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How can heat stress affect chicken meat quality? – a review

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Abstract

Heat stress is one of the most important environmental stressors for the poultry industry in the world. Reduced growth rate, low feed efficiency, impaired immunological responses, changes in intestinal microflora, and deterioration of meat quality are the consequences of acute or chronic heat stress. In terms of meat quality, 3 primary mechanisms have been suggested to explain this phenomenon: 1) rapid drop in pH during and after slaughter due to the glycogen conversion to increase in lactic acid accumulation especially when the muscle temperature is high, a combination of high temperature and low pH that facilitates the denaturation of sarcoplasmic proteins resulting in lower water-holding capacity of muscle; 2) acceleration of panting to dissipate body heat, which increases CO2exhalation and pH drop in blood, initiates metabolic acidosis in skeletal muscle. Increase in panting is also associated with a high release of corticosteroid hormones; 3) the reactive oxygen species produced by heat stress increases the oxidative stress in the birds, which can damage the structure and functions of the enzymes that regulate sarcoplasmic calcium levels in muscles. Overall, these changes in the muscle cells accentuate energy expenditure due to constant muscle contractions. This review discusses the scientific evidence about how heat stress affects the quality of chicken meat through the acid/base status, oxidative reactions, and changes in hormonal secretions.

Keywords

broiler, heat stress, meat quality, acid-base imbalance, anaerobic metabolism

Disciplines

Agriculture | Animal Sciences | Poultry or Avian Science

Comments

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How can heat stress affect chicken meat quality? – a review

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ABSTRACT Heat stress is one of the most important environmental stressors for the poultry industry in the world. Reduced growth rate, low feed efficiency, impaired immunological responses, changes in intestinal microflora, and deterioration of meat quality are the consequences of acute or chronic heat stress. In terms of meat quality, 3 primary mechanisms have been suggested to explain this phenomenon: 1) rapid drop in pH during and after slaughter due to the glycogen conversion to increase in lactic acid accumulation especially when the muscle temperature is high, a combination of high temperature and low pH that facilitates the denaturation of sarcoplasmic proteins resulting in lower water-holding capacity of muscle; 2) acceleration of panting to dissipate body heat, which increases CO_2 exhalation and pH drop in blood, initiates metabolic acidosis in skeletal muscle. Increase in panting is also associated with a high release of corticosteroid hormones; 3) the reactive oxygen species produced by heat stress increases the oxidative stress in the birds, which can damage the structure and functions of the enzymes that regulate sarcoplasmic calcium levels in muscles. Overall, these changes in the muscle cells accentuate energy expenditure due to constant muscle contractions. This review discusses the scientific evidence about how heat stress affects the quality of chicken meat through the acid/base status, oxidative reactions, and changes in hormonal secretions.

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INTRODUCTION

Meat-type birds are the primary protein resource for human in the most areas of the world (Havenstein et al., 2003; Flock et al., 2005). Over the past 5 decades, the poultry industry made dramatic improvements in meat production through the intensive genetic selection for the growth rate and the improvement in nutrition and management of broilers. However, the development of thermoregulatory systems could not match to the rapid growth rate of muscles (Havenstein et al., 2003), resulting in inability of the modern birds to control their body heat with the fluctuating environmental temperature and high metabolic rates (Settar et al., 1999; Deeb et al., 2002). In addition, birds are more sensitive to high temperatures than other monogastric animals due to feather coverage and the absence of sweat glands (Loyau et al., 2013). The total annual economic loss to the US poultry industry due to heat stress in birds was estimated at $128 to $165 million (St-Pierre et al., 2003). In addition, heat stress impairs animal welfare, an issue that should be considered by broiler producers during the growth and production stages (Scanes, 2016; Slimen et al., 2016).

