (5.4 ± 2.79 °C) had a lower temperature at 24 hours post mortem than animals from the pasture (6.7 ± 1.97 °C).

Age per se caused differences between the age classification groups for carcass temperature ($p < 0.05$). AB age classification group (temp1: 36.0 ± 4.84 °C, temp2: 27.8 ± 4.40 °C, temp3: 21.7 ± 3.40 °C, temp4: 18.0 ± 2.94 °C and temp24: 5.5 ± 3.22 °C) constantly had the lowest temperature and the B age classification group (temp1: 36.1 ± 1.99 °C, temp2: 28.2 ± 3.66 °C, temp3: 23.1 ± 3.99 °C, temp4: 19.7 ± 3.50 °C and temp24: 6.7 ± 3.02 °C) constantly showed the highest temperatures. These effects can be explained by difference in conditioning, carcass weight, metabolic rate and enzyme activity (discussed later in dissertation).

The effects of breed on carcass temperature at 1, 2, 3, 4 and 24 hours post mortem is summarised in Table 4.6. Breed per se showed that there were differences between breeds for carcass temperature at 2, 3 and 4 hours post mortem ($p < 0.05$). Brahman cross bred animals had significantly higher carcass temperatures at 2, 3 and 4 hours post mortem (29.4 °C, 23.6 °C and

19.8 °C respectively) than Nguni (27.3 °C, 21.6 °C and 18.4 °C respectively) and Simmental cross bred animals (27.1 °C, 21.9 °C and 18.2 °C respectively). This can be explained by their larger body weight (Thompson, Perry, *et al.*, 2006).

Table 4.6 Effect of breed on the carcass temperature 1, 2, 3, 4 and 24 hours post mortem

Breed (n=180)	Temp ₁ (°C) ($\bar{X} \pm SD$)	Temp ₂ (°C) ($\bar{X} \pm SD$)	Temp ₃ (°C) ($\bar{X} \pm SD$)	Temp ₄ (°C) ($\bar{X} \pm SD$)	Temp ₂₄ (°C) ($\bar{X} \pm SD$)
Br-X	36.1±5.46	29.4±4.66 ^b	23.6±4.82 ^b	19.8±4.11 ^b	6.4±2.62
Ng-X	36.1±2.77	27.3±4.44 ^a	21.6±4.75 ^a	18.4±4.64 ^a	6.1±2.43
Si-X	36.2±2.82	27.1±5.36 ^a	21.9±4.96 ^a	18.2±4.41 ^a	6.0±2.14

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

Temp₁ ~ average temperature 1 hour after slaughter, Temp₂ ~ average temperature 2 hours after slaughter, Temp₃ ~ average temperature 3 hours after slaughter, Temp₄ ~ average temperature 4 hours after slaughter, Temp₂₄ ~ average temperature 24 hours after slaughter.

Br-X ~ Breed average for Brahman cross bred animals, Ng-X ~ Breed average for Nguni cross bred animals, Si-X ~ Breed average for Simmentaler cross bred animals.

The effect of the interaction between breed and production system on carcass temperature is summarised in Table 4.7. The interaction between production systems and breed had an effect on the carcass temperature at 1, 2, 4 and 24 hours post mortem ($p < 0.05$). Brahman animals from the AP production system (33.0 °C) had the lowest temperature at 1 hour post mortem, Nguni animals from the AP production system had the lowest temperature at 2, 3 and 4 hours post mortem (20.6 °C, 15.3 °C and 12.4 °C respectively) and Nguni animals from the ABF production system (2.8 °C) had the lowest temperature at 24 hours post mortem.

Nguni animals from the AF production system (39.2°C) had the highest carcass temperature at 1 hour post mortem, Brahman animals from the AF production system had the highest temperature at 2 and 3 hours post mortem (33.5°C and 33.5°C respectively), Nguni animals from the AF production system (24.6°C) at 4 hours post mortem had the highest temperature and Brahman animals from the ABP production system (8.2°C) had the highest temperature at 24 hours post mortem.

Table 4.7 Effect of the interaction between breed and production system on carcass temperature at 1, 2, 3, 4 and 24 hours post mortem.

Production system (n=180)	Breed	Temp ₁ (°C) ($\bar{X} \pm SD$)	Temp ₂ (°C) ($\bar{X} \pm SD$)	Temp ₃ (°C) ($\bar{X} \pm SD$)	Temp ₄ (°C) ($\bar{X} \pm SD$)	Temp ₂₄ (°C) ($\bar{X} \pm SD$)
AF	Br-X	38.9±1.12 ^b	33.6±1.91 ^g	33.5±2.98 ^e	24.4±1.87 ^f	7.2±1.25 ^{cd}
	Ng-X	39.2±0.75 ^b	32.1±2.84 ^{fg}	27.7±2.31 ^{de}	24.6±2.02 ^f	7.1±1.13 ^{cd}
	Si-X	38.9±0.38 ^b	33.1±2.02 ^g	28.2±2.17 ^{de}	23.9±1.77 ^f	7.3±0.83 ^{cd}
ABF	Br-X	35.6±11.00 ^{ab}	33.1±2.67 ^g	28.6±2.51 ^{de}	20.8±2.10 ^{ef}	5.0±4.58 ^{abc}
	Ng-X	37.5±2.02 ^{ab}	28.6±2.49 ^{def}	21.4±2.05 ^{bc}	17.6±2.14 ^{cde}	2.8±1.00 ^a
	Si-X	38.7±1.09 ^b	30.8±2.17 ^{efg}	23.9±2.00 ^{cd}	19.6±1.52 ^{de}	2.9±0.75 ^{ab}
AP	Br-X	33.0±4.78 ^a	23.8±3.23 ^{abc}	17.7±2.26 ^{ab}	14.2±1.91 ^{abc}	5.9±0.63 ^{cd}
	Ng-X	34.0±1.77 ^{ab}	20.6±3.03 ^a	15.3±3.42 ^a	12.4±3.11 ^a	5.6±0.91 ^{abcd}
	Si-X	33.1±2.02 ^a	22.5±2.86 ^{ab}	16.6±2.33 ^a	13.6±2.35 ^{ab}	5.8±1.12 ^{bcd}
ABP	Br-X	36.1±2.15 ^{ab}	27.6±1.97 ^{cddef}	22.1±2.55 ^{bc}	19.4±1.68 ^{de}	8.2±3.12 ^d
	Ng-X	34.2±2.41 ^{ab}	25.5±2.09 ^{bcd}	19.6±2.23 ^{abc}	16.7±2.53 ^{bcd}	7.9±2.22 ^{cd}
	Si-X	34.3±1.89 ^{ab}	22.1±4.60 ^{ab}	18.2±3.25 ^{ab}	14.8±3.14 ^{abc}	6.4±0.90 ^{cd}
BP	Br-X	36.4±1.39 ^{ab}	28.5±4.35 ^{def}	23.1±4.66 ^c	19.9±3.97 ^{de}	6.0±0.96 ^{cd}
	Ng-X	35.7±2.46 ^{ab}	28.9±2.91 ^{def}	23.5±3.89 ^c	20.3±3.63 ^e	7.5±1.64 ^{cd}
	Si-X	36.4±2.10 ^{ab}	27.2±3.55 ^{cde}	22.6±3.49 ^c	18.8±2.81 ^{de}	6.8±2.90 ^{cd}

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

Temp₁ ~ average temperature 1 hour after slaughter, Temp₂ ~ average temperature 2 hours after slaughter, Temp₃ ~ average temperature 3 hours after slaughter, Temp₄ ~ average temperature 4 hours after slaughter, Temp₂₄ ~ average temperature 24 hours after slaughter.

Br-X ~ breed average for Brahman cross bred animals, Ng-x ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmentaler cross bred animals.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

4.3 Effect of production systems on the water holding capacity and drip loss

Water holding capacity and drip loss of the muscle are influenced by lactic acid and pH (Choe, Choi, Lee, Shin, Ryn, Hong and Kim, 2008). Therefore if there were a significant effect on pH and lactic acid (Thompson, Perry, *et al.*, 2006), there should be a significant effect on water holding capacity and drip loss. According to Muchenje, Dzama, *et al.* (2009b) water holding capacity and drip loss is influenced by pH. Effect of production system on the water holding capacity and drip loss are summarised in Table 4.8.

Table 4.8 Effect of production system on water holding capacity and drip loss

Production system (n=180)	Water holding capacity ($\bar{X} \pm SD$)	Drip loss (%) ($\bar{X} \pm SD$)
AF	0.38 \pm 0.033 ^a	2.23 \pm 0.784 ^b
ABF	0.43 \pm 0.030 ^{bc}	1.61 \pm 0.592 ^a
AP	0.42 \pm 0.060 ^{bc}	1.56 \pm 0.710 ^a
ABP	0.40 \pm 0.040 ^{ab}	1.69 \pm 0.768 ^a
BP	0.44 \pm 0.055 ^c	1.75 \pm 0.617 ^a
Total	0.42 \pm 0.050	1.77 \pm 0.727

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) with the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

Water holding capacity ~ average water holding capacity of a fresh meat sample, Drip loss ~ average amount of fluid loss over time.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

There were differences between production systems for water holding capacity and drip loss ($p < 0.05$). There were numerically small differences between the production systems for water holding capacity, meat from the BP production system had the highest water holding capacity and meat from the AF production system had the lowest water holding capacity. Drip loss of meat from the AF production system differed ($p < 0.05$) from meat from the ABF, ABP, BP and AP production systems (Table 4.8).

Considering the effect of feeding system and age, both had an influence on water holding capacity ($p < 0.05$). Meat from pasture animals resulted in a lower water holding capacity (this is a big difference) compared to meat from feedlot animals (feedlot: 0.74 ± 0.039 and pasture: 0.42 ± 0.054). There were no differences between the animals from the feedlot (1.92 ± 0.76) and animals from the pasture (1.68 ± 0.695) for drip loss ($p > 0.05$). According to Sami, Augustini and *et al.* (2004) feeding system has no effect on water holding capacity ($p > 0.05$). Anderson, Oksberg, Young and Therkildsen (2005) also state that there are no effects on water holding capacity and drip loss caused by the feeding system.

Meat from the B age classification group (0.44 ± 0.054) had a higher water holding capacity than A (0.40 ± 0.051) and AB age classification groups (0.42 ± 0.037). Numerically meat from the AB age classification group (0.42 ± 0.037) had a higher water holding capacity than A age classification group (0.40 ± 0.051). Meat from the B age classification group (1.65 ± 0.682 %) had the lowest drip loss and the A age classification group (1.90 ± 0.816 %) the highest drip loss ($p > 0.05$).

Effect of breed on water holding capacity and drip loss are summarised in Table 4.9. When only the effect of breed is considered, there were no differences in water holding capacity and drip

loss observed between the breeds ($p>0.05$). According to Muchenje, Dzama, *et al.* (2009b) pH influence water holding capacity and drip loss, because there is no breed effect on pH there should be no effect on water holding capacity and drip loss. Frylinck and Heinze (2003) reported that breed had no significant effect on the water holding capacity. These authors reported that Nguni cross bred animals had a lower drip loss than Brahman and Simmental cross bred animals.

Table 4.9 Effect of breed on water holding capacity and drip loss

Breed (n=180)	Water holding capacity ($\bar{X} \pm SD$)	Drip loss (%) ($\bar{X} \pm SD$)
Br-X	0.42±0.043	1.79±0.765
Ng-X	0.41±0.047	1.78±0.692
Si-X	0.42±0.058	1.74±0.735

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

Water holding capacity ~ average water holding capacity of a fresh meat sample, Drip loss ~ average amount of fluid loss over time.

Br-X ~ Breed average for Brahman cross bred animals, Ng-X ~ Breed average for Nguni cross bred animals, Si-X ~ Breed average for Simmentaler cross bred animals.

Effect of the interaction between breed and production system on water holding capacity and drip loss are summarised in Table 4.10. There were no differences between the interactions of production system and breed for drip loss ($p>0.05$), but the interactions between breed and production system had an effect on water holding capacity ($p<0.05$).

Table 4.10 Effect of the interaction between breed and production system on water holding capacity and drip loss

Production system (n=180)	Breed	Water holding capacity ($\bar{X} \pm SD$)	Drip loss ($\bar{X} \pm SD$)
AF	Br-X	0.38±0.028 ^a	2.26±0.767
	Ng-X	0.38±0.035 ^a	2.14±0.920
	Si-X	0.39±0.036 ^{ab}	2.27±0.728
ABF	Br-X	0.42±0.029 ^{abc}	1.74±0.799
	Ng-X	0.43±0.025 ^{abc}	1.62±0.463
	Si-X	0.43±0.039 ^{abc}	1.47±0.513
AP	Br-X	0.44±0.043 ^{abc}	1.53±0.794
	Ng-X	0.42±0.072 ^{abc}	1.78±0.898
	Si-X	0.41±0.063 ^{abc}	1.41±0.408
ABP	Br-X	0.39±0.034 ^{ab}	1.64±0.569
	Ng-X	0.41±0.037 ^{abc}	1.64±0.672
	Si-X	0.41±0.048 ^{abc}	1.81±1.0152
BP	Br-X	0.44±0.041 ^{bc}	1.73±0.762
	Ng-X	0.43±0.048 ^{abc}	1.83±0.480
	Si-X	0.46±0.071 ^c	1.70±0.593

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

Water holding capacity ~ average water holding capacity of a fresh meat sample, Drip loss ~ average amount of fluid loss over time.

Br-X ~ breed average for Brahman cross bred animals, Ng-x ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmentaler cross bred animals.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

4.4 Effect of production system on the sarcomere lengths at 1 and 3 days post mortem

Effect of production system on sarcomere length at 1 and 3 days post mortem is summarised in Table 4.11. Muscles from the AP production system (1.66 μm) had shorter sarcomere lengths at 1 day post mortem than muscles from the AF (1.75 μm), ABF (1.75 μm), ABP (1.73 μm) and BP production systems (1.72 μm) ($p < 0.05$). Sarcomere lengths at 3 days post mortem range from 1.69 μm (AP production system) to 1.76 μm (AF production system) ($p < 0.05$).

Table 4.11 Effect of production system on sarcomere length at 1 and 3 days post mortem

Production system (n=180)	Sarcomere length 1 (μm) ($\bar{X} \pm \text{SD}$)	Sarcomere length 3 (μm) ($\bar{X} \pm \text{SD}$)
AF	1.75 \pm 0.08 ^b	1.76 \pm 0.07 ^{bc}
ABF	1.75 \pm 0.07 ^b	1.80 \pm 0.07 ^c
AP	1.66 \pm 0.09 ^a	1.69 \pm 0.11 ^a
ABP	1.73 \pm 0.06 ^b	1.74 \pm 0.09 ^{ab}
BP	1.72 \pm 0.08 ^b	1.74 \pm 0.08 ^{ab}
Total	1.72 \pm 0.08	1.75 \pm 0.09

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm \text{SD}$ ~ mean \pm standard deviation.

Sarcomere length 1 ~ average sarcomere length at 1 day after slaughter, Sarcomere length 3 ~ average sarcomere length 3 days after slaughter.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

The study of Martínez-Cerezo, Sanudo, Panea, Medel, Delfa, Sierra, Beltran, Cepero and Olleta, (2005) as well as Bruce, Stark and Beilken (2003) show that the feeding system has an effect on sarcomere lengths, while other studies show no effect (Sami, Augustini and *et al*, 2004; Lowe, Peachey and *et al.*, 2002). Feeding system and age per se showed differences for the sarcomere lengths at 1 and 3 days post mortem ($p < 0.05$). Muscles from the pasture (day 1: 1.70 \pm 0.08 μm and day 3: 1.72 \pm 0.09 μm) had constantly shorter lengths than the muscles from the feedlot (day 1: 1.75 \pm 0.08 μm and day 3: 1.78 \pm 0.07 μm).

Muscles from the AB age classification group (day 1: 1.74 \pm 0.17 μm and day 3: 1.77 \pm 0.09 μm) had the longest sarcomere length and A age classification group (day 1: 1.71 \pm 0.10 μm and day 3: 1.73 \pm 0.10 μm) the shortest. There were no significant differences between A (day 1: 1.71 \pm 0.10 μm and day 3: 1.73 \pm 0.10 μm). B age classification groups (day 1: 1.72 \pm 0.08 μm and day 3: 1.74 \pm 0.08 μm) and AB age classification groups (day 1: 1.74 \pm 0.17 μm and day 3: 1.77 \pm 0.09 μm) at 1 and 3 days post mortem ($p > 0.05$). Sami, Augustini and *et al* (2004) as well as Culler, Parrish, Smith and Cross (1978), state that the animals age does not affect sarcomere lengths.

Effect of breed on the sarcomere length at 1 and 3 days post mortem is summarised in Table 4.12. When only the effect of breed is considered; there were differences ($p < 0.05$). Numerically it does not seem much but Nguni cross bred animals (day 1: 1.71 μm and day 3: 1.72 μm) had a significantly shorter length than Brahman cross bred animals (day 1: 1.74 μm and day 3: 1.77 μm). On the other hand, there were no significant differences between Nguni (day 1: 1.71 μm and day 3: 1.72 μm) and Simmental cross bred animals (day 1: 1.72 μm and day 3: 1.74 μm), and Brahman (day 1: 1.74 μm and day 3: 1.77 μm) and Simmental cross bred animals (day 1: 1.72 μm and day

3: 1.74 μm). According to the results reported by Frylinck and Heinze (2003) Simmental cross bred animals had shorter sarcomere lengths at 1 and 3 days post mortem than the Nguni and Brahman cross bred animals, and according to Frylinck, Van Wyk, *et al.* (2009) Nguni cross bred animals have the shortest sarcomere lengths.

Table 4.12 Effect of breed on sarcomere length at 1 and 3 days post mortem

Breed (n=180)	Sarcomere lengths 1 (μm) ($\bar{X} \pm \text{SD}$)	Sarcomere lengths 3 (μm) ($\bar{X} \pm \text{SD}$)
Br-X	1.74 \pm 0.08 ^b	1.77 \pm 0.08 ^b
Ng-X	1.71 \pm 0.09 ^a	1.72 \pm 0.10 ^a
Si-X	1.72 \pm 0.08 ^{ab}	1.74 \pm 0.09 ^{ab}

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm \text{SD}$ ~ mean \pm standard deviation.

Sarcomere lengths 1 ~ average sarcomere length at 1 day after slaughter, Sarcomere lengths 3 ~ average sarcomere length 3 days after slaughter.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmentaler cross bred animals.

The effect of the interaction between breed and production system of sarcomere lengths are summarised in Table 4.13. Interaction between production system and breed had an effect on the sarcomere lengths at 1 and 3 day post mortem ($p < 0.05$). Numerically there were relatively small effects between the interactions, Nguni animals from the AP production system (1.62 μm and 1.63 μm respectively) had the shortest sarcomere length at 1 and 3 days post mortem and Brahman animals from the ABF production system (1.79 μm and 1.84 μm respectively) had the longest sarcomere length at 1 and 3 hours post mortem.

Table 4.13 Effect of the interaction between breed and production system on sarcomere lengths at 1 and 3 days post mortem

Production system (n=180)	Breed	Sarcomere length ₁ (μm) ($\bar{X} \pm \text{SD}$)	Sarcomere length ₃ (μm) ($\bar{X} \pm \text{SD}$)
AF	Br-X	1.76 \pm 0.087 ^{bc}	1.77 \pm 0.073 ^{bc}
	Ng-X	1.73 \pm 0.094 ^{abc}	1.76 \pm 0.089 ^{bc}
	Si-X	1.75 \pm 0.077 ^{bc}	1.76 \pm 0.049 ^{bc}
ABF	Br-X	1.79 \pm 0.076 ^c	1.84 \pm 0.092 ^c
	Ng-X	1.72 \pm 0.070 ^{abc}	1.76 \pm 0.036 ^{bc}
	Si-X	1.76 \pm 0.056 ^{bc}	1.80 \pm 0.065 ^{bc}
AP	Br-X	1.69 \pm 0.060 ^{abc}	1.74 \pm 0.058 ^{abc}
	Ng-X	1.62 \pm 0.132 ^a	1.63 \pm 0.153 ^a
	Si-X	1.67 \pm 0.066 ^{ab}	1.70 \pm 0.083 ^{ab}
ABP	Br-X	1.75 \pm 0.055 ^{bc}	1.78 \pm 0.040 ^{bc}
	Ng-X	1.72 \pm 0.061 ^{abc}	1.74 \pm 0.079 ^{abc}
	Si-X	1.71 \pm 0.066 ^{abc}	1.70 \pm 0.116 ^{abc}
BP	Br-X	1.71 \pm 0.084 ^{abc}	1.74 \pm 0.078 ^{abc}
	Ng-X	1.72 \pm 0.065 ^{abc}	1.72 \pm 0.058 ^{ab}
	Si-X	1.72 \pm 0.102 ^{abc}	1.74 \pm 0.110 ^{abc}

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm \text{SD}$ ~ mean \pm standard deviation.

Sarcomere length₁ ~ average sarcomere length at 1 day post mortem, Sarcomere length₃ ~ average sarcomere length 3 days post mortem.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmentaler cross bred animals.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

4.5 Effect of production systems on the muscle glycolytic potential, lactic acid, glucose, glycogen, glucose-6-phosphate, ATP and creatine phosphate at 1, 2, 4 and 24 hours post mortem

It seems that a threshold value (approximately 100 $\mu\text{mol/g}$) at slaughter for glycolytic potential occurs. A high ultimate pH is associated with a glycolytic potential below this threshold value and above this value no effect on pH can be observed (Wulf, Emmett, *et al.*, 2002). According to Wulf, Emmett, *et al.* (2002) a higher glycolytic potential is associated with increased

tenderness, and low glycolytic potential is associated with DFD meat and results in substantially less palatable cooked steaks.

Effect of production system on muscle glycolytic potential at 1, 2, 4 and 24 hours post mortem is summarised in Table 4.14. There were differences ($p < 0.05$) between the production systems for the muscle glycogen potential at 1, 2 and 24 hours post mortem but not at 4 hours post mortem ($p > 0.05$). Muscles from ABF (156.76 $\mu\text{mol/g}$, 15.53 $\mu\text{mol/g}$, 152.90 $\mu\text{mol/g}$ and 153.00 $\mu\text{mol/g}$ respectively) and AP (154.00 $\mu\text{mol/g}$, 153.80 $\mu\text{mol/g}$, 152.90 $\mu\text{mol/g}$ and 153.00 $\mu\text{mol/g}$ respectively) production systems had higher glycolytic potential than muscles from AF (142.40 $\mu\text{mol/g}$, 147.62 $\mu\text{mol/g}$, 143.64 $\mu\text{mol/g}$ and 138.46 $\mu\text{mol/g}$ respectively), ABP (140.53 $\mu\text{mol/g}$, 142.00 $\mu\text{mol/g}$, 144.13 $\mu\text{mol/g}$ and 145.02 $\mu\text{mol/g}$ respectively) and BP (134.16 $\mu\text{mol/g}$, 133.44 $\mu\text{mol/g}$, 134.32 $\mu\text{mol/g}$ and 132.38 $\mu\text{mol/g}$ respectively) production systems at 1, 2, 4 and 24 hours post mortem. Muscles from the BP production system (134.16 $\mu\text{mol/g}$, 133.44 $\mu\text{mol/g}$, 134.32 $\mu\text{mol/g}$ and 132.38 $\mu\text{mol/g}$ respectively) had the lowest potential.

Table 4.14 Effect of production system on muscle glycolytic potential at 1, 2, 4 and 24 hour post mortem

Production system (n=180)	Glycolytic Potential ₁ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	Glycolytic Potential ₂ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	Glycolytic Potential ₄ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	Glycolytic Potential ₂₄ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)
AF	142.40 \pm 20.87 ^{ab}	147.62 \pm 25.17 ^{ab}	143.64 \pm 25.64	138.46 \pm 21.80 ^{ab}
ABF	156.76 \pm 21.30 ^b	150.53 \pm 29.75 ^{ab}	150.47 \pm 26.37	145.33 \pm 27.42 ^{ab}
AP	154.00 \pm 28.54 ^b	153.80 \pm 31.02 ^b	152.90 \pm 34.38	153.00 \pm 34.04 ^b
ABP	140.53 \pm 26.29 ^{ab}	142.00 \pm 36.64 ^{ab}	144.13 \pm 26.24	145.02 \pm 26.62 ^{ab}
BP	134.16 \pm 30.65 ^a	133.44 \pm 33.96 ^a	134.32 \pm 33.18	132.38 \pm 23.53 ^a
Total	145.08 \pm 27.08	143.98 \pm 29.30	144.61 \pm 29.89	142.31 \pm 27.40

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm \text{SD}$ ~ mean \pm standard deviation.

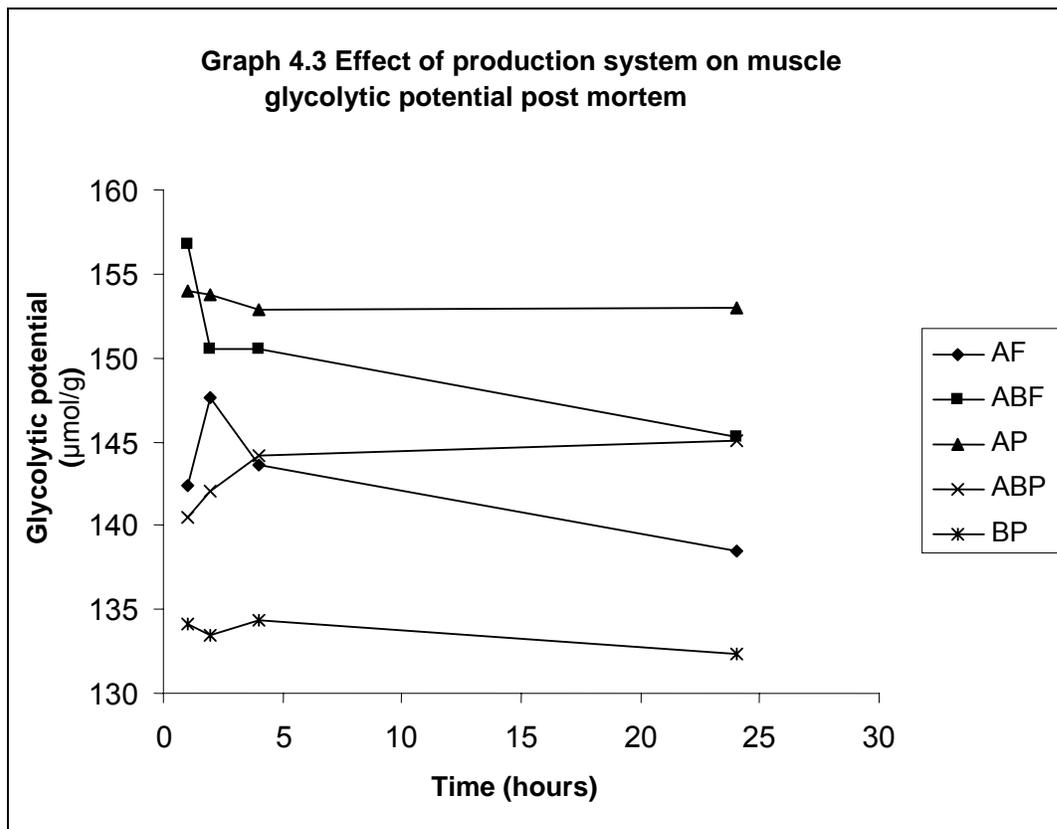
Glycolytic Potential₁ ~ muscle glycogen potential at 1 hour post mortem, Glycolytic Potential₂ ~ muscle glycogen potential at 2 hours post mortem, Glycolytic Potential₄ ~ muscle glycogen potential at 4 hours post mortem and Glycolytic Potential₂₄ ~ muscle glycogen potential at 24 hours post mortem.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

Effect of the production system on the glycolytic potential over time is illustrated in Graph 4.3. Muscles from the AF and ABF production systems showed a higher loss of glycolytic potential than the muscles from the AP, ABP and BP production systems. It seems that the glycolytic potential increased as time progressed for the muscles from the ABP production system. It seems that the

muscles from BP production system experience a slow decrease in glycolytic potential. Muscles from the AF production system seem to gain glycolytic potential before the glycolytic potential decreases. It seems that muscles from the AP production system experience an initial decrease before the potential stays constant. According to Immonen and Puolanne (2000) glycolytic potential increases as muscle pH value decrease, the pH decrease as time progress.

Glycolytic potential is calculated as follows; glycolytic potential = 2 * (glucose-6-phosphate + glucose + glycogen) + lactic acid. As discussed later in the dissertation muscle glucose-6-phosphate, glucose and lactic acid increase as time progresses after slaughter. Therefore if these three concentrations increase as time progress, glycolytic potential should also increase as time progresses. The values of the different production systems are above the threshold value (100 $\mu\text{mol/g}$); therefore a problem with DFD did not occur.



Considering the effect of feeding system only; there were no differences ($p > 0.05$) between the feeding systems for glycolytic potential. Immonen, Schaefer, *et al.*, (2000), reported no significant difference between feedlot fed animals and pasture fed animal's muscle glycolytic potential.

When age per se is considered; there were differences ($p < 0.05$) between the ages for glycolytic potential. Muscles from the B age classification group ($134.16 \pm 30.65 \mu\text{mol/g}$, $133.44 \pm 33.96 \mu\text{mol/g}$, $134.32 \pm 33.18 \mu\text{mol/g}$ and $132.38 \pm 23.53 \mu\text{mol/g}$ respectively) had lower ($p < 0.05$) glycolytic potential than muscles from the A ($147.94 \pm 25.33 \mu\text{mol/g}$, $147.97 \pm 28.47 \mu\text{mol/g}$, $148.07 \pm 30.27 \mu\text{mol/g}$ and $145.41 \pm 29.04 \mu\text{mol/g}$ respectively) and AB age classification groups ($148.76 \pm 25.09 \mu\text{mol/g}$, $146.32 \pm 25.88 \mu\text{mol/g}$, $147.35 \pm 26.31 \mu\text{mol/g}$ and $145.18 \pm 26.83 \mu\text{mol/g}$ respectively) at 1, 2, 4 and 24 hours post mortem.

Effect of the breed on muscle glycolytic potential at 1, 2, 4 and 24 hours post mortem is summarised in Table 4.15. There were no differences ($p > 0.05$) between the breeds for glycolytic potential observed at 1, 2, 4 and 24 hours post mortem.

Table 4.15 Effect of the breed on muscle glycolytic potential at 1, 2, 4 and 24 hours post mortem

Breed (n=180)	Glycolytic Potential ₁ ($\bar{X} \pm \text{SD}$)	Glycolytic Potential ₂ ($\bar{X} \pm \text{SD}$)	Glycolytic Potential ₄ ($\bar{X} \pm \text{SD}$)	Glycolytic Potential ₂₄ ($\bar{X} \pm \text{SD}$)
Br-X	146.84 \pm 28.85	145.79 \pm 30.26	145.34 \pm 29.92	144.93 \pm 25.96
Ng-X	142.07 \pm 24.68	139.45 \pm 26.47	140.81 \pm 29.34	139.17 \pm 26.17
Si-X	146.58 \pm 27.89	147.09 \pm 31.11	148.07 \pm 30.50	143.08 \pm 30.17

$\bar{X} \pm \text{SD}$ ~ mean \pm standard deviation..

Glycolytic Potential₁ ~ muscle glycogen potential at 1 hour post mortem, Glycolytic Potential₂ ~ muscle glycogen potential at 2 hours post mortem, Glycolytic Potential₄ ~ muscle glycogen potential at 4 hours post mortem and Glycolytic Potential₂₄ ~ muscle glycogen potential at 24 hours post mortem.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmentaler cross bred animals.

Effect of the interaction between breed and production system on glycolytic potential at 1, 2, 4 and 24 hours post mortem is summarised in Table 4.16. There were only effects at 1 hour post mortem between the interactions between breed and production system ($p < 0.05$) and not at 2, 4 and 24 hours post mortem ($p > 0.05$) for glycolytic potential. Muscles from the Brahman cross bred animals from the AP production system ($168.79 \mu\text{mol/g}$, $163.18 \mu\text{mol/g}$, $162.97 \mu\text{mol/g}$ and $162.95 \mu\text{mol/g}$ respectively) constantly had the highest glycolytic potential. Muscles from the Brahman cross bred animals from the ABP production system ($126.14 \mu\text{mol/g}$) had the lowest potential at 1 hour post mortem, Nguni cross bred animals from the BP production system ($129.58 \mu\text{mol/g}$) had the lowest potential at 2 hours post mortem, and Simmental cross bred animals from the BP production system ($126.90 \mu\text{mol/g}$ and $126.55 \mu\text{mol/g}$ respectively) had the lowest potential at 4 and 24 hours post mortem.

Table 4.16 Effect of the interaction between breed and production system on muscle glycolytic potential at 1, 2, 4 and 24 hours post mortem.

Production system	Breed	GlycP ₁ ($\bar{X} \pm SD$)	GlycP ₂ ($\bar{X} \pm SD$)	GlycP ₄ ($\bar{X} \pm SD$)	GlycP ₂₄ ($\bar{X} \pm SD$)
AF	Br-X	141.09±20.05 ^{ab}	139.90±29.27	139.65±28.73	137.33±18.43
	Ng-X	140.95±17.62 ^{ab}	138.96±21.76	144.62±24.27	142.92±24.57
	Si-X	145.16±25.71 ^{ab}	149.01±24.86	146.64±27.39	135.14±23.11
ABF	Br-X	154.26±23.44 ^{ab}	150.37±27.94	154.49±29.33	151.95±29.18
	Ng-X	160.81±16.57 ^{ab}	153.11±18.70	143.97±29.33	144.30±20.73
	Si-X	154.09±25.67 ^{ab}	147.14±30.00	154.97±26.77	138.84±34.00
AP	Br-X	168.79±21.87 ^b	163.18±23.57	162.97±26.00	162.95±25.87
	Ng-X	137.32±29.47 ^{ab}	138.14±31.07	137.98±39.51	138.91±25.87
	Si-X	154.32±27.42 ^{ab}	158.27±34.23	156.11±35.11	155.61±37.60
ABP	Br-X	126.14±22.33 ^a	131.48±17.41	129.61±16.73	131.04±16.71
	Ng-X	139.15±24.89 ^{ab}	137.01±26.92	142.36±24.18	142.07±26.29
	Si-X	155.37±25.38 ^{ab}	157.90±27.84	159.59±29.16	161.48±27.44
BP	Br-X	143.20±36.37 ^{ab}	148.66±39.31	139.80±36.24	141.43±27.34
	Ng-X	130.60±25.41 ^a	129.58±30.63	135.33±37.07	128.10±24.64
	Si-X	127.55±28.31 ^a	129.82±30.15	126.90±25.33	126.55±14.18

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmentaler cross bred animals.

GlycP₁ ~ muscle glycogen potential at 1 hour post mortem, GlycP₂ ~ muscle glycogen potential at 2 hours post mortem, GlycP₄ ~ muscle glycogen potential at 4 hours post mortem and GlycP₂₄ ~ muscle glycogen potential at 24 hours post mortem.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

According to Thompson, Perry, *et al.* (2006), age influences the glycolytic rate, and therefore lactate, glucose, glycogen, glucose-6-phosphate, as well as ATP and creatine phosphate. Lactic acid is produced during anaerobic respiration. After death the metabolism in the muscles does not stop immediately. Because of the circulation stopping, there is no oxygen transported to the muscles and anaerobic respiration occurs (Lawrie, 1974; Varnam and Sutherland, 1996; Munchenje, Dzama, *et al.*, 2009b)

According to Thompson (2002) there is a relationship between pH and lactic acid concentrations in muscle post mortem. Effect of the production system on the lactic acid concentration in muscle post mortem is summarised in Table 4.17. Average lactic acid

concentrations recorded at 1, 2, 4, and 24 hours post mortem were; 42.91 $\mu\text{mol/g}$, 49.56 $\mu\text{mol/g}$, 59.97 $\mu\text{mol/g}$ and 76.25 $\mu\text{mol/g}$. There were only a differences at 24 hours post mortem ($p < 0.05$) between the production systems for the muscle lactic acid concentration. Muscles from the AF production system had the tendency for higher lactic acid concentrations compared to ABF, AP, ABP and BP production systems. Muscles from the BP production system had the tendency for lower lactic acid concentrations compared to AF, ABF, AP and ABP production systems (Table 4.17).

Table 4.17 Effect of production system on muscle lactic acid concentrations at 1, 2, 4 and 24 hours post mortem

Production system (n=180)	Lactate ₁ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	Lactate ₂ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	Lactate ₄ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	Lactate ₂₄ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)
AF	44.22 \pm 7.10	52.35 \pm 8.61	64.70 \pm 11.72	83.13 \pm 10.21 ^b
ABF	45.62 \pm 10.80	51.35 \pm 11.50	60.97 \pm 11.08	77.53 \pm 13.60 ^a
AP	43.11 \pm 10.17	50.05 \pm 10.25	59.58 \pm 12.27	75.74 \pm 11.99 ^a
ABP	41.78 \pm 11.03	48.29 \pm 9.08	57.58 \pm 12.27	73.53 \pm 13.60 ^a
BP	40.38 \pm 10.81	47.60 \pm 11.81	57.37 \pm 12.40	72.18 \pm 9.86 ^a
Total	42.91 \pm 10.17	49.56 \pm 10.58	59.97 \pm 12.45	76.25 \pm 11.59

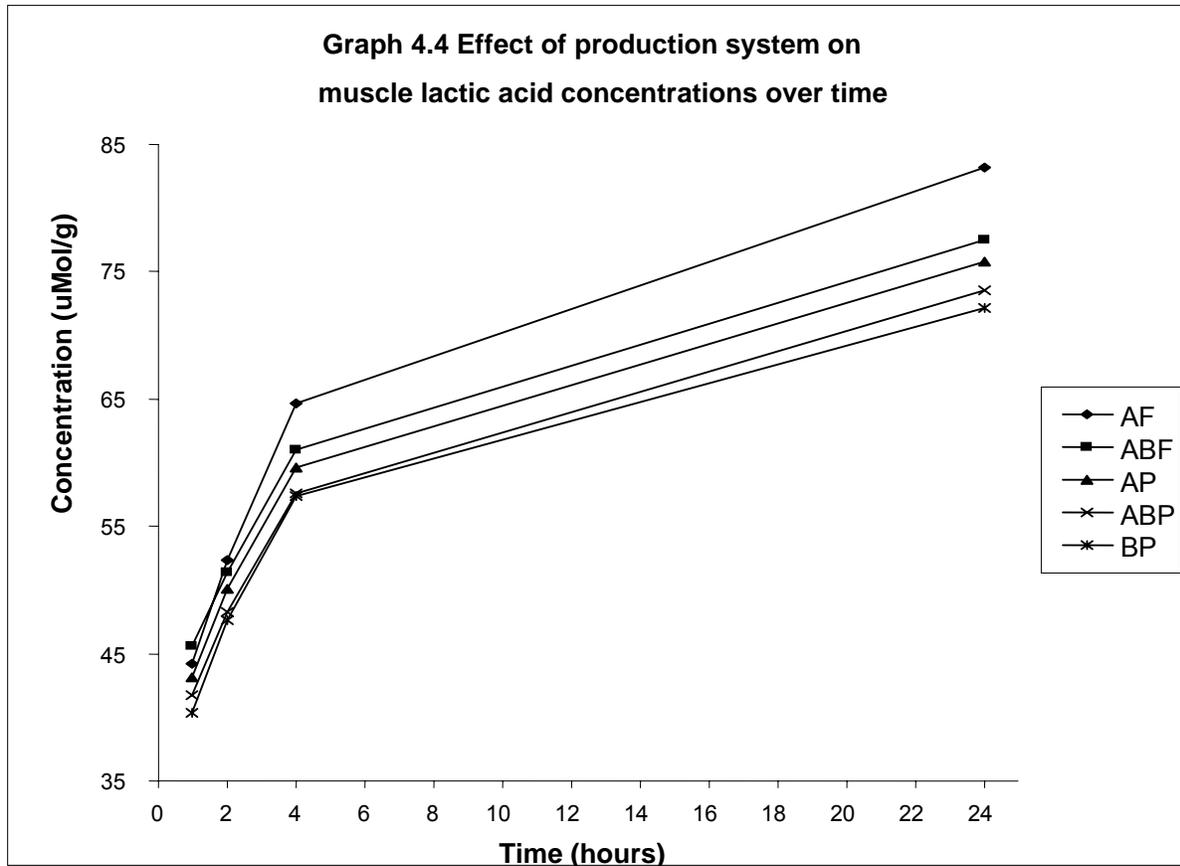
^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm \text{SD}$ ~ mean \pm standard deviation.

