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Review Article

Heat Stress Management Strategies using Plant Extracts in Poultry

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Abstract

Heat stress becomes a consistent concern in animal production as the global temperature rises due to climate change. This circumstance has led to significant economic losses, especially in poultry production and industry. Different methods have been extensively studied to address issues associated with heat stress, such as using various plant extracts. Although several environmental, genetic and nutritional strategies have gained traction, many researchers remain interested in using plant extracts as it offers a safe, accessible and low-cost solution to the problem. This review aimed to explore the existing studies on plant extracts that might be used to combat the adverse effects of heat stress, such as metabolic alterations, oxidative stress, and immune suppression. Plant extracts deemed potential to improve the production performance of affected birds were also reviewed.

Keywords: Chicken; Feed additive; Immune suppression; Metabolic alteration; Oxidative stress; Phytochemical

Introduction

The global temperature is estimated to rise by 2°C to 6°C in 2100 (Singh and Singh 2012). This massive increase affects every living organism in several ways, one of which is its negative consequence on human and animal health, be it direct or indirect (Rabinowitz and Conti 2013). The indirect impacts involve the biological alteration and distribution of vector-borne diseases (Lacetera 2018). To contrast, the direct effects may be associated with increased temperatures and heat waves (Ebi *et al.* 2009). Heat stress plays an essential role in mediating these direct effects. However, heat stress may cause oxidative stress, metabolic alterations, immune suppression, and even death, depending on the severity of infection (Lacetera 2018).

Heat stress occurs when the amount of heat created by animals reaches the capacity to disperse the heat to their surrounding (Akbarian *et al.* 2016). The inequality can be caused by multiple environmental factors such as thermal, sunlight, irradiation, movement, temperature, and humidity, and internal factors of animals, including species, sex and metabolism (Naga and Narendra 2018). Poultry meat and egg can provide considerable protein sources with a high amount of nutrients and low levels of fat as well as fatty acid, making it one of the most widely consumed animal-source food worldwide. As the global population increases, the demand for poultry products is also on the rise. As a corollary, genetic improvement of broiler and layer chickens is continuously undertaken. These improved birds often have higher metabolic rates and production performances. Higher metabolic rates produce more body heat, predisposing the birds to heat stress (Wasti *et al.* 2020). Heat stress is defined as the imbalance between heat production and heat loss in the animal body. The studies of heat stress in poultry have been examined extensively, significantly influencing poultry production. When animals experience heat stress, they will manage to decrease their heat production by restricting feed consumption, which potentially declines growth rate and products qualities (Rashamol *et al.* 2020). Hence, animal heat stress has been a pivotal issue among researchers and producers for decades, especially in tropical arid areas, and regions with harsh climates due to the stark difference in temperatures between spring and summer seasons.

Heat stress is distinguished into acute and chronic. Acute heat stress is related to a temporary and quick increase in the ambient temperature. In comparison, chronic heat stress is associated with a high ambient temperature after a prolonged period, leading to environment acclimatization. Chronic heat stress, also called "cyclic chronic heat stress," is related to a restricted time of heat exposure triggered by a comfortable temperature, or called "constant chronic heat stress," during which poultry is continuously exposed to increased ambient temperature. The heat stress affects the

physiological properties of poultry, such as increased body temperature (Liu *et al.* 2019), lower body weight (Wasti *et al.* 2020), reduced feed intake (He *et al.* 2018), immune depletion, altered electrolyte, pH balanced alteration (Ratriyanto and Mosenthin 2018), impaired endocrine and reproduction (Boni 2019), increased cortisol level, digestibility and metabolism (Yin *et al.* 2021) and changes in gut microbiota profile (Shi *et al.* 2019). Acute and chronic heat stress has a serious effect on performance, including a lower rate of egg production (Nawaz *et al.* 2021), increased chicken mortality (Barrett *et al.* 2019), reduced meat quality, altered immunological parameters and caecal microflora (Awad *et al.* 2019).

Furthermore, the poultry industries suffer a significant financial loss worldwide due to heat stress. In the U.S., heat stress caused an economic loss of $128 to $165 million to the poultry industry (Pierre *et al.* 2003; Lara and Rostagno 2013). These financial losses result from decreased production performance and increased mortality rate. Several environmental, genetic, and nutritional strategies are now being used to counter heat stress effects (Naga and Narendra 2018). One promising heat stress management strategy is using plant extracts (Akbarian *et al.* 2016) due to their beneficial compound such as an antioxidant, polyphenol, antimicrobial and antiparasite. Those compounds have been proven can mitigate heat stress in chickens (Dong *et al.* 2015; Akbarian *et al.* 2016; Ghanima *et al.* 2019). This review aimed to present and evaluate several studies on different plant extracts for mitigating heat stress on poultry.

The effect of heat stress on animal health

The average body temperature in chickens is between 41–42°C, and the ideal ambient temperature for growing poultry is 18–21°C. Studies have found that poultry may experience heat stress if the environmental temperature is higher than 35°C (Charles 2002). Heat stress commonly affects animal health by causing metabolic disorders, oxidative stress, immunosuppression, and death (Lacetera 2018).

