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Impact of Glucose to Mitigate the Heat Stress in Broiler Chickens

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Abstract:
Over a few decades, the main purpose of the progress in the poultry genes is to obtain optimum production at maximum temperature. However, these days the increase in the tremendous heat waves and the increased sensitivity of poultry genomes to the heat stress makes them more alarming to the economic poultry losses. So, the main objective of this article is to assess the importance of glucose utilized in drinking water to mitigate the bad impacts of heat stress in poultry. Though, in the latest changing environmental conditions, it is very imperative to use glucose against heat stress that is advantageous in the response to the changing environment.

Keywords: Poultry genome, mitigate heat stress, glucose, broiler birds.

INTRODUCTION

The poultry industry is a worldwide predominantly engaged among different sectors of agricultural industries (Daghir, 2009) which provide healthy meat aside from the other sources of animal protein (Hadyait *et al.,* 2018; Williams *et al.,* 2006). The growth of the poultry industry depends on the provision of physically, chemically, and microbiologically good quality water (Ashraf *et al.,* 2019). The use of organic mineral over inorganic sources in poultry is increasing very rapidly (Zafar and Fatima, 2018), and the high production rate and more efficiency of feed conversion ratio of poultry broilers make them more susceptible to diseases (Khanam *et al.,* 2016; Rasool *et al.,* 2018). Now a days, because of the increasing estimated environmental heat waves (1.4 °C to 5.8 °C), heat stress has been of main concern for poultry farmers especially in the hot environmental areas of Asia and South Africa (IPCC, 2007; Daghir, 2008). The performance of the modern poultry breeds (especially broilers) is triple than the ancestral poultry breeds (Havenstein *et al.,* 1994) as the latest poultry breeds generate more energy in their body because of the higher metabolic rate that is more susceptible to the climatic temperature changes (stress) (Deeb and Cahaner, 2002).

It's very hard to describe and recognize the stress due to its imprecise observations however stress is a response to unfavorable stimuli. According to different researchers, it is an unforced reply of an object to any order (Selye, 1976; McGrowder and Brown, 2007; Tache and Brunnhuber, 2008; Chrousos, 2009; Szabo *et al.,* 2012). Whereas the agent that causes stress at any time is called a stressor. Stressor continually disputes the homeostasis (dynamic equilibrium) of living organisms, which is sustained in intricate undesirable conditions as described by many researchers (Tache and Brunnhuber, 2008; Chrousos, 2009; Szabo *et al.,* 2012). Particularly due to public awareness and concerns the heat stress and transportation stress has rapidly turned into a massive spot of intention for the poultry industry (Renaudeau *et al.,* 2012).

Heat stress and broiler birds

Van Kämpen (1971) and Ward and Peterson (1973) explored that the ambient temperatures beyond the thermoneutral range increased the core body temperature which on the first hand neutralize the metabolic disorders and on the second side declines the body temperature. An increase in the body temperature due to heat stress leads to several responses such as increased metabolic rate, deterioration of the structure of enzymes, precipitation of proteins, and melting of lipid bilayers of cell membrane which results in the loss of cellular composition by changing its individuality and permeability (Tietz, 1970; Lehninger, 1975). These all results cause crumple and tremors of muscles and weakness of the body of broiler chickens (Odom *et al.,* 1985).

Long-term exposure to heat stress expands the peripheral blood vessels of chickens (Whittow, 1986; Bogin, 1992). Due to which the metabolic and cellular systems of birds are damaged. So the concentration of blood of different metabolites is directly and indirectly affected by the damaged metabolic and cellular systems (Röder *et al.,* 2014). It is also observed that heat stress declines the levels of glucose in the body of chickens, particularly in the kidney. In this heat stress condition the blood uric acid is increased in the kidney that results in renal failure (Boudry *et al.,* 2007; Roder *et al.,* 2014) which ultimately declines the gluconeogenic activity and along with that the renal failure of the kidney, on the other hand, disturb the heart function (Kaneko, 2008; Bogin, 1992). The glucose and uric acid are served in the chickens to survive and withstand the heat stress (Bogin *et al.,* 1996). Besides, it has been concluded that stress interrupts the growth of lymphoid organs and changes the appearance of inflammatory cytokine genes (Zhang *et al.,* 2011; Zaglool *et al.,* 2019).