Lactate₁ ~ average lactic acid concentrations in the muscle 1 hour after slaughter, Lactate₂ ~ average lactic acid concentrations in the muscle 2 hours after slaughter, Lactate₄ ~ lactic acid concentrations in the muscle 4 hours after slaughter, Lactate₂₄ ~ average lactic acid concentrations in the muscle 24 hours after slaughter.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

Effect of production system on muscle lactic acid concentrations as time progress is illustrated in Graph 4.4. The concentrations increased as time progressed, the rate of increase differed slightly from one another. Muscles from the AF production system had the highest concentrations of muscle lactic acid. Immonen and Puolanne (2000) reported that as the pH decreases, lactic acid concentration in the muscles increases, as time progress the pH of the meat decreases. Therefore as time progress, the muscle lactic acid concentration should increase.



Considering the effect of feeding system only, there was only an effect on lactic acid concentrations in the muscle at 4 and 24 hours post slaughter ($p < 0.05$). Muscles from the feedlot fed animals ($62.84 \pm 11.48 \mu\text{mol/g}$, $80.23 \pm 10.13 \mu\text{mol/g}$, $44.87 \pm 9.09 \mu\text{mol/g}$ and $52.09 \pm 10.46 \mu\text{mol/g}$ respectively) had higher muscle lactic acid concentrations post mortem than muscles from the pasture fed animals ($58.13 \pm 12.75 \mu\text{mol/g}$, $73.69 \pm 11.79 \mu\text{mol/g}$, $41.65 \pm 10.65 \mu\text{mol/g}$ and 48.56 ± 10.48 respectively). There is no agreement in the literature on effect of feeding system on lactic acid concentrations in the muscles post slaughter. Some state that there is an effect, with feedlot animals producing higher levels of lactic acid (Ferguson, Daly, Gardner and Tume, 2008; Immonen, Ruusunen, *et al.*, 2000), whereas others state that there is no effect (Lowe, Pearcey and *et al.*, 2002; Immonen, Schaefer, *et al.*, 2000).

When only the effect of age is considered, there were no differences ($p > 0.05$) between the age classification groups for muscle lactic acid concentration.

Effect of breed on muscle lactic acid concentrations at 1, 2, 4 and 24 hours post mortem is summarised in Table 4.18. Considering the effect of breed only; there were no differences between the breeds for lactic acid concentrations in the muscle post mortem ($p > 0.05$). According to Thompson (2002) there is a relationship between pH and lactic acid concentrations in muscle post

mortem. Therefore if there were no significant differences in pH, then no significant differences were expected for lactic acid concentrations in the muscle post mortem.

Table 4.18 Effects of breed on muscle lactic acid concentrations 1, 2, 4 and 24 hours post mortem.

Breed (n=180)	Lactate ₁ (μmol/g) ($\bar{X} \pm SD$)	Lactate ₂ (μmol/g) ($\bar{X} \pm SD$)	Lactate ₄ (μmol/g) ($\bar{X} \pm SD$)	Lactate ₂₄ (μmol/g) ($\bar{X} \pm SD$)
Br-X	40.20±8.70	47.14±9.99	57.75±10.71	74.90±9.59
Ng-X	44.76±10.53	51.46±9.27	60.74±12.93	77.39±13.10
Si-X	43.69±10.74	51.17±12.00	61.40±13.43	76.25±11.59

$\bar{X} \pm SD$ ~ mean ± standard deviation.

Lactate₁ ~ average lactic acid concentrations in the muscle 1 hour after slaughter, Lactate₂ ~ average lactic acid concentrations in the muscle 2 hours after slaughter, Lactate₄ ~ lactic acid concentrations in the muscle 4 hours after slaughter, Lactate₂₄ ~ average lactic acid concentrations in the muscle 24 hours after slaughter.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmentaler cross bred animals.

Effect of the interaction between breed and production system on muscle lactic acid concentration is summarised in Table 4.19. The interactions between production system and breed on the muscle lactic acid concentrations at 1, 2, 4 and 24 hours post mortem were not significant ($p > 0.05$).

Table 4.19 Effect of the interaction between breed and production system on muscle lactic acid concentrations at 1, 2, 4 and 24 hours post mortem

Production system (n=180)	Breed	Lakt ₁ (μmol/g) ($\bar{X} \pm SD$)	Lakt ₂ (μmol/g) ($\bar{X} \pm SD$)	Lakt ₄ (μmol/g) ($\bar{X} \pm SD$)	Lakt ₂₄ (μmol/g) ($\bar{X} \pm SD$)
AF	Br-X	43.01±5.87	49.63±9.07	61.24±12.54	82.16±9.04
	Ng-X	42.66±8.43	49.53±10.47	63.26±10.38	84.70±9.86
	Si-X	46.85±6.73	57.66±6.95	69.49±11.36	82.66±12.18
ABF	Br-X	40.72±9.00	49.00±9.42	59.82±8.57	72.67±9.56
	Ng-X	49.26±11.43	53.57±11.40	59.88±12.22	81.77±9.54
	Si-X	45.58±10.56	52.48±14.08	63.75±12.42	76.27±5.99
AP	Br-X	44.90±11.79	48.97±12.63	60.50±10.66	76.75±5.95
	Ng-X	38.64±10.02	48.67±8.72	57.80±16.23	73.84±18.67
	Si-X	45.19±8.15	52.19±9.51	60.25±10.72	76.41±9.80
ABP	Br-X	36.59±6.29	44.09±6.99	51.87±8.93	70.74±12.04
	Ng-X	45.98±9.60	49.84±7.97	59.99±14.27	73.01±11.81
	Si-X	41.14±14.36	50.13±11.36	59.91±16.27	76.72±17.26
BP	Br-X	36.53±7.47	44.48±10.72	55.36±10.77	72.14±7.81
	Ng-X	45.10±11.25	54.70±7.04	62.60±12.71	73.95±12.78
	Si-X	40.10±12.45	44.10±14.17	54.45±13.09	70.45±9.08

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) with the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean ± standard deviation.

Lakt₁ ~ average lactic acid concentration in the muscle 1 hour after slaughter, Lakt₂ ~ average lactic acid concentration in the muscle 2 hours after slaughter, Lakt₄ ~ lactic acid concentration in the muscle 4 hours after slaughter, Lakt₂₄ ~ average lactic acid concentration in the muscle 24 hours after slaughter.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmentaler cross bred animals.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

Effect of production system on muscle glucose concentrations at 1, 2, 4 and 24 hours post mortem are summarised in Table 4.20. There were no differences between production systems for the muscle glucose concentrations at 1, 2, 4 and 24 hours post mortem ($p > 0.05$).

Table 4.20 Effect of production system on muscle glucose concentrations at 1, 2, 4 and 24 hours post mortem.

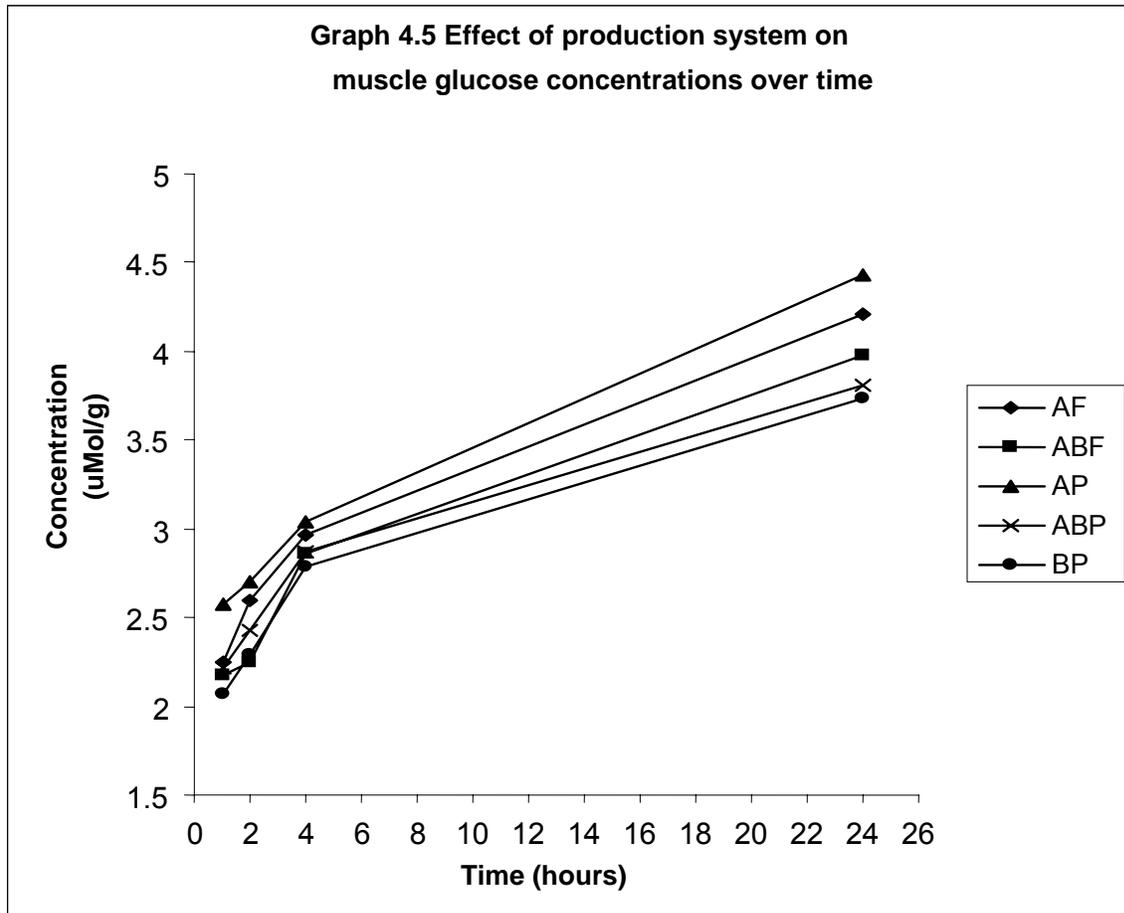
Production system (n=180)	Glucose ₁ (μmol/g) ($\bar{X} \pm SD$)	Glucose ₂ (μmol/g) ($\bar{X} \pm SD$)	Glucose ₄ (μmol/g) ($\bar{X} \pm SD$)	Glucose ₂₄ (μmol/g) ($\bar{X} \pm SD$)
AF	2.25±0.97	2.60±0.84	2.97±0.89	4.21±1.19
ABF	2.17±0.80	2.25±0.80	2.86±0.90	3.98±1.12
AP	2.58±0.96	2.70±0.94	3.04±0.93	4.43±0.98
ABP	2.22±0.77	2.43±0.76	2.87±0.76	3.81±0.78
BP	2.07±0.83	2.29±0.91	2.79±1.03	3.74±0.98
Total	2.25±0.87	2.45±0.86	2.90±0.90	4.02±1.04

$\bar{X} \pm SD$ ~ mean ± standard deviation.

Glucose₁ ~ average glucose concentrations in the muscle 1 hour after slaughter, Glucose₂ ~ average glucose concentrations in the muscle 2 hours after slaughter, Glucose₄ ~ glucose concentrations in the muscle 4 hours after slaughter, Glucose₂₄ ~ average glucose concentrations in the muscle 24 hours after slaughter.

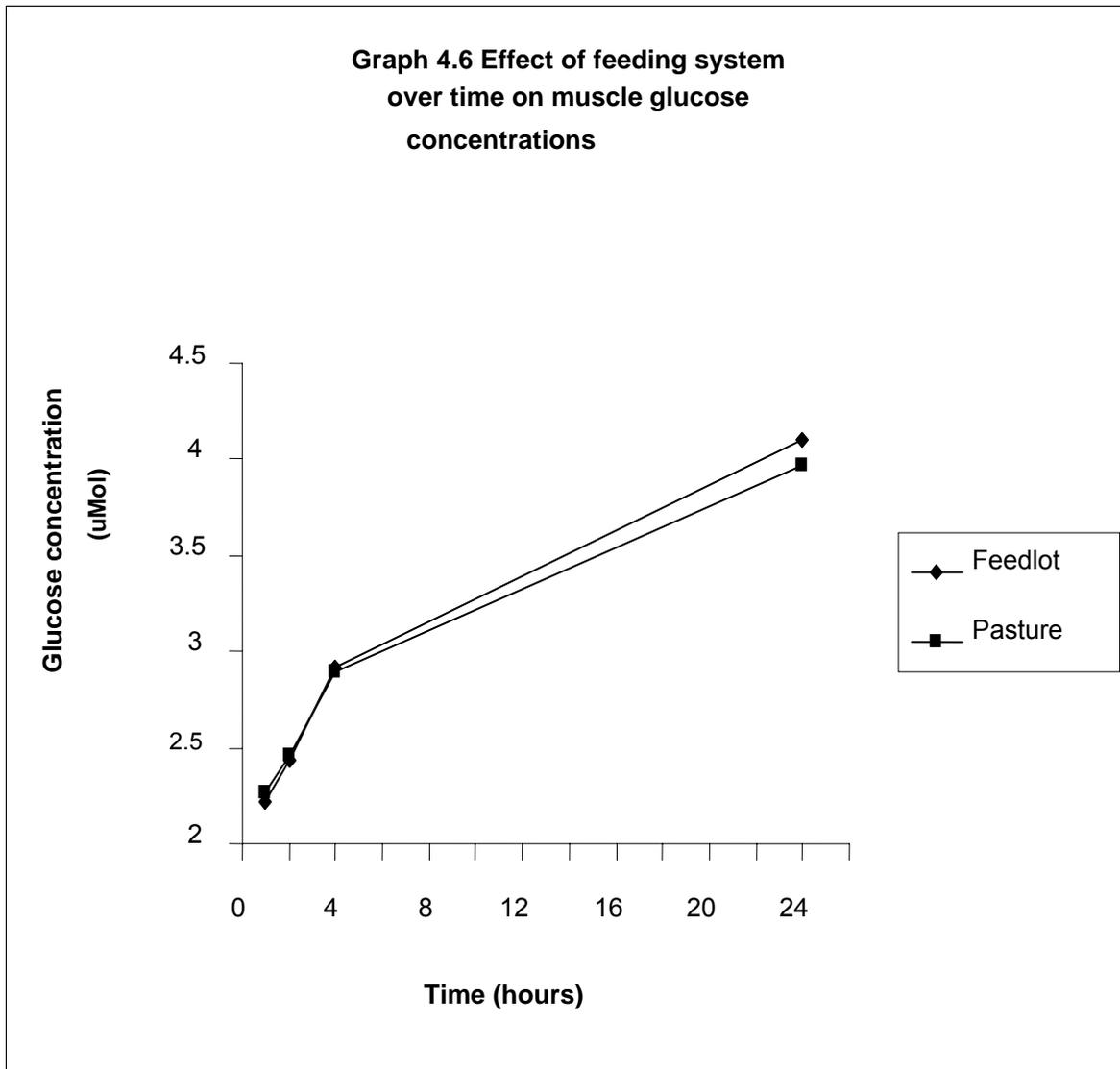
AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

To illustrate the general trend of the data in Table 4.20 it is graphically illustrated (Graph 4.5). Effect of production system on muscle glucose concentrations over time is illustrated in Graph 4.5. Concentrations of muscle glucose increased as time progressed, the rate of this increase (as indicated by the gradient in Graph 4.5) differed slightly between production systems. This increase had to do with the metabolism in the muscle after death. Muscles from the AP production system constantly had the highest concentrations of glucose in the muscle and muscles from the BP production system constantly had the lowest concentrations. Muscles from the AF production system constantly had the second highest concentrations of glucose in the muscle and muscles from the ABP production system constantly had the second lowest concentrations. These results are supported by Thompson, Perry, *et al.* (2006) as well as Immonen and Puolanne (2000). Glucose concentration increased as the pH value in the muscle decreased, therefore as time progressed glucose concentration increased (Immonen and Puolanne, 2000).



Feeding system per se showed no differences for muscle glucose concentrations at 1, 2, 4 and 24 hours post mortem ($p > 0.05$).

Graph 4.6 illustrates the effect of the feeding system on the muscle glucose concentration as time progressed. Initially muscles from pasture fed animals had a higher muscle glucose concentration, but between 2 and 4 hours post mortem there was a crossover point, after which muscles from feedlot fed animals had a higher concentration.



Age per se only differed between age classification groups at 24 hours post mortem for muscle glucose concentrations ($p < 0.05$). Muscles from the AB ($3.89 \pm 0.96 \mu\text{mol/g}$) and B age classification groups ($3.74 \pm 0.98 \mu\text{mol/g}$) had lower concentrations at 24 hours than muscles from the A age classification group ($4.32 \pm 1.09 \mu\text{mol/g}$) ($p < 0.05$). Even though there were no significant ($p > 0.05$) differences between the AB ($3.89 \pm 0.96 \mu\text{mol/g}$) and B age classification groups ($3.74 \pm 0.98 \mu\text{mol/g}$), numerically B age classification group ($3.74 \pm 0.98 \mu\text{mol/g}$) had lower concentrations than the AB age classification group ($3.89 \pm 0.96 \mu\text{mol/g}$).

Effect of breed on muscle glucose concentrations at 1, 2, 4 and 24 hours post mortem are summarised in Table 4.21. Considering the effect of breed only, there were no differences between them for the muscle glucose concentrations at 1, 2, 4 and 24 hours post mortem ($p > 0.05$).

Table 4.21 Effect of breed on muscle glucose concentrations at 1, 2, 4 and 24 hours post mortem.

Breed (n=180)	Glucose ₁ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	Glucose ₂ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	Glucose ₄ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	Glucose ₂₄ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)
Br-X	2.34 \pm 0.85	2.51 \pm 0.71	3.10 \pm 0.93	4.21 \pm 1.03
Ng-X	2.22 \pm 0.92	2.42 \pm 0.91	2.83 \pm 0.87	3.88 \pm 1.09
Si-X	2.18 \pm 0.84	2.40 \pm 0.91	2.77 \pm 0.87	3.97 \pm 0.98

$\bar{X} \pm \text{SD}$ ~ mean \pm standard deviation.

Glucose₁ ~ average glucose concentrations in the muscle 1 hour after slaughter, Glucose₂ ~ average glucose concentrations in the muscle 2 hours after slaughter, Glucose₄ ~ glucose concentrations in the muscle 4 hours after slaughter, Glucose₂₄ ~ average glucose concentrations in the muscle 24 hours after slaughter.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmentaler cross bred animals.

Effect of the interaction between breed and production system on muscle glucose concentrations post mortem is summarised in Table 4.22. The effect of the interactions between breed and production system had an effect on muscle glucose concentration at 1, 2 and 24 hours post mortem ($p < 0.05$) but no effect from the interactions between production system and breed on glucose at 4 hours post mortem ($p > 0.05$).

Muscles from the Simmental animals from ABF production system (2.17 $\mu\text{mol/g}$, 2.48 $\mu\text{mol/g}$, 2.94 $\mu\text{mol/g}$ respectively) had the tendency for the lowest glucose concentrations at 1, 2 and 4 hours post mortem and muscles from the Simmental animals from the BP production system (3.36 $\mu\text{mol/g}$) had the tendency for the lowest glucose concentrations at 24 hours post mortem. Muscles from the Brahman animals from the AP production system (2.78 $\mu\text{mol/g}$) had the tendency for the highest concentrations at 1 hour post mortem, muscles from the Simmental animals from the AP production system (3.06 $\mu\text{mol/g}$) had the tendency for the highest concentrations at 2 hours post mortem, muscles from the Simmental animals from the AP production system (3.07 $\mu\text{mol/g}$) had the tendency for the highest concentrations at 4 hours post mortem and muscles from the Brahman animals from the AP production system (4.81 $\mu\text{mol/g}$) had the tendency for the highest concentrations at 24 hours post mortem.

Table 4.22 Effect of the interaction between breed and production system on muscle glucose concentrations at 1, 2, 4 and 24 hours post mortem

Production system (n=180)	Breed	Glucose ₁ (µmol/g) ($\bar{X} \pm SD$)	Glucose ₂ (µmol/g) ($\bar{X} \pm SD$)	Glucose ₄ (µmol/g) ($\bar{X} \pm SD$)	Glucose ₂₄ (µmol/g) ($\bar{X} \pm SD$)
AF	Br-X	2.00±1.02 ^{ab}	2.42±0.73 ^{ab}	2.85±0.88	3.99±1.28 ^{ab}
	Ng-X	2.28±1.18 ^{ab}	2.56±1.04 ^{ab}	3.00±1.06	4.34±1.45 ^{ab}
	Si-X	2.47±0.68 ^{ab}	2.82±0.76 ^{ab}	3.06±0.78	4.33±0.83 ^{ab}
ABF	Br-X	2.48±0.54 ^{ab}	2.54±0.77 ^{ab}	3.25±0.76	4.58±0.78 ^{ab}
	Ng-X	2.35±0.93 ^{ab}	2.44±0.87 ^{ab}	2.80±0.94	3.80±1.24 ^{ab}
	Si-X	1.59±0.56 ^a	1.67±0.35 ^a	2.52±0.89	3.57±1.08 ^{ab}
AP	Br-X	2.78±0.98 ^{ab}	2.70±0.73 ^{ab}	3.41±0.87	4.81±0.79 ^b
	Ng-X	1.99±0.75 ^{ab}	2.27±0.91 ^{ab}	2.58±1.06	3.75±0.86 ^{ab}
	Si-X	2.88±0.95 ^b	3.06±0.04 ^b	3.07±0.78	4.65±1.01 ^{ab}
ABP	Br-X	2.49±0.53 ^{ab}	2.64±0.49 ^{ab}	3.03±0.40	3.91±0.5 ^{ab}
	Ng-X	2.07±0.96 ^{ab}	2.24±0.94 ^{ab}	2.71±0.78	3.64±0.78 ^{ab}
	Si-X	2.17±0.67 ^{ab}	2.48±0.69 ^{ab}	2.94±0.96	3.92±0.60 ^{ab}
BP	Br-X	2.07±0.88 ^{ab}	2.34±0.02 ^{ab}	3.01±1.31	3.88±1.01 ^{ab}
	Ng-X	2.39±0.83 ^{ab}	2.61±0.84 ^{ab}	3.03±0.72	3.95±1.04 ^{ab}
	Si-X	1.75±0.67 ^{ab}	1.91±0.76 ^{ab}	2.94±0.96	3.36±0.83 ^a

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

Glucose₁ ~ average glucose concentration in the muscle 1 hour after slaughter, Glucose₂ ~ average glucose concentration in the muscle 2 hours after slaughter, Glucose₄ ~ average glucose concentration in the muscle 4 hours after slaughter, Glucose₂₄ ~ average glucose concentration in the muscle 24 hours after slaughter.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmentaler cross bred animals.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

It seems that there is a muscle glycogen concentration threshold, approximately 50 µmol/g at slaughter, below which glycogen has an effect on meat quality (Immonen, Ruusunen, *et al.*, 2000). A high energy diet, as with animals in a feedlot, protects the resulting meat quality. Effect of production system on muscle glycogen concentrations at 1, 2, 4 and 24 hours post mortem is summarised in Table 4.23. There were differences between production systems for muscle glycogen concentrations at 1 and 24 hours post mortem ($p < 0.05$). Muscles from the BP production system (43.81 µmol/g) had lower glycogen concentrations than muscles from the ABF production

system (51.39 $\mu\text{mol/g}$) ($p < 0.05$), at 1 hour post mortem. At 24 hours post mortem, muscle from the AF production system (15.90 $\mu\text{mol/g}$) had the lowest concentration and muscles from the AP production system (26.12 $\mu\text{mol/g}$) had the highest concentration.

Table 4.23 Effect of production system on muscle glycogen concentrations at 1, 2, 4 and 24 hours post mortem.

Production system (n=180)	Glycogen ₁ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	Glycogen ₂ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	Glycogen ₄ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	Glycogen ₂₄ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)
AF	44.69 \pm 8.81 ^{ab}	40.09 \pm 10.53	33.53 \pm 9.79	15.90 \pm 7.18 ^a
ABF	51.39 \pm 10.86 ^b	44.93 \pm 12.78	38.32 \pm 14.13	21.81 \pm 11.45 ^{ab}
AP	51.05 \pm 14.87 ^{ab}	46.90 \pm 12.78	40.30 \pm 16.07	26.12 \pm 14.80 ^b
ABP	45.20 \pm 13.07 ^{ab}	42.21 \pm 12.75	36.96 \pm 12.63	23.46 \pm 10.04 ^b
BP	43.81 \pm 10.63 ^a	39.76 \pm 17.85	34.10 \pm 16.40	20.01 \pm 9.99 ^{ab}
Total	47.07 \pm 13.52	42.63 \pm 14.38	36.51 \pm 14.15	21.36 \pm 12.51

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

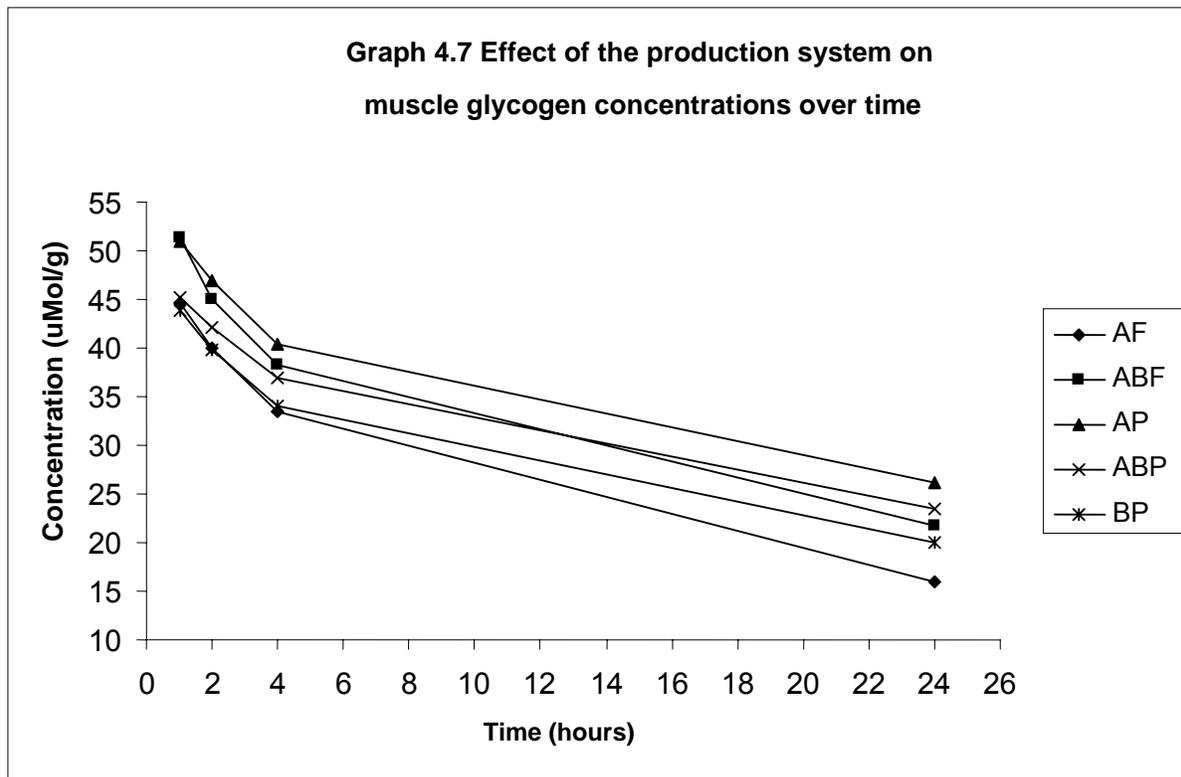
$\bar{X} \pm \text{SD}$ ~ mean \pm standard deviation.

Glycogen₁ ~ average glycogen concentrations in the muscle 1 hour after slaughter, Glycogen₂ ~ average glycogen concentrations in the muscle 2 hours after slaughter, Glycogen₄ ~ average glycogen concentrations in the muscle 4 hours after slaughter, Glycogen₂₄ ~ average glycogen concentrations in the muscle 24 hours after slaughter.

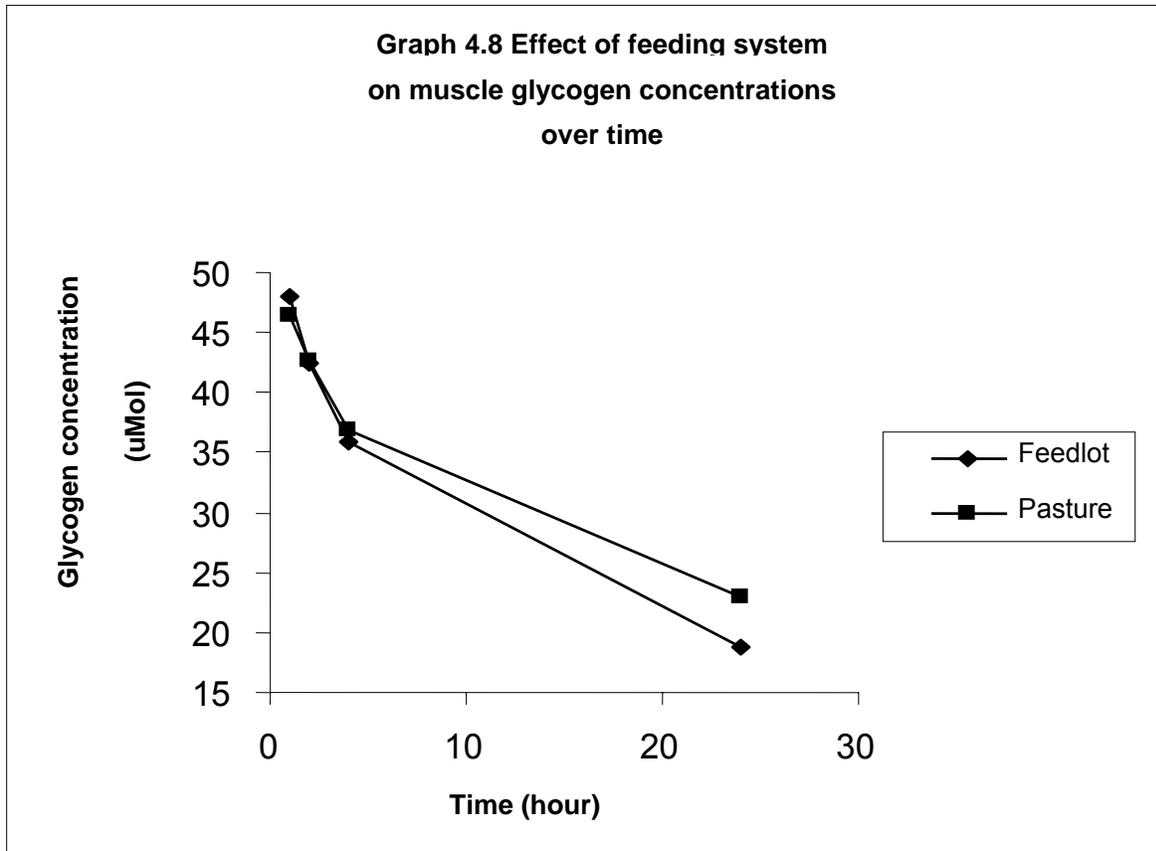
AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

Muscles from the BP production system (43.81 $\mu\text{mol/g}$, 39.76 $\mu\text{mol/g}$ and 34.10 $\mu\text{mol/g}$ respectively) had the tendency for the lowest muscle glycogen concentrations at 1, 2 and 4 hours post mortem whereas the muscles from the AF production system (15.90 $\mu\text{mol/g}$) had the tendency for the lowest concentrations at 24 hour post mortem. Muscles from the ABF production system (51.39 $\mu\text{mol/g}$) had the tendency for higher concentrations at 1 hour post mortem whereas muscles from the AP production system (46.90 $\mu\text{mol/g}$, 40.30 $\mu\text{mol/g}$ and 26.12 $\mu\text{mol/g}$ respectively) had the tendency for higher concentrations at 2, 4 and 24 hours post mortem. Immonen, Ruusunen, *et al.* (2000) states that muscles from the AF production system had significantly higher glycogen than the muscles from the AP production system.

The effect of production system on glycogen concentrations at 1, 2, 4 and 24 hours post mortem is illustrated in Graph 4.7. The concentrations decreased as time progressed. The rate of decrease differed slightly between production systems, but these differences were not significant. Muscles from the AF production system mainly had the lowest muscle glycogen concentrations and muscles from the AP production system mainly had the highest concentrations.



There is no agreement in the literature on the effect of feeding system on muscle glycogen concentrations in muscles post mortem. Some state that there is an effect, with feedlot fed animals producing higher levels of glycogen (Ferguson, Daly, Gardner and Tume, 2008; Immonen, Ruusunen, *et al.*, 2000), whereas others state that there is no effect (Lowe, Pearchey, *et al.*, 2002; Immonen, Schaefer, *et al.*, 2000). According to Immonen, Ruusunen, *et al.* (2000) the energy content in the diet influences the glycogen concentration in the muscle, therefore feedlot animals should have a higher muscle glycogen concentration. When only the effect of feeding system is considered, there were significant effects on glycogen only at 24 hours post mortem. Muscles from the feedlot ($18.85 \pm 9.94 \mu\text{mol/g}$) had lower glycogen levels at 24 hours post mortem than the muscles from the pasture ($22.97 \pm 11.84 \mu\text{mol/g}$). Muscles from the pasture animals ($46.45 \pm 15.21 \mu\text{mol/g}$, $42.71 \pm 15.84 \mu\text{mol/g}$ and $36.89 \pm 15.26 \mu\text{mol/g}$ respectively) had a tendency for lower concentrations at 1, 2 and 4 hours post mortem than the animals from the feedlot ($48.04 \pm 10.38 \mu\text{mol/g}$, $42.51 \pm 11.88 \mu\text{mol/g}$ and $35.92 \pm 12.30 \mu\text{mol/g}$ respectively) ($p > 0.05$). Effect of feeding system on muscle glycogen concentrations over time can be seen in Graph 4.8. Muscle glycogen concentration decreases as time progressed. The difference between feeding systems increased as time progressed.



Age per se did not show any difference for the muscle glycogen concentration ($p > 0.05$). Effect of breed on muscle glycogen concentrations at 1, 2, 4 and 24 hours are summarised in Table 4.24. Considering the effect of breed only; there were no differences between the breeds for glycogen ($p > 0.05$). According to Sanz, Verde, Saez and Sanudo (1996) breed does not effect the resulting muscle glycogen concentration.

Table 4.24 Effect of breed on muscle glycogen concentrations at 1, 2, 4 and 24 hours post mortem.

Breed (n=180)	Glycogen ₁ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	Glycogen ₂ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	Glycogen ₄ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	Glycogen ₂₄ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)
Br-X	49.01 \pm 14.97	44.91 \pm 15.40	37.47 \pm 14.56	22.46 \pm 11.90
Ng-X	44.87 \pm 12.36	39.64 \pm 12.81	34.45 \pm 13.66	19.84 \pm 9.34
Si-X	47.45 \pm 13.04	43.51 \pm 14.59	37.75 \pm 14.23	21.86 \pm 12.51

$\bar{X} \pm \text{SD}$ ~ mean \pm standard deviation.

Glycogen₁ ~ average glycogen concentrations in the muscle 1 hour after slaughter, Glycogen₂ ~ average glycogen concentrations in the muscle 2 hours after slaughter, Glycogen₄ ~ glycogen concentrations in the muscle 4 hours after slaughter, Glycogen₂₄ ~ average glycogen concentrations in the muscle 24 hours after slaughter.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmentaler cross bred animals.

Effect of the interaction between breed and production system on muscle glycogen concentrations is summarised in Table 4.25. There were no significant effect from the interaction between breed and production system on muscle glycogen concentrations at 1, 2, 4 and 24 hours post mortem.

Table 4.25 Effect of the interaction between breed and production system on muscle glycogen concentrations at 1, 2, 4 and 24 hours post mortem.

Production system (n=180)	Breed	Glycogen ₁ (μmol/g) ($\bar{X} \pm SD$)	Glycogen ₂ (μmol/g) ($\bar{X} \pm SD$)	Glycogen ₄ (μmol/g) ($\bar{X} \pm SD$)	Glycogen ₂₄ (μmol/g) ($\bar{X} \pm SD$)
AF	Br-X	44.65±9.49	40.42±12.58	33.23±10.69	15.98±7.30
	Ng-X	44.74±6.85	39.22±8.81	35.11±9.93	16.51±6.80
	Si-X	44.68±10.36	40.56±10.63	32.39±9.42	15.26±7.96
ABF	Br-X	51.86±12.87	46.33±14.15	40.56±13.86	26.18±11.24
	Ng-X	51.31±8.66	44.74±10.85	35.21±14.32	19.88±9.25
	Si-X	50.84±12.34	43.65±14.86	40.21±14.79	19.70±14.03
AP	Br-X	57.56±14.84	52.36±16.40	44.39±15.83	29.02±15.13
	Ng-X	45.38±16.19	40.10±14.47	34.30±15.31	22.10±10.91
	Si-X	49.81±12.47	47.57±15.05	41.54±16.75	26.82±17.56
ABP	Br-X	40.58±11.88	39.22±9.69	32.39±10.67	17.97±5.42
	Ng-X	42.56±14.25	39.08±14.71	34.82±14.07	22.75±11.39
	Si-X	52.77±9.80	48.90±10.71	43.83±10.16	29.37±8.88
BP	Br-X	49.70±18.95	45.77±19.41	36.90±17.88	23.09±13.29
	Ng-X	40.13±12.65	34.75±13.94	32.81±15.81	17.74±7.30
	Si-X	40.70±16.57	37.82±18.77	32.15±16.05	18.70±7.37

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

Glycogen₁ ~ average glycogen concentration in the muscle 1 hour after slaughter, Glycogen₂ ~ average glycogen concentration in the muscle 2 hours after slaughter, Glycogen₄ ~ glycogen concentration in the muscle 4 hours after slaughter, Glycogen₂₄ ~ average glycogen concentration in the muscle 24 hours after slaughter.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmentaler cross bred animals.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

Effect of production system on muscle glucose-6-phosphate concentrations at 1, 2, 4 and 24 hours post mortem are summarised in Table 4.26. There were no differences between production systems for the muscle glucose-6-phosphate concentrations ($p > 0.05$). Muscle from the BP production system (1 hour: 1.43 μmol/g, 2 hours: 1.54 μmol/g, 4 hours: 2.38 μmol/g and 24 hours: 6.92 μmol/g) had the tendency for lower concentrations compared to AP, ABP, AF and ABF production systems. The muscles from the AF production system (1.81 μmol/g) had the tendency for the highest concentrations at 1 hour post mortem, muscles from AP production system (2.27 μmol/g) had the tendency for the highest concentrations at 2 hours post mortem, muscles from

ABP production system (3.41 $\mu\text{mol/g}$) had the tendency for the highest concentrations at 4 hours post mortem whereas muscles from ABP production system (8.47 $\mu\text{mol/g}$) had the tendency for the highest concentrations at 24 hours post mortem.