Metabolic disorder in heat stressed poultry

The autonomic nervous system (ANS) is essential in heat stress response. During heat stress, ANS increases the heart rate and enhances blood flow to the skin. This action maximizes the heat loss to balance body temperature (Nawaz *et al.* 2021). However, birds commonly release heat through panting for evaporative cooling since they lack sweat glands (Wasti *et al.* 2020). An increased respiratory rate when the bird is panting leads to a higher rate of CO_2 excretion than its cellular production. This physiological occurrence changes blood's bicarbonate buffer system and directly impacts blood pH, gases, and others circulating metabolites. As the rate of CO_2 falls, the concentration of carbonic acid (H_2CO_3) and hydrogen ions (H^+) is also reduced. At the same time, bicarbonate ions (HCO_3^-) concentration is increased, raising blood pH. As a corollary, birds will excrete a more significant quantity of HCO_3^- and maintain H^+ from the kidney. This high H^+ affects the acid-base equilibrium and causes respiratory alkalosis and metabolic acidosis. Studies have found that maximal growth rate is achieved when blood pH ranges from 7.20 to 7.30. Changes in these elements significantly decrease growth rate and feed efficiency. During panting, pH values commonly exceed 7.25 (Borges *et al.* 2007). Additionally, metabolic acidosis triggers myoglobin and haemoglobin's unusual redox reaction (Tang *et al.* 2013). Haemoglobin plays a vital role in transporting oxygen, nutrients and wastes in the blood.

Elevated environmental temperatures also activate the hypothalamic-pituitary-adrenal (HPA) axis. Increased plasma corticosterone levels are generally observed in birds exposed to heat stress (Lara and Rostagno 2013). The secretion of corticosterone from the HPA and pituitary gland is normally detected in chronic heat stress. Extensive secretion of corticosterone can significantly affect the body and is often linked to muscle breakdown, cardiac issues, compromised immunity, and depression in broiler chickens (Nawaz *et al.* 2021). Additionally, a decrease in triiodothyronine (T3) concentration is consistently reported, while alterations in thyroxine (T4) concentration are inconsistent in affected birds. T3 and T4 are essential in regulating body temperature and metabolic activity. Changes in the poultry's neuroendocrine system due to heat stress facilitate lipid accumulation *via* raised *de novo* lipogenesis, lowered lipolysis, and improved amino acid catabolism (Lara and Rostagno 2013). Furthermore, heat stress stimulates the release of catecholamine, subsequently decomposing glycogen into glucose in muscles and reducing muscles' capacity to store energy. Catecholamine is responsible for inhibiting glycogen phosphorylase and activating muscle glycogenolysis. It works on skeletal muscles' beta androgenic receptors and triggers a series of responses, unsettling the regular enzymatic action in skeletal muscles. Moreover, heat stress leads to the discharge of glucocorticoids and causes vasodilation, proteolysis, and lipolysis in the muscle through the activation of HPA and the sympathetic-adrenal-medullar axis (SAM). Glucocorticoids improve the synthesis of glucose to ensure survival under acute conditions. In addition, heat stress also plays a crucial role in lipolysis and proteolysis. Increased lipolysis occurs when glucocorticoids trigger the hydrolysis of circulating triglycerides, intensifying lipoprotein lipase activity. It can also stimulate major proteolytic mechanisms by damaging myofibrils in skeletal muscles and negatively regulating anabolic factors such as growth factor (IGF-1), leading to increased proteolysis. Overall, HPA is regarded as a better heat stress indicator than corticosterone by sending more signals of danger or stress to an animal (Nawaz *et al.* 2021).

Oxidative stress in heat stressed poultry

Oxidative stress is an imbalance between reactive oxygen species (ROS) and the antioxidant capacity of animal cells (Mihaela *et al.* 2020). ROS delivers molecules for normal biologic processes. However, this condition also occurs when oxygen-based molecule collection contains a free radical oxidizing cellular component, leading to oxidative injury and oxidative damage of proteins and DNA, when left untreated (Auten and Davis 2009; Shokryazdan *et al.* 2017). During oxidative injury, the liver is one of the most affected tissues in the body (Saracila *et al.* 2019). Increased ROS overwhelms the buffering system of the liver and leads to oxidative deterioration of enzymes, mitochondrial membrane and cellular lipids. To maintain the redox balance, the liver needs to neutralize the excess ROS with the aid of antioxidants and antioxidative enzymes. Additionally, the liver plays an essential role in avian lipogenesis as it synthesizes up to 90% of fatty acids. These fatty acids are packaged as very-low-density lipoprotein (VLDL) molecules and serve as the primary energy source for other tissues. Furthermore, hepatic lipids are essential during egg production to nourish the embryo *via* yolk targeted VLDL (VLDLy) (Emami *et al.* 2020). It is also noteworthy that aside from its role in lipid metabolism, the liver plays a critical role in processing carbohydrate, protein, vitamin, mineral metabolism, and detoxification (Zaefarian *et al.* 2019).