Effect of heat stress on glucose level of broilers

Glucose is utilized by the different types of cells in the body for the requirement of energy (Hazelwood, 2000). As glucose provides all the energy to the brain so its accessibility persuades the psychological process. It has been studied that when the level of glucose declines the psychological process of the brain is disturbed (Fairclough and Houston, 2004; Gailliot *et al.,* 2007; Masicampo and Baumeister, 2008). From the previous studies, it has been seen that heat stress damage the composition of blood by changing its metabolic and cellular systems. So it is important to know about how different stressors particularly heat stress that exerts their bad impacts on birds. So heat stress is a convoluted multifactorial stressful and distressing event and to understand its impact on the broiler birds, stress must be well defined.

The response of birds against heat stress

As from the recent study, it has been observed that birds facing heat stress respond to gasping, panting, and even increased cannibalism (Dayyani and Bakhtiari, 2013; Pawar *et al.,* 2016). Due to the lack of sweat glands, the remaining option for birds to cooling their body is the panting system. The panting causes polypnea and impaired blood gas balance which leads to the formation of respiratory alkalosis (Darre *et al.,* 1980; Bowen and Washburn, 1984). Because of the heat stress, the blood is abstracted from interior organs to the skin, which changes the color of skin as in dark. As of the harsh heat stress the growth performance of birds is stunted and even declines (Mack *et al.,* 2013) and it is observed that more drinking of water is beneficial for broiler birds in the response to decreasing heat stress (Bahrami *et al.,* 2012; Mack *et al.,* 2013).

Heat stress causes oxidative stress in broilers

The homeostasis system in the broilers faces different stressors. Among these stressors, the heat stress in broiler birds induces the formation of reactive oxygen species (ROS) and hence produces oxidative stress in the cells (Lin *et al.,* 2000; Mahmoud and Edens, 2003). The oxidative stress in broilers destroyed the antioxidant defense system by causing lipid peroxidation (LPO) and also oxidative damage to the proteins and DNA (Droge, 2002).

Policies to conquer the consequences of heat stress

Different strategies such as nutritional supplementation of glucose are the best option to control the heat stress in broilers (Hayashi *et al.,* 2001). Heat stress minimizes the feed intake in the birds, because of which the level of glucose is reduced that caused the death of birds. But the feeding of birds with glucose which is stored in hepatic glycogen form in starvation conditions diminishes the morbidity and mortality of birds (Hayashi *et al.,* 2001; Yahav *et al.,* 2004).

Glucose is a monosaccharide and is the basic unit of carbohydrate (Domb *et al.,* 1998). Glucose is mainly formed during the process of photosynthesis by plants and algae (Bikales and Ott, 1971). Glucose is also known as blood sugar because of its presence in the blood. Glucose is a most important metabolite that is intimately narrated to the maintenance of the supply of energy for the execution of the biological purpose in the body (Klasing, 1998).

Role of glucose against oxidative heat stress

During heat stress, the feed intake is reduced. In the case of anaerobic metabolism (starvation), the walls of the intestine of the birds convert about 30% carbohydrates into lactate for the regulation of glucose homeostasis. This lactate performs in the form of buffer during the assimilation of carbohydrates, but in the case of low glucose level, the lactate is converted into glucose through gluconeogenesis. Hence the weight of the body and the level of glucose is not disturbed by the energy intake (Whittow, 1986). But within a few minutes if the oxidation of

glucose does not happen then the glucose is converted into glycogen and is stocked up in the liver and muscles. Whereas some energy is produced by the oxidation of glucose that is used for the production of glycogen, amino acids, fatty acids, vitamin C, and many other metabolites (Hernawan *et al.,* 2012).

