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Exploring the unknowns involved in the transformation of muscle to meat

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ABSTRACT

Meat quality development, or the transformation of muscle to meat, involves a myriad of biochemical pathways that are largely well-studied in living muscle tissue. However, these pathways are less predictable when homeostatic ranges are violated. In addition, there is far less known about how various management or environmental stimuli impact these pathways, either by substrate load or altered cellular environment. Likewise, it is largely accepted that oxygen plays little to no role in the conversion of muscle to meat, as anaerobic metabolism predominates in the muscle tissue. Even so, the oxygen tension within the tissues does not fall precipitously at exsanguination. Therefore, transition to an anaerobic environment may impact energy metabolism postmortem. Antemortem handling, on the other hand, clearly impacts meat quality development, yet the exact mechanisms remain a mystery. In this paper, we will attempt to review those factors known to affect postmortem energy metabolism in muscle and explore those areas where additional work may be fruitful.

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1. Introduction

Postmortem metabolism is a heavily researched topic. Many scientific and technological advances have led to improved animal welfare, feeding strategies, slaughter processes, and resulted in improved meat quality development for the consumer. Despite a myriad of advances, questions remain regarding those mechanisms controlling or impacting postmortem metabolism and how physiological and tissue-based homeostatic set points are maintained or breeched by various management practices that ultimately lead to altered meat quality development. Though the rate of postmortem metabolism is quite important in driving meat quality development, it is fairly well established. Alternatively, the biochemical mechanism(s) responsible for the cessation of postmortem metabolism, or protracted carbohydrate metabolism are particularly puzzling. The role of mitochondria and curtailed oxidative metabolism play in modulating postmortem metabolism will also be explored. Finally, we will briefly review antemortem animal handling practices in an effort to understand how these management practices alter the aforementioned.

2. Cessation of postmortem metabolism

In order to understand those mechanisms that may control an abbreviated or protracted postmortem metabolism in muscle, one must first reason why it stops. To date, this has not been unequivocally established. Though some would argue it is simply a function of glycogen abundance at harvest, this is not the case, especially when extreme deviates are removed from the population (Copenhafer, Richert, Schinckel, Grant, & Gerrard, 2006; Scheffler & Gerrard, 2007; Scheffler, Park, & Gerrard, 2011). Over sixty years have elapsed since meat scientists across the globe have known that some muscle, for whatever reason, is capable of breaching a final pH, which is otherwise relatively constant across myriad of animals managed and processed under a variety of conditions, yet little progress or even interest exists in this area. Essentially, there are two viable hypotheses, either there is a pH-mediated inactivation of glycolytic enzymes, which stops hydrogen accumulation at a constant endpoint, or there is loss of adenosine nucleotides preventing a glycolytic substrate to rephosphorylate (Dalrymple & Hamm, 1975; Greaser, 2001). In an attempt to stimulate or re-kindle an interest in this fascinating biochemical process, we will begin by reviewing the collective works of one of the great pioneers of postmortem metabolism, Robert K. Scopes.

Scopes and Lawrie (1963) first entered the area of postmortem metabolism by noting that an accelerated rate of postmortem metabolism resulted in denatured sarcoplasmic proteins and adulterated meat quality development. They predicted at that time that the antemortem 'state' of the animal likely dictated the pH decline in these muscle tissues (Scopes & Lawrie, 1963) and quickly extended these initial observations to show that differences in muscle temperature and pH combinations indeed altered the extractability, or solubility of sarcoplasmic, and myofibrillar muscle proteins (Scopes, 1964). These early data likely formed the foundation for a number of subsequent studies over the next 50 years based on the premise that enzymes, and other proteins, denature with time postmortem (Joo, Kauffman,