Cells possess two effective defensive mechanisms to maintain normal cellular processes under elevated temperatures. This mechanism includes producing heat shock proteins (HSPs) and increasing the antioxidant production inside the cell. The HSPs are produced by cells to address stress-induced conditions by regulating the heat shock factors (HSFs) gene. The HSP70 and HSP90 have cytoprotective action and perform as chaperons that assure the correct folding of proteins. An increased expression of HSP70 can stimulate the production of superoxide dismutase (SOD), glutathione (GSH) and total antioxidant capacity (TAOC). During acute heat stress endotoxins, HSFs and HSPs tend to rise (Shehata *et al.* 2020; Wasti *et al.* 2021). Moreover, to increase the production of antioxidants inside the avian cell, a redox-sensitive nuclear transcriptional factor (Nrf2) is transferred to the nucleus in response to oxidative stress. Once Nrf2 reaches the cell nucleus, it stimulates the production of different antioxidants through binding in the promoter region of genes responsible for producing antioxidants, such as GPX1, GPX3, PRDX1, SOD1, SOD2, TXN and NRF2. The predominant free radical produced in the cell is superoxide radical catalyzed by superoxide dismutases (SOD), such as SOD1 and SOD2, into hydrogen peroxide (H_2O_2). SOD1 is a copper-zinc enzyme mainly found in the cytoplasm, nucleus, lysosomes, mitochondrial intermembrane spaces, and peroxisomes. Meanwhile, SOD2, also called manganese-dependent superoxide dismutase (MnSOD), is a manganese enzyme typically found in the mitochondria. Given their function, SODs play an essential role in cells' first level of antioxidant defense. Peroxide produced by SODs is moderately more stable than superoxide and the other ROS species; however, their diffusion within the cell can oxidatively damage proteins and lipids. GPX1 and GPX3 are antioxidant-related genes that maintain peroxide at a healthy level in the avian cell. They are known to be glutathione structured selenium dependent. GPX1 is abundant in the mitochondria and cytoplasm, whereas GPX3 is abundant in the plasma. They both catalyze the glutathione to lower hydroperoxides and H_2O_2. H_2O_2 and hydroperoxides and their proximities are also reduced by PRDX1. PRDX1 is a member of the Peroxiredoxins family using thioredoxin to counteract ROS inside the cell. Thus, TXN belongs to the thioredoxin family, essential in the cellular antioxidant system involved in DNA and protein repairs, immune response and cell death endotoxins (Wasti *et al.* 2021).

Furthermore, the rate of ROS production and electron transport are inversely related. Exposure to heat stress for 3 h in broilers has been reported to repress mitochondrial respiratory complexes (I, III and IV). Mitochondrial respiratory complexes I and III are known to be the major sites of ROS production in the electron transport chain. In the mitochondria, the antioxidant enzymes, like superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GSH-Px), play a vital role. GSH-Px converts ROS to more inert species. SOD, as previously discussed, converts superoxide to hydrogen peroxide (H_2O_2) to buffer the superoxide levels, while CAT converts H_2O_2 into water and molecular oxygen to balance the H_2O_2 level. Nevertheless, despite the increased response of these antioxidant enzymes, 3 h of heat stress is usually adequate for oxidizing cellular lipids and proteins, overwhelming the buffering liver capacity. A compromised mitochondrial function affecting metabolism and energy balance is also commonly found under heat stress. Mitochondria serve as the main generator of cellular ROS but, simultaneously, denote a highly susceptible target of ROS-mediated damage. Due to the proximity of phospholipids in the mitochondrial membrane to the site of ROS generation, they are very vulnerable to oxidative damage. This leads to the production of reactive by-products, including malondialdehyde (MDA) which is commonly used as a biomarker for oxidative injury, and 4-hydroxy-trans2-nonenal (Ismail *et al.* 2013; Emami *et al.* 2020; Wasti *et al.* 2021).

Immunosuppression in heat stressed poultry

Several factors need to be taken into account when examining the immune system and its function. One of the notable factors in the immune response is environmental effects (Lacetera 2012). According to Monson *et al.* (2018), heat stress makes poultry highly vulnerable to disease as it

represses its immune response. Moreover, heat stress potentially causes pathological atrophy of primary and secondary lymphoid tissues, which reduces lymphocytes that play an essential function in immune reaction (Hirakawa *et al.* 2020). A decrease in circulating antibodies, like IgM and IgG, is generally observed in birds under heat stress. According to Mashaly *et al*. (2004), the synthesis inhibition of T and B lymphocytes leads to suppressing the phagocytic activity of blood leukocytes influenced by high temperatures. They have found that birds under heat stress have a lower rate of white blood cell (WBC) and higher heterophil/lymphocyte ratio (H/L ratio). Reduced WBC indicates a decrease in the number and activities of leukocytes, while a high H/L signals stress in poultry. Furthermore, Hirakawa *et al*. (2020) found that heat stress affects the induction of antigen-specific antibody production in broilers. This coheres with Mashaly *et al*. (2004), who document that heat-stressed birds show significantly lower antibody titers to sheep red blood cells, indicating declined antibody synthesis. They suggest that this can be associated with the growth of inflammatory cytokines underneath stress conditions which can stimulate corticotropin-releasing-factor production from the hypothalamus. This leads to increased adrenocorticotropic hormone from the pituitary, which eventually promotes corticosterone production from the adrenal gland. Corticosteroid is responsible for inhibiting antibody production.