Mechanism of glucose absorption in the small intestine

The digestion of food starts from the mouth. Salivary amylase is secreted in the oral cavity whereas the pancreatic amylase begins the digestion process but it is unable to completely convert the starch and glycogen into glucose monomers. So the enzymes are present on the apical membrane of the absorptive cell just inside the layer of the small intestine which are known as brush border enzymes. These brush border enzymes are limit dextrinase, glucoamylase, sucrose, lactase, and maltase. There is a transport protein (SGLT1) that is located near these enzymes. This transport protein helps the glucose monomers (two molecules of Na^+ and one molecule of glucose) to enter across the apical membrane. The basolateral surface enterocytes contain a glucose transporter (GLUT2). This GLUT2 permits glucose to move from the IEC into the extracellular medium near the blood capillaries (Röder *et al.,* 2014). Whereas the movement of GLUT2 from the cytoplasmic vesicles embedded in the apical membrane increases the ability of glucose uptake by the enterocytes (Affleck *et al.,* 2003; Grefner *et al.,* 2015). The mechanism of glucose absorption is shown in figure 1.

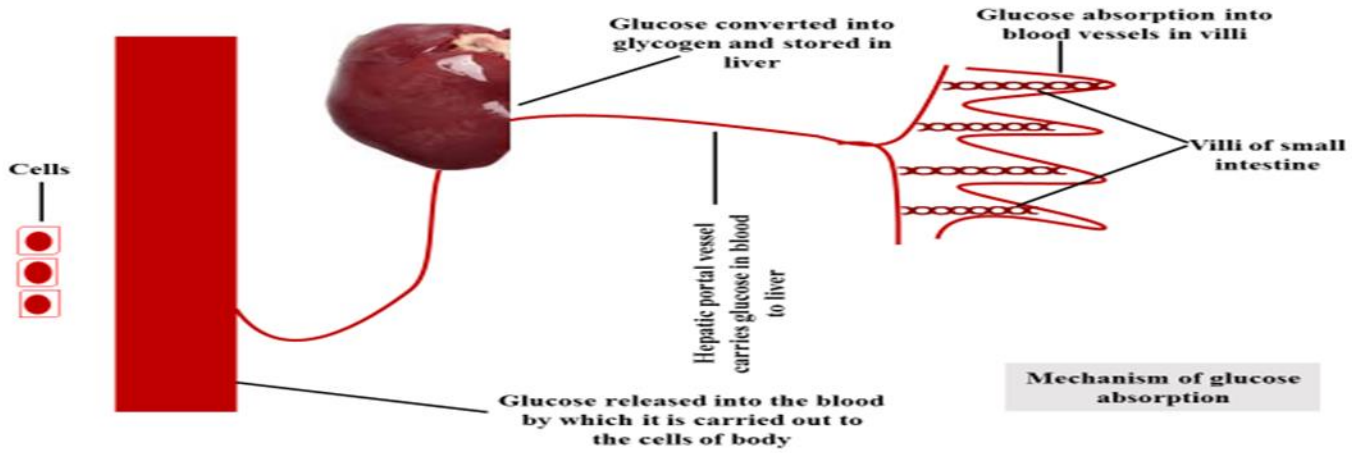

Fig. 1. The mechanism of glucose absorption.

Mechanism of Glucose regulation under heat stress

The glucose level of broiler birds is more as compared to the mammalians and varies from 180 to 250 mg/dl (Sturkie and Benzo, 1986; Hazelwood, 2000). In birds, the mechanism of glucose metabolism is similar as in the other animals, excluding the pentose phosphate pathway that is quite inactive in the liver of birds (Duncan, 1968) and also that there is no glucokinase in the liver of poultry birds (O'Neill and Langslow, 1978a,b). Due to the absence of glucokinase, the glucose is not regulated in the liver of birds. But in this condition, the uptake of glucose is controlled by the substrate cycling between glucose and glucose 6-phosphate (O'Neill and Langslow, 1978a,b).

