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Review Article

Effect of heat stress on meat quality: A review

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Abstract

One of the stressful situations for livestock and poultry is heat stress (HS), which has a detrimental effect on animal physiology, production, and product quality. Because of their quick development and metabolism, high production, and species-specific characteristics including decreased sweating, the protective layer of skin, and rumen fermentation, some varieties of livestock are more vulnerable to heat stress than others. Muscle that is pale, soft and exudative (PSE) and lacks the ability to retain water can be produced by short-term heat stress that occurs pre-slaughter. This stress increases muscle glycogenolysis. In contrast, animals that experience prolonged heat stress have lower muscle glycogen reserves, which results in meat that is dark, firm, and dry (DFD), has a high final pH and water retention capacity. Moreover, HS causes shortened shelf life, oxidative stress. According to a recent study, HS might plausibly modify the rate and amount of postmortem muscle glycolysis and the resulting pH, which can impact the physiological and metabolic disturbances that occur in live animals as well as the features of carcasses and meat quality. This review reflects the effect of heat stress on meat quality.

Introduction

Numerous meteorological factors, like excessive RH, solar radiation, wind speed & surrounding temperature, combine to cause HS, which is harmful to both animal welfare and productivity. Heat stress in cattle can occur at temperatures above 20°C, depending on the breed and species (Alam et al., 2011; Azad et al., 2021; Hashem et al., 2013; Hossain et al., 2022a, 2023a & 2023b). Warmer temperatures cause animals to become less productive because they consume 3-5% less feed for each degree Celsius as the temperature rises. Animals under heat stress exhibit altered behavior, decreased fertility, increased respiration and mortality, & weakened immunity and endocrine networks, rendering them greater susceptibility to certain illnesses. Because of the insulation of their skin and the absence of functional sweat glands, offered by their feathers and subcutaneous lipids, respectively, pigs & poultry are especially vincible to HS. For ruminants, the heat generated in the rumen during feed fermentation increases the generation of metabolic heat and compromises the animals' capacity for thermoregulation (Azad et al. 2022a; Hossain et al., 2022b & 2023c; Kim et al., 2022; Sadakuzzaman et al., 2001).

Before slaughter, acute heat stress causes a quick fall in pH of the muscle early post slaughter. Simultaneously, the remnants of the corpse are heated, which accelerates the breakdown of muscle glycogenolysis and increases the lactic acid content. (Bejaoui et al., 2023; Owens et al., 2009; Afroz et al. 2020; Islam et al. 2019). As a result, the meat becomes pale-soft-exudative (PSE), defined as a further down water holding capacity (WHC). This phenomenon has been recently noted in cattle & poultry (Oluwagbenga and Fraley, 2023; Kim et al., 2008). Typically, it affects chickens & pigs. (Adzitey and Nurul, 2011). On the other hand, livestock uncovered by prolonged HS have lower muscle glycogen stores, which cause a decrease in the formation of lactic acid. This results in DFD meat, which has a higher ultimate pH and a higher WHC. These findings are frequently seen in ruminants (Hossain et al., 2021; Kadim et al., 2008; Adzitey and Nurul, 2011), but also in pigs (D'Souza et al., 1998). In addition, hot seasons have been linked to increased breakdown of lipids and proteins in addition to decreased meat safety and shelf life due to the development and shedding of bacteria (Mujahid et al., 2007; Murshed et al., 2023).

This review's goal is to go over the scientific data on how heat stress affects livestock metabolism and physiology and how that affects the security and quality of meat from pigs, poultry, and livestock. There are gaps in the literature, and recommendations are made for future research directions. The effect of other stressors, such as feed additives, hormones, handling, and transportation, is not included in this review because heat stress is the main focus; nonetheless, it is impossible to overlook the overall effects of handling and transporting animals in hot environments on their physiology, well-being, and quality of output.

Temperature and humidity relation in heat stress

Several environmental conditions, including temperature, RH, solar radiation, air blow, can lead to heat stress. These many environmental factors are among the numerous indicators used to calculate the level of HS (Correa et al., 2004). Most research on HS in cattle has mainly emphasized RH & temperature since information on the animal's exposure to solar radiation, Air movement speed, and rainfall is not publicly available. Temperature & humidity data is available from a nearby weather station. The temperature-humidity index (THI), which was created as a gauge of animal comfort, is just a straightforward combination of temperature and humidity.