Heat stress can also negatively impact nutrient digestion and absorption. Heat stress is also reported to stimulate the hypothalamic-pituitary-adrenal (HPA) axis and therefore generate corticotropin-releasing hormone (CRH) and adrenocorticotropic hormone (ACTH). This occurrence eventually leads to increased corticosteroid levels causing fat accumulation, fatty acid synthesis, and protein catabolism. This also damages gastrointestinal functions by decreasing jejunum villi height and increasing organ permeability to microorganisms. Aside from morphological changes in the gut epithelium, the alteration in jejunal fatty acid-binding protein1 (FABP-1), glucose transporter (GLUT2), and a cluster of differentiation 36 (CD36) have also been observed in heat-stressed poultry. These factors affect nutrient absorption. Reduced digestibility induced by heat stress results from reduced digestive enzymes, like amylase, maltase, trypsin, lipase and chymotrypsin. Moreover, heat stress causes mucosal lesions in the small intestine and leaky gut syndrome (Emami *et al.* 2020), leading to intestinal inflammation, bacterial translocation, and compromised bird health and performance (Gilani *et al.* 2021). Heat stress also affects the ileum, which serves as the small intestine terminal part and is associated with the absorption of most nutrients in poultry. The gastrointestinal tract plays an essential role in animals' immune response in regulating paracellular penetration of endotoxins, pathogenic bacteria and feed-associated antigens. However, this tract can be easily affected by the intestinal epithelial tight junctional barrier. This tight junction is formed mainly of transmembrane proteins, such as occludin (OCLN) and claudin (CLDN). Occludin aids in cellular structure and barrier part, while claudin constructs the backbone of tight junctions and determines the tight junction's ability to tape the paracellular space. During heat stress, the blood's peripheral circulation is grown and this reduces blood flow in the intestinal epithelium, causing hypoxia. Hypoxia can lead to tight junction disorder, intestinal integrity reduction, and intestinal permeability increase. These circumstances contribute to the rise of circulating endotoxins (Wasti *et al.* 2021).

The effects of heat stress on chicken production

The thermoneutral zone measured by the Thermal-Humidity Index (THI) is relatively narrow in most poultries. The selection for high growth or egg production rates in commercial poultry lines, which leads to excellent metabolic activity, higher heat production and decreased thermotolerance, makes them particularly vulnerable to heat stress and its detrimental effects. Modern broilers have a high growth rate and increased ROS generation due to increased metabolic demand for oxygen induced by higher metabolic rates. (Emami *et al.* 2020). Decreased feed intake is typical in animals under heat stress as they spend more time drinking water and panting. It also aims to lower the endogenous production of body heat resulting from digestion and feed absorption (Zaboli *et al.* 2017). Reduced feed intake during panting and increased energy expense for maintenance alters the energy balance and is believed to lower body weight and mobilization of adipose tissue in heat-stressed birds (Lacetera 2018). In a study by Souza *et al*. (2016), they observed a 36 and 21% decrease in body weight in broilers exposed to continuous and cyclical heat stress, respectively. Poultry under heat stress experiences a sharp increase in body temperature, and the effort to dissipate excess heat, blood circulation, and peripheral blood flow is increased while visceral blood flow is decreased. This occurrence restricts nutrient utilization and lower production performance as well as feed conversion efficiency (Emami *et al.* 2020). Moreover, heat stress can alter hypothalamic peptides involved in appetite regulation. Studies suggest that poor feed efficiency can be associated with the reduced distribution speed of feed residue and a lower rate of chymotrypsin, trypsin, and amylase activities. Poor feed efficiency also can affect nutrient absorption and intestinal morphology. These factors significantly reduce protein digestion in broilers and decrease the digestibility of various diet components, such as proteins, starch, and fats (Souza *et al.* 2016). Meanwhile, a significant decrease in body weight, egg weight, egg production, and eggshell quality is observed in laying hens exposed to heat stress conditions caused by the alteration in the status of Ca^{2+}, acid-base balance and diminished capability of duodenal cells in calcium distribution, egg

production, and skeletal integrity (Mashaly *et al.* 2004).

Panting decreases blood bicarbonate levels, affecting calcium availability in the blood for eggshell mineralization. This condition is hypothesized to cause poor egg quality. However, its physiological mechanism is not yet fully understood. Heat-stressed birds often raise their wings, rest more and move less (Lara and Rostagno 2013). The neuroendocrine of birds is also affected during heat stress. The disruption of thyroid activity affects poultry reproduction as the thyroid gland is involved during the onset of puberty, as in the case of hens and birds (Elnagar *et al.* 2010). Overall, heat stress causes multiple serious negative effects on poultry performance, like decreased feed efficiency, growth, intestinal integrity, egg production and survival (Monson *et al.* 2018).

Poultry may even die when the body temperature exceeds 45°C, which can significantly affect normal body function. Vale *et al.* (2010) report increased mortality in broilers older than 31 days at a maximum temperature-humidity index (THI) of 30.6°C. Increased mortality in broilers aged 31-40 days has also been reported when the environmental temperature reaches 34.4°C. Al-Fataftah and Abu-Dieyeh (2006) noted a higher mortality rate at ambient temperatures over 25°C. Increased mortality by 31.7% in heat-stressed layers was also reported in a study by Mashaly *et al.* (2004).

Mitigating strategies for heat stress in poultry

Recent discussions have delved into several strategies to alleviate the negative impacts of heat stress. Different approaches to environmental management, nutritional manipulation, as well as genetic selection are being introduced. Adding feed additives and water supplementation with electrolytes are also commonly suggested.