From different studies, it has been observed that stress is activated by the choking of glucose. The blocking of glucose synchronizes the consequences of corticosteroids in the broilers which remarkably affect the metabolism of glucose and the development of muscles (Kono *et al.,* 2005; McGroeder and Brown, 2007). In the muscle tissues of chicken, the insulin at a specific level is conciliated by the progress of glucose transporter GLUT4 to the plasma membrane (Kono *et al.,* 2005). The ultimate effecter of the hypothalamic-pituitary-adrenal axis known as a glucocorticoid (GC) is engaged in managing the stress responses and the homeostatic system of the whole body. Meanwhile, another energy activator is known as AMP-activated protein kinase plays a main character in the regulation of glucose metabolism (Xue and Kahn, 2006; Hardie, 2007; Bungo *et al.,* 2011; Doustar *et al.,* 2012).

During the last days of the incubation period, the digestive system of chicks starts rapidly than on other days of incubation. So in these incubation days, there is a highly abundant requirement of energy for the normal growth of the embryo (Salmanzadeh, 2012). Hamer and Dickson (1989) stated that due to the inadequate supply of glucose to the embryos, the deterioration of protein from the breast muscles starts and by this, the amino acids start the process of gluconeogenesis. Because of this the deposition of protein declines from the muscles of the breast and ultimately decreased the organ weight (Vieira and Moran, 1999). In the case of in-ovo feeding the weight of hatchability and the rate of body growth of chicks is increased (Bhanja *et al.,* 2004). But in another study, it was observed that the percentage of hatchability of chicks was declined due to the injection of glucose in the albumin (Pedroso *et al.,* 2006; Bhanja *et al.,* 2008).

Halevy et al. (2001) encoded that the provision of a warm environment to early aged chicks enhanced the number of satellite cells. These satellite cells are important for the hypertrophy of the tissues of the muscles of early hatched chicks. It has also been recognized that the most vital growth induction factor known as plasma T3 has an optimistic association with feed intake in the broiler chicks. Under heat stress situation this plasma T3 hormone acts as a key competitor for the growth regulation in broiler chicks (Uni *et al.,* 2001). Moraes et al. (2004) observed that the introduction of heatwaves at the time of incubation of chicks has optimized the heating tolerance capacity of post-hatched chicks. But Hayashi et al. (2001) demonstrated that the provision of glucose under heat shock conditions to the broiler chicks decreased the negative aspects of heat stress. He also observed that heat stress is the main cause of the mortality of birds. As the heat stress minimizes the feed intake in birds. So in this starvation condition, the storage of glycogen in hepatic form is the only first available source of energy for balancing the metabolic system and proper growth of birds (Christensen *et al.,* 2000). Although the main target of heat shock in birds is the production of thyroid and growth hormones (Yahav *et al.,* 2001).

It is obvious that the oral provision of glucose to the newly hatched chicks that were born at 21 °C, increased the body temperature and also exposed that the metabolism of carbohydrates was responsible for the physiological regulation of their body temperature (Thaxton *et al.,* 1974). According to Iwasaki et al. (1997) the supplementation of 4% glucose in the water at 35 days aged birds decreased the mortality percentage during the heat stress period and it also increased the live body weight gain of the birds. During high environmental temperature conditions, they also concluded that the provision of glucose in water too decreased the rectal temperature of the birds. Moreover, Zhou et al. (1998) noted that when the glucose added water was given to the birds during high ambient temperature, the thermoregulatory system in birds performs more efficiently but in the case of low thermoregulatory ability of birds, the mortality of

birds was increased. They also concluded that the utilization of glucose not only vital for blood viscosity but also important for the diffusion of body heat in the birds. Along with that the provision of glucose to the birds also increased the immune system of the birds as described previously (Kazuaki and Yukio, 2002).

CONCLUSION

The current study determined the effects of glucose on poultry broiler bird's health, hatchability percentage, FI, WG, and mortality during high environmental temperature. The addition of glucose in the drinking water mitigates the harmful effects of heat stress throughout the growing and finisher stages of birds. Further study is needed to know how effectively glucose minimizes the notorious effects of heat stress under starvation conditions.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

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