NCR (1971) put out the following THI formula:

THI is equivalent to (1.8*T+32)-[(0.55-0.0055*RH) × (1.8* T-26)]

Here, T denotes temperature (°C) and RH denotes relative humidity (%). According to article, cattle under HS fall under the following THI thresholds: "comfort" if THI is less than 68, "mild discomfort" if THI is between 68 to 72, "discomfort" if THI is between 72 to 75, "awareness" if THI is between 75 to 79, "danger" if THI is between 79 to 84, and "emergency" if THI is greater than 84 (Giannone et al., 2023). Because THI considers the resultant repercussions of ambient temperature and RH, It's a practical and straightforward tool for estimating the risk of HS (Dairy Australia, 2016).

From an alternative perspective, Healthy Herd Management Report (HHMR) illustrates different temperatures & RH levels that generate a THI 72 and the time at which the dairy cow starts to exhibit symptoms of heat stress. For instance, a THI of 72 will result from 80°F temperature and a relatively low humidity value of 35%, and 76 °F and 60%, which might affect the health and productivity of dairy cows The THI remains constant at 72 across different temperature and humidity combinations (Figure 1.). From this specific data, it appears that variations in humidity or temperature have no effect on the index.

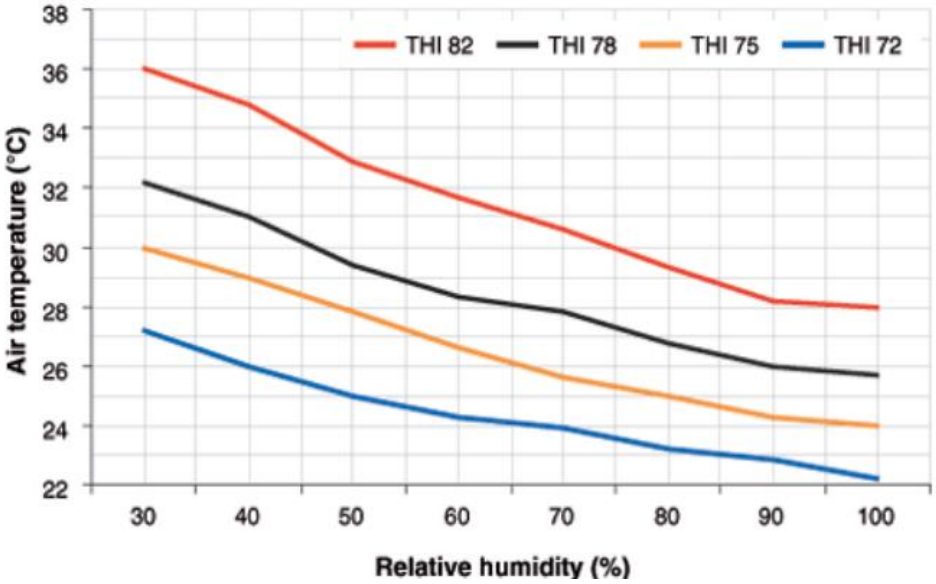

Figure 1. THI in relation to air temperature and relative humidity.

Heat stress's physiological impacts on the quality of meat

The quality of meat may be affected by the substantial physiological consequences that heat stress may have on animals. Animals that are under HS may exhibit the following physiological reactions:

- Increased Respiration Rate**:** Animals may pant or breathe rapidly to dissipate heat. This can lead to an alteration in the pH of muscle tissue, affecting meat tenderness and color.
- Reduced Feed Intake**:** Heat-stressed animals often eat less, leading to changes in their metabolism. This can affect the composition of the meat, including fat content.
- Elevated Body Temperature: Elevated body temperature has the potential to denaturize muscle tissue proteins, hence modifying the texture and water-holding ability of meat
- Changes in Blood Flow: Heat stress can alter blood flow patterns in animals, affecting nutrient delivery to muscles. This can influence the composition and quality of the meat.
- Hormonal Changes: Stress hormones such as cortisol may increase during heat stress. These hormones can influence the metabolism of nutrients and may affect the flavor and tenderness of meat.
- Electrolyte Imbalance: Heat-stressed animals may experience electrolyte imbalances, affecting muscle function and meat quality.
- Increased Fat Oxidation: Heat stress can lead to increased fat oxidation, which may impact the flavor and aroma of meat.
- Dehydration: Heat stress can cause dehydration, leading the decrease in the water content of muscle tissue. This can affect meat juiciness and tenderness.

