PS8.03 Colour stability of fresh meat 435.00

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I. Introduction and Purpose

Colour significantly influences consumer purchasing decisions because discolouration is considered indicative of product spoilage (Jeremiah, Carpenter & Smith, 1972; Kropf, 1980). This has major economic consequences that cause an annual loss of 1 billion USD to the meat industry (Smith et al., 2000). Reclaiming profit via improved colour stability relies on the proper application of the fundamental principles of myoglobin chemistry, including two often overlooked factors: oxygen consumption and NADH regeneration as they impact metmyoglobin reduction (Fig. 1). There are numerous reviews of this area (Bekhit & Faustman, 2005; Cornforth, 1994; Mancini & Hunt, 2005; McMillin, 2008) and some that emphasis future research investigations by using a systems approach (Andersen, Oksbjerg, Young & Therkildsen, 2005). Our objective is not to provide extensive coverage of factors effecting colour, rather we will present several practical applications of the science for metmyoglobin reducing activity (MRA) and oxygen consumption (OC).

II. Meat colour chemistry

Myoglobin is a sarcoplasmic protein that determines meat colour. Its centrally located heme iron reversibly and preferentially binds ligands such as oxygen, water, and carbon monoxide. The ligand present at myoglobin's 6th coordination site and the redox state of the iron critically affect the formation of deoxy-, oxy-, carboxy-, or metmyoglobin. Thus, factors that influence "oxygen and electron management" during meat processing will significantly impact colour stability (Mancini & Hunt, 2005).

III. Oxygen consumption

To maximize the colour life of fresh meat products, emphasis must be placed on oxygen management at nearly every step in production and marketing. The purplish-red colour of deoxymyoglobin results from an unoccupied (no ligand) 6th coordination site on ferrous heme iron. Oxygenation of deoxymyoglobin produces bright cherry-red oxymyoglobin. However, postmortem competition for oxygen, primarily between mitochondria and myoglobin, is the principal determinant of oxymyoglobin. Oxygen consumption by mitochondria can out-compete myoglobin and result in a relatively dark meat colour (Egbert & Cornforth, 1986).

Increases in postmortem age promote pigment oxygenation because mitochondrial activity decreases. However, this relatively brighter cherry-red colour is not very stable because increased postmortem age also depletes NADH and metmyoglobin reducing activity. Postmortem muscle has the necessary functional machinery, but not necessarily the fuel to make it run. In support of this, Bodwell, Pearson & Fennell (1965) concluded that the limiting factor in postmortem NADH production is the amount of substrates available to dehydrogenase enzymes. Enhancing meat with lactate and other intermediates is a practical example of utilizing meat's enzymatic pathways to improve colour, either by promoting reduction of metmyoglobin or by lowering tissue oxygen consumption (stability and darkening, respectively).

Fig. 1: Schematic of NADH regeneration in metmyoglobin reduction and oxygen consumption

NADH regeneration to replenish NADH pool
$\downarrow$
Metmyoglobin reduction
$\downarrow$
MYOGLOBIN COLOUR STABILITY
$\uparrow$
Mitochondrial oxygen consumption
$\uparrow$
Electron transport
$\uparrow$
NADH regeneration

The practical implication of postmortem competition for oxygen in a diverse population of muscle fiber types is evident in pale- and dark-cutting meat, the result of pre-harvest stress. Short-term stress often causes a rapid drop of pH early postmortem that denatures cellular components and reduces oxygen consumption (Sammel, Hunt, Kropf, Hachmeister, Kastner & Johnson, 2002). Although this meat will often have superior postmortem oxygenation, the colour life is shortened because processes involved in pigment reduction are impaired (Lindahl, Enfalt, von Seth, Joseli, Hedebro-Velander & Andersen, 2004). Long-term stress continuously depletes muscle glycogen and limits substrates needed for postmortem glycolysis (Rosenvold & Andersen, 2003). This causes an elevated ultimate muscle pH, a factor that promotes mitochondrial respiration, competition for oxygen, and a darkening of meat colour. These low oxygen partial pressures caused by increased mitochondrial activity help to maintain deoxymyoglobin and conserve pathway intermediates by limiting cyclical inter-conversions of myoglobin. However, myoglobin fails to oxygenate and does not form the bright cherry-red colour of freshly-cut meat. Unfortunately, colour is often detrimentally affected when muscle's ability to consume oxygen is too high or too low, rather than some intermediate level typical of "normal" postmortem meat (Renerre, 1993; McMillin, 2008).

