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Genetic and environmental effects on the muscle structure response post-mortem

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Abstract

This paper reviewed the mechanisms by which glycolytic rate and pre-rigor stretching of muscle impact on meat quality. If muscle is free to shorten during the rigor process extremes in glycolytic rate can impact negatively on meat quality by inducing either cold or rigor shortening. Factors that contribute to variation in glycolytic rate include the glycogen concentration at slaughter and fibre type of the muscle. Glycolysis is highly sensitive to temperature, which is an important factor in heavy grain fed carcasses. An alternative solution to controlling glycolysis is to stretch the muscle pre-rigor so that it cannot shorten, thus providing an insurance against extremes in processing conditions. Results are presented which show a large reduction in variance (both additive and phenotypic) in tenderness caused by pre-rigor stretching. Whilst this did not impact on the heritability of shear force, it did reduce genotype differences. The implications of these results on the magnitude of genotype effects on tenderness is discussed.
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Keywords: Tenderness; Glycolytic rate; Tenderstretch; pH decline; Cold shortening; Rigor shortening

1. Introduction

Koohmaraie, Kent, Shackelford, Veiseth, and Wheeler (2002) proposed that the tenderness of muscle can be considered as a function of three main components; the connective tissue content/composition, sarcomere length and the extent of proteolysis of the myofibrillar proteins. The relative contribution of these three components to ultimate tenderness will vary with muscle, animal, pre- and post-slaughter factors and the length of time and temperature at which the product is stored post-mortem. The consensus of several reviews (e.g. Ouali, Demeyer, & Raichon, 1992; Sentandreu, Coulis, & Ouali, 2002) was that the amount and chemical composition of connective tissue was largely a function of the age of the animal at the time of slaughter and should be considered as 'background toughness'. Whilst Purslow (2005) has challenged this view in the post-genomic era, there are currently few treatments that allow the contribution of connective tissue to tenderness to be manipulated.

An optimal slaughter process could be considered as one that during the post-mortem period maximized the degree of proteolysis and/or minimized shortening, or even

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stretched the muscle, during the rigor process. Whilst there has been much documented about the mechanisms involved in proteolysis (Hopkins & Thompson, 2002) and the effect of minimizing shortening, or stretching muscle pre-rigor (Tornberg, 1996), there has been little discussion of the magnitude of either environmental and genetic manipulations on these processes. This paper will briefly review the mechanisms by which these two events impact on tenderness and examine the evidence of genetic or environmental effects on these processes. It is by understanding these interactions that progress will be made in the improvement of tenderness.

2. Early post-mortem metabolism

After death, the muscle continues to metabolise. Initially energy is supplied by ATP and creatine phosphate within the muscle. With the cessation of blood circulation at death the muscle quickly becomes anaerobic and the muscle glycogen is metabolized to replace ATP reserves. Circulatory failure means that waste products can no longer leave the tissue and the subsequent build up of lactate and the associated hydrogen ions gradually lower the pH of the muscle from neutrality to mildly acidic (Marsh, 1993). Glycolysis is eventually halted by either depletion of the substrate glycogen, or when the pH of the muscle becomes acidic enough to deactivate the enzymes associated with post-mortem glycolysis (Lawrie, 1992).

2.1. The onset of rigor

With the cessation of glycolysis, ATP reserves within the muscle are no longer replenished and as the muscle continues to metabolise these ATP reserves will be depleted. Rigor mortis in the muscle is defined as the stage when ATP supplies are depleted (Bendall, 1969). As discussed by Hwang, Devine, and Hopkins (2003) the onset of rigor does not occur simultaneously across all muscle fibres, but rather in individual fibres as they become depleted in ATP. This produces successive rigor contractions in individual fibres that gradually increase overall muscle stiffness (Honikel, Roncales, & Hamm, 1983; Jeacocke, 1984). As the number of fibres entering rigor increases the stiffness of the muscle as a whole increases and is significant when the muscle reaches a pH of *ca.* 6.

The temperature at which the muscle enters rigor will impact on the degree of muscle shortening. The classic study by Locker and Hagyard (1963) showed that minimal shortening occurred when muscle samples were held at approximately 15 °C. As discussed by Hwang et al. (2003), for temperatures above this optimum, fibre contracture occurs at rigor, whilst at temperatures below optimal the fibre contracture occurs before rigor. Thus, shortening above 15 °C is a consequence of rigor shortening and occurs when the muscle fibres are depleted of ATP, whereas at temperatures below 15 °C pre-rigor contracture takes place until rigor is completed.

2.2. Cold shortening

Cold shortening caused by pre-rigor contracture is driven by increased cellular calcium as falling temperatures cause increased inactivation of the ATP-driven calcium pumps of the sarcoplasmic reticulum (Bendall, 1978; Honikel & Hamm, 1978) and increased release of Ca^{2+} through leakage from the sarcoplasmic reticulum (Kanda, Pearson, & Merkel, 1977; Pearson, Carse, Davey, Locker, & Hagyard, 1973). The increased cellular calcium stimulates the binding between actin and myosin whilst activating the Ca^{2+}-dependant myosin ATPase, which hydrolyses ATP to energise the muscle contraction.

2.3. Rigor shortening

At the other extreme, muscles maintained at higher temperatures for long periods post-mortem tend to have greater glycolytic rates due to increased activity of glycolytic enzymes and, thus, a faster rate of pH decline. Accelerated pH decline combined with high muscle temperature can induce a form of shortening known as heat shortening or rigor shortening (Lee & Ashmore, 1985). At the higher muscle temperatures increased cellular calcium, induced by the onset of rigor, initiates muscle contraction in a similar fashion to that of cold shortening. However, mitochondrial release of calcium ions will not occur if ATP is still available (Mickelson, 1983) and therefore the contraction associated with heat shortening occurs at the onset of rigor rather than before (Hwang et al., 2003). Due to a lack of ATP to energise the contraction it is far less severe than that of cold shortening.

2.4. Post-mortem proteolytic activity

The activity of cysteine protease enzymes, in particular the calpains, has been shown to be sensitive to the prevailing pH and temperature of the meat (Dransfield, 1994b). Calpains are Ca^{2+} dependent proteases that specifically attack certain proteins of the Z-line and are considered primarily responsible for the degradative changes which occur during post-rigor conditioning at low temperatures (Gault, 1992). Calpains not only digest important structural proteins (Taylor, Geesink, Thompson, Koohmaraie, & Goll, 1995) but also themselves and their inhibitor calpastatin and the extent of tenderisation is dependant upon net proteolysis resulting from the activity and inactivation of calpains (Tornberg, 1996).

It has been shown that the combination of low pH and high temperature conditions pre-rigor favour the autolysis and reduction of μ-calpains, which subsequently reduces ageing potential (Dransfield, 1994a; Ducastaing, Valin, Schollmeyer, & Cross, 1985; Geesink, van Laack, Barnier, & Smulders, 1994; Hwang & Thompson, 2001). When chilling was slow, the activity of μ-calpain and calpastatin decreased with a rapid pH decline, whereas when the chilling was rapid, the activity of the μ-calpain was largely

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