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The Role of Vitamin C in Mitigating Stress in Poultry: A Comprehensive Review

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ABSTRACT

Stress refers to the adverse physiological effects induced by different environmental and management conditions on animal health and performance. These challenges induce oxidative stress by disrupting antioxidant homeostasis, a condition exacerbated by the depletion of plasma antioxidants, including vitamins C and E, folic acid, and zinc. Prevalent stressors encompass transport-related variables, including thermal and humidity fluctuations, handling deficiencies, feed and water withdrawal, vehicular inadequacies, motion, and noise, as well as pathological states such as coccidiosis, vaccination, and confinement. Live birds of all ages are mostly transported by road across ecological zones increases, so does associated mortality, with outcomes ranging from diminished welfare to severe morbidity and death. The present review synthesized current knowledge on the role of vitamin C in mitigating stress in poultry, highlighting its potential for future research. Evidence indicated that dietary supplementation of 100-200 mg/kg vitamin C effectively alleviates stress-related impairments, enhancing poultry productivity.

Keywords: Ascorbic Acid, Heat Stress, Immune Function, Poultry Stress, Vitamin C.

INTRODUCTION

The transport of live poultry is an integral component of commercial production systems, yet it imposes a multitude of interrelated stressors that significantly challenge avian homeostasis and welfare. These stressors—including thermal fluctuations, feed and water deprivation, crowding, noise, and handling elicit a complex physiological stress response. This response is characterized by the activation of the hypothalamic-pituitary-adrenal (HPA) axis, leading to the release of corticotropin-releasing hormone (CRH), adrenocorticotropic hormone (ACTH), and ultimately glucocorticoids like corticosterone. Consequently, transport stress is a major contributor to economic losses, manifesting as increased morbidity, reduced live weight, poorer meat quality, and

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mortality, with studies attributing up to 40% of dead-on-arrival (DOA) incidents to transport-related factors [1-3].

A critical consequence of this stress response, particularly under heat stress, is the disruption of metabolic and oxidative balance. High ambient temperature and humidity, common during transport in tropical regions, suppress voluntary feed intake. This reduction directly limits the dietary intake and absorption of essential micronutrients, including the potent antioxidants vitamins A, C, and E [4]. Concurrently, stress potentiates the metabolic generation of reactive oxygen species (ROS), creating a state of oxidative stress where ROS production overwhelms the bird's antioxidant defenses [5]. This oxidative damage to lipids, proteins, and DNA impairs cellular function and is a key mediator of the negative outcomes observed, including immunosuppression, reduced growth rates, and poor meat quality. The severity of these effects is often compounded in arid regions, where extreme climatic conditions and frequent inadequacies in water supply during transit create a particularly challenging environment [6].

While hematological parameters, such as an elevated heterophil-to-lymphocyte (H:L) ratio, provide a reliable biomarker for quantifying this stress load [7], nutritional strategies to mitigate these effects are paramount. A promising intervention is the supplementation of vitamin C (L-ascorbic acid, AA). Although poultry synthesize AA endogenously from glucose via the renal enzyme L-gulonolactone oxidase, this intrinsic production becomes severely insufficient under duress. Conditions such as extreme thermal load, high productivity demands, and parasitic infections drastically increase the metabolic demand for antioxidants, depleting circulating AA levels [8].

Exogenous dietary supplementation of ascorbic acid addresses this deficit through multiple mechanistic pathways. As a potent water-soluble antioxidant, AA directly scavenges free radicals, regenerates vitamin E, and reduces oxidative damage. Furthermore, it demonstrates endocrine-modulating properties, evidenced by its capacity to lower circulating levels of corticosterone and ACTH, thereby blunting the HPA axis response to stressors [9]. By mitigating both the physiological and oxidative components of stress, vitamin C supplementation emerges as a critical strategy for enhancing resilience, improving welfare, and safeguarding productivity in modern poultry production systems exposed to transport challenges.