All these physiological changes can collectively result in meat quality defects such as tougher texture, altered color, reduced water-holding capacity, and changes in flavor. Additionally, the length of time and intensity of heat stress can influence extent of

these effects on meat quality. Proper management practices, including providing shade, adequate ventilation, and access to water, can help mitigate the effects of heat stress in animals & consequently, on the quality of meat. (Gonzalez-Rivas et al., 2020).

Most anxiety responses to elevated temperatures and humidity are autonomic. This is because catecholamines (adrenaline and noradrenaline) stimulate the autonomic nerve system (ANS). Catecholamines are neurotransmitters and hormones that play a crucial role in the "fight or flight" response, which is a component of the ANS's sympathetic branch.

Here's a step-by-step breakdown:

Triggering Stress Response: Catecholamines are released in response to stressors such as the perceived threat or rising room temperature. Let's concentrate on the reaction to heat stress concerning your earlier query.

- Activation of the Sympathetic Nervous System (SNS): The ANS's sympathetic division is in charge of the "fight or flight" reaction. When catecholamines are released, they bind to adrenergic receptors (specific receptors for adrenaline and noradrenaline) located throughout the body, especially in various target tissues and organs.
- The Effects on Target Tissues: The binding of catecholamines to adrenergic receptors on target tissues leads to a cascade of physiological effects:
- Heart: Increases heart rate and the force of each heartbeat.
- Lungs: Dilates the airways, leading to increased respiration rate.
- Blood Vessels: Causes vasoconstriction in certain blood vessels, directing blood flow to vital organs and muscles.
- Skin: Induces vasodilation in blood vessels of the skin, facilitating heat dissipation.
- Metabolism: Stimulates the release of glucose from the liver, providing an energy boost.
- Mobilization of Resources: The overall effect is a mobilization of resources to prepare the body for action. This can include increased alertness, enhanced physical performance, and rerouting blood flow to areas that use it most.

It also stimulates the body to release stored energy (Yousef et al., 2023). Enhancing muscle glycogenolysis while reducing energy reserves. Catecholamines' influence on β2-receptors in muscles sets off a chain reaction (cascade response) that accelerates muscle glycogenolysis by producing cyclic adenosine monophosphate (cAMP). Muscle glycogenolysis is triggered by this process, which breaks down glycogen and activates glycogen phosphorylase (Franch et al., 1999) and it prevents the synthesis of glycogen. Acute and long-term heat stress also raises levels of glucocorticoids in plasma through the HPA axis activation. But compared to chronic heat stress, acute heat stress causes a higher rise in glucocorticoids (Kadim et al., 2006; Collier et al., 1982). Glucocorticoids change lipid metabolism and boost proteolysis in addition to improving heat loss through vasodilation (Kuo et al., 2013). Increased myofibrillar protein breakdown in skeletal muscle is mediated by the autophagy-lysosome system, the ubiquitin-proteasome, and the Ca^{2+} -dependent system (Braun and Marks, 2015; Schiaffino et al., 2013; Kayali et al., 1987; Bell et al., 2016). Additionally, anabolic regulators like insulin and insulin-like growth factor-1 (IGF-I) are inhibited by glucocorticoids, which exacerbates the loss of muscle mass (Braun and Marks, 2015). According to (Campbell et al., 2009; Peckett et al., 2011) glucocorticoids cause lipolysis through increased lipoprotein lipase activity that hydrolyzes circulating triglycerides. They also cause lipogenesis in hepatocytes and adipocytes by upregulating the expression of fatty acid synthase (Figure 2 and 3).