Heat stresses can be divided into either acute or chronic depending on the duration and severity (Froning et al., 1978; Lu et al., 2007, 2017; Petracci et al., 2015; Wang et al., 2017). Lu et al. (2017) showed that chronic heat stress could adversely affect the meat quality by changing the aerobic metabolism, glycolysis, and intramuscular fat deposition, resulting in low customer acceptability (Lara and Rostagno, 2013) due to pale meat color (Petracci et al., 2004; Akşit et al., 2006; Zhang et al., 2012; Wang et al., 2017) with low water-holding capacity (**WHC**) (Mckee and Sams, 1997; Channon et al., 2000; Deng et al., 2002; Molette et al., 2003; Akşit et al., 2006; Feng et al., 2008; Wang et al., 2009) and increased cook and drip losses (Woelfel et al., 2002; Wang et al., 2017). The loss of WHC in pale, soft and exudative (**PSE**) meat is especially detrimental because a substantial portion of the broiler meats is used for further processing that uses margination, tumbling, and cooking processes.

Heat stress can increase the occurrence of PSE meat conditions (Owens et al., 2000; Alvarado and Sams, 2002; Wilhelm et al., 2010; Wang et al., 2017) and

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the affected meat had lower protein content and higher fat deposition than the control (Lu et al., 2007; Zhang et al., 2012). The adverse effects of heat stress on the growth performance of broilers have been reviewed recently by Song and King (2015), but little attention was paid to the relationships between heat stress and meat quality.

Heat Stress-Related Rapid pH Drop on Meat Quality

Among the wide range of significant factors affecting meat quality, pH is one of the most broadly accepted chemical indicators that influence meat quality (El Rammouz et al., 2004). Muscle pH could be rapidly dropped due to accelerating anaerobic glycolysis in muscle during and/or after slaughtering. Accelerated anaerobic glycolysis causes a cascade of chemical reactions to decrease pH rapidly (Sales and Mellett, 1996; Young et al., 2004), due to the conversion of glycogen to lactic acid in muscle (Zhang et al., 2012). It has been documented that rapid drop in pH might be associated with low redness, high drip, and cooking losses in chicken breast meat (Wang et al., 2009; Hao and Gu, 2014). This phenomenon has also been observed in heat-stressed turkeys where pH at different storage hours after slaughter (i.e., pH_0, pH_2, and pH_{24}) decreased, while lightness, drip loss, and cooking loss increased (Mckee and Sams, 1997). Postmortem glycolysis led to an accumulation of H^+ in muscle from adenosine triphosphate (**ATP**) hydrolysis (Sandercock et al., 2001; Wang et al., 2009), which is governed primarily by 2 critical enzymes including pyruvate kinase and lactate dehydrogenase in anaerobic conditions that converts pyruvate to lactate. The rapid postmortem glycolysis while carcass temperature is still high results in the rapid drop in pH, protein denaturation, pale meat color, reduced WHC, and poor texture (Alvarado and Sams, 2002; Wilhelm et al., 2010).

Panting and Acid/Base Status on PSE

Panting is a physiological response in birds to adjust their body temperature through the respiratory dissipation of heat by evaporative cooling to maintain thermal homeostasis under heat stress conditions (Calder and Schmidt-Nielsen, 1966; Yahav et al., 2005). Panting increases the exhalation of CO_2 that decreases the concentration of bicarbonate in the blood, resulting in lower concentration of hydrogen ions that increases plasma pH (Mongin, 1968), which referred to as respiratory alkalosis. This situation induces body water imbalance (Sandercock et al., 2001, 2006). All of these changes accelerate the rate of metabolism and energy expenditure and the depletion of ATP (Korte et al., 1999; Yahav et al., 2005). Glycolysis and creatine kinase (**CK**) activity are stimulated under heat stress conditions, and are the key for ATP generation in muscle (Mitchell and Sandercock, 2004; Zhang et al., 2012). The metabolic acidosis converts more pyruvate to lactate (anaerobic metabolism) and induces higher dependency to anaerobic metabolism to generate energy in the muscle, which will continue during the early stage of postmortem. Thus, in heat-stressed chickens, the rate of anaerobic glycolysis to generate energy (ATP) by breaking down muscle glycogen is faster than normal birds. The accelerated lactic acid production induces rapid pH decline while the body temperature is high, which results in PSE-like conditions in meat (Warriss and Brown, 1987; Fernandez et al., 1994; Santos et al., 1994; Wang et al., 2017). A similar increase in plasma CK activity has been reported with hyperthermia-associated stress in pigs (Shibata, 1996). In fact, the increased activity of CK is an indicator of muscle damage (myopathy) and the malfunction of sarcolemma in muscle cells (Ostrowski-Meissner, 1981), which could be associated with the disturbance of intracellular Ca^{2+} homeostasis in muscle. Previous research indicated that Ca^{2+}-mediated alterations in the permeability of muscle membrane could be induced either by hyperthermia or acid-base imbalance (Mitchell and Sandercock, 1995; Sandercock et al., 2001). Respiratory alkalosis due to acid/base imbalance triggered metabolic reactions in muscle tissue, ATP depletion, and increased osmotic activity in the cell membrane (Mitchell and Sandercock, 1995; Sandercock et al., 2001; Bao et al., 2004). Zhang et al. (2012) showed that the aerobic metabolism in muscle was switched to anaerobic metabolism immediately after the oxygen supply stopped, which raised lactic acid production through the conversion of pyruvate to lactate under anaerobic conditions by the relevant rate-limiting enzyme, lactate dehydrogenase (Zhang et al., 2012). Streffer (1988) speculated that heat stress might decrease the lactate/pyruvate and reduced nicotinamide adenine dinucleotide/oxidized nicotinamide adenine dinucleotide ratios in broilers. The rapid decline in muscle pH before and after slaughter speeds up protein denaturation and decreases WHC in muscle (Channon et al., 2000; Owens et al., 2000; Deng et al., 2002) but increases the light scattering (Klont et al., 1994).