Table 4.26 Effect of production system on muscle glucose-6-phosphate concentrations at 1, 2, 4 and 24 hours post mortem.

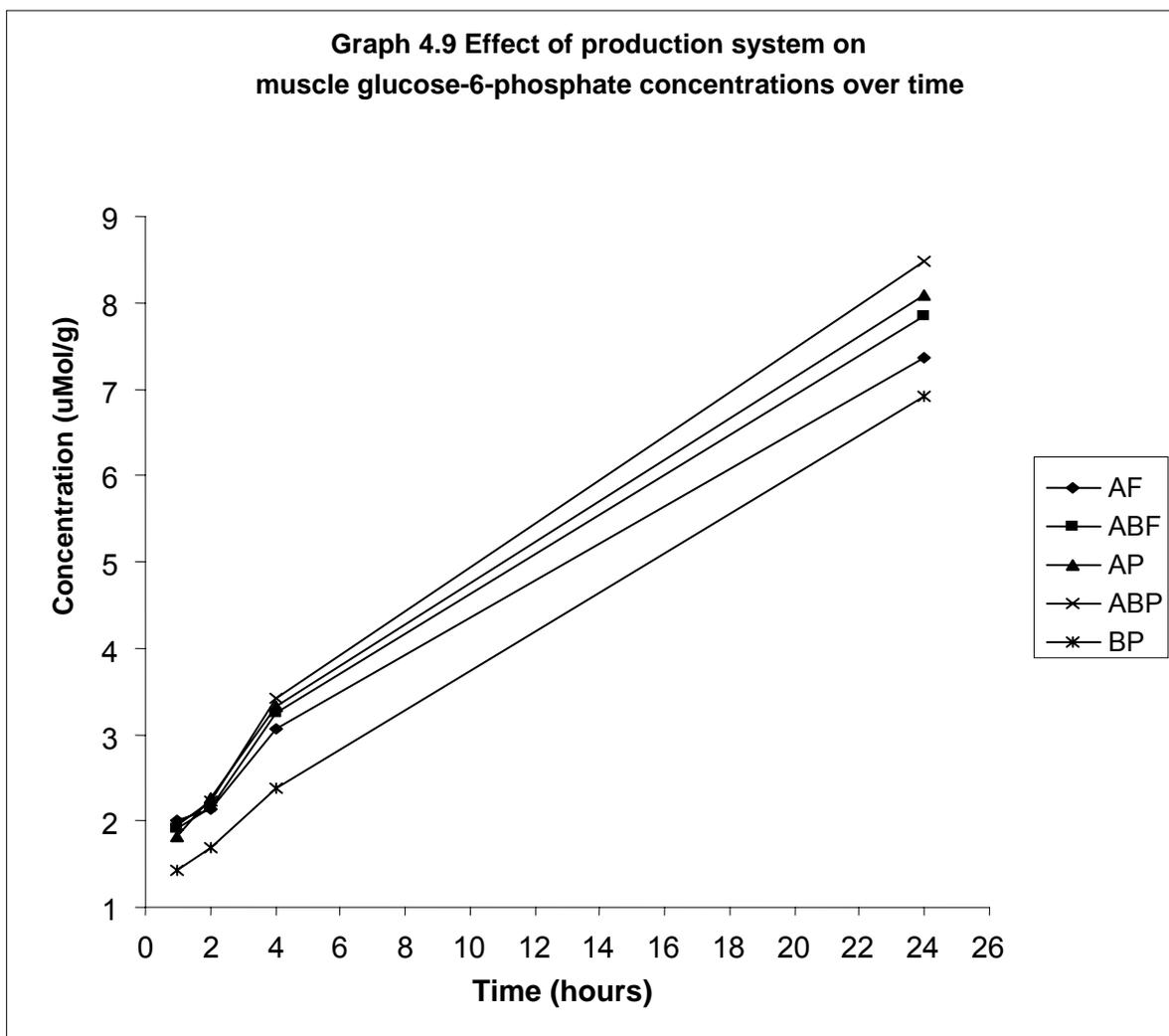
Production system (n=180)	Glucose-6-Phosphate ₁ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	Glucose-6-Phosphate ₂ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	Glucose-6-Phosphate ₄ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	Glucose-6-Phosphate ₂₄ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)
AF	2.01 \pm 1.28	2.14 \pm 1.22	3.06 \pm 1.68	7.37 \pm 2.86
ABF	1.91 \pm 0.96	2.16 \pm 1.08	3.25 \pm 1.86	7.84 \pm 2.36
AP	1.81 \pm 1.04	2.27 \pm 1.22	3.33 \pm 1.89	8.08 \pm 3.19
ABP	1.95 \pm 1.00	2.22 \pm 1.20	3.41 \pm 2.29	8.47 \pm 2.78
BP	1.43 \pm 0.76	1.54 \pm 0.96	2.38 \pm 1.57	6.92 \pm 2.95
Total	1.81 \pm 1.15	2.05 \pm 1.16	3.06 \pm 1.88	7.70 \pm 2.86

$\bar{X} \pm \text{SD}$ ~ mean \pm standard deviation.

Glucose-6-Phosphate₁ ~ average glucose-6-phosphate concentrations in the muscle 1 hour after slaughter, Glucose-6-Phosphate₂ ~ average glucose-6-phosphate concentrations in the muscle 2 hours after slaughter, Glucose-6-Phosphate₄ ~ glucose-6-phosphate concentrations in the muscle 4 hours after slaughter, Glucose-6-Phosphate₂₄ ~ average glucose-6-phosphate concentrations in the muscle 24 hours after slaughter.

AF ~ The average for the animals reared at the feedlot until A age classification group, ABF ~ The average for the animals reared at the feedlot until AB age classification group, AP ~ The average for the animals reared on the pasture until A age classification group, ABP ~ The average for the animals reared on the pasture until AB age classification group, BP ~ The average for the animals reared on the pasture until B age classification group.

Effect of production systems on muscle glucose-6-phosphate concentrations over time is illustrated in Graph 4.9. Concentrations increased as time progressed, the rate of this increase differed between production systems ($p > 0.05$). Muscles from ABP production system had the highest rate of increase, muscles from BP production system had the lowest rate of increase, muscles from AP production system had the second highest rate of increase and muscles from AF production system had the second lowest rate of increase.



Feeding system per se showed no differences between feeding systems ($p > 0.05$) for muscle glucose-6-phosphate concentration. Considering the effect of age only, there were differences between age classification groups for the glucose-6-phosphate concentrations in the muscle at 1, 2 and 4 hours post mortem ($p < 0.05$), not at 24 hours post mortem ($p > 0.05$). Muscles from the B age classification group constantly had the lowest glucose-6-phosphate concentration at 1, 2, 4 and 24 hours post mortem ($1.43 \pm 0.76 \mu\text{mol/g}$, $1.54 \pm 0.96 \mu\text{mol/g}$, $2.38 \pm 1.57 \mu\text{mol/g}$ and $6.92 \pm 2.95 \mu\text{mol/g}$ respectively). Muscles from the AB age classification group had the highest concentration at 1, 4 and 24 hours post mortem ($1.93 \pm 0.97 \mu\text{mol/g}$, $3.33 \pm 2.07 \mu\text{mol/g}$ and $8.17 \pm 2.58 \mu\text{mol/g}$ respectively).

Table 4.26 Effect of breed on muscle glucose-6-phosphate concentrations at 1, 2, 4 and 24 hours post mortem.

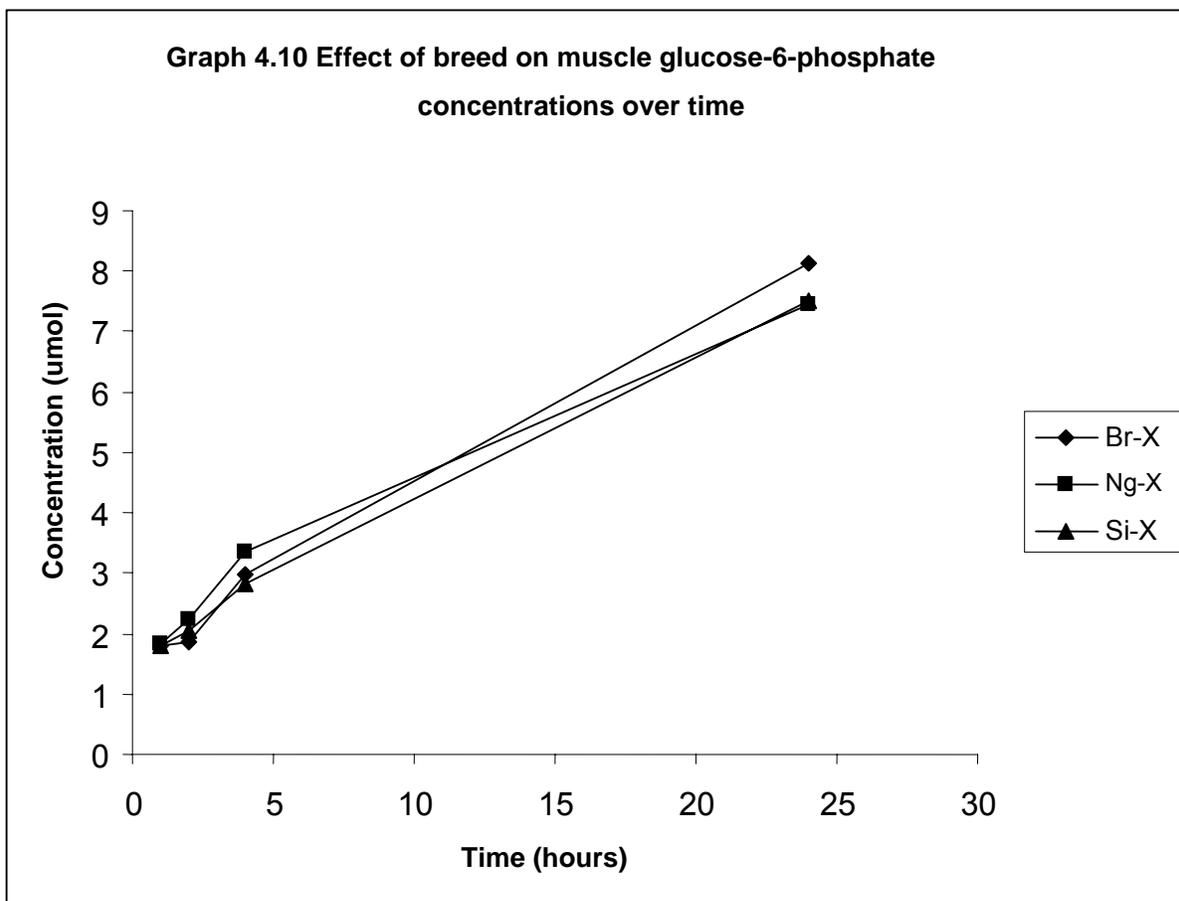
Breed (n=180)	Glucose-6-Phosphate ₁ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	Glucose-6-Phosphate ₂ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	Glucose-6-Phosphate ₄ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	Glucose-6-Phosphate ₂₄ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)
Br-X	1.80 \pm 1.06	1.87 \pm 1.10	2.99 \pm 1.85	8.13 \pm 2.95
Ng-X	1.82 \pm 0.87	2.22 \pm 1.09	3.36 \pm 2.05	7.46 \pm 2.68
Si-X	1.81 \pm 1.15	2.05 \pm 1.26	2.82 \pm 1.70	7.51 \pm 2.96

$\bar{X} \pm \text{SD}$ ~ mean \pm standard deviation.

Glucose-6-Phosphate₁ ~ average glucose-6-phosphate concentrations in the muscle 1 hour after slaughter, Glucose-6-Phosphate₂ ~ average glucose-6-phosphate concentrations in the muscle 2 hours after slaughter, Glucose-6-Phosphate₄ ~ glucose-6-phosphate concentrations in the muscle 4 hours after slaughter, Glucose-6-Phosphate₂₄ ~ average glucose-6-phosphate concentrations in the muscle 24 hours after slaughter.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmentaler cross bred animals.

Effect of breed on muscle glucose-6-phosphate concentrations at 1, 2, 4 and 24 hours post mortem are summarised in Table 4.26. When only the effect of breed is considered; there were no differences between the breeds ($p > 0.05$). It seems that there were different rates of increase in the concentrations of glucose-6-phosphate (Graph 4.10).



Effect of the interaction between breed and production system on muscle glucose-6-phosphate concentration is summarised in Table 4.28. There was no significant effect of the interaction between breed and production system on the muscle glucose-6-phosphate concentrations at 1, 2, 4 and 24 hours post mortem.

Table 4.28 Effect of the interaction between breed and production system on muscle glucose-6-phosphate concentration

Production system (n=180)	Breed	G6P ₁ (μmol/g) ($\bar{X} \pm SD$)	G6P ₂ (μmol/g) ($\bar{X} \pm SD$)	G6P ₄ (μmol/g) ($\bar{X} \pm SD$)	G6P ₂₄ (μmol/g) ($\bar{X} \pm SD$)
AF	Br-X	2.39±1.64	2.30±1.64	3.13±2.10	7.64±3.18
	Ng-X	1.61±0.80	1.80±0.81	2.93±1.67	7.72±2.64
	Si-X	2.01±1.21	2.30±1.05	3.13±1.33	6.65±2.82
ABF	Br-X	1.74±0.61	1.77±0.51	2.90±1.25	8.06±2.83
	Ng-X	2.12±0.96	2.59±1.19	3.80±2.31	7.59±1.97
	Si-X	1.81±1.26	2.01±1.25	2.88±1.66	8.02±2.53
AP	Br-X	1.60±1.09	2.05±0.98	3.44±1.84	9.28±3.08
	Ng-X	1.97±0.81	2.36±1.18	3.21±1.58	6.69±3.07
	Si-X	1.88±1.21	2.40±1.51	3.32±2.89	8.13±3.16
ABP	Br-X	1.71±0.75	1.83±0.96	3.45±2.69	8.26±2.89
	Ng-X	1.96±1.02	2.27±1.26	3.66±2.53	8.14±2.95
	Si-X	2.17±1.19	2.50±1.33	3.07±1.66	9.09±2.62
BP	Br-X	1.57±0.80	1.48±1.03	2.30±1.31	7.67±2.97
	Ng-X	1.44±0.60	2.04±0.93	3.04±1.97	6.68±2.92
	Si-X	1.27±0.89	1.13±0.74	1.80±1.20	5.99±2.93

$\bar{X} \pm SD$ ~ mean ± standard deviation.

G6P₁ ~ average glucose-6-phosphate concentration in the muscle 1 hour after slaughter, G6P₂ ~ average glucose-6-phosphate concentration in the muscle 2 hours after slaughter, G6P₄ ~ average glucose-6-phosphate concentration in the muscle 4 hours after slaughter, G6P₂₄ ~ average glucose-6-phosphate concentration in the muscle 24 hours after slaughter.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmentaler cross bred animals.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

Effect of production system on muscle ATP concentrations at 1, 2, 4 and 24 hours post mortem are summarised in Table 4.29. There were differences between production systems for muscle ATP at 1 hour post mortem ($p < 0.05$), but there were no differences between production systems at 2, 4 and 24 hours post mortem ($p > 0.05$). For muscle ATP concentration at 1 hour post mortem there were differences between muscles from the AP (7.83 μmol/g) and ABP production systems (6.59 μmol/g) ($p < 0.05$). Numerically, muscles from the ABP production system (6.59 μmol/g and 6.48 μmol/g respectively) had the lowest muscle ATP concentrations at 1 and 2 hours post mortem, muscles from AF production system (5.28 μmol/g and 2.98 μmol/g respectively) had

the lowest concentrations at 4 and 24 hours post mortem, muscles from AP production system (5.44 $\mu\text{mol/g}$ and 6.81 $\mu\text{mol/g}$ respectively) had the highest concentrations at 1 and 2 hours post mortem, muscles from the ABP production system (5.60 $\mu\text{mol/g}$) had the highest concentrations at 4 hours post mortem and muscles from the BP production system (3.27 $\mu\text{mol/g}$) had the highest concentrations at 24 hours post mortem.

Table 4.29 Effect of production system on muscle ATP concentrations at 1, 2, 4 and 24 hours post mortem.

Production system (n=180)	ATP ₁ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	ATP ₂ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	ATP ₄ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	ATP ₂₄ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)
AF	7.00 \pm 1.64 ^{ab}	6.55 \pm 1.50	5.28 \pm 1.59	2.98 \pm 1.17
ABF	7.52 \pm 1.66 ^{ab}	7.09 \pm 1.76	5.74 \pm 1.80	3.20 \pm 1.67
AP	7.83 \pm 2.03 ^b	6.81 \pm 1.62	5.44 \pm 1.51	3.26 \pm 1.46
ABP	6.59 \pm 1.74 ^a	6.48 \pm 1.58	5.60 \pm 1.93	3.10 \pm 1.24
BP	7.32 \pm 1.82 ^{ab}	6.80 \pm 1.51	5.39 \pm 1.94	3.27 \pm 1.02
Total	7.25 \pm 1.81	6.75 \pm 1.59	5.49 \pm 1.76	3.17 \pm 1.31

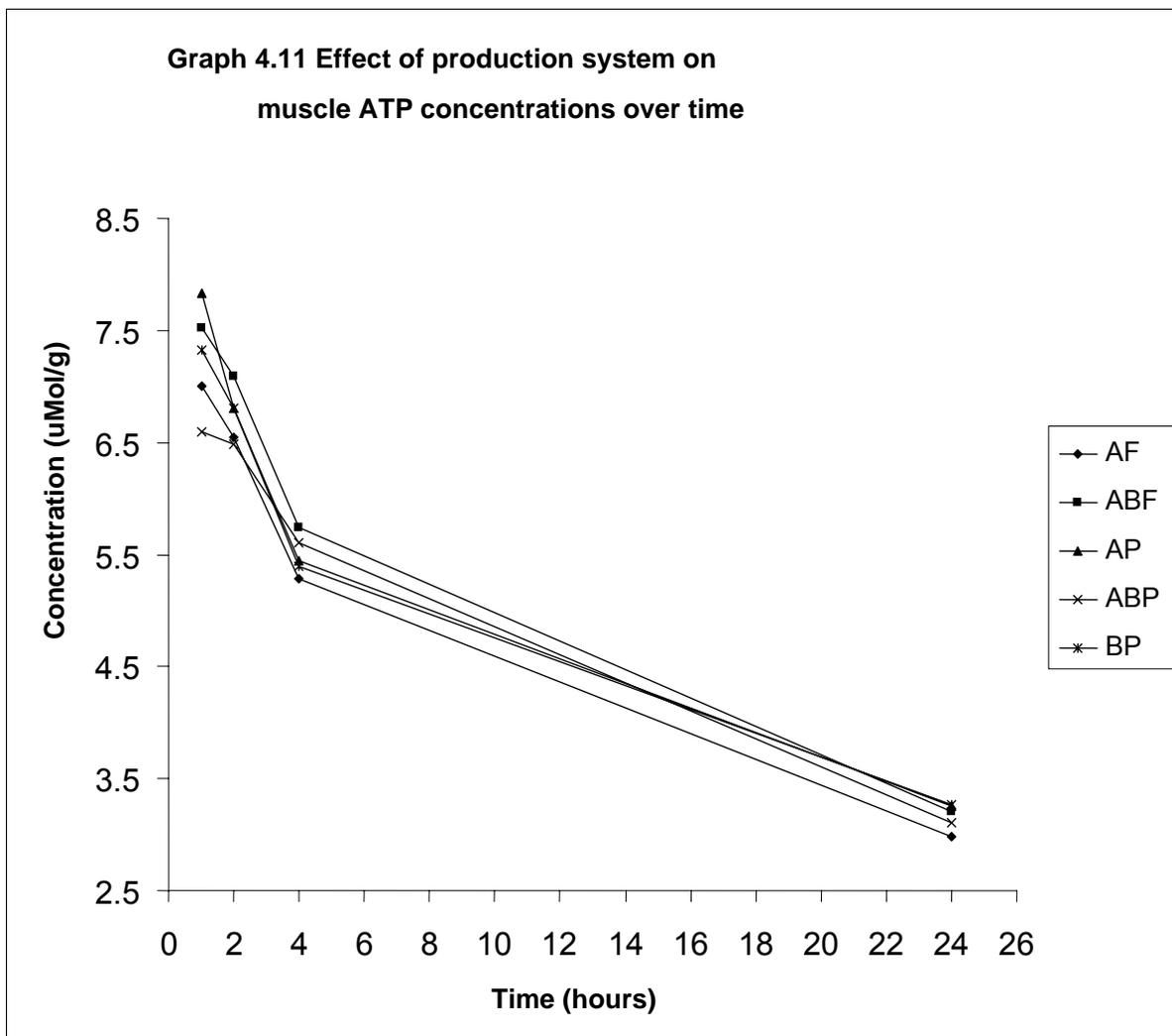
^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm \text{SD}$ ~ mean \pm standard deviation.

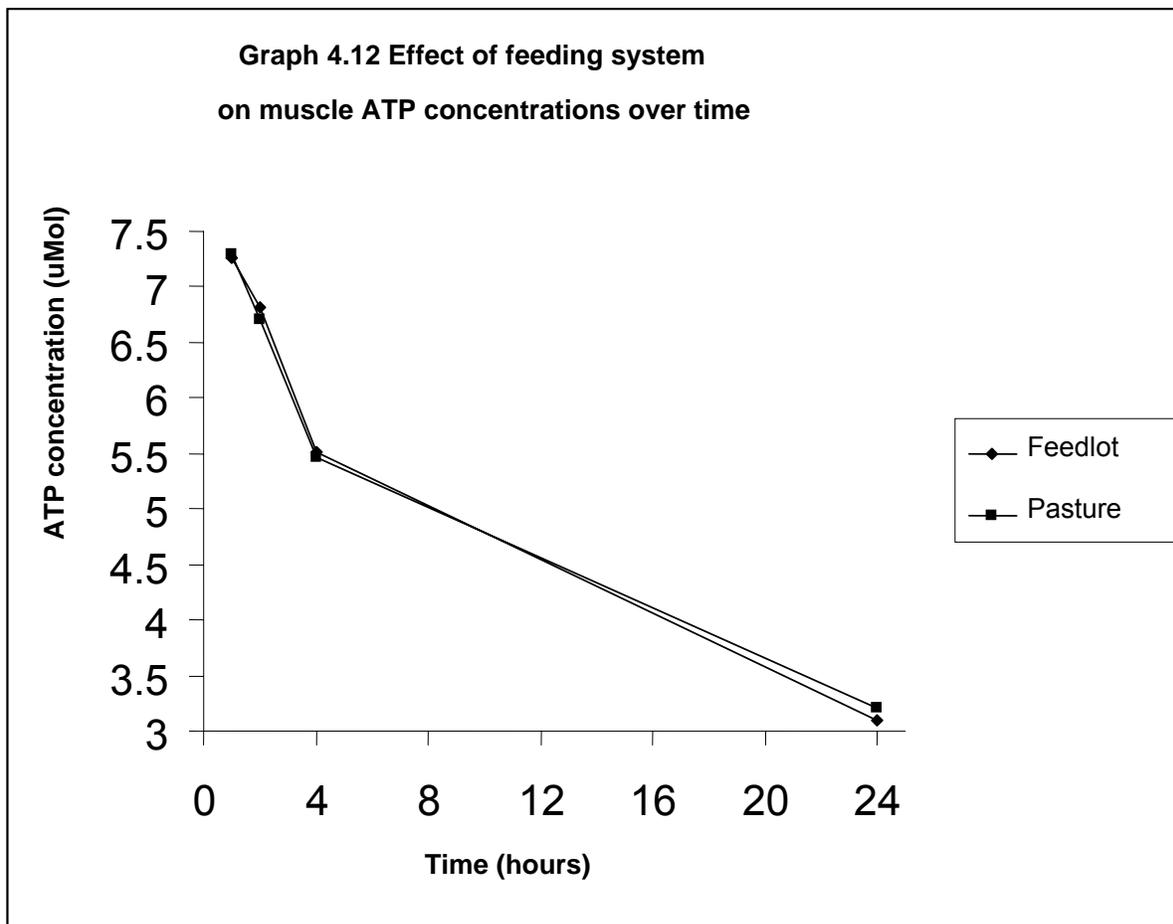
ATP₁ ~ average ATP concentrations in the muscle 1 hour after slaughter, ATP₂ ~ average ATP concentrations in the muscle 2 hours after slaughter, ATP₄ ~ ATP concentrations in the muscle 4 hours after slaughter, ATP₂₄ ~ average ATP concentrations in the muscle 24 hours after slaughter.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

Effect of production system on muscle ATP concentrations over time is illustrated in Graph 4.11. Muscle ATP concentrations decreased as time progressed; which were very similar between production systems. ATP concentration decreases over time, because ATP is not regenerated efficiently after death, due to a reduction in available energy metabolites as well as a reduction in enzyme activity post mortem (Lawrie, 1974; Varnam and Sutherland, 1996).



When only considering the effect of feeding system and age separately, there were no differences for muscle ATP concentration post mortem ($p > 0.05$). Effect of feeding system over time on muscle ATP concentration is illustrated in Graph 4.12.



The highest concentrations of ATP in the muscle were constantly from muscles of animals in the B age classification group (1 hour: $7.32 \pm 1.82 \mu\text{mol/g}$, 2 hours: $6.80 \pm 1.51 \mu\text{mol/g}$, 4 hours: $5.39 \pm 1.94 \mu\text{mol/g}$ and 24 hours: $3.27 \pm 1.02 \mu\text{mol/g}$). At 1 hour post mortem the AB age classification ($7.05 \pm 1.75 \mu\text{mol/g}$) had the tendency for lower concentrations compared to A and B aged animals. At 2, 4 and 24 hours the muscles from the A classification group ($6.68 \pm 1.56 \mu\text{mol/g}$, $5.36 \pm 1.53 \mu\text{mol/g}$ and $3.12 \pm 1.31 \mu\text{mol/g}$ respectively) had the tendency for lower concentrations compared to AB and B aged animals.

Effect of breed on muscle ATP concentrations at 1, 2, 4 and 24 hours post mortem are summarised in Table 4.30. Breed per se showed no differences between the breeds for muscle ATP concentrations ($p > 0.05$).



Table 4.30 Effect of breed on muscle ATP concentrations post mortem.

Breed (n=180)	ATP ₁ (μmol/g) ($\bar{X} \pm SD$)	ATP ₂ (μmol/g) ($\bar{X} \pm SD$)	ATP ₄ (μmol/g) ($\bar{X} \pm SD$)	ATP ₂₄ (μmol/g) ($\bar{X} \pm SD$)
Br-X	7.51±1.80	6.99±1.52	5.51±1.62	3.11±1.30
Ng-X	6.95±1.78	6.40±1.63	5.49±1.83	3.09±1.15
Si-X	7.31±1.85	6.86±1.58	5.40±1.83	3.30±1.49

$\bar{X} \pm SD$ ~ mean ± standard deviation.

ATP₁ ~ average ATP concentrations in the muscle 1 hour after slaughter, ATP₂ ~ average ATP concentrations in the muscle 2 hours after slaughter, ATP₄ ~ ATP concentrations in the muscle 4 hours after slaughter, ATP₂₄ ~ average ATP concentrations in the muscle 24 hours after slaughter.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmentaler cross bred animals.

Effect of the interaction between breed and production system on muscle ATP concentration is summarised in Table 4.31. There was no significant effect from the interaction between production system and breed for muscle ATP concentrations at 1, 2, 4 and 24 hours post mortem.

Table 4.31 Effect of the interaction between breed and production system on muscle ATP concentration at 1, 2, 4 and 24 hours post mortem.

Production system (n=180)	Breed	ATP ₁ (μmol/g) ($\bar{X} \pm SD$)	ATP ₂ (μmol/g) ($\bar{X} \pm SD$)	ATP ₄ (μmol/g) ($\bar{X} \pm SD$)	ATP ₂₄ (μmol/g) ($\bar{X} \pm SD$)
AF	Br-X	6.90±1.97	6.42±1.49	5.45±1.33	2.83±1.18
	Ng-X	7.30±1.59	6.31±1.51	5.43±1.54	2.91±0.98
	Si-X	6.82±1.43	6.90±1.57	4.97±1.90	3.19±1.37
ABF	Br-X	8.14±1.77	7.49±1.63	5.62±1.51	3.67±1.91
	Ng-X	7.20±1.58	6.91±2.01	5.90±2.02	2.80±1.07
	Si-X	7.28±1.63	6.89±1.64	5.64±1.92	3.26±2.08
AP	Br-X	8.29±1.75	7.54±1.71	6.15±1.57	3.22±1.62
	Ng-X	6.99±1.98	6.24±1.13	5.43±1.46	3.31±1.54
	Si-X	8.11±2.25	6.62±1.66	4.79±1.23	3.26±1.37
ABP	Br-X	6.21±1.14	6.10±1.19	5.01±1.75	2.73±0.86
	Ng-X	6.72±2.01	6.52±1.73	5.73±2.27	3.18±1.33
	Si-X	6.78±1.92	6.78±1.76	5.97±1.66	3.33±1.43
BP	Br-X	7.82±1.66	7.29±1.24	5.57±1.92	3.08±0.74
	Ng-X	6.59±1.87	5.94±1.48	4.87±1.68	3.31±0.85
	Si-X	7.49±1.85	7.11±1.55	5.69±2.23	3.46±1.43

$\bar{X} \pm SD$ ~ mean ± standard deviation.

ATP₁ ~ average ATP concentration in the muscle 1 hour after slaughter, ATP₂ ~ average ATP concentration in the muscle 2 hours after slaughter, ATP₄ ~ average ATP concentration in the muscle 4 hours after slaughter, ATP₂₄ ~ average ATP concentration in the muscle 24 hours after slaughter.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmentaler cross bred animals.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

Effect of production system on muscle creatine phosphate concentrations at 1, 2, 4 and 24 hours post mortem are summarised in Table 4.32. There were no differences between production systems from the muscle creatine phosphate concentrations at 1, 2, 4 and 24 hours post mortem ($p > 0.05$).

Table 4.32 Effect of production system on muscle creatine phosphate concentrations at 1, 2, 4 and 24 hours post mortem.

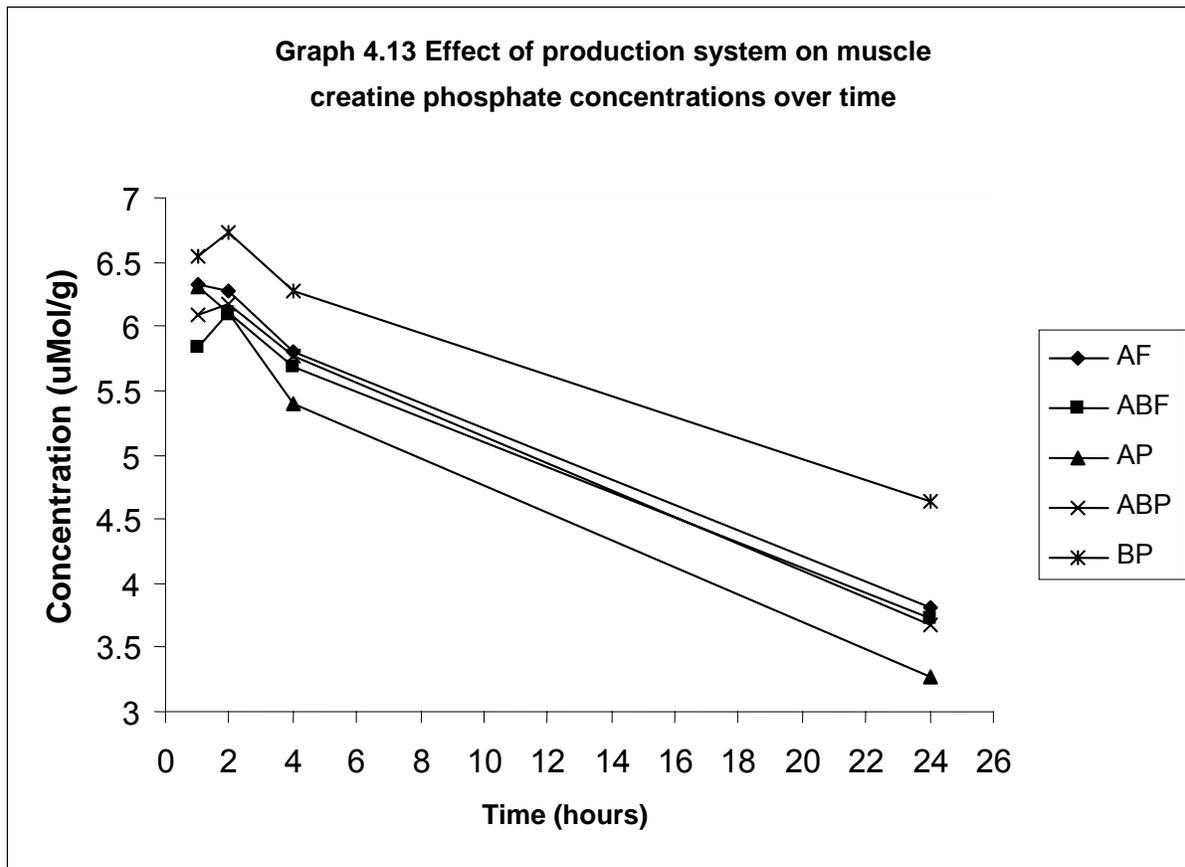
Production system (n=180)	Creatine phosphate ₁ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	Creatine phosphate ₂ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	Creatine phosphate ₄ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	Creatine phosphate ₂₄ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)
AF	6.33 \pm 1.79	6.28 \pm 1.82	5.81 \pm 2.14	3.81 \pm 1.76
ABF	5.84 \pm 1.62	6.10 \pm 1.60	5.69 \pm 1.62	3.72 \pm 1.62
AP	6.30 \pm 2.16	6.10 \pm 1.80	5.40 \pm 1.66	3.27 \pm 1.57
ABP	6.09 \pm 1.60	6.17 \pm 1.43	5.77 \pm 1.66	3.68 \pm 1.43
BP	6.55 \pm 1.40	6.73 \pm 1.64	6.27 \pm 1.76	4.64 \pm 1.46
Total	6.23 \pm 1.71	6.29 \pm 1.66	5.81 \pm 1.78	3.86 \pm 1.61

$\bar{X} \pm \text{SD}$ ~ mean \pm standard deviation.

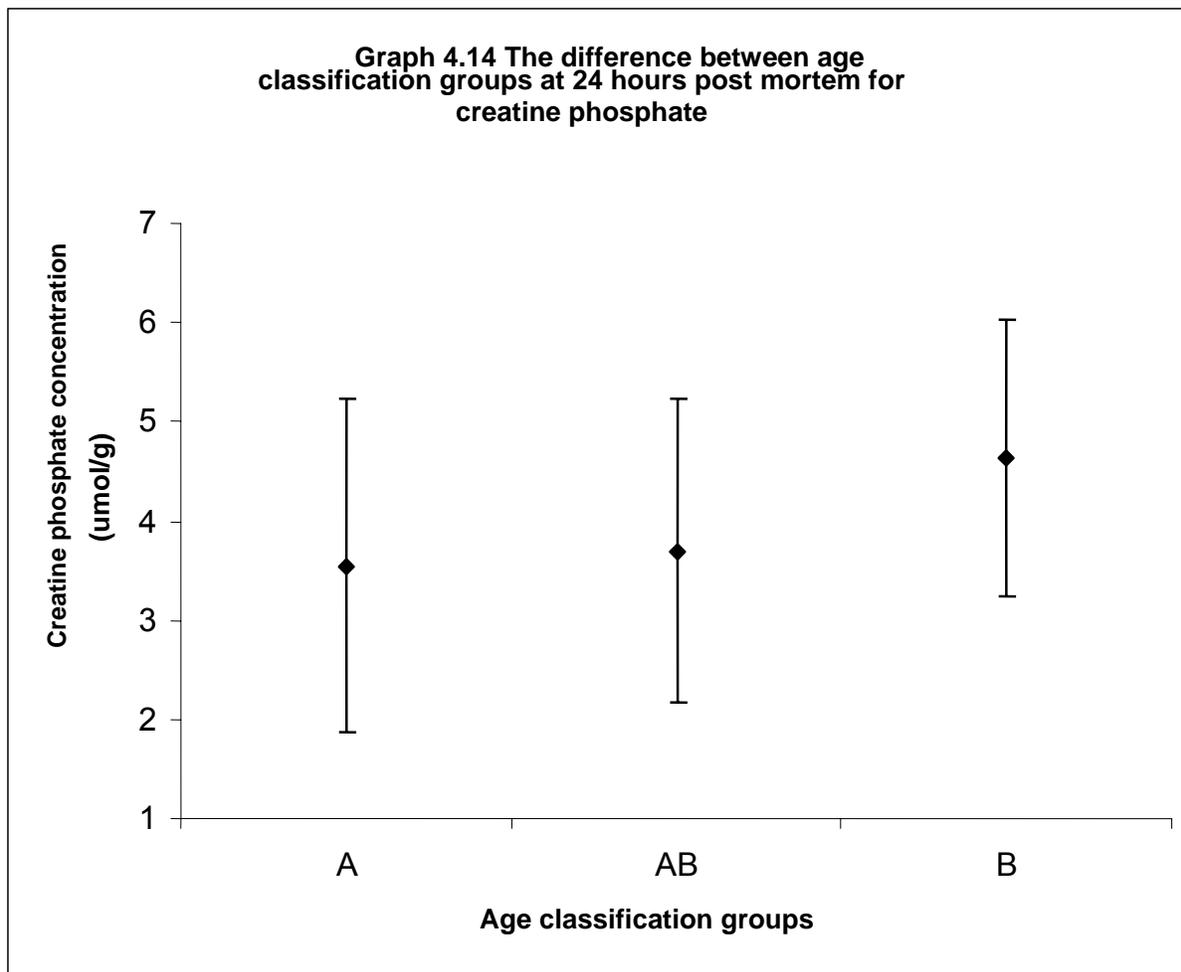
Creatine phosphate₁ ~ average creatine phosphate concentrations in the muscle 1 hour after slaughter, Creatine phosphate₂ ~ average creatine phosphate concentrations in the muscle 2 hours after slaughter, Creatine phosphate₄ ~ creatine phosphate concentrations in the muscle 4 hours after slaughter, Creatine phosphate₂₄ ~ average creatine phosphate concentrations in the muscle 24 hours after slaughter.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

Effect of production system on muscle creatine phosphate concentrations is illustrated in Graph 4.13. There was an initial increase in the muscle creatine phosphate concentrations, thereafter the concentrations decreased. Muscles from the AF and AP production systems do not show an initial increase in the muscle creatine phosphate concentration. Muscles from the BP production system constantly had the highest muscle creatine phosphate concentrations. Muscles from the ABF production system initially had the lowest concentration and muscles from the AP production system had the lowest concentration from 2 hours post mortem.



Considering the effect of feeding system only, there were no differences between feeding systems at 1, 2, 4 and 24 hours post mortem ($p > 0.05$). When only the effect of age is considered; there were only differences between age classification groups at 24 hours post mortem ($p < 0.05$). Graph 4.14 illustrates the effect of the age classification groups on the creatine phosphate at 24 hours post mortem. Muscles from B age classification group (1 hour: $6.55 \pm 1.40 \mu\text{mol/g}$, 2 hours: $6.73 \pm 1.64 \mu\text{mol/g}$, 4 hours: $6.27 \pm 1.76 \mu\text{mol/g}$ and 24 hours: $4.64 \pm 1.40 \mu\text{mol/g}$) had the tendency for higher concentrations than animals from A ($5.61 \pm 1.92 \mu\text{mol/g}$, $3.55 \pm 1.68 \mu\text{mol/g}$, $6.32 \pm 1.96 \mu\text{mol/g}$ and $3.55 \pm 1.68 \mu\text{mol/g}$ respectively) and AB age classification groups ($5.97 \pm 1.60 \mu\text{mol/g}$, $6.13 \pm 1.51 \mu\text{mol/g}$, $5.73 \pm 1.63 \mu\text{mol/g}$ and $3.70 \pm 1.52 \mu\text{mol/g}$ respectively).



