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Progress in reducing the pale, soft and exudative (PSE) problem in pork and poultry meat

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Abstract

Research in the area of the pale, soft and exudative (PSE) pork and poultry meat is reviewed in this article with an emphasis on genetic, biochemical and metabolic factors contributing to the problem. Over the past five decades, there has been much more work in the pork meat area where a few genetic markers have been identified, and are currently used to remove susceptible animals from the herd. Some of the markers are linked to aberrant calcium regulation in the early postmortem muscle. The poultry industry is still not at the point of using genetic marker(s); however, some recent work has revealed several potential markers. The review also discusses environmental factors such as antemortem stress and early postmortem processing practices (e.g. chilling rate) that can influence the development and severity of the PSE phenomenon. Some of these factors are known to cause protein denaturation at the early stage of postmortem and directly contribute to poor water-holding capacity and inferior texture in fresh meat and later in processed products. A newer hypothesis suggesting that variation in protein oxidation, in response to antemortem stress and early postmortem tissue environment, can contribute to development of PSE pork is also discussed. Finally, a few recommendations for future work are proposed.
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1. Introduction

Methods to add value to meat animal products such as selection, feeding, animal husbandry and product processing have been employed for many years. For the most part, selection of animals (e.g. pigs, poultry, cattle) within lines and between lines (crossbreeding) has been a very common procedure for genetically improving traits of economic importance to the meat industry. For producers, production traits such as growth rate, feed conversion, and carcass leanness are of great economic importance. Traits of economic importance for processors include carcass weight, carcass leanness, proportion of certain primal/sub-primal cuts and processing yields. In recent years, as processors have moved from offering "commodity pork/poultry" to branded products, meat quality has become more economically important. Thus, as a response to the growing meat quality demands of the consumer, the entire meat industry – from live animal genetics to consumer research – has taken several steps to further improve meat tenderness, juiciness, flavor and reduce and/or eliminate pale, soft, exudative (PSE) meat conditions. The latter is especially true in the pig industry where a few genetic markers have been identified and routinely used to remove animals susceptible to the PSE condition. An example is a recent survey of the

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US pork industry presented at the 2006 Mid-West meeting of the Animal Society of Animal Science indicated that only: ''3.34% of loins exhibit all three conditions of classic PSE, reporting a range from 0.1% to 10%'' (Meisinger & Berg, 2006). The poultry industry has not yet identified genetic marker(s) that can be used on a commercial scale for such a selection program; however, progress in this direction is starting to become evident.

2. Factors that contribute to PSE pork

Investigation of the factors underlying the development of pale, soft and exudative (PSE) pork date back many years (Briskey, 1959; Briskey & Wismer-Pedersen, 1961a, 1961b, 1961c; Kastenschmidt, Hoekstra, & Briskey, 1966) This is testimony to the persistence of the problem and to the elusiveness of a sustainable solution to the problem. It is generally accepted that the rate of postmortem metabolism is the major contributor to the variation in fresh pork quality and processing functionality of meat proteins. This loss of product and protein quality is attributed to protein denaturation caused by a combination of acidic conditions along with high muscle temperature in very early postmortem muscle.

A major contributor to the development of extreme cases of PSE in pork is the syndrome once characterized as ''Porcine Stress Syndrome''. Pigs with porcine stress syndrome were recognized to be at significant risk to produce PSE pork (Topel, Bicknell, Preston, Christian, & Matsushima, 1969). The well-established PSS condition is known to be linked to a single autosomal recessive gene. This gene is commonly referred to as the halothane gene because diagnosis of the mutation can be made by exposure to halothane anesthesia (Rasmusen & Christian, 1976). A point mutation in the 615 amino acid (Arg615Cys) of the sarcoplasmic reticulum Ca^{2+} release channel is responsible for the aberrant calcium metabolism observed in postmortem muscle. In pigs with this mutation, Ca^{2+} is released from the sarcoplasmic reticulum at a rate that is equivalent to twice that of normal muscle (Cheah & Cheah, 1976; Mickelson & Louis, 1996). Küchenmeister, Kuhn, Wegner, Nürnberg, and Ender (1999) demonstrated that Ca^{2+} uptake is also diminished in postmortem muscle in pigs with this stress susceptibility. This increase in sarcoplasmic Ca^{2+} is responsible for activating muscle metabolism and accelerating lactate production and subsequent accumulation in postmortem muscle.

With the advent of technologies to identify and eliminate this major cause of extreme cases of PSE, a great reduction in the incidence and severity of PSE has been realized by the industry. However, product with poor water-holding capacity and color is still observed, as previously mentioned. Much of the variation in protein functionality and fresh pork quality can still be linked to variation in early postmortem metabolism. The contributions of pH and temperature to protein denaturation and PSE development are well documented and indisputable (Briskey, 1964). An important observation is that quality features like water-holding capacity can vary so much at intermediate and low pH. This observation suggests that additional factors contribute to variation in pork quality. The primary focus of the remainder of this discussion will be other factors that influence water-holding capacity in fresh pork.

With the possible exception of rigor formation, denaturation of myosin is one of the most dramatic early postmortem events in muscle. Penny (1967) demonstrated that myosin ATPase activity is among the first to be altered under pH and temperature conditions that mimic postmortem muscle. Presumably this loss in activity is due to denaturation. Stabursvik, Fretheim, and Frøystein (1984) used differential scanning calorimetry of PSE and normal pork to determine that the HMM portion of myosin, specifically HMM S-1 is denatured in PSE pork. The consequence of denaturation of S-1 portion of myosin is likely an alteration of the rigor bonds and the spacing between filaments within the sarcomere. Indeed, Diesbourg, Swatland, and Millman (1988) used low-angle X-ray diffraction to demonstrate that low pH results in shrinkage of the myofibril and less space between myofilaments. The consequence of myofibrillar shrinkage is an increase in extramyofibrillar space within post-rigor meat and a decrease in the barrier for water to traverse out of the meat during storage. Swatland, Irving, and Millman (1989) used differential interference contrast microscopy to follow the time delays as fluid moved from the myofilament lattice to the intermyofibrillar space and finally to the extracellular space.

Myofibrillar shrinkage can also be translated into shrinkage of myofibers (cells) if the intermediate filaments and costameric connections between myofibrils and the sarcolemma are intact. Swatland (1985) showed how lateral cytoskeletal connections between myofibrils were changed during the development of rigor mortis. Having followed myofibrillar shrinkage in pork by electron microscopy, Swatland and Belfry (1985) outlined how shrinkage would be affected by desmin. A current hypothesis proposes that proteolysis of key muscle proteins (including desmin, vinculin and talin) minimizes the loss of water-holding capacity (Huff-Lonergan & Lonergan, 2005; Melody et al., 2004; Morrison, Mielche, & Purslow, 1998;) caused by lateral shrinkage of myofibrils in postmortem muscle. In a sense, early postmortem disruption of linkages between the myofibril and the sarcolemma minimizes the impact of myofibrillar shrinkage on myofiber volume. Because μ-calpain is known to degrade intermediate filament proteins and costameric proteins (including desmin, vinculin and talin) in postmortem muscle, it is suggested that factors that regulate calpain activity – calpastatin, pH, and oxidation – can influence water-holding capacity.

The calpain system includes two well characterized, ubiquitous proteinases (μ- and *m*-calpain), and an endogenous inhibitor of the calpains, calpastatin. (Goll, Thompson, Li, Wei, & Cong, 2003). Both μ- and *m*-calpain are heterodimers composed of an 80 kDa and a 28 kDa sub-

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