Hypothalamic-Pituitary-Adrenal Axis

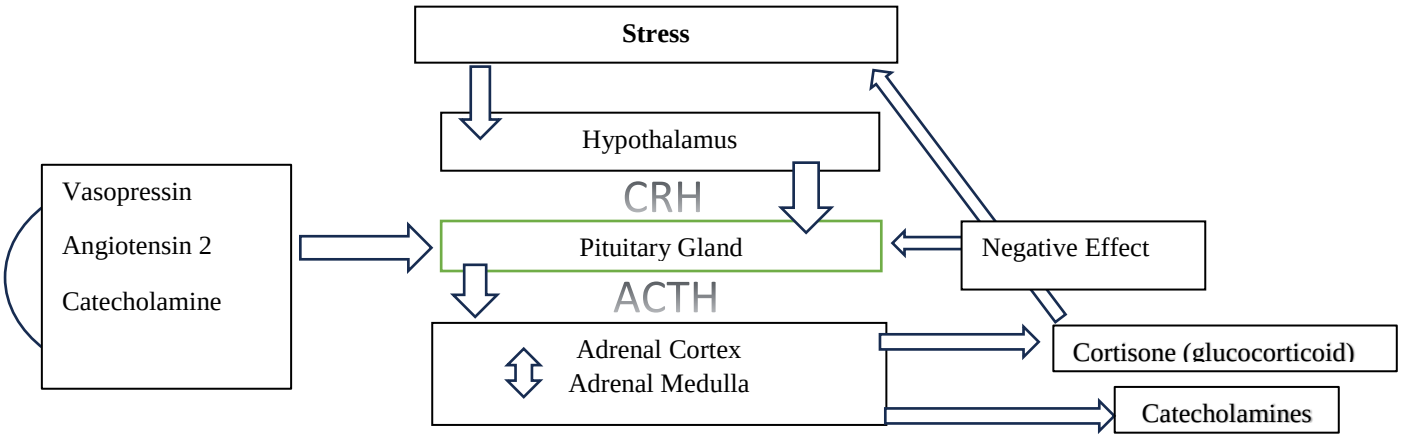

Figure 2. A Schematic overview of the Hypothalamic-Pituitary-Adrenal Axis.