Effect of breed on muscle creatine phosphate concentrations at 1, 2, 4 and 24 hours post mortem are summarised in Table 4.33. When only considering the effect of breed, there were no differences between the breeds for the creatine phosphate concentration post mortem ($p > 0.05$).

Table 4.33 Effect of breed on muscle creatine phosphate concentrations post mortem

Breed (n=180)	Creatine phosphate ₁ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	Creatine phosphate ₂ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	Creatine phosphate ₄ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)	Creatine phosphate ₂₄ ($\mu\text{mol/g}$) ($\bar{X} \pm \text{SD}$)
Br-X	6.10 \pm 1.79	6.21 \pm 1.69	5.77 \pm 1.71	3.77 \pm 1.56
Ng-X	6.08 \pm 1.47	6.09 \pm 1.44	5.64 \pm 1.73	4.01 \pm 1.52
Si-X	6.53 \pm 1.87	6.61 \pm 1.83	6.03 \pm 1.92	3.79 \pm 1.76

$\bar{X} \pm \text{SD}$ ~ mean \pm standard deviation.

Creatine phosphate₁ ~ average creatine phosphate concentrations in the muscle 1 hour after slaughter, Creatine phosphate₂ ~ average creatine phosphate concentrations in the muscle 2 hours after slaughter, Creatine phosphate₄ ~ average creatine phosphate concentrations in the muscle 4 hours after slaughter, Creatine phosphate₂₄ ~ average creatine phosphate concentrations in the muscle 24 hours after slaughter.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmentaler cross bred animals.

Effect of the interaction between breed and production system on muscle creatine phosphate concentrations is summarised in Table 4.34. There was no significant effect of the interactions between production system and breed on muscle creatine phosphate concentrations at 1, 2, 4 and 24 hours post mortem.

Table 4.34 Effect of the interaction between breed and production system on creatine phosphate concentrations at 1, 2, 4 and 24 hours post mortem

Production system (n=180)	Breed	Creatine phosphate ₁ (µmol/g) ($\bar{X} \pm SD$)	Creatine phosphate ₂ (µmol/g) ($\bar{X} \pm SD$)	Creatine phosphate ₄ (µmol/g) ($\bar{X} \pm SD$)	Creatine phosphate ₂₄ (µmol/g) ($\bar{X} \pm SD$)
AF	Br-X	6.38±2.50	6.11±2.16	6.31±2.53	4.01±1.81
	Ng-X	6.46±1.20	5.97±1.43	5.17±1.99	3.56±1.56
	Si-X	6.16±1.47	6.75±1.87	5.94±1.87	3.86±1.99
ABF	Br-X	5.28±1.78	5.75±1.90	5.07±1.60	3.36±1.87
	Ng-X	5.60±1.48	6.27±1.59	5.83±1.65	3.86±1.31
	Si-X	6.86±1.23	6.26±1.29	5.26±1.48	3.94±1.78
AP	Br-X	6.10±2.02	5.97±1.89	5.00±1.54	2.84±1.15
	Ng-X	6.05±1.96	6.02±1.91	5.97±1.71	3.99±2.17
	Si-X	6.68±2.54	6.28±1.75	5.29±1.75	3.06±1.19
ABP	Br-X	6.33±1.42	6.23±0.88	5.96±0.71	3.79±1.00
	Ng-X	5.88±1.18	5.94±1.36	5.48±1.52	3.70±1.43
	Si-X	6.15±2.22	6.42±1.93	5.96±2.40	3.56±1.84
BP	Br-X	6.39±1.04	6.81±1.38	6.35±1.33	4.56±1.32
	Ng-X	6.48±1.57	6.21±1.16	5.78±1.93	4.87±1.00
	Si-X	6.80±1.64	7.20±2.22	6.69±1.99	4.47±1.87

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

Creatine phosphate₁ ~ average creatine phosphate concentration in the muscle 1 hour after slaughter, Creatine phosphate₂ ~ average creatine phosphate concentration in the muscle 2 hours after slaughter, Creatine phosphate₄ ~ creatine phosphate concentration in the muscle 4 hours after slaughter, Creatine phosphate₂₄ ~ creatine phosphate concentration in the muscle 24 hours after slaughter.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmentaler cross bred animals.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

4.6 The effect of the production systems and breeds on the myofibrillar fragment lengths at 1, 7 and 14 days post mortem

According to Strydom, Frylinck and Smith (2005) electrical stimulation influences the resulting myofibrillar fragment length. Electrical stimulated carcasses have longer myofibrillar fragment lengths. Animals that received a growth promoter had longer myofibrillar fragment lengths than animals that did not receive a growth promoter (Hope-Jones, Strydom, Frylinck and Webb, 2010).

Effect of production system on myofibrillar fragment length at 1, 7 and 14 days post mortem are summarised in Table 4.35.

At 1 hour post mortem there were no differences between the muscles from the production systems ($p < 0.05$), the myofibrillar fragment length range from 36.39 μm (BP production system) to 40.07 μm (AF production system).

There were differences at 7 and 14 days post mortem ($p < 0.05$) for myofibrillar fragment lengths. At 7 days post mortem, no difference was observed between the muscles from the ABF (31.71 μm), AP (31.60 μm), ABP (31.73 μm) and BP production systems (30.06 μm) as well as between the muscles from the AF (32.76 μm), ABF (31.71 μm), AP (31.60 μm) and ABP production systems (31.73 μm). The muscles from the AF production system (32.76 μm) had longer myofibrillar fragment lengths than the muscles from the BP production system (30.06 μm).

At 14 days post mortem there were no difference for myofibrillar fragment lengths between the muscles from the AF (26.70 μm), ABF (26.15 μm), AP (27.81 μm) and BP production systems (25.83 μm) as well as between the muscles from the AF (26.70 μm), AP (27.81 μm) and ABP production systems (28.30 μm). Numerically there were relatively small differences between production systems for myofibrillar fragment length at 1, 7 and 14 days post mortem.

Table 4.35 Effect of production system on myofibrillar fragment length at 1, 7 and 14 days post mortem

Production system (n=180)	MFL ₁ (μm) ($\bar{X} \pm \text{SD}$)	MFL ₇ (μm) ($\bar{X} \pm \text{SD}$)	MFL ₁₄ (μm) ($\bar{X} \pm \text{SD}$)
AF	40.07 \pm 5.84	32.76 \pm 4.09 ^b	26.70 \pm 3.10 ^{ab}
ABF	36.23 \pm 6.11	31.71 \pm 6.23 ^{ab}	26.15 \pm 3.05 ^a
AP	39.67 \pm 7.16	31.60 \pm 4.61 ^{ab}	27.81 \pm 4.18 ^{ab}
ABP	38.44 \pm 5.04	31.73 \pm 4.17 ^{ab}	28.30 \pm 3.33 ^b
BP	36.39 \pm 6.57	30.06 \pm 3.09 ^a	25.83 \pm 2.80 ^a
Total	38.08 \pm 6.33	31.52 \pm 4.55	26.91 \pm 3.40

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

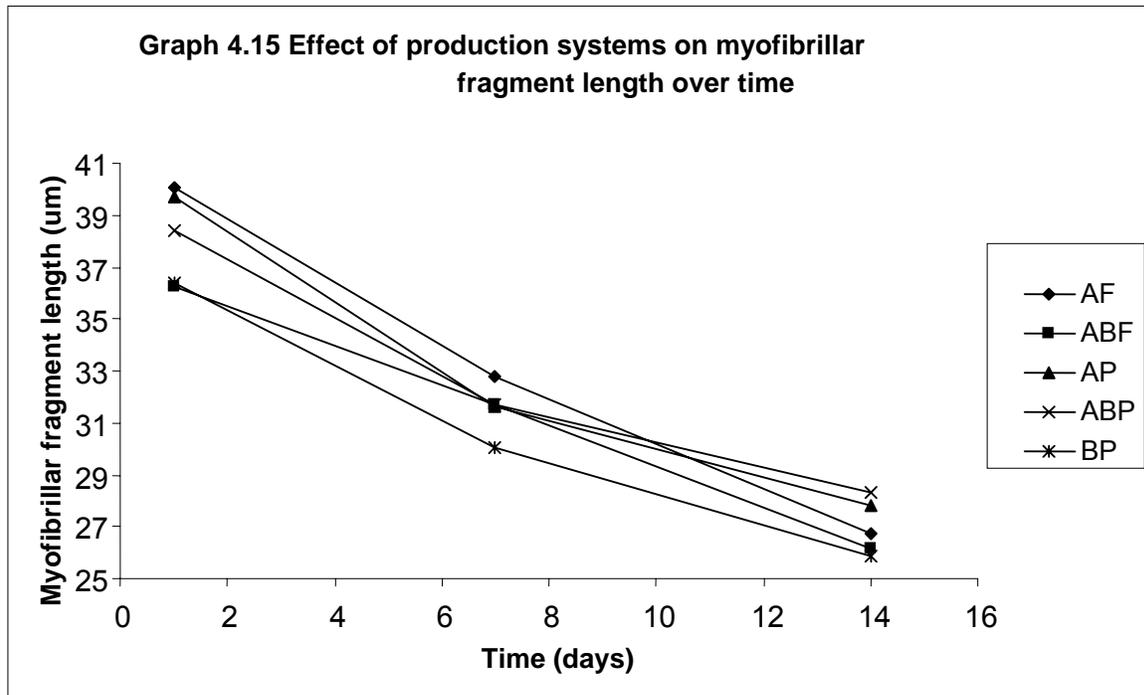
$\bar{X} \pm \text{SD}$ ~ mean \pm standard deviation.

MFL₁ ~ average myofibrillar fragment length at day 1 after slaughter, MFL₇ ~ average myofibrillar fragment length 7 days after slaughter, MFL₁₄ ~ average myofibrillar length 14 days after slaughter.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

Effect of production systems on myofibrillar fragment length over time is illustrated in Graph 4.15. Muscles from the AF production system had the longest myofibril fragment length up to approximately 7 days post mortem from where muscles from the ABF production system had the

longest fragment length. Muscles from the BP production system constantly had the shortest myofibril fragment length. According to Strydom, Frylinck, *et al.*, (2005) myofibrillar fragment length decreases with time post mortem (Also see par 2.6, Chap 2: literature review).



Considering the effect of feeding system only, there were effects on the myofibril fragment lengths at 14 days post mortem ($p < 0.05$), but not at 1 and 7 days post mortem ($p > 0.05$). Muscles from the feedlot fed animals ($26.42 \pm 3.06 \mu\text{m}$) showed shorter fragment lengths at 14 days post mortem than the muscles from the pasture fed animals ($27.22 \pm 3.58 \mu\text{m}$). Muscles from pasture fed animals ($38.04 \pm 6.41 \mu\text{m}$ and $31.06 \pm 3.99 \mu\text{m}$ respectively) had a tendency for shorter myofibril fragment lengths at 1 and 7 days post mortem in comparison with muscles from feedlot fed animals ($38.15 \pm 6.24 \mu\text{m}$ and $32.23 \pm 5.25 \mu\text{m}$ respectively). The study of Martínez-Cerezo, Sanudo, *et al.* (2005) show that feeding system has an effect on myofibril fragment lengths, whereas other studies shows no effect (Sami, Augustini and *et al.*, 2004; Lowe, Peachey and *et al.*, 2002).

Effect of age per se showed that there were differences between age classification groups at 1 and 7 days post mortem for myofibril fragment length ($p < 0.05$). At 1 and 7 days post mortem, muscles from the A age classification group ($39.88 \pm 6.47 \mu\text{m}$ and $32.19 \pm 4.36 \mu\text{m}$ respectively) had the longer myofibrillar fragment lengths than muscles from the AB ($37.33 \pm 5.67 \mu\text{m}$ and $31.77 \pm 5.26 \mu\text{m}$ respectively) and B age classification groups ($36.39 \pm 6.57 \mu\text{m}$ and $30.06 \pm 3.09 \mu\text{m}$

respectively). At 14 days post mortem there were no differences ($p>0.05$) between age classification groups. Sami, Augustini and *et al* (2004), state that animal age does not affect myofibril fragment lengths.

Effect of breed on myofibrillar fragment length is summarised in Table 4.36. According to Strydom (2008) there is a significant difference between breeds for myofibril fragment length at day 7, the author also states that Brahman cattle had the longest myofibril fragment length, at day 7. When only the breed is considered; there were differences ($p<0.05$) between the breeds at 1 and 7 days post mortem. There were no differences ($p>0.05$) between breeds for myofibril length at day 14. Muscles from Brahman cross bred animals (40.73 μm , 34.31 μm and 27.48 μm respectively) had the longest myofibril fragment length and muscles from Simmental cross bred animals (36.21 μm , 29.16 μm and 26.12 μm respectively) had the shortest myofibril fragment length, at day 1, 7 and 14. Myofibrillar fragment length decreased as time progressed.

Table 4.36 Effect of breed on myofibril fragment lengths at 1, 7 and 24 days post mortem

Breed (n=180)	MFL ₁ (μm) ($\bar{X}\pm\text{SD}$)	MFL ₇ (μm) ($\bar{X}\pm\text{SD}$)	MFL ₁₄ (μm) ($\bar{X}\pm\text{SD}$)
Br-X	40.73 \pm 6.10 ^b	34.31 \pm 5.23 ^c	27.48 \pm 3.91
Ng-X	37.32 \pm 6.43 ^a	31.07 \pm 4.29 ^b	27.11 \pm 3.27
Si-X	36.21 \pm 5.61 ^a	29.16 \pm 2.45 ^a	26.12 \pm 2.83

^{abcd} Means in a column with different superscripts differ significantly ($p<0.05$) based on the Fishers' means separation test.

$\bar{X}\pm\text{SD}$ ~ mean \pm standard deviation.

MFL₁ ~ average myofibril fragment length at day 1 after slaughter, MFL₇ ~ average myofibril fragment length 7 days after slaughter, MFL₁₄ ~ average myofibril length 14 days after slaughter.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmental cross bred animals.

Effect of the interaction between breed and production system on myofibril fragment lengths at 1, 7 and 14 days post mortem is summarised in Table 4.37. The interactions between production system and breed had an effect on myofibril fragment length 1, 7 and 14 days post mortem ($p<0.05$). Muscles of the Nguni animals from the ABF production system (33.42 μm) had the shortest myofibril fragment length at 1 day post mortem, Simmental animals from the ABP production system (27.96 μm) had the shortest myofibril fragment length at 7 days post mortem and Nguni animals from the ABF production system (25.10 μm) had the shortest length at 14 days post mortem.

Muscles of the Brahman animals from the AP production system (42.05 μm) had the longest myofibrillar fragment length at 1 day post mortem, Brahman animals from the ABF production system (32.96 μm) had the longest length at 7 days post mortem and Nguni animals from the ABP production system (26.04 μm) had the longest length at 14 days post mortem.

Table 4.37 Effect of the interaction between breed and production system on myofibril fragment lengths at 1, 7 and 14 days post mortem.

Production system (n=180)	Breed	Myofibril fragment length ₁ (μm) ($\bar{X} \pm SD$)	Myofibril fragment length ₇ (μm) ($\bar{X} \pm SD$)	Myofibril fragment length ₁₄ (μm) ($\bar{X} \pm SD$)
AF	Br-X	41.80±5.09 ^b	35.025±2.94 ^{bc}	27.97±2.78 ^{ab}
	Ng-X	41.38±7.49 ^{ab}	32.96±4.71 ^{abc}	26.52±3.30 ^{ab}
	Si-X	37.14±3.77 ^{ab}	30.30±1.93 ^{ab}	25.59±2.98 ^{ab}
ABF	Br-X	41.41±6.33 ^{ab}	37.30±6.57 ^c	27.34±4.60 ^{ab}
	Ng-X	33.42±4.20 ^a	29.20±5.17 ^a	25.10±1.52 ^a
	Si-X	34.45±4.73 ^{ab}	29.07±2.06 ^a	26.30±2.19 ^{ab}
AP	Br-X	42.05±6.61 ^b	33.56±5.83 ^{abc}	28.28±5.15 ^{ab}
	Ng-X	38.07±8.25 ^{ab}	32.23±4.18 ^{abc}	28.53±4.11 ^{ab}
	Si-X	38.83±6.71 ^{ab}	29.27±2.56 ^a	26.74±3.39 ^{ab}
ABP	Br-X	39.88±2.31 ^{ab}	35.30±3.43 ^{bc}	28.74±3.68 ^{ab}
	Ng-X	39.75±5.37 ^{ab}	32.13±3.41 ^{abc}	29.53±3.07 ^b
	Si-X	35.47±5.45 ^{ab}	27.96±2.17 ^a	26.34±2.60 ^{ab}
BP	Br-X	38.97±8.00 ^{ab}	31.44±3.06 ^{abc}	25.77±3.10 ^{ab}
	Ng-X	34.89±3.93 ^{ab}	29.44±2.77 ^{ab}	26.04±2.23 ^{ab}
	Si-X	34.91±6.38 ^{ab}	29.08±3.07 ^a	25.70±3.14 ^{ab}

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

Myofibril fragment length₁ ~ average myofibril fragment length at day 1 after slaughter, Myofibril fragment length₇ ~ average myofibril fragment length 7 days after slaughter, Myofibril fragment length₁₄ ~ average myofibril length 14 days after slaughter.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmental cross bred animals.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

4.7 The effect of production system and breed on the calpastatin and calpain activity at 1 and 24 hours post mortem.

According to Dransfield (1994), as animals grow and get older the levels of calpains increase. Growth promoters as well as muscle type have a tendency to influence the calpain system. β -agonists have the tendency to increase calpain II activity and calpastatin activity and decrease calpain I (Dransfield, 1994).

Table 4.38 Effect of production system on total calpastatin as well as the specific calpastatin activity at 1 and 24 hours post mortem

Production system (n=180)	Total Calpastatin 1 h (U/g) ($\bar{X} \pm SD$)	Total Calpastatin 24 h (U/g) ($\bar{X} \pm SD$)	Specific Calpastatin 1 h (U) ($\bar{X} \pm SD$)	Specific Calpastatin 24 h (U) ($\bar{X} \pm SD$)
AF	2.37±0.22 ^b	1.89±0.35 ^b	0.040±0.004 ^b	0.035±0.006 ^{cd}
ABF	2.36±0.24 ^b	1.99±0.28 ^b	0.041±0.005 ^b	0.037±0.006 ^d
AP	2.24±0.30 ^b	1.67±0.30 ^a	0.042±0.006 ^b	0.034±0.006 ^{bc}
ABP	2.25±0.25 ^b	1.60±0.37 ^a	0.041±0.005 ^b	0.032±0.007 ^b
BP	2.04±0.30 ^a	1.48±0.25 ^a	0.035±0.005 ^a	0.028±0.005 ^a
Total	2.24±0.29	1.72±0.36	0.039±0.005	0.033±0.007

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

Total Calpastatin 1 h ~ average unit of calpastatin at 1 hour after slaughter, Total Calpastatin 24 h ~ average unit of calpastatin at 24 hours after slaughter, Specific Calpastatin 1 h ~ average unit of specific calpastatin at 1 hour after slaughter, Specific Calpastatin 24 h ~ average unit of specific calpastatin at 24 hours after slaughter.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

According to Ferguson, Jiang, Hearnshaw, Rymill and Thompson (2000), electrical stimulation does not affect the activity of calpain I and II, but electrical stimulation decreases the activity of calpastatin. Hope-Jones, Strydom, *et al.* (2010) reported that electrical stimulation decreases calpastatin activity and increases calpain I and II activity. They also state that growth promoters increase calpastatin activity but have no effect on calpain activity. Effect of production system on total and specific calpastatin activity at 1 and 24 hours are summarised in Table 4.38. There were differences for calpastatin activity at 1 and 24 hours post mortem ($p < 0.05$). For the total and specific calpastatin activity at 1 hour post mortem the muscles from the BP (2.04 U/g and 0.035 U respectively) had lower activity than the muscles from the AF (2.37 U/g and 0.040 U respectively), ABF (2.36 U/g and 0.042 U respectively), ABP (2.25 U/g and 0.041 U respectively) and AP production systems (2.24 U/g and 0.041 U respectively) ($p < 0.05$).

Muscles from the AF (1.89 U/g), AP (1.67 U/g), ABF (1.99 U/g) and ABP production systems (1.60 U/g) had higher calpastatin activity at 24 hours post mortem than the muscles from the BP (1.48 U/g) and ABP production systems (1.60 U/g) ($p < 0.05$). The calpastatin activity decreased as time progressed. According to Kendall, Koohmaraie, Arbona, Williams and Young (1993), the calpastatin activity decreases as the pH decreases. As the time progress the pH decreases, therefore as time progresses the calpastatin activity should decrease.

Considering the effect of feeding system only, there were differences ($p < 0.05$) between the feeding systems for total calpastatin activity at 1 and 24 hours post mortem. Muscles from the pasture fed animals (2.17 ± 0.30 U/g and 1.58 ± 0.31 U/g respectively) had lower calpastatin activity than muscles from feedlot fed animals (2.37 ± 0.23 U/g and 1.94 ± 0.32 U/g respectively) at 1 and 24 hours post mortem. There were no differences in the specific calpastatin activity ($p > 0.05$). Muscles from pasture fed animals (0.039 ± 0.006 U and 0.031 ± 0.007 U respectively) had the tendency for slightly lower activity at 1 and 24 hours post mortem than the muscles from the feedlot fed animals (0.040 ± 0.004 U and 0.036 ± 0.007 U. respectively).

When only the effect of age is considered; there were differences in the calpastatin activity ($p < 0.05$) at 1 and 24 hours post mortem. Muscles from B age classification group (2.04 ± 0.30 U/g and 0.035 ± 0.005 U respectively) had lower ($p < 0.05$) calpastatin activity, at 1 hour post mortem, than muscles from AB (2.30 ± 0.25 U/g and 0.041 ± 0.005 U respectively) and A age classification group (2.31 ± 0.27 U/g and 0.041 ± 0.005 U respectively). Muscles from the B age classification group (1.48 ± 0.25 U/g and 0.028 ± 0.005 U respectively) had ($p < 0.05$) lower calpastatin activity, at 24 hours post mortem, than muscles from AB (1.80 ± 0.38 U/g and 0.034 ± 0.007 U respectively) and A age classification group (1.78 ± 0.34 U/g and 0.034 ± 0.006 U respectively).

Table 4.39 Effect of breed on total and specific calpastatin at 1 and 24 hours post mortem.

Breed (n=180)	Total Calpastatin	Total Calpastatin	Specific	Specific
	1 h (U/g) ($\bar{X} \pm SD$)	24 h (U/g) ($\bar{X} \pm SD$)	Calpastatin 1 h (U) ($\bar{X} \pm SD$)	Calpastatin 24 h (U) ($\bar{X} \pm SD$)
Br-X	2.32 ± 0.26^b	1.87 ± 0.35^b	0.041 ± 0.005^b	0.036 ± 0.007^b
Ng-X	2.26 ± 0.29^{ab}	1.66 ± 0.36^a	0.039 ± 0.005^{ab}	0.031 ± 0.007^a
Si-X	2.15 ± 0.30^a	1.63 ± 0.34^a	0.038 ± 0.006^a	0.031 ± 0.007^a

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

Total calpastatin 1 h ~ average total calpastatin activity at 1 hour post mortem, Total calpastatin 24 h ~ average total calpastatin activity at 24 hours post mortem, Specific calpastatin 1 h ~ average specific calpastatin activity at 1 hour post mortem.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmental cross bred animals.

Effect of breed on the activity of calpastatin at 1 and 24 hours post mortem is summarised in Table 4.39. Considering the effect of breed only; there were differences between the breed for calpastatin activity at 1 and 24 hours post mortem ($p < 0.05$). Muscles from Brahman cross bred animals had the highest calpastatin activity and muscles from Simmental cross bred animals the lowest activity (Table 4.39). The differences in breeds can be explained by differences in muscle energy status, genetics (not discussed in dissertation) and pH. Frylinck, van Wyk, *et al.* (2009),

reported no differences between Nguni, Simmental and Brahman cross bred animals for calpastatin activity at 1 and 24 hours post mortem. This can be explained by the fact that the origin of the Simmental cross bred animals is from an unreliable source and was probably contaminated with Brahman genetics.

Table 4.40 Effect of the interaction between breed and production system on calpastatin activity

Production system (n=180)	Breed	Total	Total	Specific	Specific
		Calpastatin 1 h (U/g) ($\bar{X} \pm SD$)	Calpastatin 24 h (U/g) ($\bar{X} \pm SD$)	Calpastatin 1 h (U) ($\bar{X} \pm SD$)	Calpastatin 24 h (U) ($\bar{X} \pm SD$)
AF	Br-X	2.43±0.22 ^{bc}	2.16±0.26 ^f	0.041±0.004 ^{bc}	0.041±0.006 ^c
	Ng-X	2.42±0.25 ^{bc}	1.82±0.27 ^{bcdef}	0.041±0.004 ^{bc}	0.033±0.006 ^{abc}
	Si-X	2.26±0.15 ^{bc}	1.68±0.35 ^{abcde}	0.038±0.002 ^{bc}	0.030±0.006 ^{ab}
ABF	Br-X	2.44±0.20 ^c	2.10±0.22 ^{ef}	0.043±0.004 ^c	0.041±0.004 ^c
	Ng-X	2.32±0.25 ^{bc}	1.87±0.28 ^{cdef}	0.039±0.004 ^{bc}	0.034±0.005 ^{bc}
	Si-X	2.33±0.26 ^{bc}	2.03±0.28 ^{def}	0.042±0.006 ^{bc}	0.038±0.006 ^{bc}
AP	Br-X	2.34±0.27 ^{bc}	1.73±0.18 ^{abcdef}	0.043±0.006 ^c	0.034±0.004 ^{bc}
	Ng-X	2.21±0.39 ^{abc}	1.70±0.41 ^{abcde}	0.041±0.007 ^{bc}	0.033±0.007 ^{abc}
	Si-X	2.17±0.24 ^{abc}	1.60±0.28 ^{abcd}	0.041±0.006 ^{bc}	0.033±0.006 ^{abc}
ABP	Br-X	2.31±0.23 ^{bc}	1.79±0.44 ^{abcdef}	0.037±0.004 ^{bc}	0.036±0.009 ^{bc}
	Ng-X	2.27±0.23 ^{bc}	1.55±0.39 ^{abc}	0.041±0.005 ^{bc}	0.030±0.008 ^{ab}
	Si-X	2.16±0.29 ^{abc}	1.50±0.23 ^{abc}	0.039±0.004 ^{bc}	0.030±0.004 ^{ab}
BP	Br-X	2.14±0.28 ^{abc}	1.61±0.25 ^{abcd}	0.037±0.005 ^{abc}	0.030±0.005 ^{ab}
	Ng-X	2.06±0.25 ^{ab}	1.40±0.22 ^a	0.035±0.004 ^{ab}	0.027±0.005 ^a
	Si-X	1.89±0.32 ^b	1.41±0.22 ^{ab}	0.032±0.005 ^a	0.026±0.005 ^a

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

Total calpastatin 1 h ~ average total calpastatin activity at 1 hour post mortem, Total calpastatin 24 h ~ average total calpastatin activity at 24 hours post mortem, Specific calpastatin 1 h ~ average specific calpastatin activity at 1 hour post mortem, Specific calpastatin 24 h ~ average specific calpastatin activity at 24 hours post mortem.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmental cross bred animals.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

Effect of the interaction between breed and production system on calpastatin activity are summarised in Table 4.40. The interactions between production system and breed had an effect

on total calpastatin and specific calpastatin activity at 1 and 24 hours post mortem ($p < 0.05$). Muscles of the Simmental animals from the BP production system (1.89 U/g) had the lowest total calpastatin activity at 1 hour post mortem, Nguni animals from the BP production system (1.40 U/g) had the lowest total calpastatin activity at 24 hours post mortem, Brahman animals from the ABF production system (2.44 U/g) had the highest total calpastatin activity at 1 hour post mortem and Brahman animals from the AF production system (2.16 U/g) had the highest total calpastatin activity at 24 hours post mortem. Muscles of the Simmental animals from the BP production system (0.032 U and 0.026 U respectively) had the lowest specific calpastatin activity at 1 and 24 hours post mortem, Brahman animals from the ABF production system (0.043 U) and Brahman animals from the AP production system (0.043 U) had the highest specific calpastatin activity at 1 hour post mortem and Brahman animals from the AF (0.041 U) and ABF production system (0.041 U) had the highest specific calpastatin activity at 24 hours post mortem.

Table 4.41 Effect of production system on calpain I activity at 1 and 24 hours post mortem

Production system (n=180)	Calpain I 1 h (U/g) ($\bar{X} \pm SD$)	Calpain I 24 h (U/g) ($\bar{X} \pm SD$)	Specific calpain I 1 h (U) ($\bar{X} \pm SD$)	Specific calpain I 24 h (U) ($\bar{X} \pm SD$)
AF	1.17 \pm 0.20 ^b	0.73 \pm 0.19 ^a	0.020 \pm 0.003 ^a	0.014 \pm 0.003 ^a
ABF	1.34 \pm 0.22 ^c	0.92 \pm 0.22 ^b	0.023 \pm 0.004 ^b	0.017 \pm 0.004 ^b
AP	1.04 \pm 0.19 ^a	0.74 \pm 0.18 ^a	0.019 \pm 0.004 ^a	0.015 \pm 0.003 ^{ab}
ABP	1.13 \pm 0.18 ^{ab}	0.66 \pm 0.22 ^a	0.021 \pm 0.003 ^a	0.013 \pm 0.005 ^a
BP	1.13 \pm 0.17 ^{ab}	0.70 \pm 0.26 ^a	0.019 \pm 0.003 ^a	0.013 \pm 0.005 ^a
Total	1.16 \pm 0.21	0.75 \pm 0.23	0.020 \pm 0.004	0.014 \pm 0.004

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

Calpain I 1 h ~ average unit of calpain I at 1 hour after slaughter, Specific calpain I 1 h ~ average unit of specific calpain I at 1 hour after slaughter. Calpain I 24 h ~ average unit of calpain I at 24 hour after slaughter, Specific calpain I 24 h ~ average unit of specific calpain I at 24 hour after slaughter.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

Calpain I activity are effected by thawing rate, with rapid thawing there is a reduction of 14 % of calpain I activity (Dransfield, 1996). Effect of production system on calpain I activity at 1 and 24 hours post mortem is summarised in Table 4.41. There were differences between production systems for calpain I activity at 1 and 24 hours post mortem ($p < 0.05$). Total calpain I activity at 1 hour post mortem was the highest in the muscles from the ABF production system (1.34 U/g) and the lowest in muscles from the AP production system (1.04 U/g). Total calpain I activity at 24 hours post mortem was the highest in the muscles from the ABF production system (0.92 U/g) and the

lowest in muscles from the ABP production system (0.66 U/g). The specific calpain I activity at 1 and 24 hours post mortem was the lowest in the muscles from the BP production system (0.019 U and 0.013 U respectively) and the muscles from the ABF production system (0.023 U and 0.017 U respectively) had the highest activity. Calpain I activity seems to decrease as the time progressed. According to Dransfield (1996) the extractability of the calpains decrease as time progress post mortem.

Considering the effect of feeding system only, muscles from the pasture fed animals (1.10 ± 0.18 U/g and 0.98 ± 0.11 respectively) showed lower total calpain I activity than muscles from feedlot fed animals (1.25 ± 0.22 U/g and 1.08 ± 0.13 respectively) at 1 and 24 hours ($p < 0.05$). Muscles from pasture fed animals (0.020 ± 0.003 U and 0.014 ± 0.004 U respectively) had slightly lower activity than muscles from feedlot fed animals (0.022 ± 0.004 U and 0.015 ± 0.004 U respectively) at 1 and 24 hours post mortem ($p < 0.05$).

When only the effect of age is considered, muscles from the AB age classification group (1.23 ± 0.22 U/g) had a higher calpain I activity, at 1 hour post mortem, than muscles from the A (1.11 ± 0.20 U/g) and B age classification group (1.13 ± 0.17 U/g) ($p < 0.05$). Muscles from the B age classification group (0.70 ± 0.26 U/g) had significant lower calpain I activity, at 24 hours, than muscles from the AB age classification group (0.79 ± 0.26 U/g) and muscles from the A age classification group (0.74 ± 0.18 U/g). All the differences that occurred were numerically small.

Effect of breed on calpain I and specific calpain I activity at 1 and 24 hours post mortem are summarized in Table 4.42. When only the effect of breed is considered, there were differences ($p < 0.05$) between the breeds for total and specific calpain I activity at 1 hour post mortem, but not ($p > 0.05$) at 24 hours post mortem. Muscles from Brahman cross bred animals (1.07 U/g and 0.019 U respectively) had lower total and specific calpain I activity at 1 hour than Simmental (1.19 U/g and 0.021 U respectively) and Nguni cross bred animals (1.23 U/g and 0.021 U respectively) ($p < 0.05$).

Muscles from Nguni cross bred animals (0.73 U/g and 0.014 U respectively) had the lower total and specific calpain I activity at 24 hours post mortem than the muscles from the Brahman (0.75 U/g and 0.015 U respectively) and Simmental cross bred animals (0.76 U/g and 0.015 U respectively) ($p > 0.05$). According to Frylinck, Van Wyk, *et al.* (2009), Nguni cross bred animals had lower calpain I activity at 1 and 24 hours post mortem than muscles from Simmental and Brahman cross bred animals.

Table 4.42 Effect of breed on calpain I and specific calpain I at 1 and 24 hours post mortem.

Breed (n=180)	Calpain I 1 h (U/g) ($\bar{X} \pm SD$)	Calpain I 24 h (U/g) ($\bar{X} \pm SD$)	Specific Calpain I 1 h (U) ($\bar{X} \pm SD$)	Specific Calpain I 24 h (U) ($\bar{X} \pm SD$)
Br-X	1.07±0.17 ^a	0.75±0.21	0.019±0.003 ^a	0.015±0.004
Ng-X	1.23±0.23 ^b	0.73±0.25	0.021±0.004 ^b	0.014±0.005
Si-X	1.19±0.20 ^b	0.76±0.24	0.021±0.004 ^b	0.015±0.004

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

Calpain 1 h ~ average unit of calpain at 1 hour after slaughter, Specific calpain 1 h ~ average unit of specific calpain at 1 hour after slaughter, Calpain I 24 h ~ average unit of calpain I at 24 hour after slaughter, Specific calpain I 24 h ~ average unit of specific calpain I at 24 hour after slaughter.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmental cross bred animals.

Effect of the interaction between breed and production system on the total and specific calpain I activity at 1 and 24 hours post mortem are summarised in Table 4.43. The interaction between breed and production systems had an effect on total calpain I activity at 1 and 24 hours post mortem and specific calpain I activity at 1 and 24 hours post mortem ($p < 0.05$).

Muscles of the Brahman animals from the AP production system (0.93 U/g) had the lowest calpain I activity at 1 hour post mortem, Nguni animals from the BP production system (0.61 U/g) had the lowest calpain I activity at 24 hours post mortem, Nguni animals from the ABF production system (1.45 U/g) had the highest calpain I activity 1 hour post mortem and Simmental animals from the ABF production system (0.98 U/g) had the highest calpain I activity at 24 hours post mortem.

Muscles of the Brahman animals from the AP production system (0.017 U) had the lowest specific calpain I activity at 1 hour post mortem, Nguni animals from the AF production system (0.012 U), Nguni animals from the BP production system (0.012 U) and Simmental animals from the BP production system (0.012 U) had the lowest specific calpain I activity at 24 hours post mortem, Nguni and Simmental animals from the ABF production system (0.025 U) had the highest specific calpain I activity at 1 hour post mortem and Simmental animals from the ABF production system (0.018 U) had the highest specific calpain I activity at 24 hours post mortem.

Table 4.43 Effect of the interaction between breed and production system on total and specific calpain I activity at 1 and 24 hours post mortem

Production system (n=180)	Breed	Calpain I 1 h (U/g) ($\bar{X} \pm SD$)	Calpain I 24 h (U/g) ($\bar{X} \pm SD$)	Specific Calpain I 1 h (U) ($\bar{X} \pm SD$)	Specific Calpain I 24 h (U) ($\bar{X} \pm SD$)
AF	Br-X	1.10±0.17 ^a	0.78±0.17 ^{abc}	0.019±0.003 ^{ab}	0.014±0.003 ^{ab}
	Ng-X	1.22±0.22 ^{ab}	0.66±0.17 ^{abc}	0.021±0.004 ^{bcd}	0.012±0.003 ^{ab}
	Si-X	1.20±0.20 ^a	0.74±0.21 ^{abc}	0.020±0.003 ^{bcd}	0.013±0.004 ^{ab}
ABF	Br-X	1.17±0.18 ^{ab}	0.86±0.23 ^{abc}	0.020±0.004 ^{abc}	0.017±0.004 ^{ab}
	Ng-X	1.45±0.18 ^b	0.92±0.20 ^{bc}	0.025±0.003 ^{cd}	0.017±0.004 ^{ab}
	Si-X	1.38±0.20 ^b	0.98±0.24 ^c	0.025±0.004 ^{cd}	0.018±0.004 ^b
AP	Br-X	0.93±0.18 ^a	0.70±0.14 ^{abc}	0.017±0.004 ^a	0.014±0.003 ^{ab}
	Ng-X	1.12±0.22 ^{ab}	0.78±0.20 ^{abc}	0.021±0.005 ^{abc}	0.015±0.004 ^{ab}
	Si-X	1.08±0.14 ^{ab}	0.75±0.21 ^{abc}	0.020±0.003 ^{ab}	0.015±0.004 ^{ab}
ABP	Br-X	1.09±0.05 ^{ab}	0.63±0.14 ^{ab}	0.020±0.001 ^{ab}	0.013±0.003 ^{ab}
	Ng-X	1.13±0.21 ^{ab}	0.66±0.30 ^{ab}	0.020±0.004 ^{abc}	0.013±0.006 ^{ab}
	Si-X	1.17±0.21 ^{ab}	0.68±0.17 ^{abc}	0.021±0.004 ^{abc}	0.014±0.004 ^{ab}
BP	Br-X	1.06±0.16 ^a	0.78±0.26 ^{abc}	0.018±0.003 ^{ab}	0.015±0.005 ^{ab}
	Ng-X	1.19±0.20 ^{ab}	0.61±0.24 ^a	0.020±0.004 ^{bc}	0.012±0.005 ^a
	Si-X	1.15±0.12 ^{ab}	0.68±0.26 ^{abc}	0.020±0.003 ^{abc}	0.012±0.005 ^{ab}

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

Total calpain I 1 h ~ average total calpain I activity at 1 hour post mortem, Total calpain I 24 h ~ average total calpain I activity at 24 hours post mortem, Specific calpain I 1 h ~ average specific calpain I activity at 1 hour post mortem, Specific calpain I 24 h ~ average specific calpain I activity at 24 hours post mortem.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmental cross bred animals.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

According to Kendall, Koohmaraie, *et al.* (1993), calpain II activity decreases as pH levels decrease. The pH levels decrease as time progress, therefore as time progress the activity of calpain II should decrease. Effect of production system on total calpain II and specific calpain II activity at 1 and 24 hours post mortem are summarised in Table 4.44. The calpain II activity at 1 hour post mortem range from 0.94 U/g (AP production system) to 1.10 U/g (ABF production system) ($p < 0.05$). At 24 hours post mortem the calpain II activity of the muscles of the BP production system had the highest activity and muscles from the AP production system had the

lowest activity. Muscles of the ABF production system had the highest specific calpain II activity at 1 and 24 hours post mortem and muscles from the BP production system had lowest activity at 24 hour post mortem. Muscles from the AP production system as well as the muscles from the BP production system had the lowest specific calpain II activity at 1 hour post mortem. The activity of calpain II decreased as time progressed for 1 hour to 24 hours post mortem.