IV. Metmyoglobin reduction

Oxidation of myoglobin produces brown discolouration as the result of metmyoglobin formation. Therefore, preservation of ferrous myoglobin via chemical reduction of metmyoglobin's ferric iron is critical. Metmyoglobin reduction involves the transfer of an electron from a donating source to metmyoglobin. The limiting factor in this process is NADH, which is depleted with time postmortem (Bekhit & Faustman, 2005; Brown & Snyder, 1969; Livingston, McLachlan, LaMar & Brown, 1985; Stewart, Hutchins, Zisper & Watts, 1965). Since the 1960s, little research has focused on identifying mechanisms that replenish the postmortem NADH pool. The key for meat colour stability is to find a balance between tissue oxygen consumption and the ability to reduce metmyoglobin.

1.Chilling and Processing

The effects of chilling on colour have been linked to genotype, diet, and chill-room parameters (Mancini & Hunt, 2005). Smaller and less-fat carcasses will chill rapidly (Bruce, Stark & Beilken, 2004). Deep portions of muscles routinely have more impaired colour chemistry than superficial portions (Sammel et al., 2002). This limits both glycolytic enzyme activity and acid production, resulting in muscle that has either an elevated ultimate pH and increased mitochondrial respiration or a near normal pH that is pale and less colour stable. During meat processing, meat is often sliced or ground and stored in containers prior to packaging. This is hard to avoid, but it significantly alters oxygen exposure and consumption, both of which can deplete substrates essential for long term metmyoglobin reduction (Madhavi & Carpenter, 1993). Managing this critical time in a product's life is often overlooked.

2. Lactate-mediated effects on MRA and OC

Numerous postmortem processes utilize NADH and as a result, limit the amount available for metmyoglobin reduction. Thus, postmortem production of NADH is critical to improved meat colour stability. Research in the 1960s suggested that enzymes involved in glycolysis, the tricarboxylic acid cycle, and the electron-transport chain, remain active in postmortem muscle, and thus could be possible sources of NADH regeneration (Bodwell et al., 1965; Watts, Kendrick, Zisper, Hutchins & Saleh, 1966; Saleh & Watts, 1968). In particular, Stewart et al. (1965) hypothesized that lactate dehydrogenase catalyzes the production of reduced pyridine nucleotides (NADH) from lactate and diphosphopyridine (NAD). These authors believed this mechanism was feasible because both lactate and lactate dehydrogenase (LDH) are present and active in postmortem muscle. More recently, Kim et al. (2006) reported nonenzymatic metmyoglobin reduction in a model system containing lactate, LDH, and NAD. Kim et al. (2006) also reported that beef *longissimus* steaks enhanced with 2.5% lactate exhibited more LDH and metmyoglobin reducing activity and contained significantly more NADH than treatments without lactate. These researchers concluded that lactate-mediated improvements in LDH activity likely contributed to increased NADH and metmyoglobin reduction.

Ramanathan, Mancini & Konda (2009) reported that combining lactate, LDH, and NAD with bovine mitochondria resulted in oxygen consumption that was comparable to the direct addition of NADH to isolated mitochondria. This suggests that lactate promotes mitochondrial production of NADH, and this NADH can be subsequently utilized for oxygen consumption. In addition, electrons generated by lactate-induced mitochondrial respiration can be used to reduce metmyoglobin (Ramanathan et al., 2009).

3. Packaging: A blend of myoglobin deoxygenation, oxidation and reoxygenation

A practical example that highlights the significant role of both oxygen consumption and metmyoglobin reduction in meat colour is the discolouration/browning of bloomed product when subsequently packaged in vacuum. A two-step process is needed to convert oxymyoglobin to deoxymyoglobin whereby (1) myoglobin is oxidized by low-oxygen partial pressures and (2) reducing activity regenerates deoxymyoglobin. Thus, meat must be capable of both oxygen consumption and metmyoglobin reduction to regenerate deoxymyoglobin. As a result, meat needs both sufficient mitochondrial activity and sufficient NADH production in order to regenerate deoxymyoglobin following oxymyoglobin formation.

V. Summary

Meat colour life is not determined solely by one factor, but by numerous factors operating in concert.

It is often difficult to incorporate the fundamental principles of myoglobin chemistry into daily meat industry operations in cleaver and effective ways. To improve our knowledge of meat colour, researchers should think "outside-the-box" to better understand myoglobin redox chemistry. It appears that oxygen consumption and NADH regeneration need more focused research to help solve practical industry colour issues.

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