Genetic strategies

Increased susceptibility to heat stress can be addressed by adjusting the development rate and feed efficiency. Fast-growing broilers have higher heat production; hence, they have a higher heat load than slower-growing ones (Yalçin *et al.* 2001). However, Gonet *et al*. (2000) have found that three different lines of hen breeders have comparable growth performance despite the exposure to a hot environment. This offered the opportunity to find genes associated with an increased growth rate with more heat tolerance. Lin *et al*. (2006) enumerated three significant genes that may be important in mitigating heat stress, including the naked neck (*Na*) gene, Frizzle (*F)* gene and Dwarf (*dw*) gene. *Na* gene is reported to reduce feather mass by 20% in heterozygous (*Na/na*) birds and 40% in homozygous (*Na/Na*) birds. They are found to have better body weight and feed efficiency during lower body temperature (na/na). On the other hand, *F* gene causes curling and a decrease in feather size, which is said to reduce heat insulation. Lastly, *dw* gene is the suspected cause of heat tolerance in dwarf broiler breeders and causes a 30–40% reduction in body size.

Previous studies also highlight the function of heat shock proteins (HSPs) in heat stress. Although birds are exposed to improved ambient temperature, synthesis of most proteins at the genetic level is decreased except for heat shock proteins (Kumar *et al.* 2021); hence, they are utilized in heat stress studies. HSPs bind with other cellular proteins to assist intracellular transport. It also facilitates protein structures and folds formation by acting as chaperones. Lastly, it prevents protein aggregation during stress (Kang and Shim 2020). A number of studies have examined HSP families, including HSP27, HSP60, HSP70 and HSP90. HSP60 and HSP70 are responsible for preventing the accumulation of synthesized polypeptides and restoring their native form. HSP70 is also the most conservative and joint constituent of the HSP family in chickens. It prevents lipid peroxidation, improves antioxidant levels and increases digestive enzyme action, which aids in the adaptive reaction to the thermal stress of birds. Meanwhile, HSP90 is associated with developing and modifying proteins pattern in older phases. HSP27 is reported to undergo accelerated phosphorylation during heat stress, resulting in actin polymerization and stress fiber formation. Studies suggest that HSP70 and HSP27 are critical in preserving protein solubility in specific cell compartments. They also protected cells against apoptosis in endothelial populations (Shehata *et al.* 2020). A significant addition in the expression of HSP27, HSP60, HSP90a, HSP70 and HSP90b has been discovered in chronic heat stress (Vinoth *et al.* 2015). Identifying single nucleotide polymorphisms (SNP) corresponding to thermotolerance also assists in selecting birds that are more resilient against high environmental temperatures. Kumar *et al.* (2021) report that some genes with a differential expression during heat stress are HSPH1, HSP25, BAG3, RB1CC, PDK, and ID1 and suggest that those genes play a significant role during acute stress in chickens (Kumar *et al.* 2021).

Environmental strategies

Several factors should be considered when planning environmental strategies for counteracting heat stress (Lin *et al.* 2006). Most of the recent studies focus on environmental temperature and relative humidity. These factors are proven to affect the evaporative cooling mechanism in birds (Ranjan *et al.* 2019). According to Lin *et al*. (2005), evaporative heat loss is better in high temperatures with wind speed. However, it reduces with higher humidity. The ventilation system also aids in heat stress management. Good ventilation can assist in the removal of ammonia, moisture, and carbon dioxide while providing oxygen in poultry houses (Ranjan *et al.* 2019). Moreover, in a study about thermal manipulation conducted by Al-Zghoul *et al.*

(2018), where broilers are exposed to thermal stress on days 10 to 18 of embryonic development, they observe improved thermotolerance acquisition in birds as reflected by lower mRNA expression of the genes of redox pathway such as NOX4, GPx2, SOD2 and catalase. This is indicative of a decrease in heat-induced oxidative stress. Lower cloacal temperature upon exposure to acute heat stress is also reported to support better adaptation to changing environmental temperatures. Meanwhile, Basilio *et al.* (2003) indicated that thermal conditioning at 40°C for 24 h of 5-day-old chicks can decrease body temperature. Aside from improving body temperature, thermal exercise improves plasma MDA and glucose levels (Oke *et al.* 2020).

Nutritional strategies

Dietary manipulations are suggested to alleviate the effects of heat stress. One of the most straightforward techniques is providing drinking water with electrolyte solutions. This technique will control the acid-base equilibrium in the heat-stressed bird. Studies also suggest that giving vitamin C, E and selenium can mitigate the harmful consequences of heat stress. Vitamin E is known to have free radical quenching activity that helps to combat oxidative injury. It contains tocopherols and tocotrienols, making it fat-soluble. The alpha-tocopherol, an active vitamin E form, is affected in the glutathione peroxidase pathway by protecting organisms from oxidative harm by responding with lipid radicals produced in the lipid peroxidation reaction. Oxidized alpha-tocopherol radicals are recycled back to their functional shape by lowering other antioxidants, such as vitamin C. An antioxidant compound like vitamin C is a water-soluble substance that can improve an animal's immune system, defends cells against oxidative deterioration, and serve as a crucial co-factor in enzyme reactions. Vitamin C can act as a co-antioxidant with different antioxidants by synergistic effects. Studies confirm that adult birds can normally synthesize vitamin C to complete their necessities. However, their needs are found to rise during stress. Both vitamins C and E are also reported to enhance feed intake, eggshell quality and body weight. Meanwhile, selenium is a vital micronutrient that acts as a co-factor for antioxidant enzymes, such as superoxide dismutase, glutathione peroxidase and thioredoxin reductase and is thought to be a co-factor for iodothyronine deiodinase.