Table 4.44 Effect of production system on calpain II and specific calpain II at 1 and 24 hours post mortem

Production system (n=180)	Calpain II 1 h (U/g) ($\bar{X} \pm SD$)	Calpain II 24 h (U/g) ($\bar{X} \pm SD$)	Specific Calpain II 1 h (U) ($\bar{X} \pm SD$)	Specific Calpain II 24 h (U) ($\bar{X} \pm SD$)
AF	1.06±0.13 ^{bc}	0.90±0.11 ^a	0.018±0.002 ^{ab}	0.017±0.002 ^a
ABF	1.10±0.14 ^c	0.95±0.09 ^b	0.019±0.002 ^b	0.021±0.002 ^c
AP	0.94±0.10 ^a	0.93±0.11 ^a	0.017±0.002 ^a	0.019±0.002 ^b
ABP	0.99±0.11 ^{ab}	0.95±0.09 ^a	0.018±0.002 ^{ab}	0.019±0.002 ^b
BP	1.00±0.12 ^{ab}	0.98±0.09 ^c	0.017±0.002 ^a	0.018±0.002 ^b
Total	1.02±0.13	0.98±0.13	0.018±0.002	0.019±0.002

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

Calpain II 1 h ~ The average unit of Calpain II at 1 hour after slaughter, Calpain II 24 h ~ The average unit of Calpain II at 24 hour after slaughter, Specific calpain II 1 h ~ The average unit of specific calpain II at 1 hour after slaughter, Specific calpain II 24 h ~ The average unit of specific calpain II at 24 hour after slaughter

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

Considering the effect of feeding system only, there were differences between feeding systems for total calpain II activity at 1 and 24 hours post mortem and specific calpain II activity at 1 hour post mortem ($p < 0.05$). This difference was small with animals from the pasture (0.98 ± 0.11 U/g and 0.96 ± 0.10 U/g respectively) showing lower activity than animals from the feedlot (1.08 ± 0.13 U/g and 1.01 ± 0.16 U/g respectively) for calpain II activity at 1 and 24 hours post mortem. Animals from the pasture (0.018 ± 0.002 U and 0.019 ± 0.002 U respectively) had lower activity than animals from the feedlot (0.018 ± 0.002 U and 0.019 ± 0.003 U respectively) for specific calpain II activity at 1 and 24 hours post mortem.

When only the effect of age is considered, muscles from the A age classification group animals (1.00 ± 0.13 U/g) had lower total calpain II activity at 1 hour ($p < 0.05$), than AB age classification group animals (1.00 ± 0.13 U/g) (there were no difference ($p > 0.05$) between B (1.00 ± 0.12 U/g) and A (1.00 ± 0.13 U/g), B (1.00 ± 0.12 U/g) and AB (1.00 ± 0.13 U/g)). Muscles from

the AB age classification group animals (0.019 ± 0.002 U) had higher specific calpain II activity at 1 hour ($p < 0.05$), than A (0.018 ± 0.002 U) and B age classification group animals (0.017 ± 0.002 U).

Muscles from the A age classification group (1.091 ± 0.11 U/g) had the lowest total calpain II activity, muscles from the B age classification group (0.98 ± 0.09 U/g) had higher activity and animals from the AB age classification group (1.03 ± 0.14 U/g) had the highest activity, at 24 hours, (there were no significant difference between A (0.018 ± 0.002 U) and B age classification group animals (0.018 ± 0.002 U) ($p < 0.05$)). All the differences that occurred are numerically small.

Effect of breed on the calpain II and specific calpain II at 1 and 24 hours post mortem are summarised in Table 4.45. Breed per se showed differences between the breeds for calpain II and specific calpain II activity at 1 and 24 hours post mortem ($p < 0.05$). Muscles from Brahman and Nguni animals had higher calpain II activity than muscles from Simmental cross bred animals at 1 and 24 hours post mortem (Table 4.45). According to Frylinck, Van Wyk, *et al.* (2009), Nguni cross bred animals had a higher calpain II activity than muscles from Simmental and Brahman cross bred animals.

Table 4.45 Effect of breeds on calpain II and specific calpain II at 1 and 24 hours post mortem

Breed (n=180)	Calpain II 1 h (U/g) ($\bar{X} \pm SD$)	Calpain II 24 h (U/g) ($\bar{X} \pm SD$)	Specific Calpain II 1 h (U) (\bar{X} $\pm SD$)	Specific Calpain II 24 h (U) (\bar{X} $\pm SD$)
Br-X	1.02 ± 0.12^{ab}	0.99 ± 0.13^b	0.018 ± 0.002^{ab}	0.019 ± 0.003^b
Ng-X	1.04 ± 0.14^b	0.99 ± 0.14^{ab}	0.018 ± 0.002^b	0.019 ± 0.002^{ab}
Si-X	0.98 ± 0.12^a	0.94 ± 0.10^a	0.017 ± 0.002^a	0.018 ± 0.002^a

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

Calpain II 1 h ~ average unit of Calpain II at 1 hour after slaughter, Calpain II 24 h ~ average unit of Calpain II at 24 hour after slaughter, Specific calpain II 1 h ~ average unit of specific calpain II at 1 hour after slaughter, Specific calpain II 24 h ~ average unit of specific calpain II at 24 hour after slaughter.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmental cross bred animals.

Effect of the interaction between breed and production system on the calpain II activity is summarised in Table 4.46. The interaction between breed and production system had an effect on calpain activity at 1 and 24 hours post mortem and specific calpain II at 24 hours post mortem ($p < 0.05$). Muscles of the Simmental animals from the ABP production system (0.91 U/g) had the lowest calpain II activity at 1 hour post mortem, Simmental animals from the AF production system (0.87 U/g) had the lowest calpain II activity at 24 hours post mortem, Nguni animals from the ABF

production system (1.13 U/g and 1.17 U/g respectively) had the highest calpain II activity 1 and 24 hours post mortem. Numerically there were very small differences between the interactions for specific calpain II at 1 and 24 hours post mortem.

Table 4.46 Effect of the interaction between breed and production system on the calpain II and specific calpain II activity at 1 and 24 hours post mortem

Production system (n=180)	Breed	Calpain II 1 h (U/g) ($\bar{X} \pm SD$)	Calpain II 24 h (U/g) ($\bar{X} \pm SD$)	Specific Calpain II 1 h (U) ($\bar{X} \pm SD$)	Specific Calpain II 24 h (U) ($\bar{X} \pm SD$)
AF	Br-X	1.10±0.10 ^{bc}	0.94±0.10 ^{ab}	0.019±0.002	0.018±0.002 ^{abc}
	Ng-X	1.08±0.16 ^{abc}	0.88±0.13 ^a	0.018±0.003	0.016±0.002 ^{ab}
	Si-X	0.99±0.08 ^{abc}	0.87±0.08 ^a	0.017±0.001	0.016±0.001 ^a
ABF	Br-X	1.06±0.17 ^{abc}	1.12±0.15 ^c	0.019±0.003	0.022±0.003 ^e
	Ng-X	1.13±0.11 ^c	1.17±0.09 ^d	0.019±0.002	0.021±0.002 ^{de}
	Si-X	1.11±0.13 ^{bc}	1.06±0.09 ^{bc}	0.020±0.002	0.020±0.002 ^{cde}
AP	Br-X	0.95±0.06 ^{ab}	0.93±0.11 ^{ab}	0.017±0.001	0.019±0.003 ^{bcd}
	Ng-X	0.95±0.16 ^{ab}	0.96±0.14 ^{ab}	0.018±0.003	0.019±0.002 ^{bcd}
	Si-X	0.92±0.06 ^a	0.90±0.08 ^a	0.017±0.001	0.019±0.002 ^{bcd}
ABP	Br-X	1.02±0.10 ^{abc}	0.98±0.11 ^{abc}	0.019±0.002	0.020±0.001 ^{cde}
	Ng-X	1.03±0.09 ^{abc}	0.95±0.08 ^{ab}	0.018±0.001	0.019±0.002 ^{bcd}
	Si-X	0.91±0.10 ^a	0.90±0.04 ^a	0.017±0.002	0.018±0.002 ^{abc}
BP	Br-X	1.00±0.10 ^{abc}	1.00±0.11 ^{abc}	0.017±0.002	0.019±0.002 ^{bcd}
	Ng-X	1.01±0.13 ^{abc}	0.96±0.08 ^{ab}	0.017±0.002	0.018±0.001 ^{abc}
	Si-X	1.00±0.12 ^{abc}	0.99±0.09 ^{abc}	0.017±0.002	0.018±0.002 ^{abc}

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

Calpain II 1 h ~ average unit of Calpain II at 1 hour after slaughter, Calpain II 24 h ~ average unit of Calpain II at 24 hour after slaughter, Specific calpain II 1 h ~ average unit of specific calpain II at 1 hour after slaughter, Specific calpain II 24 h ~ average unit of specific calpain II at 24 hour after slaughter.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmental cross bred animals.

Effect of production system on the ratio between calpastatin and calpain I at 1 and 24 hours post mortem, as well as the ratio between calpastatin and calpain I + II at 1 and 24 hours post mortem is summarised in Table 4.47. There were differences between production systems for

calpastatin/calpain I + II ratio at 1 and 24 hours post mortem ($p < 0.05$), and there were differences between production systems for calpastatin/calpain I ratio at 1 hour post mortem ($p < 0.05$). There were no differences for the ratio between calpastatin and calpain I at 1 hour post mortem between muscles from the ABF (1.82), ABP (2.03) and BP production systems (1.84) ($p > 0.05$). There were no differences for the ratio between calpastatin and calpain I at 1 hour post mortem between the muscles from the AP (2.22), AF (2.09) and ABP production systems (2.03) ($p > 0.05$).

Table 4.47 Effect of production system on the ratio between calpastatin and calpain I, as well as the ratio between calpastatin and calpain I + II at 1 and 24 hours post mortem.

Production system (n=180)	Calpastatin/Calpain I 1 h ($\bar{X} \pm SD$)	Calpastatin/Calpain I 24 h ($\bar{X} \pm SD$)	Calpastatin/Calpain I+II 1 h ($\bar{X} \pm SD$)	Calpastatin/Calpain I+II 24 h ($\bar{X} \pm SD$)
AF	2.09±0.36 ^b	2.73±0.63	1.08±0.12 ^c	1.17±0.19 ^b
ABF	1.82±0.34 ^a	2.31±0.62	0.98±0.15 ^{ab}	0.99±0.17 ^a
AP	2.22±0.48 ^b	2.36±0.45	1.14±0.18 ^c	1.01±0.15 ^a
ABP	2.03±0.30 ^{ab}	2.65±0.81	1.07±0.13 ^{bc}	1.00±0.18 ^a
BP	1.84±0.32 ^a	2.38±0.89	0.96±0.14 ^a	0.90±0.18 ^a
Total	1.99±0.39	2.48±0.72	1.04±0.16	1.01±0.19

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

Calpastatin/Calpain I 1 h ~ average ratio of calpastatin/calpain I at 1 hour after slaughter, Calpastatin/Calpain I 24 h ~ average ratio of calpastatin/calpain I at 24 hour after slaughter, Calpastatin/Calpain I+II 1 h ~ average ratio of calpastatin/calpain I+II at 1 hour after slaughter, Calpastatin/Calpain I+II 24 h ~ average ratio of calpastatin/calpain I+II at 24 hour after slaughter.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

There were no differences for the ratio between calpastatin and calpain I + II at 1 hour post mortem between the muscles from the ABF (0.98) and BP production systems (0.96) ($p > 0.05$). There were no differences for the ratio between calpastatin and calpain I + II at 1 hour post mortem between the muscles from the ABF (0.98) and ABP production systems (1.07) ($p > 0.05$). There were no differences for the ratio between calpastatin and calpain I+II at 1 hour post mortem between the muscles from the AF (1.08), AP (1.14) and ABP production systems (1.07) ($p > 0.05$). Muscles from the AF production system (1.17) had a higher ratio between calpastatin and calpain I + II at 24 hours post mortem than the muscles from the AP (1.01), ABP (1.00), ABF (0.99) and BP production systems (0.90) ($p < 0.05$). There were no differences for the ratio between calpastatin and calpain I + II at 24 hours post mortem between the muscles from the AP (1.01), ABP (1.00), BP (0.90) and ABF production systems (0.99).

Muscles from the ABF production system (1.82 and 2.31 respectively) had the tendency for a lower ratio between calpastatin/calpain I at 1 and 24 hours post mortem than the other production systems (Table 4.47) and the muscles from the AF production system (2.09 and 2.73 respectively) had the tendency for higher calpastatin/calpain I ratio at 1 and 24 hours post mortem than the other production systems. Numerically, muscles from the BP production system (0.96 and 0.90 respectively) had the lowest calpastatin, calpain I + II ratio at 1 and 24 hours post mortem and muscles from the AP production system (1.14 and 1.01 respectively) had the highest calpastatin, calpain I + II ratio at 1 and 24 hours post mortem.

When only considering the effect of feeding system, there were differences between the feeding systems for the ratio between calpastatin and calpain I and the ratio between calpastatin and calpain I + II at 1 hour post mortem ($p < 0.05$). Muscles from pasture fed animals (2.02 ± 0.40) showed a higher ratio between calpastatin and calpain I than the muscles from the feedlot fed animals (0.95 ± 0.37). Muscles from pasture fed animals (1.05 ± 0.16) showed a slightly higher ratio between calpastatin and calpain I + II than the muscles from feedlot fed animals (1.03 ± 0.14).

There were differences between feeding systems for the ratio between calpastatin and calpain I and II at 24 hours post mortem ($p < 0.05$). Muscles for pasture fed animals (0.97 ± 0.18) had a lower ratio than the muscles from feedlot fed animals (1.08 ± 0.20). There was no difference between the feeding systems for the ratio between calpastatin and calpain I at 24 hours post mortem ($p > 0.05$). Muscles for pasture fed animals (2.46 ± 0.76) had a lower ratio than muscles from feedlot fed animals (2.52 ± 0.65).

When only the effect of age is considered, there were differences in the calpains and calpastatin activity ($p < 0.05$). Muscles from A age classification group (2.15 ± 0.42) had higher calpastatin/calpain I at 1 hour ($p < 0.05$), than muscles from AB (1.92 ± 0.34) and B age classification groups (1.84 ± 0.32). Muscles from B age classification group (2.38 ± 0.89) had a lower calpastatin/calpain I ratio, at 24 hours, than muscles from A age classification group (2.55 ± 0.58) (there was no difference between A (2.55 ± 0.58) and AB (2.48 ± 0.74), AB (2.48 ± 0.74) and B (2.38 ± 0.89) ($p > 0.05$)). Muscles from the B age classification group (0.90 ± 0.18) had the lowest calpastatin/calpain I + II, at 24 hours, muscles from AB age classification group (1.00 ± 0.17) had higher levels and muscles from A age classification group (1.09 ± 0.19) had the highest levels. All the differences that occurred were numerically small.

Strydom (2008) notes that Nguni cross bred animals had a lower ratio calpastatin and calpain I than Simmental cross bred animals. Effect of breed on calpastatin/calpain I and calpastatin/calpain I + II at 1 and 24 hours post mortem are summarised in Table 4.48. Considering the effect of breed only; there were differences between breeds for the calpastatin, calpain ratios ($p < 0.05$). Muscles from Brahman cross bred animals had a higher calpastatin/calpain I ratio as well as for calpastatin/calpain I + II ratio at 1 and 24 hours post mortem than muscles from Simmental

and muscles from Nguni cross bred animals (Table 4.48). These differences between the breeds can be explained by differences in pH, energy metabolites and genetics.

There was no difference between muscles from Nguni and Simmental cross bred animals for calpastatin, calpain I ratio at 24 hours ($p>0.05$). Nguni cross bred animals had numerically higher ratios than Simmental cross bred animals. There was no difference between Brahman and Nguni cross bred animals ($p>0.05$). Brahman cross bred animals had a numerically higher ratio than Nguni cross bed animals (Table 4.48).

Table 4.48 Effect of breed on Calpastatin/Calpain I and Calpastatin/Calpain I+II at 1 and 24 hours post mortem.

Breed (n=180)	Calpastatin/Calpain I 1 h ($\bar{X}\pm SD$)	Calpastatin/Calpain I 24 h ($\bar{X}\pm SD$)	Calpastatin/Calpain I+II 1 h ($\bar{X}\pm SD$)	Calpastatin/Calpain I+II 24 h ($\bar{X}\pm SD$)
Br-X	2.23±0.40 ^b	2.67±0.79 ^b	1.12±0.15 ^b	1.08±0.21 ^b
Ng-X	1.88±0.32 ^a	2.49±0.74 ^{ab}	1.00±0.13 ^a	0.98±0.19 ^a
Si-X	1.86±0.34 ^a	2.29±0.57 ^a	1.00±0.16 ^a	0.97±0.16 ^a

^{abcd} Means in a column with different superscripts differ significantly ($p<0.05$) based on the Fishers' means separation test.

$\bar{X}\pm SD$ ~ mean ± standard deviation.

Calpastatin/Calpain I 1 h ~ average ratio of calpastatin/calpain I at 1 hour after slaughter, Calpastatin/Calpain I+II 1 h ~ average ratio of calpastatin/calpain I+II at 1 hour after slaughter, Calpastatin/Calpain I 24 h ~ average ratio of calpastatin/calpain I at 24 hour after slaughter, Calpastatin/Calpain I+II 24 h ~ average ratio of calpastatin/calpain I+II at 24 hour after slaughter.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmental cross bred animals.

Effect of the interaction between breed and production system on calpastatin and calpain ratios are summarised in Table 4.49. There were effects from the interactions between production system and breed that influenced the ratio of calpastatin and calpain I and ratio of calpastatin and calpain I + II at 1 hour post mortem and the ratio between calpastatin and calpain I + II at 24 hours post mortem ($p<0.05$) but not for the ratio between calpastatins and calpain I at 24 hours post mortem ($p>0.05$). The muscles of the Nguni animals from the ABF production system (1.61 and 2.11 respectively) had the lowest ratio of calpastatin and calpain I at 1 and 24 hours post mortem, Brahman animals from the AP production system (2.60) had the highest ratio of calpastatin and calpain I at 1 hour post mortem, and Nguni animals from the AF production system (2.97) and Brahman animals from the ABP production system (2.97) at 24 hours post mortem.

Table 4.49 Effect of the interaction between breed and production system on calpastatin and calpain ratios

Production system (n=180)	Breed	Calpastatin/Calpain I 1 h ($\bar{X} \pm SD$)	Calpastatin/Calpain I 24 h ($\bar{X} \pm SD$)	Calpastatin/Calpain I+II 1 h ($\bar{X} \pm SD$)	Calpastatin/Calpain I+II 24 h ($\bar{X} \pm SD$)
AF	Br-X	2.28±0.44 ^{cd}	2.92±0.56	1.12±0.14 ^{bc}	1.27±0.16 ^c
	Ng-X	2.03±0.19 ^{abcd}	2.97±0.76	1.07±0.17 ^{abc}	1.21±0.23 ^{bc}
	Si-X	1.96±0.32 ^{abcd}	2.32±0.29	1.05±0.12 ^{ab}	1.04±0.007 ^{abc}
ABF	Br-X	2.14±0.29 ^{cde}	2.66±0.76	1.11±0.14 ^{bc}	1.09±0.15 ^{abc}
	Ng-X	1.61±0.15 ^a	2.11±0.34	0.90±0.08 ^a	0.90±0.12 ^a
	Si-X	1.74±0.34 ^{abc}	2.21±0.66	0.95±0.15 ^{ab}	1.01±0.19 ^{ab}
AP	Br-X	2.60±0.51 ^e	2.59±0.49	1.25±0.19 ^c	1.07±0.16 ^{abc}
	Ng-X	2.00±0.44 ^{abcd}	2.24±0.38	1.07±0.08 ^{abc}	0.98±0.15 ^{ab}
	Si-X	2.05±0.25 ^{bcd}	2.25±0.41	1.10±0.12 ^{bc}	0.97±0.12 ^{ab}
ABP	Br-X	2.14±0.19 ^{cde}	2.97±0.57	1.10±0.09 ^{bc}	1.11±0.20 ^{abc}
	Ng-X	2.06±0.29 ^{bcd}	2.69±1.09	1.06±0.12 ^{ab}	0.97±0.18 ^a
	Si-X	1.89±0.36 ^{abcd}	2.31±0.43	1.05±0.18 ^{ab}	0.96±0.13 ^a
BP	Br-X	2.06±0.28 ^{bcd}	2.35±1.13	1.05±0.11 ^{ab}	0.93±0.21 ^a
	Ng-X	1.77±0.23 ^{abc}	2.47±0.57	0.95±0.10 ^{ab}	0.90±0.12 ^a
	Si-X	1.65±0.29 ^{ab}	2.34±0.91	0.88±0.15 ^a	0.87±0.22 ^a

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

Calpastatin/Calpain I 1 h ~ average ratio of calpastatin/calpain I at 1 hour after slaughter, Calpastatin/Calpain I 24 h ~ average ratio of calpastatin/calpain I at 24 hour after slaughter, Calpastatin/Calpain I+II 1 h ~ average ratio of calpastatin/calpain I+II at 1 hour after slaughter, Calpastatin/Calpain I+II 24 h ~ average ratio of calpastatin/calpain I+II at 24 hour after slaughter.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmental cross bred animals.

Muscles of the Simmental animals from the BP production system had the lowest ratio of calpastatin and calpain I + II at 1 and 24 hours post mortem (0.88 and 0.87 respectively), Brahman animals from the AP production system (1.25) had the highest ratio of calpastatin and calpain I + II at 1 hour post mortem and Brahman animals from the AF production system (1.27) at 24 hours post mortem ($p < 0.05$).

4.8 Effect of production system and breed on the collagen content and solubility

Effect of production system on soluble and insoluble collagen, total collagen and collagen solubility is summarised in Table 4.50. There were differences between production systems for soluble and insoluble collagen and total collagen content ($p < 0.05$). The soluble collagen content in the muscles ranges from 0.18 mg/g (BP production system) to 0.26 mg/g (AP production system). Muscle insoluble collagen content ranged from 1.25 mg/g (AF production system) to 1.90 mg/g (AP production system). Muscles from the AF production system (1.44) had the lowest total collagen content compared to ABF (1.79), AP (2.16), ABP (2.02) and BP (1.92) production systems. Muscles from the AP production system (2.16) had the highest total collagen compared to AF (1.44), ABF (1.79), AP (2.16) and ABP (2.02). The collagen solubility of the BP production system (9.19 %) was the lowest and the solubility from the AF production system (13.40 %) was the highest.

Table 4.50 Effect of production system on soluble and insoluble collagen, total collagen and collagen solubility

Production system (n=180)	Soluble collagen (mg/g) ($\bar{X} \pm SD$)	Insoluble collagen (mg/g) ($\bar{X} \pm SD$)	Total collagen ($\bar{X} \pm SD$)	Collagen solubility (%) ($\bar{X} \pm SD$)
AF	0.19 \pm 0.046 ^a	1.25 \pm 0.213 ^a	1.44 \pm 0.254 ^a	13.40 \pm 1.386 ^c
ABF	0.23 \pm 0.130 ^{ab}	1.57 \pm 0.292 ^b	1.79 \pm 0.395 ^b	12.13 \pm 4.003 ^{bc}
AP	0.26 \pm 0.088 ^b	1.90 \pm 0.489 ^c	2.16 \pm 0.551 ^c	11.93 \pm 2.621 ^b
ABP	0.22 \pm 0.065 ^{ab}	1.81 \pm 0.386 ^c	2.02 \pm 0.430 ^c	10.70 \pm 2.272 ^{ab}
BP	0.18 \pm 0.060 ^a	1.74 \pm 0.388 ^b	1.92 \pm 0.462 ^{bc}	9.19 \pm 1.778 ^a
Total	0.21 \pm 0.086	1.65 \pm 0.426	1.87 \pm 0.481	11.39 \pm 2.920

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

Soluble collagen ~ average soluble collagen measured, insoluble collagen ~ average insoluble collagen measured, Total collagen ~ average total collagen content, Collagen solubility ~ average collagen solubility.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

When only the effect of feeding system is considered, feeding system had an effect on collagen characteristics ($p < 0.05$). Muscles from pasture fed animals (0.21 \pm 0.078 mg/g and 10.50 \pm 2.474 % respectively) showed lower levels of soluble collagen and collagen solubility and

muscles from feedlot fed animals (1.41 ± 0.300 mg/g and 1.62 ± 0.374 respectively) showing lower levels of insoluble collagen and total collagen content.

When only the effect of age is considered, there were differences for collagen characteristics ($p < 0.05$). Muscles from animals in the B age classification group (9.19 ± 1.778 %) had the lowest collagen solubility, muscles from AB age classification group (11.42 ± 3.311 %) had a higher solubility and muscles from A age classification group (12.69 ± 2.192 %) had the highest solubility.

Muscles from animals in the B age classification group (0.18 ± 0.060 mg/g) had lower ($p < 0.05$) soluble collagen than muscles from AB (0.22 ± 0.102 mg/g) and A age classification groups (0.23 ± 0.076 mg/g). Muscles from cattle in the A age classification group (1.56 ± 0.494 mg/g) had significantly lower insoluble collagen than muscles from B age classification group (1.74 ± 0.388 mg/g). There was no difference ($p > 0.05$) between muscles from cattle in the AB age classification group (1.69 ± 0.361 mg/g) and muscles from B age classification group (1.74 ± 0.388 mg/g), muscles from AB age classification group (1.69 ± 0.361 mg/g) and muscles from A age classification group (1.56 ± 0.494 mg/g).

There was no difference between age classification groups for the total collagen content ($p > 0.05$), muscles from the A age classification group (1.79 ± 0.554) had the tendency to result in the lowest total collagen content and muscles from the B age classification group (1.92 ± 0.432) had the tendency for the highest total collagen content.

Effect of breeds on the soluble collagen content, insoluble collagen content, total collagen content and collagen solubility of the muscle is summarised in Table 4.51. Considering the effect of breed only, there were differences between the breeds for insoluble collagen and the total collagen content ($p < 0.05$), but no differences was observed for soluble collagen and collagen solubility ($p > 0.05$). Nguni cross bred animals (0.23 mg/g) had the tendency for higher soluble collagen content, and the muscles from Brahman (0.20 mg/g) as well as Simmental cross bred animals (0.20 mg/g) had the tendency for lower soluble collagen.

There were no differences ($p > 0.05$) for insoluble collagen between muscles from Brahman (1.55 mg/g) and Simmental cross bred animals (1.62 mg/g), and between muscles from Simmental (1.62 mg/g) and Nguni cross bred animals (1.77 mg/g). Muscles of Nguni cross bred animals (1.77 mg/g) had the tendency for higher insoluble collagen content and muscles from Brahman cross bred animals (1.55 mg/g) had the tendency for lower insoluble collagen content.

Muscles from Brahman cross bred animals (1.76) had no difference ($p > 0.05$) with Simmental cross bred animals (1.82) for the total collagen content. Nguni cross bred animals (2.01) had a higher total collagen than the other breeds. Numerically, muscles of Simmental cross bred animals (10.95 %) had the lowest collagen solubility, Nguni cross bred animals (11.58 %) had a higher collagen solubility and Brahman cross bred animals (11.62 %) had the highest collagen solubility. Strydom and Frylinck (2005) found no significant differences between the breeds for collagen

solubility, with Nguni cross bred animals tending to have the highest solubility compared to Brahman and Simmental cross bred animals. Simmental cross bred animals had the tendency for lower solubility. According to Frylinck and Heinze (2003), no significant difference exists between Nguni, Brahman and Simmental animals for total collagen content, soluble collagen content and collagen solubility. Simmental animals had a higher insoluble collagen ($p < 0.05$) content than Nguni and Brahman animals.

Table 4.51 Effect of breeds on the soluble collagen content, insoluble collagen content, total collagen content and collagen solubility of the muscle

Breed (n=180)	Soluble collagen (mg/g) ($\bar{X} \pm SD$)	Insoluble collagen (mg/g) ($\bar{X} \pm SD$)	Total collagen ($\bar{X} \pm SD$)	Collagen solubility (%) ($\bar{X} \pm SD$)
Br-X	0.20 \pm 0.072	1.55 \pm 0.402 ^a	1.76 \pm 0.449 ^a	11.62 \pm 2.885
Ng-X	0.23 \pm 0.104	1.77 \pm 0.442 ^b	2.01 \pm 0.504 ^b	11.58 \pm 3.142
Si-X	0.20 \pm 0.075	1.62 \pm 0.408 ^{ab}	1.82 \pm 0.459 ^a	10.95 \pm 2.702

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

Soluble collagen ~ average soluble collagen measured, insoluble collagen ~ average insoluble collagen measured, Total collagen ~ average total collagen measured, Collagen solubility ~ average collagen solubility.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmental cross bred animals.

Effect of the interaction between breed and production system on collagen content and solubility are summarised in Table 4.52. There were effects from the interactions between breed and production system for soluble and insoluble collagen, the total collagen content and collagen solubility ($p < 0.05$).

Numerically there were relatively small effects from the interactions between breed and production system on soluble collagen; Simmental animals from the AB production system (0.15 mg/g) had the lowest content and Brahman animals from the AP production system (0.27 mg/g) and Nguni animals from the ABF production system (0.27 mg/g) had the highest content. Numerically, Brahman animals from the AF production system (1.19 mg/g) had the lowest insoluble collagen content and Nguni animals from the AP production system (20.90 mg/g) had the highest content. Numerically, Brahman animals from the AF production system (1.37 mg/g) had the lowest total collagen content and Nguni animals from the AP production system (2.35 mg/g) had the highest content. Numerically, Simmental animals from the BP production system (8.79 mg/g) had the lowest collagen solubility and Nguni animals from the AF production system (14.05 mg/g) had the highest solubility.

Table 4.52 Effect of the interaction between breed and production system on soluble collagen content, insoluble collagen content, total collagen content and collagen solubility of the muscle

Production system (n=180)	Breed	Soluble collagen (mg/g)($\bar{X} \pm SD$)	Insoluble collagen (mg/g) ($\bar{X} \pm SD$)	Total collagen ($\bar{X} \pm SD$)	Collagen solubility (%) ($\bar{X} \pm SD$)
AF	Br-X	0.18±0.042 ^{ab}	1.19±0.229 ^a	1.37±0.266 ^a	12.90 ±1.389 ^{bcd}
	Ng-X	0.22±0.044 ^{ab}	1.34±0.201 ^{ab}	1.56±0.240 ^{abc}	14.05±1.218 ^d
	Si-X	0.19±0.044 ^{ab}	1.22±0.194 ^a	1.41±0.233 ^{ab}	13.30±1.398 ^{cd}
ABF	Br-X	0.21±0.074 ^{ab}	1.44±0.170 ^{abc}	1.65±0.204 ^{abc}	12.60±3.781 ^{abcd}
	Ng-X	0.27±0.175 ^b	1.68±0.329 ^{abcd}	1.95±0.480 ^{bcd}	13.28±4.525 ^{cd}
	Si-X	0.18±0.087 ^{ab}	1.55±0.312 ^{abcd}	1.73±0.380 ^{abcd}	10.09±2.882 ^{abc}
AP	Br-X	0.27±0.073 ^{ab}	1.75±0.466 ^{bcd}	2.02±0.525 ^{bcd}	13.32±2.150 ^{cd}
	Ng-X	0.26±0.098 ^{ab}	2.09±0.577 ^d	2.35±0.645 ^d	11.13±2.494 ^{abcd}
	Si-X	0.24±0.097 ^{ab}	1.87±0.412 ^{cd}	2.12±0.485 ^{cd}	11.33±2.789 ^{abcd}
ABP	Br-X	0.20±0.072 ^{ab}	1.65±0.335 ^{abcd}	1.85±0.380 ^{abcd}	10.55±3.002 ^{abcd}
	Ng-X	0.21±0.065 ^{ab}	1.84±0.460 ^{bcd}	2.05±0.515 ^{cd}	10.33±1.477 ^{abc}
	Si-X	0.24±0.057 ^{ab}	1.91±0.302 ^{cd}	2.15±0.319 ^{cd}	11.31±2.445 ^{abcd}
BP	Br-X	0.18±0.067 ^{ab}	1.72±0.435 ^{bcd}	1.90±0.487 ^{abcd}	9.35±1.696 ^a
	Ng-X	0.21±0.062 ^{ab}	1.93±0.259 ^d	2.14±0.298 ^{cd}	9.40±1.959 ^{ab}
	Si-X	0.15±0.039 ^a	1.57±0.379 ^{abcd}	1.72±0.406 ^{abc}	8.79±1.759 ^a

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

Soluble collagen ~ average soluble collagen measured, insoluble collagen ~ average insoluble collagen measured, Total collagen ~ average total collagen content, Collagen solubility ~ average collagen solubility.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmental cross bred animals.

4.9 Effect of production system and breed on the meat colour

It would seem that the glycogen in the muscle at slaughter may influence meat colour (Muir, Beaker and Brown, 1998; Mancini and Hunt, 2005). According to Strydom, Frylinck, *et al.*, (2005), electrical stimulation does not influence the resulting meat colour.

Effect of production system on meat colour is summarised in Table 4.53. There were significant differences between production systems for L*, a* and b* coordinates, hue angle and colour saturation. Fresh meat samples from the BP production system (35.31) had a darker colour ($p < 0.05$) than the meat from the ABF (38.15), AP (37.76), ABP (37.77) and AF production systems (39.00). A fresh meat sample from the AF production system (39.00) had the lightest colour. The difference between the production systems can be explained by differences in exercise, inter-muscular fat, pH, water holding capacity and drip loss (discussed later in dissertation).

The more exercise an animal gets the higher the proportion of red coloured fibers, therefore an animal from the pasture should have a higher a* coordinate value. The younger the animal, the higher the proportion of these red fibers in the muscle (Frandsen, *et al*, 2003).

Table 4.53 Effect of production system on meat colour of a fresh meat sample

Production system (n=180)	Meat L* ($\bar{X} \pm SD$)	Meat a* ($\bar{X} \pm SD$)	Meat b* ($\bar{X} \pm SD$)	Hue angle ($\bar{X} \pm SD$)	Chroma ($\bar{X} \pm SD$)
AF	39.00 \pm 1.68 ^b	15.93 \pm 1.37 ^b	7.25 \pm 0.82 ^b	2.05 \pm 0.16 ^a	17.50 \pm 1.54 ^b
ABF	38.15 \pm 2.10 ^b	14.39 \pm 1.38 ^a	6.32 \pm 0.91 ^a	2.15 \pm 0.20 ^{ab}	15.72 \pm 1.58 ^a
AP	37.76 \pm 2.54 ^b	14.95 \pm 1.35 ^{ab}	6.41 \pm 0.69 ^a	2.20 \pm 0.15 ^b	16.27 \pm 1.47 ^a
ABP	37.77 \pm 2.46 ^b	14.98 \pm 1.42 ^{ab}	6.41 \pm 0.67 ^a	2.20 \pm 0.18 ^b	16.30 \pm 1.51 ^a
BP	35.31 \pm 2.46 ^a	15.36 \pm 1.92 ^{ab}	6.59 \pm 1.13 ^a	2.21 \pm 0.17 ^b	16.72 \pm 2.19 ^{ab}
Total	37.52 \pm 2.59	15.13 \pm 1.59	6.60 \pm 0.93	2.16 \pm 0.18	16.51 \pm 1.78

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

Meat L* ~ average lightness of a fresh meat sample, Meat a* ~ average a* coordinate value for a fresh meat sample, Meat b* ~ average b* coordinate value for a fresh meat sample, Hue angle ~ average hue angle of the fresh meat sample, Chroma ~ average chroma of the fresh meat sample.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

The a* coordinate ranges from 14.39 (ABF production system) to 15.93 (AF production system). The b* coordinate ranges from 6.32 (ABF production system) to 7.25 (AF production system). Meat samples from the AF production system (2.05) had the lowest hue angle and meat

samples from the BP production system (2.21) had the highest hue angle ($p < 0.05$). Colour saturation from the ABF production system (15.72) was the lowest and colour saturation from the AF production system (17.50) was the highest ($p < 0.05$).

Considering the effect of feeding system only, there were differences between feeding systems for the L^* and b^* coordinate value as well as hue angle ($p < 0.05$). There was no difference ($p > 0.05$) for the a^* coordinate value and colour saturation between the feeding systems. Meat from feedlot fed animals (38.57 ± 1.94 , 6.78 ± 0.98 and 2.10 ± 0.18 respectively) had a lower L^* coordinate value, higher b^* coordinate value and lower hue angle than meat from pasture fed animals (38.84 ± 2.73 , 6.48 ± 0.87 and 2.20 ± 0.17 respectively). Even though there was no difference ($p > 0.05$) in the value of the a^* coordinate, meat from pasture fed animals (15.11 ± 1.61) had a tendency for lower a^* value than meat from feedlot fed animals (15.16 ± 1.57). Meat from pasture fed animals (16.45 ± 1.78) had a lower chroma than meat from feedlot fed animals (16.61 ± 1.79). According to French, O'Riordan, Monahan, Caffrey, Mooney, Troy and Moloney (2001) the diet the animals were fed have no effect on the resulting meat colour. Bruce, Stark, *et al.* (2003) reported that the diet affected the resulting L^* , a^* and b^* coordinates. Feedlot fed animal's meat had a higher L^* value, higher a^* value and higher b^* value than the meat from the pasture fed animals. Farouk and Wieliczko (2003) reported no significant difference between the feeding systems for L^* and b^* coordinates. They reported that feedlot fed animal's meat had a higher a^* value, lower spectral colour and higher colour saturation than the pasture fed animals.

When only considering the effect of age; there were differences between the age classification groups for meat colour ($p < 0.05$). Meat samples from the B age classification group (35.31 ± 2.46) had lighter ($p < 0.05$) meat than meat samples from the A (38.40 ± 2.22) and AB age classification group (37.96 ± 2.28). Meat samples from the A age classification group (15.45 ± 1.44 and 6.84 ± 0.86 respectively) had a higher a^* and b^* coordinates value than meat samples from the AB age classification group (14.68 ± 1.42 and 6.36 ± 0.79 respectively) (there were no difference ($p > 0.05$) between A (15.45 ± 1.44 and 6.84 ± 0.86 respectively) and B (15.36 ± 1.92 and 6.59 ± 1.13 respectively), B (15.36 ± 1.92 and 6.59 ± 1.13 respectively) and AB (14.68 ± 1.42 and 6.36 ± 0.79 respectively)).

Meat samples from the B age classification group (2.21 ± 0.17) had higher ($p < 0.05$) hue angle than meat samples from the A age classification group (2.12 ± 0.17) (there were no difference ($p < 0.05$) between AB (2.17 ± 0.19) and A (2.12 ± 0.17), AB (2.17 ± 0.19) and B (2.21 ± 0.17)) and meat samples from the AB age classification group (16.01 ± 1.56) had lower ($p < 0.05$) chroma value than meat samples from the A age classification group (16.90 ± 1.62) (there were no difference ($p > 0.05$) between B (16.72 ± 2.19) and AB (16.01 ± 1.56), B (16.72 ± 2.19) and A (16.90 ± 1.62)). According to Boles and Swan (2002) B age classification group animals have significantly greener meat than the AB age classification group animals.