Heat Stress Affects Meat Quality Through Oxidative Damages

Modern rapid-growing broiler strains are more susceptible to heat stress and oxidative damages than their ancestors (Altan et al., 2003; Tan et al., 2010; Sihvo et al., 2014). Oxidative stress in the body occurs when the oxidants exceed the antioxidants, including superoxide dismutase, catalase, glutathione peroxidase, ascorbate, and vitamin E in cells (Sahin et al., 2005; Star et al., 2009). In living animals, oxidation is typically initiated by reactive oxygen species (**ROS**), which are generated by cellular metabolism as well as external sources, including feed that contain oxidized

fats and lipids (Cadenas and Davies, 2000). Therefore, the addition of antioxidants such as vitamin E in animal feed can be a tool to alleviate the adverse effect of heat stress on live animals to a certain degree (Zhang et al., 2011; Habibian et al., 2016). However, the primary source of ROS in broiler muscles is the leakage of electrons from the respiratory chain in mitochondria during the conversion of molecular oxygen to water (Mujahid et al., 2007). Also, it was hypothesized that elevated body temperature increased oxygen reaction possibly by disrupting the electron transport assemblies of the membrane (Ando et al., 1997). Reactive oxygen species can alter the functions of muscle components such as enzymes, cause aging and loss of protein functions, and inactivate protein, deoxyribonucleic acid, and ribonucleic acid (Zabłocka and Janusz, 2008). Küchenmeister et al. (2005) reported that acute stress decreased sarcoplasmic reticulum Ca^{2+} transport before slaughter, but Favero et al. (1995) indicated that the oxidative damages of sarcoplasmic reticulum stimulated Ca^{2+} release. Oxidation of thiol groups in the ryanodine receptor can change the calcium sensitivity of this calcium channel (Zissimopoulos and Lai, 2006). Klebl et al. (1998) also demonstrated that sarcoendoplasmic reticulum Ca^{2+}-ATPase (**SERCA**), an enzyme that removes calcium from sarcoplasm (Klebl et al., 1998; Moreau et al., 1998; Adachi et al., 2002), is not able to adjust the sarcoplasmic calcium concentration when the enzyme was damaged by ROS. As a result, the calcium levels increased in the sarcoplasm, which induced uncontrollable muscle contraction. Consequently, the pH of muscle dropped and the WHC of meat decreased. It is well known that heat stress increases the accumulation of lactate and lowers the pH; pH decline could accelerate ROS production and result in protein oxidation (Srinivasan et al., 1996; Estévez, 2011).