The activation or inactivation of these enzymes release hormone T4 to T3 or reverses triiodothyronine (rT3) with the aforesaid indirect influence on the principle of T3 and T4 production of selenium, and it can influence the protein synthesis and the metabolism of fat, protein, carbohydrate, and vitamins, and animal basal metabolic rate. It is expected that the thyroid hormone synthesis is defective under stress, and providing the selenium helps restrain the thyroid hormone synthesis and restore body homeostasis (Shakeri *et al.* 2020; Goel 2021). Adding fat to the diet pattern is another technique to mitigate heat stress in poultry. Fat also produces lower heat compared to carbohydrates and protein during metabolism. A lower rate of food passage, which increases nutrient utilization on the GI tract and increases the energy value of food constituents, is observed in birds given fat supplementation. Moreover, providing a 5% fat diet benefits both layers and broilers and increases feed intake by 17% in heat-stressed laying hens. Another advantage of adding fat is the improved performance of broilers. Improving oil supplementation in higher protein levels is acknowledged to relieve the harmful impacts of chronic heat stress on meat lipids, chicken production, and its physiological and immunological characteristics (Wasti *et al.* 2020). Htin *et al.* (2007) mentioned that the addition of fat improves the palatability of feed and may be considered as the contributing factor to higher feed consumption and better performance of birds.

Another common practice to decrease heat rate in poultry is reducing metabolic rate through feed restriction during hot periods. Unfortunately, this may harm the growth rate, hence introducing a dual feeding regime. In this practice, a high amount of protein is supplied during cooler periods, while a high amount of energy is supplied during the warmer periods of the day. This method takes into consideration the fact that protein produces higher metabolic heat than carbohydrates. Lastly, wet feeding may also be considered. This practice aims to improve water consumption and reduce the viscosity in the gut, resulting in a faster feeding passage. In addition, wet feeding stimulates pre-digestion, enhances the absorption of nutrients in the gut, and accelerates digestive enzyme activity. Improved performances associated with wet feedings, such as better body weight, feed consumption and GI tract weight, are reported in broilers, while increased dry matter consumption, egg weight, and egg production are found in layers. However, this practice remains underexplored, as it poses the threat of fungal growth in feed, causing mycotoxicosis (Wasti *et al.* 2021).

Plant extracts used in mitigating the effects of heat stress

Nutrition management is more economically feasible than genetic and environmental strategies when addressing heat stress (Saracila *et al.* 2021). Furthermore, several investigations have proven that certain plant extracts can be used to alleviate the harmful results of heat stress. Plant extracts are also cheap and widely accessible (Shokryazdan *et al.* 2017). The following discussion will discuss the effect of plant extract in alleviating heat stress problems.

Effect of plant extract on metabolic disorder

In dealing with metabolic disruptions due to heat stress, most studies focus on alleviating the apparent signs of heat stress, such as raising wings and panting, which significantly affects the maintenance of the acid-base

balance in affected birds. The assessment of hematologic parameters is also commonly done. In a study by Zmrhal *et al.* (2018), panting and weightlifting were significantly decreased in broiler chickens provided with *Scutellaria baicakensis* L. in comparison with the control group, indicating the association with the anxiolytic result of plant's functional compounds, such as wogonin, baicalin, and baicalein. These compounds pose diverse influences on the nervous system, affecting the behavior of heat-stressed birds. Additionally, although *Scutellaria baicakensis* L. does not significantly impact feed and water consumption, an improved feed conversion ratio has been observed in experimental birds. El-Shoukary *et al.* (2014) also observed a significant decrease in panting behavior in heat-stressed chickens given black seed (*Nigella sativa*). The authors suggest that it may be due to some biological value of the chemical composition of black seed. Likewise, Iraqi *et al.* (2013) also observed that broilers with ginkgo (*Gingko Biloba*) and peppermint (*Mentha piperita*) showed a decrease in panting and weightlifting. This can be associated with the ability of *Gingko Biloba* to promote vasodilation in the brain, which improves oxygenation in brain tissue and acts on the hypothalamic level. Furthermore, it helps to reduce the expression and secretion of corticotropic releasing hormone. Meanwhile, peppermint is known to improve circulation, dispel fevers, and cool the skin and mucosa through stimulating cold receptors. These interventions help to reduce the negative effects of heat stress on the behavior of affected birds.

Moringa oleifera is another plant helpful to combat the negative metabolic effect of increased environmental temperature. When given to broiler chicks under heat stress, a significant increase in hemoglobin concentration was observed compared to the control group. Given the function of hemoglobin to transport oxygen, nutrients and waste, a higher value may indicate a more significant potential in addressing the negative impacts and better health status (Hassan *et al.* 2015). Black grape (*Vitis vinifera*) is another highly valuable plant for birds suffering from heat stress. It is reported to decrease the concentration of serum glucose. This action is associated with its high flavonoid content that inhibits renal glucose reabsorption by inhibiting sodium-glucose symporters found in the proximal renal tubule. Stress hormones alter energy, protein, lipid and mineral metabolisms, blood gases, acid-base and electrolyte balances, and haemoglobin concentration. The increased blood glucose level in the presence of a stressor is commonly observed during stress. By extension, it helps to mobilize or produce glucose for energy to retain homeostasis; thus, the hypoglycaemic activity of grape seed helps alleviate the negative effects of heat stress (Hajati *et al.* 2015).