Effect of breed on meat colour post mortem is summarised in Table 4.54. Considering the effect of breed only, breeds only had effect on hue angle ($p < 0.05$). Meat samples from Nguni cross bred animals (2.22) had a higher ($p < 0.05$) hue angle than meat samples from Brahman (2.13) and Simmental cross bred animals (2.14). Meat samples from Nguni cross bred animals (14.91 and 6.38 respectively) had a lower a^* value and lower b^* value than that of the other breeds (Brahman (15.23 and 6.71 respectively) and Simmental cross bred animals (15.27 and 6.72 respectively)). Insausti, Beriain, Purroy, Alberti, Lizaso and Hernandez (1999) reported that the breed does play a role in the resulting meat colour. Blanco, Villalba, *et al.* (2009) reported that the breed does not affect the meat lightness but effect the hue angle.

Table 4.54 Effect of breed on meat colour of fresh meat samples

Breed (n=180)	Meat L* ($\bar{X} \pm SD$)	Meat a* ($\bar{X} \pm SD$)	Meat b* ($\bar{X} \pm SD$)	Hue angle ($\bar{X} \pm SD$)	Chroma ($\bar{X} \pm SD$)
Br-X	37.97±2.34	15.23±1.59	6.71±0.92	2.13±0.16 ^a	16.64±1.80
Ng-X	37.23±2.51	14.91±1.45	6.38±0.93	2.22±0.19 ^b	16.23±1.66
Si-X	37.38±2.88	15.27±1.73	6.72±0.90	2.14±0.18 ^a	16.69±1.89

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

Meat L* ~ average lightness of a fresh meat sample, Meat a* ~ average a^* coordinate value for a fresh meat sample, Meat b* ~ average b^* coordinate value for a fresh meat sample, Hue angle ~ average hue angle of the fresh meat sample, Chroma ~ average chroma of the fresh meat sample.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmental cross bred animals.

Effect of the interaction between breed and production system on meat colour is summarised in Table 4.55. The interaction between production system and breed had an effect on meat colour (L^* , a^* and b^*) ($p < 0.05$). There were no significant effect of the interaction on hue angle and chroma. Simmental animals from the BP production system (34.77) had the darkest meat colour, Brahman animals from the AF production system (39.74) had the lightest meat, Simmental animals from the ABF production system (13.95) had the lowest a^* coordinate value, Simmental animals from the AF production system (16.38) had the highest a^* coordinate value, Nguni animals from the ABF production system (6.11) had the lowest b^* coordinate value and Simmental animals from the AF production system (7.45) had the highest b^* value. Numerically, Brahman and Simmental animals from the AF production system (2.05) had the lowest hue angle, Nguni animals from the AP production system (2.26) had the highest hue angle, Simmental animals from the ABF production system (15.32) had the lowest colour saturation and Simmental animals from the AF production system (18.00) had the highest colour saturation.

Table 4.55 Effect of the interaction between breed and production system on meat colour

Production system	Breed	Meat L* ($\bar{X} \pm SD$)	Meat a* ($\bar{X} \pm SD$)	Meat b* ($\bar{X} \pm SD$)	Hue angle ($\bar{X} \pm SD$)	Chroma ($\bar{X} \pm SD$)
AF	Br-X	39.74±1.16 ^d	15.82±1.75 ^{ab}	7.20±0.95 ^{ab}	2.05±0.15	17.38±1.95
	Ng-X	39.13±1.97 ^d	15.55±1.12 ^{ab}	7.08±0.79 ^{ab}	2.06±0.16	17.09±1.31
	Si-X	38.14±1.57 ^{bcd}	16.38±1.12 ^b	7.45±0.73 ^b	2.05±0.17	18.00±1.24
ABF	Br-X	38.18±1.87 ^{bcd}	14.87±1.54 ^{ab}	6.59±1.04 ^{ab}	2.13±0.18	16.27±1.81
	Ng-X	37.58±1.41 ^{abcd}	14.33±1.19 ^{ab}	6.11±0.88 ^a	2.22±0.20	15.59±1.40
	Si-X	38.93±2.95 ^{cd}	13.95±1.40 ^a	6.31±0.81 ^{ab}	2.07±0.19	15.32±1.54
AP	Br-X	37.86±1.99 ^{abcd}	15.00±1.18 ^{ab}	6.47±0.46 ^{ab}	2.18±0.16	16.35±1.20
	Ng-X	37.16±2.91 ^{abcd}	14.88±1.63 ^{ab}	6.24±0.87 ^{ab}	2.26±0.16	16.14±1.82
	Si-X	38.16±2.78 ^{bcd}	14.96±1.35 ^{ab}	6.50±0.72 ^{ab}	2.16±0.14	16.31±1.49
ABP	Br-X	38.98±2.03 ^{cd}	14.51±0.91 ^{ab}	6.28±0.49 ^{ab}	2.19±0.14	15.81±0.97
	Ng-X	37.28±2.08 ^{abcd}	14.94±1.51 ^{ab}	6.29±0.75 ^{ab}	2.24±0.15	16.21±1.65
	Si-X	37.29±3.04 ^{abcd}	15.46±1.64 ^{ab}	6.68±0.67 ^{ab}	2.18±0.23	16.85±1.67
BP	Br-X	35.82±2.30 ^{abc}	15.66±1.97 ^{ab}	6.88±1.15 ^{ab}	2.14±0.15	17.11±2.24
	Ng-X	35.27±2.82 ^{ab}	15.00±1.67 ^{ab}	6.26±1.12 ^{ab}	2.28±0.20	16.26±1.94
	Si-X	34.77±2.32 ^a	15.39±2.19 ^{ab}	6.59±1.13 ^{ab}	2.20±0.14	16.74±2.44

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

Meat L* ~ average lightness of a fresh meat sample, Meat a* ~ average a* coordinate value for a fresh meat sample, Meat b* ~ average b* coordinate value for a fresh meat sample, Hue angle ~ average hue angle of the fresh meat sample, Chroma ~ average chroma of the fresh meat sample.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmental cross bred animals.

4.10 Effect of production system on the shear force values at 1, 7 and 14 days post mortem

When the shear force is measured on raw meat, the value reflects the effect of collagen, while shear force measured on cooked meat reflect the effect of myofibrillar fragment length (Torrenscano, Sanchez-Escalante, Gimenez, Roncales and Beltran, 2003).

Hope-Jones, Strydom, *et al.* (2010) reported that animals that received a growth promoter had less tender meat than the animals that did not receive a growth promoter. Effect of production system on shear force values at 1, 7 and 14 days post mortem are summarised in Table 4.56. There were differences between production systems for shear force values at 1, 7 and 14 days

post mortem ($p < 0.05$). Shear force value of meat from the ABF production system was the lowest at 1, 7 and 14 days post mortem while meat from the AP production system was the highest at 1, 7 and 14 days post mortem.

Table 4.56 Effect of production system on shear force values at 1, 7 and 14 days post mortem.

Production system (n=180)	WBSF ₁ (kg) ($\bar{X} \pm SD$)	WBSF ₇ (kg) ($\bar{X} \pm SD$)	WBSF ₁₄ (kg) ($\bar{X} \pm SD$)
AF	6.70 ± 1.40 ^{ab}	5.29 ± 1.12 ^{ab}	4.19 ± 0.86 ^a
ABF	5.97 ± 0.90 ^a	4.82 ± 1.00 ^a	3.85 ± 0.70 ^a
AP	7.45 ± 1.60 ^b	6.04 ± 2.00 ^b	5.19 ± 1.94 ^b
ABP	6.64 ± 1.34 ^{ab}	5.51 ± 1.37 ^{ab}	4.56 ± 0.93 ^{ab}
BP	6.60 ± 1.91 ^{ab}	5.26 ± 1.55 ^{ab}	3.97 ± 0.81 ^a
Total	6.66 ± 1.54	5.37 ± 1.48	4.33 ± 1.20

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean ± standard deviation.

WBSF₁ ~ average shear force value at 1 day after slaughter, WBSF₇ ~ average shear force value 7 days after slaughter, WBSF₁₄ ~ average shear force value 14 days after slaughter.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared on the pasture until AB age classification, BP ~ average for the animals reared on the pasture until B age classification.

When only the effect of feeding system is considered, meat from feedlot fed animals (6.34 ± 1.23 kg, 5.06 ± 1.08 kg and 4.02 ± 0.79 kg respectively) showed lower shear force values at 1, 7 and 14 days post mortem, than meat from pasture fed animals (6.34 ± 1.23 kg, 5.06 ± 1.08 kg and 4.02 ± 0.79 kg respectively) ($p < 0.05$).

When only the effect of age is considered, there were differences ($p < 0.05$) between ages at 1 and 14 days post mortem for shear force. Meat from the A age classification group (7.07 ± 1.54 kg) had a higher shear force value, at day 1, than meat from the AB age classification group (6.30 ± 1.18 kg) ($p < 0.05$) (there was no difference ($p > 0.05$) between AB (6.30 ± 1.18 kg) and B (6.60 ± 1.91 kg), B (6.60 ± 1.91 kg) and A (7.07 ± 1.54 kg)). Meat from the A age classification group (4.67 ± 1.56 kg) had a higher shear force value, at day 14, than animals from the AB (4.21 ± 0.89 kg) and B age classification groups (3.97 ± 0.81 kg) ($p < 0.05$). At 14 days post mortem, the meat from the B age classification group (3.97 ± 0.81 kg) had a tendency for the lower shear force values. At 7 days post mortem there were no differences ($p > 0.05$) between age classification groups, animals from the AB age classification group (5.16 ± 1.24 kg) had the tendency to have the lowest shear force values and meat from the A age classification group (5.66 ± 1.64 kg) had the tendency to

have the highest shear force values. The meat from the B age classification group (5.26 ± 1.55 kg) had an intermediate shear force at 7 days post mortem.

Table 4.57 summarise effects of breed on shear force of meat sample. Considering the effect of breed only, there were differences ($p < 0.05$) between the breeds for WBSF on day 7. Brahman cross bred animals (5.74 kg) had less tender meat than Nguni (5.24 kg) and Simmental cross bred animals (5.14 kg) at 7 days post mortem. Strydom (2008) state that there are breed differences for Warner-Bratzler shear force (WBSF), where Brahman cross bred animals had less tender meat than Nguni cross bred animals, but the same as Simmental cross bred animals. Even if there were no differences ($p > 0.05$) at day 1 and 14 post mortem, there were numerical differences. Meat from Brahman cross bred animals (6.96 kg, 5.74 kg and 4.52 kg respectively) had the tendency for the highest shear force value at day 1, 7 and 14 post mortem. Meat from Nguni cross bred animals (6.46 kg) had the tendency for the lowest shear force value at day 1, meat from Simmental cross animals (5.14 kg and 4.15 kg respectively) had the lowest shear force value at day 7 and 14 post mortem. The shear force value decreased as time progressed, this is supported by Strydom (2008). Frylinck and Heinze (2003) reported that Nguni had constantly lower shear force values than those of Brahman and Simmental cross bred animals ($p < 0.05$).

Table 4.57 Effect of breed on shear force values of meat samples

Breed (n=180)	WBSF ₁ (kg) ($\bar{X} \pm SD$)	WBSF ₇ (kg) ($\bar{X} \pm SD$)	WBSF ₁₄ (kg) ($\bar{X} \pm SD$)
Br-X	6.96 \pm 1.22	5.74 \pm 1.43 ^b	4.52 \pm 1.00
Ng-X	6.46 \pm 1.62	5.24 \pm 1.55 ^a	4.32 \pm 1.59
Si-X	6.57 \pm 1.71	5.14 \pm 1.42 ^a	4.15 \pm 0.86

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

WBSF₁ ~ average shear force value at 1 day after slaughter, WBSF₇ ~ average shear force value 7 days after slaughter, WBSF₁₄ ~ average shear force value 14 days after slaughter.

Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmental cross bred animals.

Effect of the interaction between breed and production system on the shear force of meat samples at 1, 7 and 14 days post mortem is summarised in Table 4.58. There were no effects from the interactions between production system and breed on shear force values at 7 days post mortem ($p > 0.05$). There was an effect from the interaction between breed and production system on shear force at 1 and 14 days post mortem ($p < 0.05$). Meat of the Nguni animals from the BP production system (5.64 kg) had the tendency for a lower shear force value at 1 day post mortem and meat of the Nguni animals from the ABF production system (4.48 kg and 3.83 kg respectively) had the tendency for a lower shear force value at 7 and 14 days post mortem. Meat from the Nguni

animals from the AP production system (8.15 kg and 6.26 kg respectively) had a tendency for higher shear force values at 1 and 14 days post mortem and meat from the Brahman animals from the AP production system (6.39 kg) had a tendency for higher shear force value at 7 days post mortem.

Table 4.58 Effect of the interaction between breed and production system on the shear force of meat samples at 1, 7 and 14 days post mortem

Production system (n=180)	Breed	WBSF ₁ (kg) ($\bar{X} \pm SD$)	WBSF ₇ (kg) ($\bar{X} \pm SD$)	WBSF ₁₄ (kg) ($\bar{X} \pm SD$)
AF	Br-X	7.03±1.34 ^{ab}	5.98±1.04	4.60±0.69 ^{ab}
	Ng-X	7.10±1.22 ^{ab}	5.39±0.81	4.14±1.15 ^{ab}
	Si-X	6.01±1.45 ^{ab}	4.53±1.01	3.83±0.52 ^{ab}
ABF	Br-X	6.28±0.89 ^{ab}	5.42±1.30	4.08±0.87 ^{ab}
	Ng-X	5.70±0.87 ^{ab}	4.48±0.64	3.57±0.45 ^a
	Si-X	6.00±0.92 ^{ab}	4.64±0.81	4.00±0.69 ^{ab}
AP	Br-X	7.45±1.20 ^{ab}	6.39±1.65	5.19±1.22 ^{bc}
	Ng-X	8.14±2.38 ^b	6.31±3.07	6.26±2.94 ^c
	Si-X	6.88±0.87 ^{ab}	5.49±0.98	4.29±0.77 ^{ab}
ABP	Br-X	7.23±0.87 ^{ab}	5.78±1.09	4.98±0.87 ^{abc}
	Ng-X	6.27±1.41 ^{ab}	5.37±1.35	4.24±0.85 ^{ab}
	Si-X	6.58±1.52 ^{ab}	5.43±1.68	4.59±0.99 ^{ab}
BP	Br-X	6.88±1.45 ^{ab}	5.29±1.73	3.97±0.85 ^{ab}
	Ng-X	5.64±0.83 ^a	4.95±0.61	3.90±0.36 ^{ab}
	Si-X	7.25±2.73 ^{ab}	5.53±2.00	4.06±1.09 ^{ab}

^{abcd} Means in a column with different superscripts differ significantly ($p < 0.05$) based on the Fishers' means separation test.

$\bar{X} \pm SD$ ~ mean \pm standard deviation.

WBSF₁ ~ average shear force value at 1 day after slaughter, WBSF₇ ~ average shear force value 7 days after slaughter, WBSF₁₄ ~ average shear force value 14 days after slaughter.

AF ~ average for the animals reared at the feedlot until A age classification, ABF ~ average for the animals reared at the feedlot until AB age classification, AP ~ average for the animals reared on the pasture until A age classification, ABP ~ average for the animals reared
Br-X ~ breed average for Brahman cross bred animals, Ng-X ~ breed average for Nguni cross bred animals, Si-X ~ breed average for Simmental cross bred animals.

4.11 The effect of the production systems and breeds on the DFD phenomenon

The frequency of the DFD phenomenon (pH>5.8) detected among three breeds and five production systems are summarised in Table 4.59. Nguni cross bred animals had the highest percentage of DFD occurrences, at 10.94 % and Brahman cross bred animals the lowest percentage, at 5.00 %. The AP production system had the highest DFD occurrence, at 15.15 %. AF and ABF had the lowest percentage DFD, at 2.78 %. Nguni animals from the AP production system had the highest occurrence of DFD, at 40 %.

Table 4.59 Frequency of DFD phenomenon detected among the three breeds and five production systems

	Total population	Brahman cross breeds	Simmental cross breeds	Nguni cross breeds
Total	182	60	58	64
All production systems	7.69 % (14/182)	5.00 % (3/60)	6.90 % (4/58)	10.94 % (7/64)
AF	2.78 % (1/36)	0.00 % (0/12)	0.00 % (0/12)	8.33 % (1/12)
ABF	2.78 % (1/36)	0.00 % (0/12)	10.00 % (1/10)	0.00 % (0/14)
AP	15.15 % (5/33)	9.09 % (1/11)	0.00 % (0/12)	40 % (4/10)
ABP	5.71 % (2/35)	0.00 % (0/10)	9.09 % (1/11)	7.14 % (1/14)
BP	11.90 % (5/42)	13.33 % (2/15)	15.38 % (2/13)	7.14 % (1/14)

AF ~ A age classification animals fed at a feedlot, ABF ~ AB age classification animals fed at a feedlot, AP ~ A age classification animals fed on pasture, ABP ~ AB age classification animals fed on pasture, BP ~ B age classification animals fed on pasture

It is important to know how many cases of the DFD phenomenon occurred because it will influence the end results. DFD will increase the ultimate carcass pH, influence muscle energy metabolite concentration, increase water binding capacity, decrease drip loss, influence tenderness and cause a darker coloured meat. It will therefore influence the means and standard deviations. Fortunately a very small percentage of DFD cases were reported in this study. DFD carcasses are darker, have higher pH values and the tenderness variation is larger compared to normal carcasses meat (see par 2.6, Chap 2: literature study).

4.12 Correlation of energy metabolites, muscle pH and muscle temperature

Table 4.60 summarises the correlations between the energy metabolites in the muscle post mortem and muscle pH post mortem.

Table 4.60 Correlation-matrix showing simple correlation coefficients of how muscle energy metabolites correlated with muscle pH in the production systems evaluated

	pH1	pH2	pH3	pH4	pH24
Lakt1	-0.476*	-0.377*	-0.437*	-0.421*	-0.209*
Lakt2	-0.473*	-0.340*	-0.439*	-0.362*	-0.174*
Lakt4	-0.396*	-0.355*	-0.416*	-0.359*	-0.159*
Lakt24	-0.172*	-0.153*	-0.254*	-0.241*	-0.172*
Glu1	-0.409*	-0.170*	-0.346*	-0.312*	-0.258*
Glu2	-0.375*	-0.200*	-0.402*	-0.341*	-0.299*
Glu4	-0.461*	-0.238*	-0.472*	-0.435*	-0.351*
Glu24	-0.300*	-0.072	-0.290*	-0.284*	-0.306*
Glyc1	0.232*	0.229*	0.178*	0.138	-0.007
Glyc2	0.239*	0.219*	0.200*	0.159*	0.010
Glyc4	0.253*	0.211*	0.239*	0.204*	0.054
Glyc24	0.100	0.114	0.140	0.131	0.073
G6P1	-0.121	-0.060	-0.145	-0.156*	-0.157*
G6P2	-0.185*	-0.090	-0.179*	-0.179*	-0.134
G6P4	-0.233*	-0.141	-0.258*	-0.194*	-0.054
G6P24	-0.086	-0.099	-0.065	-0.046	-0.069
ATP1	0.137	0.184*	0.122	0.088	-0.014
ATP2	0.196*	0.216*	0.178*	0.090	0.033
ATP4	0.320*	0.272*	0.285*	0.210*	0.078
ATP24	0.134	0.168*	0.127	0.088	0.057
CP1	0.170*	0.098	0.249*	0.196*	0.100
CP2	0.147*	0.102	0.245*	0.232*	0.067
CP4	0.175*	0.158*	0.220*	0.205*	-0.021
CP24	0.049	0.125	0.116	0.073	0.075

*. Correlation is significant at the 0.05 level (2-tailed).

pH 1 ~ muscle pH at 1 hour post mortem, pH 2 ~ muscle pH at 2 hours post mortem, pH 3 ~ muscle pH at 3 hours post mortem, pH 4 ~ muscle pH at 4 hours post mortem, pH 24 ~ muscle pH at 24 hours post mortem, Lakt 1 ~ muscle lactic acid concentration at 1 hour post mortem, Lakt 2 ~ muscle lactic acid concentration at 2 hours post mortem, Lakt 4 ~ muscle lactic acid concentration at 4 hours post mortem, Lakt 24 ~ muscle lactic acid concentration at 24 hours post mortem, Glu 1 ~ muscle glucose concentration at 1 hour post mortem, Glu 2 ~ muscle glucose concentration at 2 hours post mortem, Glu 4 ~ muscle glucose concentration at 4 hours post mortem, Glu 24 ~ the muscle glucose concentration at 24 hours post mortem, Glyc 1 ~ muscle glycogen concentration at 1 hour post mortem, Glyc 2 ~ the muscle glycogen concentration at 2 hours post mortem, Glyc 4 ~ the muscle glycogen concentration at 4 hours post mortem, Glyc 24 ~ muscle glycogen concentration at 24 hours post mortem, G6P 1 ~ muscle glucose-6-phosphat concentration at 1 hour post mortem, G6P 2 ~ muscle glucose-6-phosphat concentration at 2 hours post mortem, G6P 4 ~ muscle glucose-6-phosphat concentration at 4 hours post mortem, G6P 24 ~ muscle glucose-6-phosphat at 24 hours post mortem, ATP 1 ~ muscle ATP concentration at 1 hour post mortem, ATP 2 ~ the muscle ATP concentration at 2 hours post mortem, ATP 4 ~ muscle ATP concentration at 4 hours post mortem, ATP 24 ~ muscle ATP concentration at 24 hours post mortem, CP 1 ~ muscle creatine phosphate concentration at 1 hour post mortem, CP 2 ~ muscle creatine phosphate concentration at 2 hours post mortem, CP 4 ~ muscle creatine phosphate concentration at 4 hours post mortem, CP 24 ~ muscle creatine phosphate concentration at 24 hours post mortem.

There was a negative correlation between muscle lactic acid concentrations and muscle pH ($p < 0.05$) (Table 4.60), therefore the higher the lactic acid concentration the lower the pH. Serra, Gil, *et al.* (2004) support the finding that lactic acid and pH have a negative correlation.

There was a negative correlation between muscle glucose concentrations and muscle pH ($p < 0.05$), therefore the higher the glucose concentration the lower the carcass pH. Muscle glycogen concentrations and muscle pH had a positive correlation ($p < 0.05$) (Table 4.60), therefore the higher the glycogen concentration the higher the muscle pH. Lahucky, Palanska, Mojto, Zaujec and Huba (1998) and Immonen, Ruusunen, *et al.* (2000) also concluded that there was a positive correlation between glycogen and pH, but according to Muchenje, Dzama, *et al.* (2009a) and Immonen, Ruusunen, *et al.* (2000) there was a negative correlation. This difference can be explained by the fact that the relationship between pH and glycogen is not linear but curvilinear (Immonen and Puolanne, 2000).

Muscle glucose-6-phosphate concentrations had a negative correlation with muscle pH ($p < 0.05$) (Table 4.60), therefore the higher the glucose-6-phosphate the lower the muscle pH. There was a positive correlation between muscle ATP concentrations and muscle pH ($p < 0.05$) (Table 4.60), therefore the lower the muscle ATP concentration the lower the pH. Dransfield (1994), Dransfield (1993) and Olsson, Hertzman, and Tornberg (1994) concluded that there is a positive correlation between ATP and pH.

Muscle creatine phosphate concentrations correlate positively with muscle pH ($p < 0.05$), therefore the lower the muscle creatine phosphate concentration the lower the pH. Sterten, Oksbjerg, Froystein, Ekker and Kjos (2010) describe a negative correlation between pH and glycolytic potential; this is supported by Wulf, Emmett, *et al.* (2002). It can be concluded that the energy status in the muscle will influence the carcass pH.

The correlation between the energy metabolites and the muscle temperature is summarised in Table 4.61. ATP, creatine phosphate and glucose did not correlate with muscle temperature post mortem ($p > 0.05$) (Table 4.61). Muscle lactic acid concentrations had a weak positive correlation with muscle temperature ($p < 0.05$), therefore the higher the muscle lactic acid concentration the higher the muscle temperature. Muscle glycogen concentrations had a weak negative correlation with muscle temperature ($p < 0.05$) (Table 4.61), therefore the lower the muscle glycogen concentration the higher the muscle temperature.

Only a weak negative correlation between muscle ATP concentrations at 1 and 2 hours post mortem with muscle temperature at 3, 4 and 24 hours post mortem was detected ($p < 0.05$) (Table 4.61), therefore the lower the muscle ATP concentration the higher the muscle temperature.

Glycolytic potential had a weak negative correlation with muscle temperature at 4 and 24 hours post mortem ($p < 0.05$) (Table 4.61), therefore the lower the glycolytic potential the higher the

muscle temperature. In conclusion, the muscle lactic acid, glycogen concentration and glycolytic potential influence the carcass temperature.

Table 4.61 Correlation-matrix showing simple correlation coefficients of how muscle energy metabolites correlated with muscle temperature in the production systems evaluated

	Temp1	Temp2	Temp3	Temp4	Temp24
Lakt1	0.061	0.161*	0.129	0.104	0.025
Lakt2	0.070	0.113	0.148*	0.087	0.015
Lakt4	0.111	0.172*	0.209*	0.159*	0.01
Lakt24	0.155*	0.170*	0.195*	0.163*	-0.045
Glyc1	0.005	-0.127	-0.161*	-0.191*	-0.273*
Glyc2	-0.027	-0.171*	-0.207*	-0.217*	-0.268*
Glyc4	-0.051	-0.181*	-0.208*	-0.229*	-0.227*
Glyc24	-0.042	-0.202*	-0.230*	-0.254*	-0.155*
ATP1	-0.083	-0.074	-0.123	-0.162*	-0.245*
ATP2	-0.034	-0.007	-0.045	-0.076	-0.182*
ATP4	-0.091	-0.057	-0.084	-0.099	-0.091
ATP24	-0.046	-0.060	-0.099	-0.107	-0.060
CP1	0.028	-0.143	-0.094	-0.088	-0.165*
CP2	-0.112	-0.074	-0.025	-0.039	-0.041
CP4	-0.052	-0.019	0.005	0.014	-0.048
CP24	0.054	0.058	0.072	0.108	-0.025
GlyP1	0.034	-0.075	-0.121	-0.163*	-0.270*
GlyP2	0.003	-0.133	-0.153*	-0.189*	-0.258*
GlyP4	0.007	-0.091	-0.102	-0.151*	-0.205*
GlyP24	0.031	-0.104	-0.114	-0.163*	-0.158*

*. Correlation is significant at the 0.05 level (2-tailed).

Temp 1 ~ muscle temperature at 1 hour post mortem, Temp 2 ~ muscle temperature at 2 hours post mortem, Temp 3 ~ muscle temperature at 3 hours post mortem, Temp 4 ~ muscle temperature at 4 hours post mortem, Temp 24 ~ muscle temperature at 24 hour post mortem, Lakt 1 ~ muscle lactic acid concentration at 1 hour post mortem, Lakt 2 ~ muscle lactic acid concentration at 2 hours post mortem, Lakt 4 ~ muscle lactic acid concentration at 4 hours post mortem, Lakt 24 ~ muscle lactic acid concentration at 24 hours post mortem, Glyc 1 ~ muscle glycogen concentration at 1 hour post mortem, Glyc 2 ~ the muscle glycogen concentration at 2 hours post mortem, Glyc 4 ~ the muscle glycogen concentration at 4 hours post mortem, Glyc 24 ~ muscle glycogen concentration at 24 hours post mortem, ATP 1 ~ muscle ATP concentration at 1 hour post mortem, ATP 2 ~ muscle ATP concentration at 2 hours post mortem, ATP 4 ~ muscle ATP concentration at 4 hours post mortem, ATP 24 ~ muscle ATP concentration at 24 hours post mortem, CP 1 ~ muscle creatine phosphate concentration at 1 hour post mortem, CP 2 ~ muscle creatine phosphate concentration at 2 hours post mortem, CP 4 ~ muscle creatine phosphate concentration at 4 hours post mortem, CP 24 ~ muscle creatine phosphate concentration at 24 hours post mortem, GlyP 1 ~ glycolytic potential at 1 hour post mortem, GlyP 2 ~ glycolytic potential at 2 hours post mortem, GlyP 4 ~ glycolytic potential at 4 hours post mortem, GlyP 24 ~ glycolytic potential at 24 hours post mortem

The correlation between muscle energy metabolism and glycogen is summarised in Table 4.62. Muscle lactic acid concentrations had a weak negative correlation with muscle glycogen concentrations ($p < 0.05$) (Table 4.62), therefore the lower the muscle lactic acid concentration the higher the muscle glycogen concentration.

Table 4.62 Correlation-matrix showing simple correlation coefficients of how muscle energy metabolites correlated with muscle glycogen concentration in the production systems evaluated

	Glyc1	Glyc2	Glyc4	Glyc24
Lakt1	-0.262*	-0.287*	-0.319*	-0.211*
Lakt2	-0.225*	-0.245*	-0.279*	-0.185*
Lakt4	-0.135	-0.174*	-0.184*	-0.132
Lakt24	0.005	-0.008	-0.059	-0.101
Glu1	0.089	0.194*	0.181*	0.201*
Glu2	0.013	0.071	0.044	0.193*
Glu4	0.082	0.116	0.110	0.230*
Glu24	0.277*	0.312*	0.330*	0.330*
G6P1	0.020	0.030	0.006	-0.004
G6P2	0.119	0.115	0.076	0.178*
G6P4	-0.104	-0.091	-0.131	-0.010
G6P24	0.189*	0.202*	0.200*	0.186*
ATP1	0.360*	0.425*	0.382*	0.300*
ATP2	0.319*	0.370*	0.348*	0.234*
ATP4	0.326*	0.371*	0.370*	0.226*
ATP24	0.140	0.194*	0.190*	0.196*
GlyP1	0.914*	0.830*	0.733*	0.662*
GlyP2	0.836*	0.912*	0.810	0.709*
GlyP4	0.735*	0.797*	0.870	0.706*
GlyP24	0.661*	0.689*	0.687*	0.846*

*. Correlation is significant at the 0.05 level (2-tailed).

Lakt 1 ~ muscle lactic acid concentration at 1 hour post mortem, Lakt 2 ~ muscle lactic acid concentration at 2 hours post mortem, Lakt 4 ~ muscle lactic acid concentration at 4 hours post mortem, Lakt 24 ~ muscle lactic acid concentration at 24 hours post mortem, Glu 1 ~ muscle glucose concentration at 1 hour post mortem, Glu 2 ~ muscle glucose concentration at 2 hours post mortem, Glu 4 ~ muscle glucose concentration at 4 hours post mortem, Glu 24 ~ the muscle glucose concentration at 24 hours post mortem, Glyc 1 ~ muscle glycogen concentration at 1 hour post mortem, Glyc 2 ~ the muscle glycogen concentration at 2 hours post mortem, Glyc 4 ~ the muscle glycogen concentration at 4 hours post mortem, Glyc 24 ~ muscle glycogen concentration at 24 hours post mortem, G6P 1 ~ muscle glucose-6-phosphate concentration at 1 hour post mortem, G6P 2 ~ muscle glucose-6-phosphate concentration at 2 hours post mortem, G6P 24 ~ muscle glucose-6-phosphate at 24 hours post mortem, ATP 1 ~ muscle ATP concentration at 1 hour post mortem, ATP 2 ~ the muscle ATP concentration at 2 hours post mortem, ATP 4 ~ muscle ATP concentration at 4 hours post mortem, ATP 24 ~ muscle ATP concentration at 24 hours post mortem, GlyP 1 ~ glycolytic potential at 1 hour post mortem, GlyP 2 ~ glycolytic potential at 2 hours post mortem, GlyP 4 ~ glycolytic potential at 4 hours post mortem, GlyP 24 ~ glycolytic potential at 24 hours post mortem

There was a positive correlation between muscle glucose concentrations at 24 hours post mortem and muscle glycogen concentrations at 1 hour post mortem, muscle glucose concentrations at 1 and 24 hours post mortem and muscle glycogen concentrations at 2 and 4

hours post mortem, and muscle glucose concentrations at 1, 2, 4 and 24 hours post mortem and muscle glycogen at 24 hours post mortem ($p < 0.05$) (Table 4.62), therefore the lower the muscle glucose concentration the higher the muscle glycogen concentration.

Muscle ATP concentration had a positive correlation with muscle glycogen concentration ($p < 0.05$), therefore the lower the muscle ATP concentration the lower the muscle glycogen concentration. Muscle glucose-6-phosphate at 24 hours post mortem had a weak positive correlation with muscle glycogen concentrations at 1, 2, 4 and 24 hours post mortem ($p < 0.05$) (Table 4.62), therefore the lower glucose-6-phosphate concentration the lower the muscle glycogen concentration.

Glycolytic potential had a strong positive correlation with glycogen concentration ($p < 0.05$) (Table 4.62), therefore the lower the glycolytic potential the lower the muscle glycolytic potential. This is expected because glycogen is used to calculate the glycolytic potential ($GlyP = 2 * (G6P + Glu + Glyc) + Lakt$).

Table 4.63 summarises the correlation between energy metabolites and muscle glucose-6-phosphate. Muscle lactic acid concentrations had a weak positive correlation with muscle glucose-6-phosphate ($p < 0.05$), therefore the lower the muscle lactic acid concentration the lower the muscle glucose-6-phosphate concentration. Muscle glucose concentrations at 2 and 4 hours post mortem had a weak positive correlation with muscle glucose-6-phosphate concentrations at 2 and 4 hours post mortem, and muscle glucose concentrations at 24 hours post mortem had a weak positive correlation with glucose-6-phosphate concentrations at 24 hours post mortem ($p < 0.05$) (Table 4.63), therefore the lower the muscle glucose concentration, the lower the muscle glucose-6-phosphate concentration.

Muscle glycogen concentrations at 1, 2, 4 and 24 hours post mortem had a weak positive correlation with muscle glucose-6-phosphate concentrations at 24 hours post mortem ($p < 0.05$), therefore the lower the muscle glycogen concentration the lower the muscle glucose-6-phosphate concentration. There was a weak negative correlation between muscle ATP and creatine phosphate concentrations, and glucose-6-phosphate ($p < 0.05$), therefore the lower the ATP and the creatine phosphate concentration the higher the muscle glucose-6-phosphate concentration. Glycolytic potential had a strong positive correlation with glucose-6-phosphate ($p < 0.05$) (Table 4.63). This is expected because glucose-6-phosphate is used to calculate glycolytic potential (see Chapter 3, paragraph 3.4).

Table 4.63 Correlation-matrix showing simple correlation coefficients of how muscle energy metabolites correlated with muscle glucose-6-phosphate concentration in the production systems evaluated

	G6P1	G6P2	G6P4	G6P24
Lakt1	0.234*	0.230*	0.374*	0.166*
Lakt2	0.282*	0.313*	0.442*	0.179*
Lakt4	0.265*	0.253*	0.400*	0.203*
Lakt24	0.234*	0.212*	0.299*	0.223*
Glu1	-0.020	0.088	0.118	0.038
Glu2	0.050	0.235*	0.213*	0.016
Glu4	0.031	0.149*	0.218*	0.068
Glu24	-0.006	0.116	0.042	0.158*
Glyc1	0.020	0.119	-0.104	0.189*
Glyc2	0.030	0.115	-0.091	0.202*
Glyc4	0.006	0.076	-0.131	0.200*
Glyc24	-0.004	0.178*	-0.010	0.186*
ATP1	-0.129	-0.087	-0.198*	-0.126
ATP2	-0.173*	-0.184*	-0.202*	-0.123
ATP4	-0.122	-0.150*	-0.280*	-0.064
ATP24	-0.225*	-0.174*	-0.176*	-0.271*
CP1	-0.188*	-0.179*	-0.266*	-0.194*
CP2	-0.198*	-0.241*	-0.258*	-0.230*
CP4	-0.096	-0.177*	-0.270*	-0.132
CP24	-0.234*	-0.259*	-0.232*	-0.329*
GlyP1	0.182*	0.271*	0.081	0.283*
GlyP2	0.197*	0.320*	0.134	0.297*
GlyP4	0.180*	0.267*	0.179*	0.334*
GlyP24	0.175*	0.330*	0.214*	0.471*

*. Correlation is significant at the 0.05 level (2-tailed).

Lakt 1 ~ muscle lactic acid concentration at 1 hour post mortem, Lakt 2 ~ muscle lactic acid concentration at 2 hours post mortem, Lakt 4 ~ muscle lactic acid concentration at 4 hours post mortem, Lakt 24 ~ muscle lactic acid concentration at 24 hours post mortem, Glu 1 ~ muscle glucose concentration at 1 hour post mortem, Glu 2 ~ muscle glucose concentration at 2 hours post mortem, Glu 4 ~ muscle glucose concentration at 4 hours post mortem, Glu 24 ~ muscle glucose concentration at 24 hours post mortem, Glyc 1 ~ muscle glycogen concentration at 1 hour post mortem, Gluc 2 ~ muscle glycogen concentration at 2 hours post mortem, Glyc 4 ~ the muscle glycogen concentration at 4 hours post mortem, Gluc 24 ~ muscle glycogen concentration at 24 hours post mortem, G6P 1 ~ muscle glucose-6-phosphate concentration at 1 hour post mortem, G6P 2 ~ muscle glucose-6-phosphate concentration at 2 hours post mortem, G6P 24 ~ muscle glucose-6-phosphate at 24 hours post mortem, ATP 1 ~ muscle ATP concentration at 1 hour post mortem, ATP 2 ~ the muscle ATP concentration at 2 hours post mortem, ATP 4 ~ muscle ATP concentration at 4 hours post mortem, ATP 24 ~ muscle ATP concentration at 24 hours post mortem, CP 1 ~ muscle creatine phosphate concentration at 1 hour post mortem, CP 2 ~ muscle creatine phosphate concentration at 2 hours post mortem, CP 4 ~ muscle creatine phosphate concentration at 4 hours post mortem, CP 24 ~ muscle creatine phosphate concentration at 24 hours post mortem, GlyP 1 ~ glycolytic potential at 1 hour post mortem, GlyP 2 ~ glycolytic potential at 2 hours post mortem, GlyP 4 ~ glycolytic potential at 4 hours post mortem, GlyP 24 ~ glycolytic potential at 24 hours post mortem

The correlations between the energy metabolites and the muscle ATP concentration are summarised in Table 4.64. Muscle lactic acid concentrations had a weak negative correlation with muscle ATP concentrations ($p < 0.05$), therefore the lower the muscle ATP concentration the higher the lactic acid concentration. Muscle glucose concentrations at 1 and 24 hours post mortem had a

weak positive correlation with muscle ATP concentrations at 1 hour post mortem, and muscle glucose concentrations at 1 and 4 hours post mortem had a weak negative correlation with muscle ATP concentrations at 4 hours post mortem ($p < 0.05$) (Table 4.64), therefore the lower the muscle glucose concentration the lower the muscle ATP concentration.

Table 4.64 Correlation-matrix showing simple correlation coefficients of how muscle energy metabolites correlated with muscle ATP concentration in the production systems evaluated

	ATP1	ATP2	ATP4	ATP24
Lakt1	-0.232*	-0.280*	-0.339*	-0.233*
Lakt2	-0.202*	-0.319*	-0.419*	-0.221*
Lakt4	-0.172*	-0.225*	-0.394*	-0.209*
Lakt24	-0.062	-0.084	-0.138	-0.186*
Glu1	0.186*	0.057	-0.077	0.103
Glu2	0.083	-0.089	-0.194*	0.083
Glu4	0.096	0.012	-0.220*	0.035
Glu24	0.225*	0.103	-0.032	0.039
Glyc1	0.360*	0.319*	0.326*	0.140
Glyc2	0.425*	0.370*	0.371*	0.194*
Glyc4	0.382*	0.348*	0.370*	0.190*
Glyc24	0.300*	0.234*	0.226*	0.196*
G6P1	-0.129	-0.173*	-0.122	-0.225*
G6P2	-0.087	-0.184*	-0.150*	-0.174*
G6P4	-0.198*	-0.202*	-0.280*	-0.176*
G6P24	-0.126	-0.123	-0.064	-0.271*
CP1	0.268*	0.169*	0.243*	0.297*
CP2	0.200*	0.183*	0.205*	0.290*
CP4	0.009	0.107	0.215*	0.108
CP24	0.040	0.121	0.121	0.357*
GlyP1	0.277*	0.206*	0.186*	0.043
GlyP2	0.345*	0.230*	0.191*	0.103
GlyP4	0.275*	0.215*	0.142	0.075
GlyP24	0.212*	0.139	0.112	0.029

*. Correlation is significant at the 0.05 level (2-tailed).