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Kim, & Park, 1999; Warner, Kauffman, & Greaser, 1997), and represent events now commonly known as the conversion of muscle to meat. Most likely because of animal to animal variation, Scopes then modified his approach and used minced muscle to study postmortem metabolism. Using this approach, Newbold and Scopes (1971) showed that varying the concentrations of inorganic phosphate (P_i) up to 50 mM reduced the ultimate pH (pH_u) of the mince, yet concentrations greater than 50 mM did not further facilitate greater declines in the metabolizing tissue preparation. Based on these observations, they proposed that P_i may induce greater glycogen phosphorylase (GP) activity, thereby explaining the lower pH_u observed in some muscles. This hypothesis is particularly intriguing given the intimate relationship between the phosphagen system and energy metabolism in exercising muscle (Robergs, Ghiasvand, & Parker, 2004). Recall, GP is present in muscle in two forms, *a* and *b*, the former being more active than the latter (Berg, Tymoczko, & Stryer, 2007). GP is activated by AMP and inhibited by ATP and glucose 6-phosphate. Both calcium and epinephrine are capable of shifting the inactive form *b* to the active *a* form by stimulating phosphorylase kinase. Of particular significance is that though active, GP cannot generate glucose 1-phosphate without inorganic phosphate (Morgan & Parmeggianiani, 1964). Given that added inorganic phosphate to a muscle mince caused a lower pH_u in glycolysing muscle preparations (Newbold & Scopes, 1971), it is possible to argue that inorganic phosphate may be rate-limiting during particular times postmortem metabolism. Addition of inorganic phosphate, on the other hand, would undoubtedly raise the pH of the mince early and ultimately could result in a higher pH of the reaction. After all, liberation of free phosphate via ATP hydrolysis, especially in heavily exercising muscle, is known to buffer muscle cells against massive hydrogen accumulation (Robergs, 2001). Regardless, defining the role of phosphate, especially free phosphate in postmortem muscle, may be quite enlightening.

Scopes further refined his in vitro system to include a glycolysing mixture (glycogen, ATP, NAD, P_i, etc.), organic buffers (TRIS, acetate), and purified glycolytic enzymes (Scopes, 1973). The composition of the buffer is particularly germane to the issue at hand, as buffer capacity of muscle can dramatically impact the pH_u and quality of meat (Kylä-Puhju, Ruusunen, Kivikari, & Puolanne, 2004; van Laack, Kauffman, & Greaser, 2001). Using this system, Scopes (1973) documented the extent resting muscle could rephosphorylate creatine dependent upon available inorganic phosphate, GP *a* concentration, and ATPase activity. He then showed that the rate of glycolysis is directly proportional to the amount of ATP consumed (Scopes, 1974a). Specifically, when ATPase activity was stimulated, lactate formation was increased proportionally. In addition, he noted that glycolysis stopped once adenonucleotides were metabolized. In contrast, when ATPase, or ATP consumption, was reduced or minimized, the entire system was capable of maintaining ATP concentrations in a steady-state condition, where minimal AMP is detected. As a result, a slower metabolism ensued. These results are particularly interesting as they argue that energy levels such as: phosphocreatine, ATP or the ability of ATP to be rephosphorylated (see discussion below) in the muscle tissue at harvest may shift the time at which glycolysis may begin, or even reach maximal levels. Changes in the time at which these events occur postmortem could have dramatic effects on ultimate meat quality development, as protein denaturation again is a pH-temperature phenomenon (Offer, 1991; Wismer-Pedersen, 1959). Furthermore, these data show theoretically, that removal of adenonucleotides from glycolysing muscle will arrest metabolism raising another point of control that will be discussed briefly below.

His final and arguably the most important data using this in vitro system, were those directly targeted at understanding the enzymes responsible for pacing both early postmortem metabolism and that responsible for extending carbohydrate metabolism in skeletal muscle (Scopes, 1974b). These data showed that regardless of enzymes present, ATPase concentration, or ATP consumption, drives the rate of metabolism. These findings formed the basis by which many understand the role ATPase plays in controlling the rate of postmortem metabolism (Bowker, Grant, Swartz, & Gerrard, 2004; Hamm, 1977), especially where inherent differences in ATP consumption found between muscles of different fiber types change postmortem metabolism (Fernandez & Tornberg, 1991; Klont, Brocks, & Eikelenboom, 1998). Moreover, early postmortem consumption of ATP in muscle is hallmark of halothane-positive pigs containing a mutated calcium channel protein that allows cellular calcium concentrations to rise to a point where corresponding downstream ATPases force an aggressive metabolism and aberrant meat quality development (Cheah, Cheah, Crosland, Casey, & Webb, 1984; Greaser, Cassens, Briskey, & Hoekstra, 1969; Monin, Sellier, Ollivier, Goutepongea, & Girard, 1981). Results of these studies also raised the idea that GP *a*, and to a lesser extent AMP deaminase concentrations may dictate the pH at which metabolism stops (Scopes, 1974b).