Effect of plant extract on oxidative stress

Few strategies have been suggested when combatting oxidative stress in exposed heat-stress conditions on poultry, such as reducing membrane potential, increasing electron transport chain efficiency, and improving ROS detoxifying capacity, which remains the principal target in heat-stressed birds. Scavenging ROS can improve ROS detox capacity by interfering with enzymatic processes that lead to ROS development, chelating trace elements affected in ROS development, or upregulating and shielding endogenous antioxidant defense (Akbarian *et al.* 2016). In a study by Wasti *et al*. (2021), the supplementation of dried plum (*Prunus domestica*) significantly improves the expression of NRF2, GPX1, GPX3, SOD1, SOD2, PRDXN and TXN genes. They claimed that Nerf2-mediated antioxidant could be activated by dried plum, resulting in the upregulation of the antioxidant gene in heat-stressed birds and lower ROS and lipid peroxidation within the cell. On the other hand, Ghanima *et al*. (2019) reports that supplementing boldo (*Peumus boldus Molina)* to heat-stressed birds decreases MDA, GPx, and SOD levels. Boldo leaves are known to have a high content of catechin and boldine, associated with the potent antioxidant activity and free radical scavenging of this plant. Boldine and catechin are reported to counteract scavenging and neutralizing the overabundance of free radicals that subsequently increase the antioxidant system of heat-stressed birds and reduce the oxidative stress biomarkers to the normal level. Similarly, birds under heat stress that were given both ginger (*Zingiber officiale*) and thyme (*Thymus vulgaris*) showed reduced liver MDA concentration. Increased serum total antioxidant capacity (TAC) and total superoxide dismutase (TSOD) were also observed in birds supplemented with ginger compared to the control group. Ginger root contains several antioxidant compounds, such as shogaol, gingerol, zingerone, and diarylheptanoids (Habibi *et al.* 2014). Meanwhile, the ability of thyme to act as a natural antioxidant is associated with phenolic hydroxyl groups functioning as hydrogen donors to the proxy radicals produced during the initial stage of lipid oxidation. This method effectively inhibits the formation of hydroxyl peroxide (Abdel-Ghaney *et al.* 2017). Plants rich in vitamin C, such as *Moringa oleifera* and *Glycyrrhiza glabra*, also help to relieve the negative effects of heat stress through their antioxidant activity (Al-Daraji *et al.* 2012; Hassan *et al.* 2015).

Furthermore, the induction of HSPs is also essential in combatting oxidative stress. These molecules enhance cell stability and help develop thermotolerance during heat stress states. They also promote cell survival and prevent apoptotic functions in different cell types (Shehata *et al.* 2020). Tang *et al*. (2018) reported that rosemary (*Salvia rosmarinus*) could preinduce HSP70 expression before stress occurs. They also note that during acute stress, HSP70 level is decreased in birds given rosemary, suggesting sufficient HSP70 in myocardial cells under heat stress. By contrast, HSP70 levels are reported to increase in birds under heat stress conditions without rosemary supplementation, followed by a decrease in HSP70 levels when exposed to

heat for a longer period. This decrease can be associated with the inability of the body to tolerate the severe damage from heat.

Effect of plant extract on immunosuppression

Several factors affect the immune response of heat-stressed birds, thus allowing different approaches to be implemented. Many studies focus on the health of the gastrointestinal tract when dealing with an immune response, as it is regarded as one of the main targets of heat stress. *Nigella sativa,* or black cumin, has long been used as traditional medicine for various diseases in the Middle East and has proven to have a wide range of pharmacological benefits. The antimicrobial activity of black cumin can be very helpful to help birds combat different infectious diseases. Thymoquinone (TQ) is attributed to the antimicrobial effect of *Nigella sativa.* TQ is effective to fight against *Listeria monocytogenes*, methicillin-resistant *Staphylococcus aureus* (MRSA), *Streptococcus* spp., *Pseudomonas aeruginosa*, *Proteus vulgaris, Klebsiella penumoniae, Bacillus subtillis* and *Escherichia coli.* It is also effective against viruses, parasites, and fungi (Forouzanfar *et al.* 2014). Other plants that may help mitigate the negative effects of heat stress through their antimicrobial property include rosehip (*Rosa canina* L.) and willow bark (*Salix alba*). Rosehip inhibits the development of *Escherichia coli* colony and is effective against *Clostridium perfringes* (Criste *et al.* 2017). On the other hand, the phenolic compounds of willow bark have bactericide and bacteriostatic properties for depressing the adhesion of pathogens, such as *E. coli* and *Clostridium* spp. What is more, these plants can improve nutrient utilization and animal performance (Saracila *et al.* 2019). Al-Daraji (2012) have documented the potential antiviral action of licorice (*Glycyrrhiza glabra*) against Newcastle disease virus (NDV), and it was also noted for its immunomodulatory activity. Given to poultry, licorice can improve a bird's humoral immunity by inducing antibody titers against non-specific and specific antigens. Its glycyrrhiza polysaccharide is said to have a sturdy immune action and is recognized to be involved in immune regulation. It increases WBC counts and ultimately boosts interferon levels; hence it can improve immunity. Licorice is also reported to improve the cellular immunity of layers through the increased phagocytic capacity of mononuclear cells and granulocytes of chicken. This is seen as very beneficial as heat stress is documented to inhibit B and T lymphocyte production and ultimately suppresses phagocytic activities. Additionally, decreased H/L ratio is observed in birds given licorice compared with the control group. An increase in H/L ratio usually indicates that a chicken suffers from acute stress. Another plant with a similar effect on the immune system of chickens is peppermint (*Mentha piperita*). Peppermint helps to protect lymphocytes against damages brought by free radicals produced during heat stress. It can also stimulate the production of interferon and enhance phagocytosis. Peppermint supplementation also stimulates increased TIg, IgM, and IgG titer against SRBC. Increased albumin: globulin ratio is also reported, indicating improved liver function (Arab-Ameri *et al.* 2016).