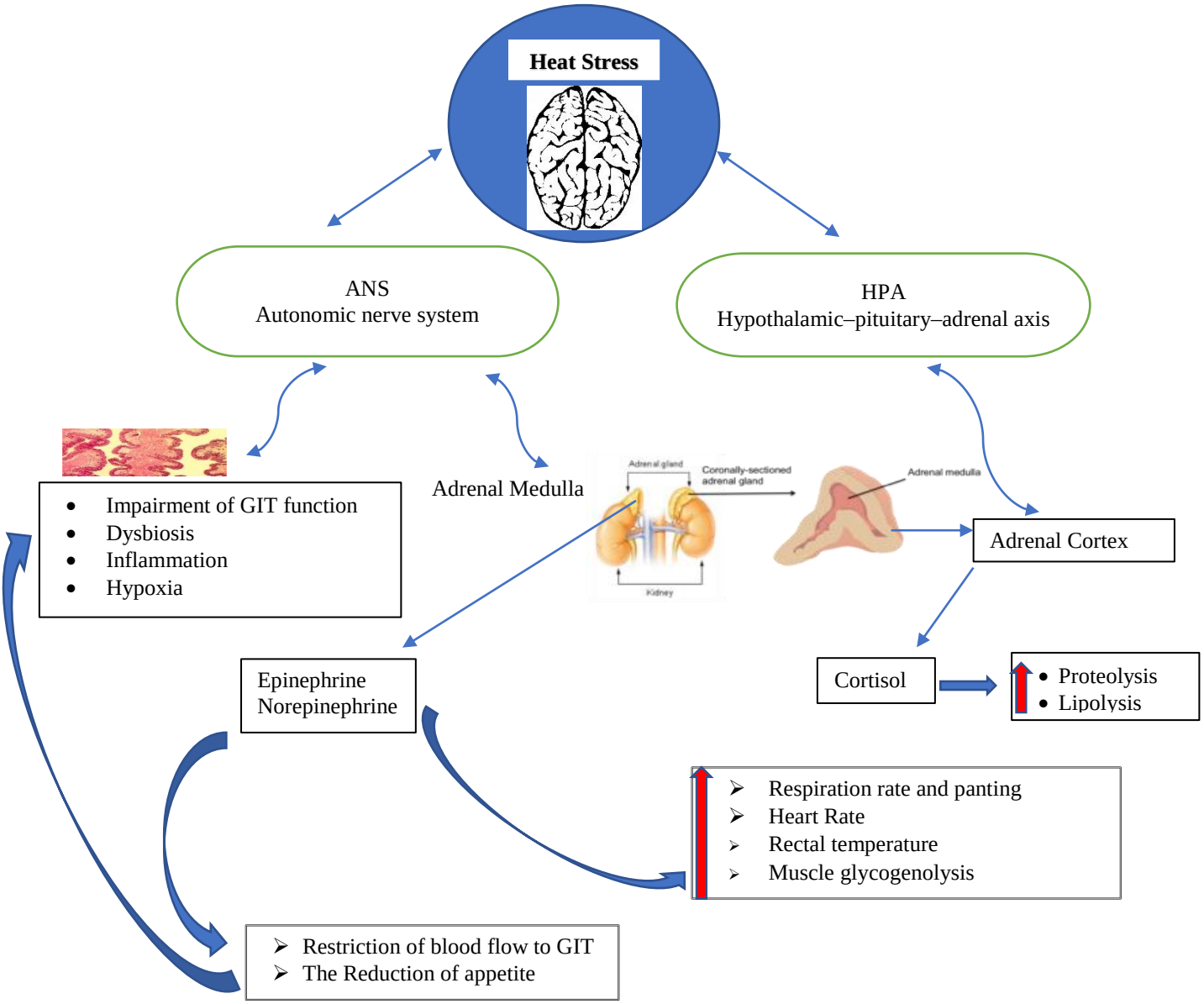

Figure 3. Heat stress's physiological impact.

Heat Stress's (HS) Effect on Cellular Level

Heat stress can cause modifications to cells by disrupting protein structures, affecting membrane integrity, and triggering cellular responses like heat shock protein production. This can impact cell function and may lead to various physiological responses or adaptations. Heat stress can disrupt protein structures through a process known as denaturation. Proteins are large, complex molecules that have specific three-dimensional structures crucial for their function. This structure is preserved via a number of weak bonds, such as ionic, hydrophobic, disulfide, and H-bonds. When proteins are exposed to elevated temperatures, the increased kinetic energy of the molecules can overcome the forces that maintain the protein's structure (Khan et al., 2020). This results in the unfolding or denaturation of the protein, where it loses its specific three-dimensional shape and, consequently, its purpose. The disruption of the protein structure can occur in several ways below (Zhao et al., 2021):

- Breaking Bonds: Elevated temperatures can break the weak bonds that stabilize the protein structure. For example, hydrogen bonds and disulfide bonds may be disrupted, leading to the unfolding of the protein.
- Increased Molecular Motion**:** Heat increases the molecular motion of atoms and molecules in the protein structure. As a result, the protein may vibrate more vigorously, leading to the disruption of the interactions that maintain its structure.
- The Loss of Hydrophobic Interactions**:** Proteins often have hydrophobic regions that are crucial for their stability. Heat stress can disrupt these hydrophobic interactions, leading to a loss of stability and protein denaturation.
- Unfolding of Protein Domains**:** Proteins often consist of distinct structural domains that contribute to their overall function. Elevated temperatures can lead to the unfolding of these domains, compromising the protein's ability to carry out its specific biological activities.
- Aggregation**:** In some cases, denatured proteins may aggregate or clump together. This can further impair cellular function and result in the creation of protein aggregation which is associated with various diseases.

The specific effects of heat stress on protein structure can vary depending on the protein's characteristics, the degree and duration of heat exposure, and the cellular environment. Cells have mechanisms to repair or remove damaged proteins, but severe or prolonged heat stress can overwhelm these protective mechanisms, leading to cellular dysfunction and, in extreme cases, cell death. Understanding how heat stress affects protein structure is essential in many different disciplines, encompassing medicine, molecular biology, and biochemistry.