Oxidation of proteins by ROS can occur in protein backbone as well as the side chains, and induces backbone fragmentation or produces carbonyls, thiols, sulfoxides, and sulfones depending upon the side chains oxidized (Shacter, 2000). Furthermore, ROS can lead to diverse functions, including inhibition of enzyme activities, aging, loss of protein functions, and development of PSE conditions (Estévez, 2015). The primary mechanisms of ROS actions may comprise the collaborative control of 2 proteins: 1) inactivation of proteolytic enzymes linked to meat tenderization, and 2) the oxidative changes of the muscle proteins, mainly actin and myosin, that lead to the decreased proteolytic susceptibility and result in PSE-like meat (Rowe et al., 2004; Carlin et al., 2006; Lonergan et al., 2010). Other parameters related to WHC, such as drip loss and cooking loss, were also negatively affected especially under PSE conditions. Furthermore, protein oxidation lowers their solubility and ability to bind water, resulting in increased drip and cooking losses (Wang et al., 2009). Myoglobin oxidation results in lower redness (Zhang et al., 2012) and increases lipid oxidation, which increases the production of malondialdehyde and reduces shelf-life (Lin et al., 2000; Feng et al., 2008; Wang et al., 2009).

Myoglobin contains iron that could trigger lipid peroxidation by promoting the generation of hydroxyl radicals through the Fenton reaction (Min and Ahn, 2005). Wang et al. (2009) reported that acute heat stress significantly increased the amounts of malondialdehyde and carbonyls in muscle. They also indicated that the increased lipid and protein oxidation were closely associated with the increased cooking and drip losses and decreased pH and WHC. Altan et al. (2003) reported that heat stress at 38°C for 3 h accelerated fat oxidation, catalase activity, superoxide dismutase, and glutathione reductase in blood.

Secretion of Corticosterone Hormone on Meat Quality

Heat stress stimulates the hypothalamic–pituitary–adrenal axis in poultry and increases the concentration of circulating corticosterone hormone (Sapolsky et al., 2000), which has a significant impact to the protein and lipid metabolism, body composition, and meat quality (Scanes, 2016). Imik et al. (2012) and Lu et al. (2007) demonstrated that heat stress is associated with chemical changes in broiler meat. High concentration of corticosterone hormone (glucocorticoid) increased fat accumulation in abdominal, cervical, and thigh adipose tissues (Cai et al., 2009; Wang et al., 2012a,b), but stimulated protein degradation and breakdown of skeletal muscle (Scanes, 2016), possibly through the expression of fatty acid transport protein I and insulin receptor in the pectoralis major (Wang et al., 2012b). Song et al. (2011) reported that corticosterone hormones consistently increased the concentration of urate/uric acid in the blood, indicating increased protein catabolism. However, a decrease in thyroid hormone (T3) during heat stress is involved in reduced basal metabolic and physical activities, and redirecting available energy for growth (Zaboli et al., 2017). This extra energy is primarily stored as abdominal and subcutaneous fats in chickens (Ain Baziz et al., 1996). Furthermore, corticosteroid hormones can accelerate production of ROS (Sato et al., 2010), which are involved in the incidence of PSE conditions. The changes in body composition due to the increased corticoid hormones can negatively influence the processing quality, which includes higher drip loss, lower marinade uptake, lower protein solubility, higher shear force, and lower cooking yields, of broiler meat (Van Laack et al., 2000; Barbut et al., 2005).

CONCLUSION

Heat stress is one of the most critical environmental stressors in meat-type birds worldwide. The negative impact of heat stress on meat-type birds ranges from reduced growth to decreased meat quality. However, the

primary concern is the adverse effect of heat stress on meat quality that affects consumer acceptability. The negative influence of heat stress to meat quality is due to the combined effects of the following 3 factors: 1) panting that accelerates anaerobic glycolysis (metabolic acidosis), which increases muscle temperature and the accumulation of lactic acid in the muscle. When the birds are slaughtered under the metabolic acidosis conditions, the meat will become PSE because the muscles carry high contractility and activation of enzymes involved in anaerobic glycolysis, which induce rapid pH drop while the temperature of the muscle is high; 2) increasing the level of ROS and damages to SERCA and results in high sarcoplasmic calcium content and uncontrolled muscle contraction; and 3) increase in the concentration of corticosterone results in the changes of metabolic rates and the production of ROS, which promote the incidence of PSE conditions in broiler meat. Extensive works have been done to determine the effects of heat stress on broiler meat quality over the past several decades, but more studies are needed to determine the combined effects of heat stress and oxidative stress on the incidence of PSE.

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