Aside from enhancing the immune system of birds under heat stress, improving gut health can be another beneficial option. Oregano (*Origanum vulgare*) and rosehip (*Rosa canina* L.) were both documented to have a favorable influence on the colonization of beneficial bacteria in the gastrointestinal tract as it suppresses the development of pathogenic bacteria, like *E. coli* (Criste *et al.* 2017). Gut microbiome plays a vital function in birds' intestinal health and development. In the same vein, according to Wasti *et al*. (2021), *Prunus domestica* can significantly increase the expression of the CLDN1 and OCLN in the ileum of heat-stressed birds, suggesting improved integrity of the digestive tract. This approach can be attributed to the flavonoid component of dried plums used in the experiment. Flavonoids are reported to indicate protective and promotive results on intestinal tight junction barrier functions. Additionally, an increase in IL4 and MUC2 expression was also observed in affected birds supplemented with dried plum. Interleukine 4 (IL4) is a cytokine that plays a critical function in controlling the immune system and cellular homeostasis; thus, its significant IL4 gene expression suggests an enhanced immune response. Another gene, MUC2, also associated with mucin, plays an essential role in defending the gut from acidic chyme, pathogens, and digestive enzymes and in affecting nutrient absorption and digestion.

Effect of plant extract on production performance

Combatting the negative effects of heat stress on production performance, such as decreased feed efficiency, growth, intestinal integrity, egg production and survival, is also crucial as it not only affects the wellbeing of birds but also the farmers. Dried plum has an auspicious effect on the production performance of broilers under heat stress. Wasti *et al.* (2021) acknowledges that the supplementation of dried plum leads to a significant increase in average daily gain (ADG), total body weight, average daily feed intake (ADFI), and feed conversion ratio (FCR) compared to birds without dried plum supplementation. As an antioxidant, dried plums are proven effective to combat the negative effects of heat stress due to their tight junction-related genes and capacity to improve the relative abundance of beneficial bacteria to support nutrient absorption and the overall health of heat-stressed birds. Similarly, *Moringa oleifera* helps heat-stressed birds to attain better body weight gain and FCR, presumably due to the improvement in crude protein digestibility and nutrient utilization in the presence of flavonoids which serve as antibacterial and antioxidants. It can also be associated with the positive effect of *Moringa oleifera* on the gut's microbial environment, leading to

enhanced digestion, absorption, and utilization of nutrients (Hassan *et al.* 2015). Meanwhile, rosemary, dill, and chicory extracts are reported to improve egg index, eggshell weight, Haugh unit and yolk index in layers suffering from heat stress compared to the control group (Torki *et al.* 2018). Despite these promising findings, further studies need to check for replicability and explain the mechanism behind these results.

Plant extracts with potential benefit against heat stress in the philippines

Increased ambient temperature is the primary aspect that negatively impacts animal performance and production in tropical, subtropical, and arid regions (Habeeb 2020). In the Philippines, heat stress caused by high environmental temperature is a common issue in poultry farms, especially during summer. The poultry industry in the country is dominated by smallholders (Chang 2004). As such, offering safe, accessible and low-cost alternatives such as plant extract to mitigate heat stress's negative effect is a highly beneficial solution. One of the most abundant plants in the Philippines for addressing heat stress is *Moringa oleifera.* It is notable for its ability to boost the immune response of heat-stressed birds. Turmeric (*Curcuma longa*) is also reported to improve the productive performance and health of broilers reared under heat stress conditions. It enhances affected birds' immune response and antioxidant systems (Sugiharto 2020). Furthermore, *Psidium guajava* is another common plant in the country that can mitigate the adverse impacts of heat stress. Ngoula *et al.* (2017) found that using guava can significantly counteract the negative impacts on cavies' reproductive system, such as sperm quantity and quality. Lemongrass (*Cymbopogon citratus*) is also found to alleviate the harmful effects of heat stress on rabbits due to its antioxidant activity (Daader *et al.* 2018).

Conclusion

Heat stress can result in massive financial loss to farmers due to a decrease in growth, reproduction, meat and egg production, health through metabolic disorders, oxidative stress and immunosuppression. Genetic improvement, environmental modification, and nutrient intervention can help mitigate the heat stress in poultry. Adding plant extracts can decipher the effects of heat stress in poultry while improving its overall performance. The effective method and dosage of plant extract supplementation need further investigation. Comprehensive *in vitro* or *in vivo* studies are required to identify the best approach to heat stress and examine the potential of other plant extracts in strengthening poultry defense against heat. stress.

Author Contribution

MMM and JSDC conducted this study, writing and literature review. HK, DC and LP as co-author performed the manuscript writing.

Conflicts of Interest

The author declares no conflict of interest of any sort.

Data Availability

Not Applicable in this paper.

Ethics Approval

This study does not involve human subjects. Thus, ethics approval is not required.

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