HS can alter the cell membranes' fluidity, affecting their integrity and permeability. This change can impact the transport of molecules across the membrane and disrupt normal cellular communication. Indeed, heat stress can significantly alter meat's molecules. Proteins, lipids, and water content undergo the most significant alterations. Now let's explore each:

- Proteins: Denaturation: When animal proteins are subjected to HS, They forfeit their inherent composition and performance. The meat's color and texture may alter as a result of this. (Martin et al., 1992; Parvin et al., 2017)
- Maillard Browning: The Maillard process between reducing sugars and amino acids takes place at higher temperatures, which causes the meat's exterior to turn brown. The taste and the color of cooked meat are influenced by this response. (Miranda et al., 2012; Rahman et al., 2023)
- Collagen and Connective Tissue: Heat also affects collagen, a protein responsible for the connective tissue in meat. Through a process called collagen denaturation, heat helps convert collagen into gelatin, contributing to the tenderness of cooked meat. (Purslow, 2018).
- Lipids Oxidation: Lipids in meat are susceptible to oxidation under heat stress. This process can result in the creation of off-flavors and odor in the muscle, affecting its overall quality. (Toghyani et al., 2012)
- Hydrolysis: Elevated temperatures can promote the hydrolysis of lipids into free fatty acids. This can contribute to the development of rancidity in meat. (Zaboli et al., 2019)
- Water: Loss of Moisture: Heat stress causes the meat to lose water through evaporation. This can result in dehydration and a reduction in juiciness.

The "Journal of Food Science" and "Meat Science" are two journals that frequently publish research on these subjects.

Heat stress and small heat shock protein's effects on the color, juiciness, taste, and softness of meat

When muscle is turned into meat, intricate interplay between biological characteristics and biochemical processes determine the meat's quality for consumption. It was postulated that after exsanguination, muscles experience apoptosis as a result of the cessation of oxygen and nutritional delivery Factors that govern the process of muscle cells undergoing apoptosis ultimately influence the quality of meat. Different research has connected the regulation of sHSPs to several characteristics of meat quality, including taste, juiciness, color, and tenderness. Due of their chaperone and anti-apoptotic properties, it has been proposed that sHSPs are associated with the quality of meat that is fit for human consumption.

Small Heat Shock Protein (sHSPs)

sHSPs are members of the heat shock protein family. Based on the size of their monomeric molecules, heat shock proteins (HSP) are divided into five types. The molecular weights of four of these classes—designated as HSP60, HSP70, HSP90, and HSP100, respectively—are around 60 kDa, 70 kDa, 90 kDa, and 100 kDa (Hu et al., 2022). sHSPs are a member of the fifth class of heat shock proteins. Despite being the smallest in the HSP family, sHSPs exhibit the greatest degree of size variability, with molecular weights ranging from 12-43 kDa (Haslbeck et al., 2005). They have been isolated from bacteria, eukaryotes, and archaea, and are found in all species (Gusev et al., 2002). It's interesting to note that different organisms express different numbers of single-stranded polymers (sHSPs). For example, the yeast Saccharomyces cerevisiae expresses just two sHSPs, whereas the plant Arabidopsis thaliana expresses 19 sHSPs (Haslbeck et al., 2005). Ten sHSPs have been found in mammals to date, although not all of them are expressed consistently in mammalian cells (Taylor and Benjamin, 2005). Mammalian sHSPs were categorized by Taylor and Benjamin (2005) based on where they are expressed throughout the organism: Class I sHSPs are those that are expressed everywhere, whereas Class II sHSPs are those that are exclusively expressed in particular tissues. At the moment, αβ-crystallin, HSP20, and HSP27 are related to meat quality.

The growing significance of sHSPs in the meat quality

Initial reviews by Ouali et al. (2006) suggested that muscle fiber inherently undergoes apoptosis after post-mortem, suggesting a potential function for sHSPs in meat quality. To preserve cellular homeostasis, sHSPs are varyingly recruited to chaperone unfolding muscle proteins and prevent the initiation of apoptotic cell death. Because of their protective properties. It lessens the deterioration of muscular tissue and lessens the breakdown of myofibrillar proteins. It examines how much is now known about the expression of sHSPs in post-mortem muscle and potential pathways via which They could be crucial in the evolution of meat quality.