Understanding how Scopes settled on GP *a* as a driver of pH_u in porcine skeletal muscle is logical though difficult to explain in living, or dying muscle. In his studies (Scopes, 1974b), GP *a* concentrations were included at sub- and supra-physiological levels. While the increased levels of GP *a* resulted in a reduced pH_u in vitro, it also accelerated the rate of pH decline, begging the question of whether phosphorylase differs between pigs with altered muscle metabolism. GP *a* activity in resting halothane positive pig muscle is lower compared to wild-type pigs (Fernandez, Neyraud, Astruc, & Sante, 2002), yet increases above that of wild-type animals during postmortem metabolism (Monin, Talmant, Laborde, Zabari, & Sellier, 1986). This increase in phosphorylase *a* content is likely due to the calcium-mediated activation of phosphorylase kinase, the enzyme responsible for the conversion of GP *b* to *a* form (Meyer, Fischer, & Krebs, 1964). Alternatively, it could be due to increased catecholamine release and action at harvest (Althen, Ono, & Topel, 1977). However, classically halothane pigs do not differ in pH_u (Copenhafer et al., 2006; De Smet et al., 1996; Fernandez et al., 2002; Klont, Lambooy, & van Logtestijn, 1993; Kocwin-Podsiadla, Przybylski, Kuryl, Talmant, & Monin, 1995) which argues against GP *a* content driving the pH_u of meat. Even so, a number of investigators have shown that meat of halothane positive pigs results in a lower pH_u (Fisher, Mellett, & Hoffman, 2000; Hamilton, Ellis, Miller, McKeith, & Parrett, 2000; Klont & Lambooy, 1995; Klont, Lambooy, & van Logtestijn, 1994; Monin et al., 1981). The latter issue makes it difficult to study cessation of postmortem metabolism in halothane-sensitive pigs. In comparison, no differences have been noted in GP *a* activity between wild-type and Rendement Napole (RN) pigs (Estrade, Ayoub, Talmant, & Monin, 1994). Recall, RN pigs possess a gene mutation that somehow allows for a breech in the normal postmortem set points and results in lower pH_u and a type of 'acid meat'. Regardless, the aforementioned data strongly support the notion that GP may be involved in controlling the extent of postmortem metabolism and should be closer scrutinized.

Though not specifically addressed by Scopes' work, the enzyme most frequently implicated as responsible for the cessation of postmortem metabolism is phosphofructokinase (PFK) (Bendall, 1973; Hamm, 1977), due in part, to its rate-limiting status in glycolysis and its complex control in living tissues (Berg et al., 2007). Moreover, glucose 6-phosphate increases late postmortem (Copenhafer et al., 2006; Kastenschmidt, Hoekstra, & Briskey, 1968) suggesting a loss in PFK activity sometime earlier. Data to directly support this hypothesis are scant. However, a recent study comparing normal and RN pig muscle showed a number of sarcoplasmic and myofibrillar proteins experience phosphorylation events postmortem (Lametsch et al., 2011). To that end, the increased phosphorylation of PFK in RN pig muscle may increase its stability and subsequent pH inhibition during the postmortem period (Sola-Penna, Da Silva, Coelho, Marinho-Carvalho, & Zancan, 2010). This may help explain the lower pH_u of fresh pork derived from this genotype.

Schwägele and Honikel (1988) quantified a host of glycolytic enzyme activities over a wide range of pH values (5.3 to 6.8) found in postmortem tissue. Results showed the activities of PFK, glyceraldehyde 3-phosphate dehydrogenase, phosphoglycerate kinase, pyruvate kinase,

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