Lakt 1 ~ muscle lactic acid concentration at 1 hour post mortem, Lakt 2 ~ muscle lactic acid concentration at 2 hours post mortem, Lakt 4 ~ muscle lactic acid concentration at 4 hours post mortem, Lakt 24 ~ muscle lactic acid concentration at 24 hours post mortem, Glu 1 ~ muscle glucose concentration at 1 hour post mortem, Glu 2 ~ muscle glucose concentration at 2 hours post mortem, Glu 4 ~ muscle glucose concentration at 4 hours post mortem, Glu 24 ~ the muscle glucose concentration at 24 hours post mortem, Glyc 1 ~ muscle glycogen concentration at 1 hour post mortem, Gluc 2 ~ the muscle glycogen concentration at 2 hours post mortem, Glyc 4 ~ the muscle glycogen concentration at 4 hours post mortem, Gluc 24 ~ muscle glycogen concentration at 24 hours post mortem, G6P 1 ~ muscle glucose-6-phosphate concentration at 1 hour post mortem, G6P 2 ~ muscle glucose-6-phosphate concentration at 2 hours post mortem, G6P 24 ~ muscle glucose-6-phosphate at 24 hours post mortem, ATP 1 ~ muscle ATP concentration at 1 hour post mortem, ATP 2 ~ the muscle ATP concentration at 2 hours post mortem, ATP 4 ~ muscle ATP concentration at 4 hours post mortem, ATP 24 ~ muscle ATP concentration at 24 hours post mortem, CP 1 ~ muscle creatine phosphate concentration at 1 hour post mortem, CP 2 ~ muscle creatine phosphate concentration at 2 hours post mortem, CP 4 ~ muscle creatine phosphate concentration at 4 hours post mortem, CP 24 ~ muscle creatine phosphate concentration at 24 hours post mortem, GlyP 1 ~ glycolytic potential at 1 hour post mortem, GlyP 2 ~ glycolytic potential at 2 hours post mortem, GlyP 4 ~ glycolytic potential at 4 hours post mortem, GlyP 24 ~ glycolytic potential at 24 hours post mortem

Muscle glycogen concentrations had a weak positive correlation with muscle ATP concentrations ($p < 0.05$), therefore the lower the muscle glycogen concentration the lower the muscle ATP concentration. There was a weak negative correlation between muscle glucose-6-phosphate concentrations at 1 hour post mortem and muscle ATP concentrations at 2 and 24 hours post mortem ($p < 0.05$), muscle glucose-6-phosphate concentrations at 2 hours post mortem and muscle ATP concentrations at 2, 4 and 24 hours post mortem ($p < 0.05$), muscle glucose-6-phosphate concentrations at 4 hours post mortem and muscle ATP concentrations at 1, 2, 4 and 24 hours post mortem ($p < 0.05$), and muscle glucose-6-phosphate concentrations at 24 hours post mortem and muscle ATP concentrations at 24 hours post mortem ($p < 0.05$) (Table 4.64), therefore the lower the muscle glycogen concentration the higher the muscle ATP concentration.

The correlations between energy metabolites and muscle creatine phosphate are summarised in Table 4.65. Muscle lactic acid concentrations had a weak negative correlation with muscle creatine phosphate ($p < 0.05$) (Table 4.65), therefore the lower the muscle lactic acid concentration the higher the muscle creatine phosphate concentration.

Muscle glucose concentrations at 1, 2, 4 and 24 hours post mortem had a weak negative correlation with muscle creatine phosphate concentrations at 4 hours post mortem ($p < 0.05$), therefore the lower the muscle glucose concentration the higher the muscle creatine phosphate concentration. Muscle glycogen concentrations did not correlate with muscle creatine phosphate concentrations ($p > 0.05$) (Table 4.65).

Muscle glucose-6-phosphate concentrations at 1 and 24 hours post mortem had a weak negative correlation with muscle creatine phosphate at 1, 2 and 24 hours post mortem ($p < 0.05$), therefore the lower the muscle glucose-6-phosphate concentration the higher the muscle creatine phosphate concentration. There was a weak negative correlation between muscle glucose-6-phosphate concentrations at 2 and 4 hours post mortem and muscle creatine phosphate concentration at 1, 2, 4 and 24 hours post mortem ($p < 0.05$), therefore the lower the muscle glucose-6-phosphate concentration the higher the muscle creatine phosphate concentration. Glycolytic potential at 1, 2, 4 and 24 hours post mortem had a weak negative correlation with creatine phosphate concentration at 24 hours post mortem ($p < 0.05$) (Table 4.65), therefore the lower the glycolytic potential the higher the muscle creatine phosphate concentration. In conclusion (as expected) the different energy metabolites influence each other.

Table 4.65 Correlation-matrix showing simple correlation coefficients of how muscle energy metabolites correlated with muscle creatine phosphate concentration in the production systems evaluated

	CP1	CP2	CP4	CP24
Lakt1	-0.144	-0.117	-0.139	-0.093
Lakt2	-0.216*	-0.148*	-0.230*	-0.163*
Lakt4	-0.157*	-0.150*	-0.211*	-0.221*
Lakt24	-0.142	-0.129	-0.149*	-0.185*
Glu1	-0.041	-0.058	-0.150*	-0.051
Glu2	-0.046	-0.052	-0.198*	-0.052
Glu4	-0.115	-0.114	-0.187*	-0.050
Glu24	-0.110	-0.123	-0.178*	-0.138
G6P1	-0.188*	-0.198*	-0.096	-0.234*
G6P2	-0.179*	-0.241*	-0.177*	-0.259*
G6P4	-0.266*	-0.258*	-0.270*	-0.232*
G6P24	-0.194*	-0.230*	-0.132	-0.329*
ATP1	0.268*	0.200*	0.009	0.040
ATP2	0.169*	0.183*	0.107	0.121
ATP4	0.243*	0.205*	0.215*	0.121
ATP24	0.297*	0.290*	0.108	0.357*
GlyP1	-0.006	-0.048	-0.004	-0.186*
GlyP2	0.029	-0.038	-0.017	-0.195*
GlyP4	0.015	-0.059	-0.041	-0.239*
GlyP24	-0.038	-0.077	-0.098	-0.200*

*. Correlation is significant at the 0.05 level (2-tailed).

Lakt 1 ~ muscle lactic acid concentration at 1 hour post mortem, Lakt 2 ~ muscle lactic acid concentration at 2 hours post mortem, Lakt 4 ~ muscle lactic acid concentration at 4 hours post mortem, Lakt 24 ~ muscle lactic acid concentration at 24 hours post mortem, Glu 1 ~ muscle glucose concentration at 1 hour post mortem, Glu 2 ~ muscle glucose concentration at 2 hours post mortem, Glu 4 ~ muscle glucose concentration at 4 hours post mortem, Glu 24 ~ the muscle glucose concentration at 24 hours post mortem, G6P 1 ~ muscle glucose-6-phosphate concentration at 1 hour post mortem, G6P 2 ~ muscle glucose-6-phosphate concentration at 2 hours post mortem, G6P 24 ~ muscle glucose-6-phosphate at 24 hours post mortem, ATP 1 ~ muscle ATP concentration at 1 hour post mortem, ATP 2 ~ the muscle ATP concentration at 2 hours post mortem, ATP 4 ~ muscle ATP concentration at 4 hours post mortem, ATP 24 ~ muscle ATP concentration at 24 hours post mortem, CP 1 ~ muscle creatine phosphate concentration at 1 hour post mortem, CP 2 ~ muscle creatine phosphate concentration at 2 hours post mortem, CP 4 ~ muscle creatine phosphate concentration at 4 hours post mortem, CP 24 ~ muscle creatine phosphate concentration at 24 hours post mortem, GlyP 1 ~ glycolytic potential at 1 hour post mortem, GlyP 2 ~ glycolytic potential at 2 hours post mortem, GlyP 4 ~ glycolytic potential at 4 hours post mortem, GlyP 24 ~ glycolytic potential at 24 hours post mortem

4.13 Correlation of energy metabolites, muscle pH and muscle temperature with muscle contraction (sarcomere lengths)

The energy metabolites had no significant correlation with sarcomere lengths. The correlation between pH and sarcomere length as well as between temperature and sarcomere length is summarised in Table 4.66. There was a weak to medium negative correlation between pH and sarcomere length ($p < 0.05$), therefore the lower the carcass pH the longer the sarcomere length. Muscle temperature at 24 hours post mortem had no effect on the sarcomere lengths at 1 and 3

days post mortem. Muscle temperature had a medium to strong positive correlation with sarcomere lengths ($p < 0.05$) (Table 4.66), therefore the lower the carcass temperature the shorter the sarcomere length. Long sarcomere length is important for muscle tenderness (discussed later in dissertation), therefore carcass pH and temperature is important for tenderness.

Table 4.66 Correlation between sarcomere lengths and muscle pH as well as muscle temperature

	Sar1	Sar3
pH1	-0.188*	-0.205*
pH2	-0.238*	-0.296*
pH3	-0.277*	-0.277*
pH4	-0.357*	-0.351*
pH24	-0.238*	-0.237*
Temp1	0.302*	0.244*
Temp2	0.417*	0.435*
Temp3	0.414*	0.415*
Temp4	0.414*	0.395*
Temp24	0.025	-0.006

*. Correlation is significant at the 0.05 level (2-tailed).

sar1 ~ sarcomere length at 1 day post mortem, sar3 ~ sarcomere length at 3 days post mortem, pH1 ~ muscle pH at 1 hour post mortem, pH2 ~ muscle pH at 2 hours post mortem, pH3 ~ muscle pH at 3 hours post mortem, pH4 ~ muscle pH at 4 hours post mortem, pH24 ~ muscle pH at 24 hours post mortem, temp1 ~ muscle temperature at 1 hour post mortem, temp2 ~ muscle temperature at 2 hours post mortem, temp3 ~ muscle temperature at 3 hours post mortem, temp4 ~ muscle temperature at 4 hours post mortem, temp24 ~ muscle temperature at 24 hours post mortem

4.14 Correlation of energy metabolites, muscle pH and muscle temperature with collagen content

The correlation between muscle temperature and collagen content is summarised in Table 4.67. Muscle temperature at 24 hours post mortem had no effect on the collagen content. There was a weak to medium negative correlation between muscle temperature and collagen content ($p < 0.05$), therefore the lower the muscle temperature the higher the collagen content. There was no significant correlation between muscle temperature and collagen solubility (Table 4.67).

Table 4.67 Correlation between muscle temperature and collagen content

	Temp1	Temp2	Temp3	Temp4	Temp24
Sol	-0.208*	-0.188*	-0.192*	-0.255*	-0.062
Insol	-0.348*	-0.452*	-0.431*	-0.463*	0.069
Sollnsol	-0.345*	-0.434*	-0.416*	-0.456*	0.050
ColSolper	0.023	0.148*	0.133	0.088	-0.101

*. Correlation is significant at the 0.05 level (2-tailed).

Sol ~ soluble collagen content, Insol ~ insoluble collagen content, Sollnsol ~ total collagen content, ColSolper ~ percentage soluble collagen, temp1 ~ muscle temperature at 1 hour post mortem, temp2 ~ muscle temperature at 2 hours post mortem, temp3 ~ muscle temperature at 3 hours post mortem, temp4 ~ muscle temperature at 4 hours post mortem, temp24 ~ muscle temperature at 24 hours post mortem

4.15 Correlation of energy metabolites, muscle pH, muscle temperature with myofibrillar fragment lengths as well as calpain system activity

Correlations between the activity of the calpain systems and energy metabolites are summarised in Table 4.68. Muscle glycogen concentrations had a weak positive correlation with calpastatin activity ($p < 0.05$) (Table 4.68), therefore the lower muscle glycogen concentration the higher the calpastatin activity.

Muscle glucose-6-phosphate concentrations had a weak positive correlation with calpastatin activity at 1 hour post mortem ($p < 0.05$), therefore the lower the muscle glucose-6-phosphate concentration the lower the calpastatin activity at 1 hour post mortem (Table 4.68).

Muscle creatine phosphate had a weak negative correlation with calpastatin activity at 1 hour post mortem ($p < 0.05$), therefore the lower muscle creatine phosphate concentrations, the higher the calpastatin activity at 1 hour post mortem. There was a weak positive correlation between glycolytic potential and calpastatin activity at 24 hours post mortem ($p < 0.05$) (Table 4.68), therefore the lower the glycolytic potential the lower the calpastatin activity at 24 hour post mortem.

Muscle glucose concentrations had a weak negative correlation with calpain I activity at 24 hours post mortem ($p < 0.05$) (Table 4.68), therefore the lower the muscle glucose concentration the higher the calpain I activity at 24 hours post mortem.

Muscle glycogen concentrations at 1, 2 and 4 hours post mortem had a weak positive correlation with calpain I activity at 24 hours post mortem ($p < 0.05$), therefore the lower the muscle glycogen concentration the lower the calpain I activity at 24 hour post mortem. Glycolytic potential had a weak positive correlation with calpain I activity at 24 hours post mortem ($p < 0.05$) (Table 4.68), therefore the lower the glycolytic potential the lower the calpain I activity at 24 hours post mortem.

Muscle lactic acid concentrations had a weak positive correlation with calpain II activity at 1 hour post mortem ($p < 0.05$), therefore the lower the muscle lactic acid concentration the lower the calpain II activity at 1 hour post mortem. Muscle glycogen concentrations had a weak negative

correlation with calpain II activity at 1 hour post mortem ($p < 0.05$), therefore the lower the muscle glucose concentration the higher the calpain II activity at 1 hour post mortem (Table 4.68).

Table 4.68 Correlation-matrix showing simple correlation coefficients of how muscle energy metabolites correlated with calpastatin and calpain activity in the production systems evaluated

	TOT IN1h	Spes IN1h	CAI1h	Spes CAI1h	CAII1h	Spes CAII1h	INCAI1h	INCAIII1h
Lakt1	-0.014	-0.030	0.127	0.105	0.199*	0.175*	-0.105	-0.148*
Lakt2	0.004	-0.002	0.161*	0.155*	0.173*	0.166*	-0.131	-0.147*
Lakt4	0.045	-0.011	0.083	0.038	0.294*	0.234*	-0.031	-0.113
Lakt24	0.057	-0.034	0.024	-0.040	0.185*	0.093	0.036	-0.029
Glu1	0.015	0.045	-0.045	-0.031	0.008	0.035	0.042	0.031
Glu2	0.057	0.091	0.000	0.020	-0.068	-0.030	0.022	0.069
Glu4	0.151*	0.14	-0.034	-0.046	0.085	0.087	0.124	0.114
Glu24	0.140	0.150*	-0.132	-0.109	-0.084	-0.066	0.216*	0.241*
Glyc1	0.119	0.115	-0.092	-0.077	-0.170*	-0.179*	0.170*	0.227*
Glyc2	0.114	0.131	-0.094	-0.066	-0.219*	-0.211*	0.158*	0.237*
Glyc4	0.072	0.108	-0.101	-0.058	-0.237*	-0.211*	0.133	0.209*
Glyc24	0.090	0.184*	-0.086	-0.003	-0.223*	-0.131	0.127	0.212*
G6P1	0.084	0.097	0.081	0.095	0.017	0.041	-0.008	0.011
G6P2	0.181*	0.235*	0.075	0.123	-0.021	0.051	0.064	0.122
G6P4	0.175*	0.185*	0.024	0.032	0.103	0.132	0.117	0.103
G6P24	0.198*	0.226*	-0.032	0.008	0.056	0.097	0.174*	0.173*
ATP1	0.081	0.078	0.043	0.05	-0.012	-0.016	0.006	0.045
ATP2	0.092	0.044	0.039	0.004	0.029	-0.018	0.028	0.042
ATP4	0.079	0.050	0.061	0.042	-0.018	-0.049	-0.018	0.027
ATP24	-0.002	-0.008	0.001	0.000	-0.048	-0.062	-0.027	0.010
CP1	-0.115	-0.083	-0.013	0.004	-0.069	-0.049	-0.097	-0.081
CP2	-0.284*	-0.246*	-0.015	0.005	-0.071	-0.056	-0.197*	-0.208*
CP4	-0.160*	-0.162*	0.040	0.040	-0.089	-0.104	-0.155*	-0.133
CP24	-0.160*	-0.144	0.026	0.040	-0.050	-0.050	-0.159*	-0.153*
GlyP1	0.122	0.114	-0.041	-0.033	-0.095	-0.109	0.134	0.176*
GlyP2	0.131	0.153*	-0.029	0.002	-0.159*	-0.146*	0.115	0.195*
GlyP4	0.119	0.130	-0.062	-0.039	-0.087	-0.083	0.136	0.173*
GlyP24	0.151*	0.196*	-0.077	-0.026	-0.100	-0.053	0.173*	0.217*

*. Correlation is significant at the 0.05 level (2-tailed).

Lakt 1 ~ muscle lactic acid concentration at 1 hour post mortem, Lakt 2 ~ muscle lactic acid concentration at 2 hours post mortem, Lakt 4 ~ muscle lactic acid concentration at 4 hours post mortem, Lakt 24 ~ muscle lactic acid concentration at 24 hours post mortem, Glu 1 ~ muscle glucose concentration at 1 hour post mortem, Glu 2 ~ muscle glucose concentration at 2 hours post mortem, Glu 4 ~ muscle glucose concentration at 4 hours post mortem, Glu 24 ~ the muscle glucose concentration at 24 hours post mortem, Glyc 1 ~ muscle glycogen concentration at 1 hour post mortem, Glyc 2 ~ the muscle glycogen concentration at 2 hours post mortem, Glyc 4 ~ the muscle glycogen concentration at 4 hours post mortem, Glyc 24 ~ muscle glycogen concentration at 24 hours post mortem, G6P 1 ~ muscle glucose-6-phosphate concentration at 1 hour post mortem, G6P 2 ~ muscle glucose-6-phosphate concentration at 2 hours post mortem, G6P 24 ~ muscle glucose-6-phosphate at 24 hours post mortem, ATP 1 ~ muscle ATP concentration at 1 hour post mortem, ATP 2 ~ muscle ATP concentration at 2 hours post mortem, ATP 4 ~ muscle ATP concentration at 4 hours post mortem, ATP 24 ~ muscle ATP concentration at 24 hours post mortem, CP 1 ~ muscle creatine phosphate concentration at 1 hour post mortem, CP 2 ~ muscle creatine phosphate concentration at 2 hours post mortem, CP 4 ~ muscle creatine phosphate concentration at 4 hours post mortem, CP 24 ~ muscle creatine phosphate concentration at 24 hours post mortem, GlyP 1 ~ glycolytic potential at 1 hour post mortem, GlyP 2 ~ glycolytic potential at 2 hours post mortem, GlyP 4 ~ glycolytic potential at 4 hours post mortem, GlyP 24 ~ glycolytic potential at 24 hours post mortem, TOT IN1h ~ total calpastatin at 1 hour post mortem, Spes IN1h ~ specific calpastatin at 1 hour post mortem, CAI1h ~ calpain I activity 1 hour post mortem, Spes CAI1h ~ specific calpain I activity 1 hour post mortem, CAII1h ~ calpain II activity 1 hour post mortem, Spes CAII ~ specific calpain II activity 1 hour post mortem, INCAI1h ~ the ratio between calpastatin and calpain I 1 hour post mortem, INCAIII1h ~ the ratio between calpastatin and calpain I + II 1 hour post mortem

Table 4.68 (continue) Correlation-matrix showing simple correlation coefficients of how muscle energy metabolites correlated with calpastatin and calpain activity in the production systems evaluated

	TOT IN24h	Spes IN24h	CAI24h	Spes CAI24h	CALII 24h	Spes CAII24h	INCAI 24 h	INCAIII 24 h
Lakt1	-0.083	-0.122	-0.126	-0.162*	0.067	-0.021	0.066	-0.041
Lakt2	-0.049	-0.058	-0.118	-0.135	0.009	-0.020	0.055	0.004
Lakt4	-0.044	-0.086	-0.185*	-0.223*	0.012	-0.070	0.146*	0.062
Lakt24	0.158*	0.115	0.000	-0.035	0.034	-0.038	0.088	0.172*
Glu1	-0.094	-0.070	-0.122	-0.101	-0.053	-0.018	0.058	-0.012
Glu2	-0.106	-0.067	-0.190*	-0.159*	-0.100	-0.038	0.110	0.036
Glu4	-0.019	0.007	-0.171*	-0.146*	-0.030	0.011	0.173*	0.093
Glu24	0.039	0.086	-0.064	-0.032	-0.057	0.016	0.089	0.098
Glyc1	0.212*	0.258*	0.273*	0.307*	0.061	0.150*	-0.149*	0.028
Glyc2	0.175*	0.220*	0.260*	0.294*	0.027	0.112	-0.152*	0.011
Glyc4	0.123	0.187*	0.183*	0.230*	-0.026	0.087	-0.084	0.030
Glyc24	0.007	0.092	0.036	0.098	0.013	0.160*	-0.023	-0.021
G6P1	0.124	0.116	0.090	0.093	0.078	0.072	-0.038	0.034
G6P2	0.107	0.131	0.090	0.113	0.069	0.123	-0.069	0.010
G6P4	0.032	0.062	-0.102	-0.080	0.034	0.092	0.090	0.072
G6P24	0.050	0.108	0.006	0.044	0.078	0.165*	0.029	0.020
ATP1	0.067	0.071	0.166*	0.171*	0.079	0.083	-0.131	-0.072
ATP2	0.08	0.055	0.191*	0.177*	0.097	0.053	-0.146*	-0.078
ATP4	0.081	0.061	0.227*	0.219*	0.059	0.030	-0.153*	-0.070
ATP24	-0.046	-0.052	-0.052	-0.054	-0.058	-0.064	0.045	-0.007
CP1	-0.060	-0.044	0.106	0.125	-0.091	-0.054	-0.148*	-0.105
CP2	-0.144	-0.135	-0.022	-0.010	-0.084	-0.071	-0.055	-0.100
CP4	-0.067	-0.074	0.036	0.030	-0.019	-0.039	-0.059	-0.080
CP24	-0.106	-0.127	-0.113	-0.134	-0.053	-0.086	0.069	-0.031
GlyP1	0.185*	0.218*	0.226*	0.249*	0.089	0.147*	-0.124	0.015
GlyP2	0.157*	0.203*	0.209*	0.241*	0.029	0.111	-0.129	0.015
GlyP4	0.102	0.151*	0.075	0.109	-0.018	0.066	0.002	0.070
GlyP24	0.086	0.154*	0.026	0.072	0.037	0.152*	0.031	0.067

*. Correlation is significant at the 0.05 level (2-tailed).

Lakt 1 ~ muscle lactic acid concentration at 1 hour post mortem, Lakt 2 ~ muscle lactic acid concentration at 2 hours post mortem, Lakt 4 ~ muscle lactic acid concentration at 4 hours post mortem, Lakt 24 ~ muscle lactic acid concentration at 24 hours post mortem, Glu 1 ~ muscle glucose concentration at 1 hour post mortem, Glu 2 ~ muscle glucose concentration at 2 hours post mortem, Glu 4 ~ muscle glucose concentration at 4 hours post mortem, Glu 24 ~ the muscle glucose concentration at 24 hours post mortem, Glyc 1 ~ muscle glycogen concentration at 1 hour post mortem, Gluc 2 ~ the muscle glycogen concentration at 2 hours post mortem, Glyc 4 ~ the muscle glycogen concentration at 4 hours post mortem, Gluc 24 ~ muscle glycogen concentration at 24 hours post mortem, G6P 1 ~ muscle glucose-6-phosphate concentration at 1 hour post mortem, G6P 2 ~ muscle glucose-6-phosphate concentration at 2 hours post mortem, G6P 24 ~ muscle glucose-6-phosphate at 24 hours post mortem, ATP 1 ~ muscle ATP concentration at 1 hour post mortem, ATP 2 ~ the muscle ATP concentration at 2 hours post mortem, ATP 4 ~ muscle ATP concentration at 4 hours post mortem, ATP 24 ~ muscle ATP concentration at 24 hours post mortem, CP 1 ~ muscle creatine phosphate concentration at 1 hour post mortem, CP 2 ~ muscle creatine phosphate concentration at 2 hours post mortem, CP 4 ~ muscle creatine phosphate concentration at 4 hours post mortem, CP 24 ~ muscle creatine phosphate concentration at 24 hours post mortem, GlyP 1 ~ glycolytic potential at 1 hour post mortem, GlyP 2 ~ glycolytic potential at 2 hours post mortem, GlyP 4 ~ glycolytic potential at 4 hours post mortem, GlyP 24 ~ glycolytic potential at 24 hours post mortem, TOT IN24h ~ total calpastatin at 24 hours post mortem, Spes IN24h ~ specific calpastatin at 24 hours post mortem, CAI24h ~ calpain I activity 24 hours post mortem, Spes CAI24h ~ specific calpain I activity 24 hours post mortem, CAII24h ~ calpain II activity 24 hours post mortem, Spes CAII 24 ~ specific calpain II activity 24 hours post mortem, INCAI24h ~ the ratio between calpastatin and calpain I 24 hours post mortem, INCAIII24h ~ the ratio between calpastatin and calpain I + II 24 hours post mortem

Muscle glycogen concentrations had a weak positive correlation with the ratio between calpastatin/calpain I at 1 hour post mortem ($p < 0.05$), therefore the lower the muscle glycogen

concentration the lower the ratio between calpastatin/calpain I at 1 hour post mortem. Muscle glycogen concentrations had a weak negative correlation with the ratio between calpastatin/calpain I at 24 hours post mortem ($p < 0.05$), therefore the lower the muscle glycogen concentration the higher the ratio between calpastatin/calpain I at 24 hours post mortem. Muscle ATP concentrations had a weak negative correlation with the ratio between calpastatin/calpain I at 24 hours post mortem ($p < 0.05$) (Table 4.68), therefore the lower the muscle ATP concentration the higher the ratio between calpastatin/calpain I + II.

Muscle creatine phosphate had a weak negative correlation with the ratio between calpastatin/calpain I ($p < 0.05$), therefore the lower the muscle creatine phosphate the higher the ratio between calpastatin/calpain I + II (Table 4.68).

There was a weak negative correlation between muscle lactic acid concentrations and the ratio between calpastatin/calpain I + II at 1 hour post mortem ($p < 0.05$), therefore the lower the muscle lactic acid concentration the higher the ratio between the calpastatin/calpain I + II at 1 hour post mortem. Muscle glycogen concentrations had a weak positive correlation with the ratio between calpastatin/calpain I + II at 1 hour post mortem ($p < 0.05$), therefore the lower the muscle glycogen concentration the lower the calpastatin/calpain I + II at 1 hour post mortem. Glycolytic potential had a weak positive correlation with the ratio between calpastatin/calpain I + II at 1 hour post mortem ($p < 0.05$) (Table 4.68), therefore the lower the glycolytic potential the lower the ratio between calpastatin/calpain I + II.

The correlations between muscle pH and the calpain systems activity is summarised in Table 4.69. Muscle pH had a weak negative correlation with total calpastatin activity at 1 hour post mortem ($p < 0.05$), therefore the lower the muscle pH the higher the total calpastatin activity at 1 hour post mortem (Table 4.69).

Muscle pH had a weak negative correlation with calpain I and II activity at 1 hour post mortem ($p < 0.05$), therefore the lower the muscle pH the higher the calpain I and II activity at 1 hour post mortem (Table 4.69).

Table 4.69 Correlation between muscle pH and the calpain system activity

	pH1	pH2	pH3	pH4	pH24
TOTIN1h	-0.046	0.011	-0.155*	-0.237*	-0.226*
SpesIN1h	-0.019	0.097	-0.013	-0.081	-0.071
CAI1h	-0.185*	-0.223*	-0.201*	-0.221*	-0.121
SpesCAI1h	-0.157*	-0.142	-0.09	-0.104	-0.011
CAII1h	-0.228*	-0.263*	-0.259*	-0.268*	-0.217*
SpesCAII1h	-0.206*	-0.165*	-0.121	-0.121	-0.071
INCAI1	0.132	0.184*	0.079	0.045	-0.039
INCAIII1	0.155*	0.222*	0.089	0.038	-0.039
TOTIN24	0.143	0.072	0.024	-0.041	-0.031
SpesIN24	0.155*	0.131	0.095	0.037	0.033
CAI24	0.132	0.009	0.137	0.093	0.069
SpesCAI24	0.143	0.060	0.191*	0.153*	0.114
CAII24	-0.018	-0.113	0.010	-0.016	0.059
SpesCAII24	0.022	0.007	0.143	0.130	0.171*
INCAI24	-0.032	0.000	-0.127	-0.129	-0.095
INCAIII24	0.083	0.094	-0.072	-0.105	-0.116

*. Correlation is significant at the 0.05 level (2-tailed).

pH1 ~ muscle pH at 1 hour post mortem, pH2 ~ muscle pH at 2 hours post mortem, pH3 ~ muscle pH at 3 hours post mortem, pH4 ~ muscle pH at 4 hours post mortem, pH24 ~ muscle pH at 24 hours post mortem, TOT IN1h ~ total calpastatin at 1 hour post mortem, Spes IN1h ~ specific calpastatin at 1 hour post mortem, CAI1h ~ calpain I activity 1 hour post mortem, Spes CAI1h ~ specific calpain I activity 1 hour post mortem, CAII1h ~ calpain II activity 1 hour post mortem, Spes CAII ~ specific calpain II activity 1 hour post mortem, INCAI1h ~ the ratio between calpastatin and calpain I 1 hour post mortem, INCAIII1h ~ the ratio between calpastatin and calpain I + II 1 hour post mortem, TOT IN24h ~ total calpastatin at 24 hours post mortem, Spes IN24h ~ specific calpastatin at 24 hours post mortem, CAI24h ~ calpain I activity 24 hours post mortem, Spes CAI24h ~ specific calpain I activity 24 hours post mortem, CAII24h ~ calpain II activity 24 hour post mortem, Spes CAII 24 ~ specific calpain II activity 24 hours post mortem, INCAI24h ~ the ratio between calpastatin and calpain I 24 hours post mortem, INCAIII24h ~ the ratio between calpastatin and calpain I + II 24 hours post mortem

The correlation between muscle temperature and the calpain system activity is summarised in Table 4.70. There was a weak positive correlation between muscle temperature and total calpastatin activity, except that muscle temperature at 24 hours post mortem had a weak negative correlation with calpastatin activity ($p < 0.05$). There was no significant correlation between muscle temperature and specific calpastatin activity (Table 4.70).

Calpain I and II activity at 1 hour post mortem had a weak positive correlation with muscle temperature at 1, 2, 3 and 4 hours post mortem ($p < 0.05$), there were a weak negative correlation between muscle temperature at 24 hours post mortem and calpain I and II activity at 1 hour post mortem ($p < 0.05$) (Table 4.70).

Muscle temperature at 1 hour post mortem had a weak negative correlation with the ratio between calpastatin and calpain at 1 hour post mortem ($p < 0.05$) and muscle temperature at 24 hours post mortem had a weak positive correlation with the ratio between calpastatin and calpain ($p < 0.05$) (Table 4.70).

Table 4.70 Correlation between muscle temperature and calpain system activity

	Temp1	Temp2	Temp3	Temp4	Temp24
TOTIN1h	0.120	0.264*	0.203*	0.209*	-0.042
SpesIN1h	0.000	0.029	-0.023	-0.030	-0.036
CAI1h	0.235*	0.179*	0.151*	0.154*	-0.213*
SpesCAI1h	0.152*	0.017	-0.006	-0.014	-0.215*
CAII1h	0.329*	0.371*	0.315*	0.276*	-0.150*
SpesCALII1h	0.217*	0.150*	0.098	0.045	-0.143
INCAI1	-0.155*	-0.005	-0.025	-0.022	0.158*
INCALIII1	-0.198*	-0.035	-0.057	-0.039	0.148*
TOTIN24	0.148*	0.267**	0.225*	0.178*	-0.264*
SpesIN24	0.033	0.158*	0.120	0.053	-0.225*
CAI24	0.069	0.017	-0.023	-0.074	-0.378*
SpesCAI24	-0.012	-0.067	-0.104	-0.168*	-0.351*
CALII24	0.128	0.090	-0.025	-0.084	-0.410*
SpesCAII24	-0.042	-0.094	-0.208*	-0.295*	-0.367*
INCAI24	0.056	0.179*	0.196*	0.228*	0.226*
INCAIII24	0.080	0.259*	0.293*	0.298*	0.106

*. Correlation is significant at the 0.05 level (2-tailed).

TOT IN1h ~ total calpastatin at 1 hour post mortem, Spes IN1h ~ specific calpastatin at 1 hour post mortem, CAI1h ~ calpain I activity 1 hour post mortem, Spes CAI1h ~ specific calpain I activity 1 hour post mortem, CAII1h ~ calpain II activity 1 hour post mortem, Spes CAII ~ specific calpain II activity 1 hour post mortem, INCAI1h ~ the ratio between calpastatin and calpain I 1 hour post mortem, INCAIII1h ~ the ratio between calpastatin and calpain I + II 1 hour post mortem, TOT IN24h ~ total calpastatin at 24 hours post mortem, Spes IN24h ~ specific calpastatin at 24 hours post mortem, CAI24h ~ calpain I activity 24 hours post mortem, Spes CAI24h ~ specific calpain I activity 24 hours post mortem, CAII24h ~ calpain II activity 24 hour post mortem, Spes CAII 24 ~ specific calpain II activity 24 hours post mortem, INCAI24h ~ the ratio between calpastatin and calpain I 24 hours post mortem, INCAIII24h ~ the ratio between calpastatin and calpain I + II 24 hours post mortem, temp1 ~ muscle temperature at 1 hour post mortem, temp2 ~ muscle temperature at 2 hours post mortem, temp3 ~ muscle temperature at 3 hours post mortem, temp4 ~ muscle temperature at 4 hours post mortem, temp24 ~ muscle temperature at 24 hours post mortem

4.16 Correlation between energy metabolites, muscle pH and muscle temperature with tenderness (WBSF), water binding capacity and drip loss

The correlation-matrix showing simple correlation coefficients of how tenderness (Warner-Bratzler shear force) correlated with various carcass characteristics in the production systems evaluated is summarised in Table 4.71.

There was a positive correlation between shear force at 1, 7 and 14 days post mortem and muscle pH at 1, 2, 3, 4 and 24 hours post mortem ($p < 0.05$) (Table 4.71). Muchenje, Dzama, *et al.* (2009a) concluded that there was no significant correlation between shear force and pH, whereas Jelenikova, Pipek, *et al.* (2008) concluded that there was a positive correlation between shear force and pH. There was a weak negative correlation between shear force and muscle temperature ($p < 0.05$) (Table 4.71).

Table 4.71 Correlation-matrix showing simple correlation coefficients of how tenderness (Warner-Bratzler shear force) correlated with various carcass characteristics in the production systems evaluated

	WBSF 1 day (kg)	WBSF 7 days (kg)	WBSF 14 days (kg)
pH 1 h	0.355*	0.313*	0.332*
pH 2 h	0.359*	0.298*	0.286*
pH 3 h	0.397*	0.385*	0.358*
pH 4 h	0.393*	0.410*	0.379*
pH 24 h	0.186*	0.289*	0.269*
Temp 1 h	-0.157*	-0.241*	-0.179*
Temp 2 h	-0.257*	-0.245*	-0.263*
Temp 3 h	-0.268*	-0.227*	-0.240*
Temp 4 h	-0.267*	-0.224*	-0.218*
Temp 24 h	0.029	0.127	0.054

*. Correlation is significant at the 0.05 level (2-tailed).

WBSF 1 day ~ shear force at 1 day post mortem, WBSF 7 days ~ shear force at 7 days post mortem, WBSF 14 days ~ shear force at 14 days post mortem, pH 1 h ~ muscle pH at 1 hour post mortem, pH 2 h ~ muscle pH at 2 hours post mortem, pH 3 h ~ muscle pH at 3 hours post mortem, pH 4 h ~ muscle pH at 4 hours post mortem, pH 24 h ~ muscle pH at 24 hours post mortem, Temp 1 h ~ the correlation with muscle temperature at 1 hour post mortem, Temp 2 h ~ muscle temperature at 2 hours post mortem, Temp 3 h ~ muscle temperature at 3 hours post mortem, Temp 4 h ~ muscle temperature at 4 hours post mortem, Temp 24 h ~ muscle temperature at 24 hours post mortem

Thompson, Perry, *et al.* (2006) concluded that there is no correlation between energy in the muscle and shear force. The correlation-matrix showing simple correlation coefficients of how tenderness (Warner-Bratzler shear force) correlated with muscle energy status in the production systems evaluated is summarised in Table 4.72. There was a negative correlation between shear force at 1 day post mortem and muscle lactic acid concentrations ($p < 0.05$) (Table 4.72).

Shear force at 1 day post mortem had a weak positive correlation with muscle glycogen ($p < 0.05$) (Table 4.72).

Table 4.72 Correlation-matrix showing simple correlation coefficients of how tenderness (Warner-Bratzler shear force) correlated with muscle energy status in the production systems evaluated

	WBSF 1 day (kg)	WBSF 7 days (kg)	WBSF 14 days (kg)
Lakt1	-0.322*	-0.180*	-0.203*
Lakt2	-0.229*	-0.088	-0.132
Lakt4	-0.296*	-0.057	-0.087
Lakt24	-0.213*	-0.084	-0.033
Glu1	-0.121	-0.146*	-0.104
Glu2	-0.098	-0.117	-0.113
Glu4	-0.155*	-0.148*	-0.121
Glu24	-0.017	-0.066	-0.088
Glyc1	0.149*	0.063	0.026
Glyc2	0.166*	0.070	0.044
Glyc4	0.151*	0.057	0.026
Glyc24	0.094	0.060	0.083

*. Correlation is significant at the 0.05 level (2-tailed).

WBSF 1 day ~ shear force at 1 day post mortem, WBSF 7 days ~ shear force at 7 days post mortem, WBSF 14 days ~ shear force at 14 days post mortem, Lakt1 ~ lactic acid concentration at 1 hour post mortem, Lakt2 ~ muscle lactic acid concentration at 2 hours post mortem, Lakt4 ~ muscle lactic acid concentration at 4 hours post mortem, Lakt24 ~ muscle lactic acid concentration at 24 hours post mortem, Glu1 ~ muscle glucose concentration at 1 hour post mortem, Glu2 ~ muscle glucose concentration at 2 hours post mortem, Glu4 ~ muscle glucose concentration at 4 hours post mortem, Glu24 ~ muscle glucose concentration at 24 hours post mortem, Glyc1 ~ muscle glycogen concentration at 1 hour post mortem, Glyc2 ~ muscle glycogen concentration at 2 hours post mortem, Glyc4 ~ muscle glycogen concentration at 4 hours post mortem, Glyc24 ~ muscle glycogen concentration at 24 hours post mortem

The correlation between the muscle energy metabolites and water holding capacity as well as drip loss is summarised in Table 4.73. Muscle lactic acid had a weak negative correlation with water holding capacity and a weak positive correlation with drip loss ($p < 0.05$) (Table 4.73).

Muscle glycogen concentrations at 1, 2, 4 and 24 hours post mortem had a weak positive correlation with water binding capacity ($p < 0.05$) and a weak negative correlation with drip loss ($p < 0.05$). Muscle ATP concentrations at 2 and 4 hours post mortem had a weak positive correlation with water holding capacity ($p < 0.05$). Glycolytic potential at 1 and 4 hours post mortem had a weak negative correlation with water holding capacity ($p < 0.05$) (Table 4.73).