Apoptosis occurs in muscle cells post-mortem

As a result of oxygen and nutrition supply of muscle cells ceasing, muscle acidifies and ischemias progressively after death. In the process of turning muscle into meat, muscle cells experience apoptotic cell death prior to rigor mortis because ischemia conditions and food deprivation are known to trigger apoptosis. (Herrera-Mendez et al., 2006). When apoptosis starts, the oppositely charged leaflets on the inside and outside of the cellular membrane invert, isolating apoptotic cells from nearby cells.

Meat quality characteristics using biomarkers

For the meat business, it is advantageous to predict the final result of a certain quality attribute early after death. Proteins regulate some characteristics of meat quality, enabling researchers to identify novel biomarkers for predicting meat quality through the application of proteomics in meat science. Meat softness, juiciness, and flavor were shown to be negatively correlated with HSP27 and αβ-crystallin concentrations, according to Bernard et al. (2007). Furthermore, Kim et al. (2008) found a negative correlation among the concentration of HSP27 and color quality (L* and a* values) of beef meat. These results imply the possibility of the meat business being able to forecast the final result for any certain grade of meat characteristic post-mortem

through the creation of models based on statistics these biomarkers' expression. Nevertheless, it is unclear how sHSPs avail to the evolution of different meat quality characteristics.

Meat softness and proteases

A measure of meat's softness is its ultimate pH (pHu). Meat with an intermediate pH of 5.8 to 6.19 is often firmer than meat with a low pH (pHu less than 5.79) or a high pH (pH greater than or equal to 6.2) (Bouton et al., 1971; Jeremiah et al., 1991; Lomiwes et al., 2011). Meat with an intermediate pH is likewise less consistently tender, and the biochemical causes of this are mostly unclear (Pulford et al., 2008). Post-mortem meat tenderization is generally acknowledged to be an enzymatic process. At the moment, two protease systems that have been significantly linked to the breakdown of myofibrillar proteins that result in meat tenderization are μ-calpains and cathepsins. Ca^{2+} dependent proteases, known as calpains, function best at physiological pH. As we age, sHSPs facilitate the breakdown of myofibrillar proteins, causing the hardness of meat.

Heat Stress and Meat Safety

Even though high ambient humidity and temperature on meat safety has not been well studied, it is widely acknowledged that these factors create ideal conditions for the colonization of pathogens, thus posing a danger to the safety of meat and its byproducts. Chronic stress can affect an animal's susceptibility to a microorganism and/or the course of an infection (Ali t al., 2022; Boby et al., 2021). It can also change the susceptibility of the host to pathogenic bacteria through the intestinal barrier and enhance bacterial luminal adhesion and intestinal permeability (Sayeed et al. 2023). Increased production of catecholamines and glucocorticoids, or stress hormones, could affect the operation of animals' intestinal barriers and environmental microbiology (Azad et al., 2022b). Norepinephrine secretion also quickens colonic transit, intestinal motility, and transepithelial ion transfer, all of which can have an impact on the populations of microbes in the gut. Other than the immediate consequences of stress, the elevated pHu brought on by Heat Stress provides the favorable setting the proliferation of microorganisms. After vacuum packing and 35 days at 4 °C, DFD meat had overall aerobic mesophilic presumed LAB populations.

One of the biggest problems with livestock production is heat stress, which will probably get worse as a result of increasing the global temperatures brought on by climate change. The development of Heat Stress mollification measures to lessen the financial impairment suffered worldwide cattle sector during the summer has attracted considerable attention from the viewpoints of animal welfare & productivity. Understanding how HS impacts an animal physiological, metabolic state and how that affects animal performance has advanced significantly. The impact of HS on the quality of animal products & suitable strategies to mitigate these adverse consequences have been the focus of recent investigations. Certain data suggest that Heat Stress pre-slaughter may increase the incidence of DFD in ruminants and PSE in pigs and poultry (Mujahid et al., 2005 and 2006). These effects, however, varied according to the length and severity of HS exposure and were not constant between investigations. Few carefully planned controlled environmental studies compare the consequences of high temperatures on livestock meat quality. Closing these knowledge gaps would be the first step in creating appropriate mitigation methods for the effects of HS on meat quality. However, many dietary strategies, such as betaine, flavonoids, vitamin E, electrolytes are useful in reducing some detrimental impacts of HS on the metabolic processes and physiology of animals.

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