Table 4.73 Correlation-matrix showing simple correlation coefficients of how muscle energy metabolites correlated with water holding capacity and drip loss in the production systems evaluated

	WBC	Drip loss
Lakt1	-0.151*	0.044
Lakt2	-0.214*	0.124
Lakt4	-0.104	0.186*
Lakt24	-0.049	0.173*
Glu1	-0.094	-0.011
Glu2	-0.155*	0.066
Glu4	-0.095	0.063
Glu24	-0.096	0.051
Glyc1	0.216*	-0.176*
Glyc2	0.228*	-0.148*
Glyc4	0.224*	-0.145
Glyc24	0.188*	-0.151*
ATP1	0.127	-0.063
ATP2	0.181*	-0.082
ATP4	0.175*	-0.126
ATP24	0.127	-0.063
GlyP1	0.151*	-0.163*
GlyP2	0.136	-0.098
GlyP4	0.157*	-0.049
GlyP24	0.135	-0.058

*. Correlation is significant at the 0.05 level (2-tailed).

Lakt 1 ~ muscle lactic acid concentration at 1 hour post mortem, Lakt 2 ~ muscle lactic acid concentration at 2 hours post mortem, Lakt 4 ~ muscle lactic acid concentration at 4 hours post mortem, Lakt 24 ~ muscle lactic acid concentration at 24 hours post mortem, Glu 1 ~ muscle glucose concentration at 1 hour post mortem, Glu 2 ~ muscle glucose concentration at 2 hours post mortem, Glu 4 ~ muscle glucose concentration at 4 hours post mortem, Glu 24 ~ the muscle glucose concentration at 24 hours post mortem, Glyc 1 ~ muscle glycogen concentration at 1 hour post mortem, Glyc 2 ~ the muscle glycogen concentration at 2 hours post mortem, Glyc 4 ~ the muscle glycogen concentration at 4 hours post mortem, Glyc 24 ~ muscle glycogen concentration at 24 hours post mortem, ATP 1 ~ muscle ATP concentration at 1 hour post mortem, ATP 2 ~ the muscle ATP concentration at 2 hours post mortem, ATP 4 ~ muscle ATP concentration at 4 hours post mortem, ATP 24 ~ muscle ATP concentration at 24 hours post mortem, GlyP 1 ~ glycolytic potential at 1 hour post mortem, GlyP 2 ~ glycolytic potential at 2 hours post mortem, GlyP 4 ~ glycolytic potential at 4 hours post mortem, GlyP 24 ~ glycolytic potential at 24 hours post mortem, WBC ~ water holding capacity measured, drip loss ~ drip loss measured

Correlation of muscle temperate with water holding capacity and drip loss is summarised in Table 4.74. There was a weak negative correlation between muscle temperature and water holding capacity ($p < 0.05$). Muscle temperature had a weak positive correlation with drip loss ($p < 0.05$) (Table 4.74).

Table 4.74 Correlation matrix of muscle temperature with water holding capacity and drip loss

	Temp1	Temp2	Temp3	Temp4	Temp24
WBC	-0.095	-0.127	-0.153*	-0.172*	-0.246*
Drip loss	0.230*	0.219*	0.284*	0.272*	0.171*

*. Correlation is significant at the 0.05 level (2-tailed).

WBC ~ water holding capacity measured, drip loss ~ drip loss measured, Temp 1 ~ the correlation with muscle temperature at 1 hour post mortem, Temp 2 ~ muscle temperature at 2 hours post mortem, Temp 3 ~ muscle temperature at 3 hours post mortem, Temp 4 ~ muscle temperature at 4 hours post mortem, Temp 24 ~ muscle temperature at 24 hours post mortem

4.17 Correlation of energy metabolites, muscle pH and muscle temperature with meat colour

The correlation-matrix showing simple correlation coefficients of how meat colour correlated with muscle pH and temperature is summarised in Table 4.75.

Table 4.75 Correlation-matrix showing simple correlation coefficients of how meat colour correlated with various muscle characteristics in the production systems evaluated

	L*	a*	b*	Hue angle	Chroma
pH 1 h	-0.226*	-0.180*	-0.111	-0.048	-0.171*
pH 2 h	-0.230*	-0.210*	-0.169*	0.017	-0.207*
pH 3 h	-0.255*	-0.306*	-0.238*	0.015	-0.299*
pH 4 h	-0.246*	-0.301*	-0.242*	0.031	-0.296*
pH 24 h	-0.325*	-0.377*	-0.396*	0.222*	-0.390*
Temp 1 h	0.147*	0.160*	0.272*	-0.312*	0.187*
Temp 2 h	0.161*	0.058	0.171*	-0.221*	0.084
Temp 3 h	0.186*	0.132	0.245*	-0.260*	0.159*
Temp 4 h	0.131	0.130	0.231*	-0.237*	0.155*
Temp 24 h	0.003	0.063	-0.010	0.126	0.049

*. Correlation is significant at the 0.05 level (2-tailed).

L* ~ meat lightness of a fresh meat sample, a* ~ meat redness of a fresh meat sample, b* ~ meat blueness of a fresh meat sample, Hue angle ~ hue angle of a fresh meat sample, Chroma ~ chroma of a fresh meat sample, pH 1 h ~ muscle pH at 1 hour post mortem, pH 2 h ~ muscle pH at 2 hours post mortem, pH 3 h ~ muscle pH at 3 hours post mortem, pH 4 h ~ muscle pH at 4 hours post mortem, pH 24 h ~ muscle pH at 24 hours post mortem, Temp 1 h ~ muscle temperature at 1 hour post mortem, Temp 2 h ~ muscle temperature at 2 hours post mortem, Temp 3 h ~ muscle temperature at 3 hours post mortem, Temp 4 h ~ muscle temperature at 4 hours post mortem, Temp 24 h ~ muscle temperature at 24 hours post mortem

There was a weak negative correlation between L* coordinate and muscle pH, a* coordinate and muscle pH, b* coordinate and muscle pH, and chroma and muscle pH ($p < 0.05$) (Table 4.75). Wulf and Wise (1999) also concluded that there was a significant negative correlation with muscle

pH, and redness and blueness has a negative correlation with muscle pH. Serra, Gil, *et al.* (2004) and Muchenje, Dzama, *et al.* (2008) concluded that there is a negative correlation between lightness and ultimate pH, but it is not significant.

b* coordinate, hue angle and chroma had a weak positive correlation with muscle temperature ($p < 0.05$). Meat lightness had no correlation with muscle temperature at 24 hours post mortem, and meat redness had no correlation with muscle temperature at 1, 2, 3, 4 and 24 hours post mortem ($p > 0.05$). There was a weak positive correlation between meat lightness and muscle temperature ($p < 0.05$). Meat blueness and chroma had a weak positive correlation with muscle temperature ($p < 0.05$). Hue angle had a weak negative correlation with muscle temperature ($p < 0.05$) (Table 4.75).

Table 4.76 Correlation-matrix showing simple correlation coefficients of how meat colour correlated with various muscle energy status in the production systems evaluated

	L*	a*	b*	Hue angle	Chroma
Glu1	0.048	0.239*	0.138	0.065	0.223*
Glu2	0.106	0.268*	0.167*	0.047	0.253*
Glu4	0.144	0.221*	0.153*	0.016	0.212*
Glu24	0.149*	0.206*	0.186*	-0.048	0.208*
G6P1	0.085	0.093	0.057	0.014	0.088
G6P2	0.134	0.150*	0.133	-0.055	0.150*
G6P4	0.142	0.017	-0.017	0.042	0.010
G6P24	0.129	0.039	0.067	-0.071	0.046
ATP1	-0.075	0.051	0.088	-0.116	0.060
ATP2	-0.120	-0.045	0.021	-0.118	-0.033
ATP4	-0.165*	-0.058	-0.005	-0.077	-0.049
ATP24	-0.166*	0.045	0.021	0.013	0.041

*. Correlation is significant at the 0.05 level (2-tailed).

L* ~ meat lightness of a fresh meat sample, a* ~ meat redness of a fresh meat sample, b* ~ meat blueness of a fresh meat sample, Hue angle ~ hue angle of a fresh meat sample, Chroma ~ chroma of a fresh meat sample, Glu1 ~ muscle glucose concentration at 1 hour post mortem, Glu2 ~ muscle glucose concentration at 2 hours post mortem, Glu4 ~ muscle glucose concentration at 4 hours post mortem, Glu24 ~ muscle glucose concentration at 24 hours post mortem, G6P1 ~ muscle glucose-6-phosphate concentration at 1 hour post mortem, G6P2 ~ muscle glucose-6-phosphate concentration at 2 hours post mortem, G6P4 ~ muscle glucose-6-phosphate concentration at 4 hours post mortem, G6P24 ~ muscle glucose-6-phosphate concentration at 24 hours post mortem, ATP1 ~ muscle ATP concentration at 1 hour post mortem, ATP2 ~ muscle ATP concentration at 2 hours post mortem, ATP4 ~ muscle ATP concentration at 4 hours post mortem, ATP24 ~ muscle ATP concentration at 24 hours post mortem

The correlation-matrix showing simple correlation coefficients of how meat colour correlated with various muscle energy status in the production systems evaluated is summarised in Table 4.76. Serra, Gil, *et al.* (2004) concluded that b* coordinate and hue angle had a significant negative correlation with lactic acid. a* and b* coordinate as well as chroma had a weak positive correlation with muscle glucose concentrations ($p < 0.05$) (Table 7.76).

4.18 Correlation between Warner-Bratzler shear force (meat tenderness) and various meat characteristics

The correlation-matrix showing simple correlation coefficients of how tenderness (Warner-Bratzler shear force) correlated with various carcass characteristics in the production systems evaluated is summarised in Table 4.77.

Shear force had a weak negative correlation with sarcomere length at 1 and 3 days post mortem ($p < 0.05$), therefore the lower the shear force the longer the sarcomere length. Shear force had a weak positive correlation with myofibrillar fragment length at 1, 7 and 14 days post mortem ($p < 0.05$) (Table 4.79), therefore the lower the shear force the shorter the myofibrillar fragment length. Muchenje, Dzama, *et al.* (2008), Muchenje, Dzama, *et al.* (2009a) and Pflander and de Felicio (2009) also concluded that there is a negative correlation between shear force and sarcomere length. Muchenje, Dzama, *et al.* (2008), Muchenje, Dzama, *et al.* (2009a) and Ilian, Bekhit and Bickerstaffe (2004) concluded that there is a positive correlation between shear force and myofibrillar fragment length. Shear force at 1, 7 and 14 days post mortem had no correlation with soluble collagen, insoluble collagen, the total collagen content, and collagen solubility ($p > 0.05$). Silva, Patarata, *et al.* (1999) also concluded that there is no significant correlation between collagen and shear force (Table 4.77).

There was no correlation between shear force at 1, 7 and 14 days post mortem and water holding capacity ($p > 0.05$), and shear force at 1 and 14 days post mortem and drip loss ($p > 0.05$) (Table 4.77). However, Muchenje, Dzama, *et al.* (2009a) concluded that there was a positive correlation between drip loss and shear force. This difference can be explained by a low occurrence of DFD meat in current study (discussed earlier in the dissertation).

Table 4.77 Correlation-matrix showing simple correlation coefficients of how tenderness (Warner-Bratzler shear force) correlated with various carcass characteristics in the production systems evaluated

	WBSF 1 day (kg)	WBSF 7 days (kg)	WBSF 14 days (kg)
WC	-0.096	-0.148*	-0.156*
CC	-0.104	-0.155*	-0.160*
PML	0.320*	0.296*	0.223*
WBC	-0.033	-0.081	-0.052
Drip loss	0.008	0.218*	0.122
Sar1	-0.290*	-0.287*	-0.276*
Sar3	-0.326*	-0.289*	-0.312*
MFL1	0.260*	0.369*	0.268*
MFL7	0.265*	0.393*	0.342*
MFL14	0.248*	0.447*	0.394*

*. Correlation is significant at the 0.05 level (2-tailed).

WBSF 1 day ~ shear force at 1 day post mortem, WBSF 7 days ~ shear force at 7 days post mortem, WBSF 14 days ~ shear force at 14 days post mortem, WC ~ warm carcass weight, CC ~ cold carcass weight, PML ~ percentage weight loss, WBC ~ water binding capacity of fresh meat, Drip loss ~ drip loss post mortem, Sar1 ~ sarcomere lengths at 1 day post mortem, Sar3 ~ sarcomere lengths at 3 days post mortem, MFL1 ~ myofibril fragment length at 1 day post mortem, MFL7 ~ myofibril fragment length at 7 days post mortem, MFL14 ~ myofibril fragment length at 14 days post mortem

The correlation-matrix showing simple correlation coefficients of how tenderness (Warner-Bratzler shear force) correlated with various muscle characteristics in the production systems evaluated is summarised in Table 4.78. Strydom, Frylinck, *et al.* (2009) and Zamora, Debiton, Lepetit, Lebert, Dransfield and Ouali (1996) concluded that there is a positive correlation between shear force and calpastatin and no correlation between shear force and calpain activity. Neath, Del Barrio, Lapitan, Herrera, Cruz, Fujihara, Muroya, Chikuni, Hirabayashi and Kanai (2007) and Ilian, Bekhit, *et al.* (2004) concluded that shear force has a correlation with calpain activity. Ouali and Talmant (1990) concluded that the ratio between calpastatin and calpain have a positive correlation with shear force.

Shear force at 1 day post mortem had a weak negative correlation with calpain I activity at 1 hour post mortem ($p < 0.05$), therefore the lower the shear force the higher the calpain I activity at 1 hour post mortem. Shear force at 1 day post mortem had a weak negative correlation with total calpain II activity at 1 hour post mortem ($p < 0.05$), therefore the lower the shear force the higher the calpain II activity at 1 hour post mortem. Shear force at 1 day post mortem had a weak positive correlation with the ratio between calpastatin/calpain I at 1 hour post mortem ($p < 0.05$), the ratio between calpastatin/calpain I + II at 1 and 24 hours post mortem ($p < 0.05$), and calpastatin activity

at 24 hours post mortem ($p < 0.05$) (Table 4.78), therefore the lower the shear force the lower the ratios.

Table 4.78 Correlation-matrix showing simple correlation coefficients of how tenderness (Warner-Bratzler shear force) correlated with various enzyme characteristics in the production systems evaluated

	WBSF 1 day (kg)	WBSF 7 days (kg)	WBSF 14 days (kg)
TOT IN1h	0.064	0.124	0.183*
TOT IN24h	0.193*	0.164*	0.169*
Spes IN1h	0.152	0.197*	0.253*
Spes IN24h	0.240*	0.233*	0.211*
CAI 1 h	-0.313*	-0.304*	-0.246*
CAI 24 h	0.094	-0.067	-0.090
Spes CAI 1 h	-0.243*	-0.244*	-0.183*
Spes CAI 24 h	0.143	-0.012	-0.054
CAII 1 h	-0.167*	-0.046	0.008
CAII 24 h	-0.079	-0.132	-0.068
Spes CAII 1 h	-0.065	0.042	0.104
Spes CAII 24 h	0.026	-0.012	0.020
INCAI 1 h	0.326*	0.379*	0.359*
INCAI 24 h	0.038	0.165*	0.156*
INCAIII 1 h	0.323*	0.348*	0.326*
INCAIII24 h	0.183*	0.275*	0.254*

*. Correlation is significant at the 0.05 level (2-tailed).

WBSF 1 day ~ shear force at 1 day post mortem, WBSF 7 days ~ shear force at 7 days post mortem, WBSF 14 days ~ shear force at 14 days post mortem, TOT IN1h ~ total calpastatin at 1 hour post mortem, Spes IN1h ~ specific calpastatin at 1 hour post mortem, CAI1h ~ calpain I activity 1 hour post mortem, Spes CAI1h ~ specific calpain I activity 1 hour post mortem, CAII1h ~ calpain II activity 1 hour post mortem, Spec CAII 1 h ~ specific calpain II activity 1 hour post mortem, INCAI1h ~ the ratio between calpastatin and calpain I 1 hour post mortem, INCAIII1h ~ the ratio between calpastatin and calpain I + II 1 hour post mortem, TOT IN24h ~ total calpastatin at 24 hours post mortem, Spes IN24h ~ specific calpastatin at 24 hours post mortem, CAI24h ~ calpain I activity 24 hours post mortem, Spes CAI24h ~ specific calpain I activity 24 hours post mortem, CAII24h ~ calpain II activity 24 hour post mortem, Spes CAII 24 h ~ specific calpain II activity 24 hours post mortem, INCAI24h ~ the ratio between calpastatin and calpain I 24 hours post mortem, INCAIII24h ~ the ratio between calpastatin and calpain I + II 24 hours post mortem

Shear force at 7 and 14 days post mortem had a negative correlation with calpain I activity at 1 hour post mortem ($p < 0.05$), therefore the lower the shear force at 7 and 14 days post mortem the higher the calpain I activity. Shear force at 7 days post mortem had a weak positive correlation with specific calpastatin at 1 and 24 hours post mortem ($p < 0.05$), total calpastatin activity at 24 hours post mortem ($p < 0.05$), the ratio between calpastatin/calpain I at 1 and 24 hours post mortem

($p < 0.05$), and the ratio between calpastatin/calpain I + II at 1 and 24 hours post mortem ($p < 0.05$) (Table 4.78), therefore the lower the shear force at 7 days post mortem the lower the calpastatin activity and ratios.

Shear force at 14 days post mortem had a weak positive correlation with calpastatin activity, the ratio between calpastatin and calpain I ($p < 0.05$), and the ratio between calpastatin and calpain I + II ($p < 0.05$) (Table 4.78), therefore the lower the shear force at 14 days post mortem the lower the activity of calpastatin and ratios.

4.19 Correlation between meat colour and various carcass characteristics

The correlation-matrix showing simple correlation coefficients of how meat colour correlated with various muscle characteristics in the production systems evaluated is summarised in Table 4.79.

a^* coordinate and chroma had no correlation with sarcomere length at 3 days post mortem ($p > 0.05$). Hue angle had a weak negative correlation with sarcomere length ($p < 0.05$). L^* coordinate, a^* coordinate, b^* coordinate and chroma had a weak positive correlation with sarcomere length at 1 day post mortem ($p < 0.05$). L^* and b^* coordinates had a weak positive correlation with sarcomere length at 3 days post mortem ($p < 0.05$) (Table 4.79). Muchenje, Dzama, *et al.* (2008) and Muchenje, Dzama, *et al.* (2009a) concluded that lightness has no significant correlation with sarcomere length and myofibrillar fragment length.

a^* coordinate, b^* coordinate, hue angle and chroma had no correlation with collagen solubility ($p > 0.05$). Meat lightness had a weak positive correlation with collagen solubility ($p < 0.05$) (Table 4.79).

a^* coordinate, b^* coordinate and chroma had a weak negative correlation with shear force at 7 days post mortem ($p < 0.05$). a^* coordinate and chroma had a weak negative correlation with shear force at 14 days post mortem ($p < 0.05$) (Table 4.79). Muchenje, Dzama, *et al.* (2008) and Muchenje, Dzama, *et al.* (2009a) concluded that lightness has no significant correlation with shear force.

Table 4.79 Correlation-matrix showing simple correlation coefficients of how meat colour correlated with various muscle characteristics in the production systems evaluated

	L*	a*	b*	Hue angle	Chroma
WC	-0.093	0.121	0.233*	-0.274*	0.147*
CC	-0.087	0.122	0.236*	-0.277*	0.149*
PML	-0.149*	-0.06	-0.147*	0.181*	-0.080
WBC	-0.421*	-0.084	-0.122	0.120	-0.094
Drip loss	0.178*	0.052	0.102	-0.138	0.063
MFL1	0.088	0.077	0.047	0.008	0.072
MFL7	0.121	-0.109	-0.087	0.001	-0.107
MFL14	-0.038	-0.143	-0.191*	0.136	-0.158*
WBSF 1 d	-0.112	-0.097	-0.096	0.039	-0.100
WBSF 7 d	-0.065	-0.222*	-0.203*	0.060	-0.224*
WBSF 14 d	-0.047	-0.171*	-0.171*	0.070	-0.175*
Sol	0.159*	-0.145	-0.148*	0.079	-0.148*
Insol	-0.015	0.010	-0.100	0.183*	-0.013
Sol/Insol	0.016	-0.017	-0.115	0.176*	-0.038
ColSolper	0.251*	-0.158*	-0.069	-0.072	-0.038

*. Correlation is significant at the 0.05 level (2-tailed).

L* ~ meat lightness of a fresh meat sample, a* ~ meat redness of a fresh meat sample, b* ~ meat blueness of a fresh meat sample, Hue angle ~ hue angle of a fresh meat sample, Chroma ~ chroma of a fresh meat sample, WC ~ warm carcass weight, CC ~ cold carcass weight, PML ~ percentage weight loss, WBC ~ water binding capacity of a fresh meat sample, Drip loss ~ drip loss of a fresh meat sample, MFL1 ~ myofibril fragment length at 1 day post mortem, MFL7 ~ myofibril fragment length at 7 days post mortem, MFL14 ~ myofibril fragment length at 14 days post mortem, WBSF 1 d ~ shear force at 1 day post mortem, WBSF 7 d ~ shear force at 7 days post mortem, WBSF 14 d ~ shear force at 14 days post mortem, Sol ~ soluble collagen, Insol ~ insoluble collagen, Sol/insol ~ total collagen, ColSolper ~ collagen solubility

L* and b* coordinates had a weak negative correlation with water binding capacity ($p < 0.05$). There was a weak positive correlation between hue angle and water holding capacity ($p < 0.05$) (Table 4.79). Muchenje, Dzama, *et al.* (2008) concluded that there is no significant correlation between lightness and drip loss. There was a positive correlation between drip loss and L* coordinate ($p < 0.05$). Serra, Gil, *et al.* (2004) concluded that hue angle, chroma and b* coordinate had a negative correlation with water binding capacity.

The correlation-matrix showing simple correlation coefficients of how meat colour correlated with various muscle enzyme and inhibitors activity in the production systems evaluated is summarised in Table 4.80.

Table 4.80 Correlation-matrix showing simple correlation coefficients of how meat colour correlated with various muscle enzyme and inhibitors activity in the production systems evaluated

	L*	a*	b*	Hue angle	Chroma
TOT IN1h	0.222*	-0.035	0.095	-0.224*	-0.009
TOT IN24h	0.161*	-0.186*	-0.070	-0.129	-0.167*
Spes IN1h	0.310*	-0.092	0.055	-0.242*	-0.064
Spes IN24h	0.229*	-0.215*	-0.095	-0.126	-0.196*
CAI 1 h	0.068	0.009	0.029	-0.039	0.013
CAI 24 h	-0.080	-0.123	-0.07	-0.031	-0.115
Spes CAI 1 h	0.163*	-0.043	0.006	-0.08	-0.034
Spes CAI 24 h	-0.124	-0.104	-0.053	-0.034	-0.096
CAII 1 h	0.088	-0.027	0.011	-0.049	-0.020
CAII 24 h	-0.043	-0.153*	-0.109	0.002	-0.148*
Spes CAII 1 h	0.219*	-0.083	-0.012	-0.097	-0.070
Spes CAII 24 h	0.096	-0.188*	-0.128	-0.02	-0.180*
INCAI 1 h	0.111	-0.056	0.021	-0.121	-0.042
INCAI 24 h	0.258*	0.002	0.024	-0.054	0.007
INCAIII 1 h	0.126	-0.047	0.048	-0.157*	-0.028
INCAIII24 h	0.289*	-0.07	0.012	-0.133	-0.054

*. Correlation is significant at the 0.05 level (2-tailed).

L* ~ meat lightness of a fresh meat sample, a* ~ meat redness of a fresh meat sample, b* ~ meat blueness of a fresh meat sample, Hue angle ~ hue angle of a fresh meat sample, Chroma ~ chroma of a fresh meat sample, TOT IN1h ~ total calpastatin at 1 hour post mortem, Spes IN1h ~ specific calpastatin at 1 hour post mortem, CAI1h ~ calpain I activity 1 hour post mortem, Spes CAI1h ~ specific calpain I activity 1 hour post mortem, CAII1h ~ calpain II activity 1 hour post mortem, Spes CAII 1h ~ specific calpain II activity 1 hour post mortem, INCAI1h ~ the ratio between calpastatin and calpain I 1 hour post mortem, INCAIII1h ~ the ratio between calpastatin and calpain I + II 1 hour post mortem, TOT IN24h ~ total calpastatin at 24 hours post mortem, Spes IN24h ~ specific calpastatin at 24 hours post mortem, CAI24h ~ calpain I activity 24 hours post mortem, Spes CAI24h ~ specific calpain I activity 24 hours post mortem, CAII24h ~ calpain II activity 24 hour post mortem, Spes CAII 24h - specific calpain II activity 24 hours post mortem, INCAI24h ~ the ratio between calpastatin and calpain I 24 hours post mortem, INCAIII24h ~ the ratio between calpastatin and calpain I + II 24 hours post mortem

L* coordinate had a weak positive correlation with calpastatin activity at 1 and 24 hours post mortem, specific calpain I at 1 hour post mortem, specific calpain II activity at 1 hour post mortem, the ratio between calpastatin and calpain I at 24 hours post mortem and the ratio between calpastatin and calpain I + II at 24 hours post mortem ($p < 0.05$) (Table 4.80).

a* coordinate had a weak positive correlation with calpastatin activity at 24 hours post mortem and calpain II activity at 24 hours post mortem ($p < 0.05$) (Table 4.80).

Hue angle did not correlate with calpain I activity at 1 and 24 hours post mortem, calpain II activity at 1 and 24 hours post mortem, the ratio between calpastatin and calpain I at 1 and 24 hours post mortem, and total calpastatin activity at 24 hours post mortem. Hue angle had a weak negative correlation with total calpastatin at 1 hour post mortem, specific calpastatin activity at 1 and 24 hours post mortem, and the ratio between calpastatin and calpain I + II at 1 and 24 hours post mortem ($p>0.05$) (Table 4.80).

Chroma had no correlation with calpastatin activity at 1 hour post mortem, calpain I activity at 1 and 24 hours post mortem, the ratio between calpastatin and calpain I at 1 and 24 hours post mortem, and the ratio between calpastatin and calpain I + II at 1 and 24 hours post mortem ($p>0.05$). Chroma had a weak negative correlation with calpastatin activity at 24 hours post mortem and calpain II activity at 24 hours post mortem ($p<0.05$) (Table 4.80).

CHAPTER 5

CONCLUSION

The aim of the study was to determine the effect of beef production system on muscle energy status post mortem and its effects on meat tenderness and colour. This study showed that the production system had a small effect on the muscle energy status of meat from cattle slaughtered under ideal commercial conditions in South Africa, while production system had a significant effect on meat tenderness and colour.

This study was done under optimal slaughter conditions according to results from previous studies on ideal slaughter conditions. Both feedlot and pasture animals received non-aggressive implants (Zeraplix-Intervet) at specified finishing periods to further stimulate standard practices in commercial feedlots and pasture practices. The results of this experiment should not be influenced by this practice. Stress before slaughter was minimized which decreased the occurrences of extreme results. Electrical stimulation for optimal meat quality was used as determined by previous studies. Electrical stimulation is normally used to deplete some of the glucose in the muscles to prevent poor meat quality as a result of cold shortening. Due to the use of electrical stimulation there were probably smaller differences between the muscle energy values in different production systems.

It would seem that Nguni cross bred animals had the highest occurrence of DFD (dark, firm dry) ($\text{pH} > 5.8$) phenomena compared to Brahman and Simmental cross bred animals, although the overall incidence of DFD in this study was low. AP production system animals showed a higher percentage of DFD compared to the other production systems (ABP, BP, AF and ABF).

Out of this study it can be concluded that the production system has an effect on the muscle energy status post mortem, with younger animals from the feedlot having a higher glycolytic potential, muscle lactic acid concentration, muscle glucose-6-phosphate concentration and muscle ATP concentration, compared to older animals from the pasture ($p < 0.05$). Younger animals from the pasture had the highest muscle glucose concentrations and muscle glycogen concentration post mortem compared to older animals from the feedlot ($p < 0.05$). Oldest animals from the pasture had the highest muscle creatine phosphate concentrations compared to younger animals from the feedlot ($p < 0.05$).

Breed had no effect on the muscle energy status for muscle glycolytic potential, muscle lactic acid concentration, muscle glucose concentration, muscle glycogen concentration, muscle glucose-6-phosphate concentration, muscle ATP concentration and creatine phosphate concentration ($p > 0.05$).

Nguni cross bred animals had the tendency for the lowest glycolytic potential and Simmental cross bred animals had the tendency for the highest glycolytic potential ($p>0.05$). Nguni cross bred animals had the tendency for the highest muscle lactic acid concentrations and muscle glucose-6-phosphate concentrations ($p>0.05$). Brahman cross bred animals had the tendency for the highest muscle glucose concentrations, muscle glycogen concentrations and muscle ATP concentrations ($p>0.05$). Simmental cross bred animals had the highest muscle creatine phosphate concentrations ($p>0.05$).

Simmental cross bred animals from the ABF production system had the lowest muscle glucose concentration. Brahman and Simmental cross bred animals from the AP production system had the highest muscle glucose concentration. There were only differences between Simmental cross bred animals from the ABF production system and the Brahman and Simmental cross bred animals from the AP production system ($p<0.05$).

Production system had an effect on the tenderness of the meat ($p<0.05$). Feeding system as well as age of the animals had an influence on meat tenderness. Breed only had an influence on the tenderness at 7 days post mortem ($p<0.05$). Animals from the ABF production system had the most tender meat and the animals from the AP production system had the toughest meat ($p<0.05$). Overall Nguni and Simmental cross bred animals had the most tender meat as was expected for *Bos taurus Africanus* and *Bos taurus* breeds.

Production system had an effect on a^* coordinate, b^* coordinate and colour saturation, but not on L^* coordinate and hue angle ($p<0.05$). The effect of the production system on the b^* coordinate was very small, meat from the AF production system had a higher value than the other production systems (ABF, AP, ABP and BP) ($p<0.05$). Feeding system influenced the b^* coordinate, L^* coordinate, colour saturation and hue angle ($p<0.05$), but not a^* coordinate ($p>0.05$). Age of the animals influenced the a^* coordinate, L^* coordinate, b^* coordinate, colour saturation and hue angle ($p<0.05$). Breed only influenced hue angle ($p<0.05$).

Older animals from the pasture had a darker colour meat, animals from the AF production system had a higher a^* and b^* value, animals from the ABF production system had a lower a^* and b^* value, older animals from the pasture had the highest hue angle and youngest animals from the feedlot had the highest colour saturation ($p<0.05$). Simmental cross bred animals had the lowest L^* and b^* coordinate values, and Brahman cross bred animals the highest L^* and a^* values. Nguni cross bred animals had the lowest b^* value and Brahman cross bred animals had the highest b^* value ($p<0.05$). Brahman and Simmental cross bred animals had the lowest hue angle ($p<0.05$). Simmental cross bred animals had the highest colour saturation and Nguni cross bred animals the lowest chroma ($p<0.05$).

This study showed that the energy status in the muscle post mortem does not influence the tenderness of the meat nor the colour ($p>0.05$). Shear force had a medium positive correlation with

muscle pH and a medium negative correlation with muscle temperature ($p < 0.05$). Muscle lactic acid concentrations, muscle glucose concentrations and sarcomere length had a weak negative correlation with shear force ($p < 0.05$). Muscle glycogen concentrations and myofibrillar fragment length had a medium positive correlation with shear force ($p < 0.05$). Shear force had a weak positive correlation with calpastatin activity and a weak negative correlation with calpain I activity ($p < 0.05$). The ratio of calpastatin: calpain I and the ratio of calpastatin: calpain I + II had a medium positive correlation with shear force ($p < 0.05$).

Muscle pH had a weak negative correlation with meat lightness, a^* coordinate, b^* coordinate and chroma ($p < 0.05$).

There was a weak positive correlation between meat lightness and muscle temperature ($p < 0.05$). b^* coordinate and chroma had a weak positive correlation with muscle temperature. Muscle temperature had a weak negative correlation with hue angle ($p < 0.05$).

Shear force had a weak to medium negative correlation with a^* coordinate, b^* coordinate and chroma ($p < 0.05$), therefore shear force and colour influence one another to a small degree, because the same factors that influence colour also influence the shear force.

The absence of correlations between the calpain and calpastatin measurements and muscle energy status could mean that under these slaughter conditions, the calpain system tenderisation was not influenced by the energy status of the muscle. The correlation of muscle energy status and meat colour with sarcomere length and shear force could suggest a relationship with the muscle contraction tenderness.

If electrical stimulation was not used in this study, the difference between the production systems in terms of muscle energy status and colour would have been more prominent. The conclusion is that if animals are slaughtered under “ideal” circumstances, in terms of stress being kept to a minimum before slaughter, and the carcasses are electrically stimulated in order to prevent cold shortening, the production system shows a small effect on the energy status of the animal. This has the consequence that the meat quality characteristics such as tenderness and colour, levels out. For more dramatic results and academic value it would have been more useful to include more variations of non-ideal slaughter conditions and non-electrical stimulation, as well as more breeds. A follow up study with no electrical stimulation can be helpful to explain some uncertainties. Such a follow up study will present its own challenges for example a higher frequency of DFD.

A follow up study on the effects (namely the tenderness and colour of the meat) of a larger variety of breeds can help to determine the exact effect of muscle energy metabolites in the different breeds.

CHAPTER 6

CRITICAL EVALUATION

The experiment was designed to study all factors associated with production systems that influence meat quality, specifically focusing on meat tenderness and colour. The following variables were included, namely, body temperature, muscle pH, sarcomere length, myofibrillar fragment length, collagen content and solubility, calpain and calpastatin activity, water holding capacity, drip loss, muscle lactic acid concentration, muscle glucose concentration, muscle glycogen concentration, muscle glucose-6-phosphate concentration, muscle ATP concentration and muscle creatine phosphate concentration.

Brahman and Simmental cross bred animals are used mostly in the South African feedlot industry for beef production. The Nguni cross bred animals were included in this study as an example of an indigenous breed. From an academic point of view it would have been more interesting to use the pure breeds instead of the cross bred animals in order to draw clear conclusions about breed effects. This experiment was designed in the interest of the meat industry, which predominantly uses cross bred animals.

The methods used to determine or measure the different variables are the same as those used by other researchers. The results found in this study agree with those reported in the literature, and adds to our current understanding of the effects of production system on muscle energy status post mortem and resulting effects on meat tenderness and colour of beef cattle.

Other variables such as the effect of non-aggressive beta-agonist supplementations (which is used on regular basis in the South African feedlots), should also be evaluated.

CHAPTER 7

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APPENDIX A



Classification of Red Meat*

A key to more effective marketing



Classification provides a sound basis for:

- Meat traders to describe their specific requirements in simple terms when purchasing carcasses.
- Utilisation of variety in the market with a view to optimum consumer satisfaction.
- Utilisation of price differences.
- Determining selling prices.

CLASSIFICATION CHARACTERISTICS OF: Beef, Lamb, Sheep and Goat Meat

AGE	CLASS	CONFORMATION	CLASS
0 Teeth	A	Very flat	1
1-2 Teeth	AB	Flat	2
3-6 Teeth	B	Medium	3
More than 6 Teeth	C	Round	4
		Very round	5
FATNESS	CLASS	DAMAGE	CLASS
No fat	0	Slight	1
Very lean	1	Moderate	2
Lean	2	Severe	3
Medium	3		
Fat	4		
Slightly overfat	5		
Excessively overfat	6		

SEX
The carcass of a ram or a bull as well as of a hamel, a kapater or an ox showing signs of late castration of the AB, B or C age classes, are identified.

CLASSIFICATION CHARACTERISTICS OF: Pork

% MEAT*	mm**	CLASS	CONFORMATION	CLASS
≥70	≤12	P	Very flat	1
68-69	13-17	O	Flat	2
66-67	18-22	R	Medium	3
64-65	23-27	C	Round	4
62-63	28-32	U	Very round	5
≤61	>32	S		
% MEAT*		DAMAGE		CLASS
No specifications in respect of % meat apply in the case of Rough, Sucking pig (≤20 kg) and Sausage pig (≥100.1 kg).		Slight		1
** In case of Intrascopie.		Moderate		2
		Severe		3
FAT THICKNESS**		SEX		
% MEAT*		The carcass of a boar as well as of a barrow showing signs of late castration, are identified.		
Only in the case of the Hennessy classification apparatus.				

MARKS FOR CLASSIFICATION CHARACTERISTICS ON: Beef, Lamb, Sheep and Goat Carcasses

TRAIT	MARK	WHERE ON THE CARCASS
Age (A, AB, B, C)		One mark on each quarter of beef carcass.
Fatness* (0 to 6)		Only one mark on the carcass for lamb, sheep and goat carcasses.
Conformation (1 to 5)		One mark on each side for beef carcasses. No mark for lamb, sheep and goat carcasses.
Damage** (1 to 3)		Taking into account the area of damage, one mark on each side for beef carcasses. Only one mark on the carcass for lamb, sheep and goat carcasses.
Sex.		One mark on each side for beef carcasses. Only one mark on the carcass for lamb, sheep and goat carcasses.

* In case of a sheep carcass with a fat tail, a double impression of the mark.

** Damage, if it occurs, is indicated on a scale of one to three for the areas concerned, viz B (buttock), L (loin) and F (forequarter).

EXAMPLES OF THE ROLLER-MARK COMPOSITIONS FOR: Beef, Lamb, Sheep and Goat Carcasses*

AAA	ABAB	BBB	CCC	- Age class of the animal as an indication of tenderness.
ZWZ	ZWZ	ZWZ	ZWZ	The A age class is roller-marked in purple (most tender), AB carcasses are in green (tender), B in brown (less tender) and C in red (least tender).
AAA	ABAB	BBB	CCC	
OOO	OOO	OOO	OOO	- Fatness class** of the carcass.
ZWZ	ZWZ	ZWZ	ZWZ	This symbol can be replaced in the roller-mark by 111, 222, 333, 444, 555 or 666.
AAA	ABAB	BBB	CCC	
OOO	OOO	OOO	OOO	
ZWZ	ZWZ	ZWZ	ZWZ	- Abattoir-identification code.

* All goat carcasses are roller-marked in orange, taking into account the age of the animal (AAA, ABAB, BBB or CCC).

** The amount of visible fat can be evaluated by the consumer and selected according to preference.

MARKS FOR CLASSIFICATION CHARACTERISTICS ON: Pork Carcasses

TRAIT	MARK	WHERE ON THE CARCASS
Conformation (1 to 5)		One mark on each side.
Damage* (1 to 3)		Taking into account the area of damage, only one mark on the carcass.
Sex		One mark on each side.

* Damage, if it occurs, is indicated on a scale of 1 to 3 for the areas concerned, viz B (buttock), L (loin) and F (forequarter).

MARKS FOR CLASSES OF PORK:*

CLASS	MARK	WHERE ON THE CARCASS
Sucking pig	S	One mark on forehead.
P,O,R,C,U & S	P,O,R,C,U & S	One mark on each side.
Sausage Pig	W	One mark on each buttock.
Rough	RU	One mark on each side.

* The class of a pig carcass is not roller-marked on it. Some pig carcasses may be roller-marked in purple ink with a specific abattoir-identification code/trademark.

SOUTH AFRICAN MEAT INDUSTRY COMPANY (SAMIC)

318 The Hillside, Lynnwood, Pretoria 0081

PO Box 36802, Menlo Park, 0102

Tel: +27 (12) 361 4545 • Fax: +27 (12) 361 9837

* Meat Classification Regulations No. R. 863 in Government Gazette of The Republic of South Africa, 1 September 2006.