ROLES OF VITAMIN C

Vitamin C in combating heat stress

Dietary supplementation of L-ascorbic acid (AA) at levels between 100-200 mg/kg of feed has been established as an effective nutritional strategy to ameliorate the adverse effects of stress in poultry, enhancing both productivity and physiological resilience. Its efficacy is demonstrated across multiple performance parameters. In broilers, AA supplementation under thermal stress conditions has been shown to significantly improve body weight gain and feed efficiency by mitigating the hypophagia and metabolic disruptions induced by high temperatures. This is coupled with a marked improvement in thermoregulatory capacity, as evidenced by significant reductions in rectal temperature and respiratory panting rates, allowing birds to better dissipate metabolic heat and maintain homeostasis [10,11].

The benefits extend to the immune system, where AA acts as an immunomodulator. It enhances both humoral and cell-mediated immune responses, leading to improved antibody titers following vaccination and greater resistance to opportunistic pathogens, which are more prevalent during stressful periods [11]. In laying hens, the positive impacts are equally pronounced. Research indicates that ascorbic acid supplementation can increase egg production by 11-23%, a critical improvement during heat stress when production typically declines. This is accompanied by enhanced eggshell quality, as AA is involved in the metabolism of calcium and the formation of the organic matrix of the shell, leading to stronger shells and reduced breakage. Furthermore, improved overall livability in supplemented flocks underscores its role in promoting general health and reducing stress-induced mortality [12].

The antioxidant potential of AA is significantly potentiated when administered in combination with other antioxidants, such as vitamin E. This synergistic relationship is well-documented; vitamin E, a lipid-soluble antioxidant, quenches free radicals within the cell membrane, and in the process, becomes oxidized. Ascorbic acid, residing in the aqueous phase, efficiently regenerates reduced vitamin E, thereby reinforcing the overall antioxidant defense system. This synergistic action has been shown to more effectively lower lipid peroxidation markers (e.g., malondialdehyde) and enhance antioxidant enzyme activity in pullets and quails compared to either vitamin administered alone [13].

It is important to note that the scientific literature presents some heterogeneity in results. A minority of studies have reported no significant effect of AA supplementation on core body temperature (e.g., cloacal temperature) under certain experimental conditions [14]. These discrepancies may be attributed to factors such as the genetic line of the birds, the severity and duration of the stress challenge, the basal composition of the diet, and the specific timing and dosage of the AA administration. This underscores the necessity for further research to precisely optimize supplementation protocols for varying production environments.

Vitamin C in cold stress and disease resistance

Exposure to cold stress presents a significant metabolic challenge to poultry, precipitating a substantial depletion of plasma ascorbic acid (AA) levels. This depletion occurs as the vitamin is mobilized to support essential physiological functions, including catecholamine synthesis for thermogenesis and to counteract elevated oxidative stress. Dietary intervention with ascorbic acid, typically at a dosage of 150 mg/kg of diet, has been demonstrated to effectively counter this depletion. By restoring circulating AA, supplementation mitigates the adverse effects of cold stress, leading to measurable enhancements in key performance metrics such as improved feed efficiency, sustained weight gain, and superior livability [15].

Beyond its role in mitigating abiotic stress, ascorbic acid is a critical modulator of immune competence and disease resistance. Its potent antioxidant properties protect immune cells from oxidative damage during activation, thereby preserving their functional integrity. This immunomodulatory effect translates into significantly improved outcomes following pathogenic challenges. For instance, AA supplementation has been shown to reduce mortality associated with Salmonella Gallinarum infection (fowl typhoid) by enhancing macrophage activity and bolstering the overall immune response [13]. Furthermore, research indicates that ascorbic acid enhances specific immunity against major respiratory pathogens such as Mycoplasma gallisepticum and viral infections like Newcastle disease. It contributes to this defense by supporting the proliferation of lymphocytes, improving antibody production (higher titers), and potentiating the cell-mediated immune response, leading to reduced pathogen load and milder clinical manifestations [16].

The mechanism by which AA confers these benefits is multifaceted. It is integral to the synthesis and maintenance of collagen, a crucial structural component of epithelial barriers, thereby enhancing the first line of defense against invading pathogens. Its role as a water-soluble antioxidant ensures the protection of immune cells from free-radical-mediated damage, allowing for a more robust and effective immune reaction. By mitigating the immunosuppressive effects of stress-induced corticosterone, ascorbic acid helps maintain immune homeostasis, making it a vital nutritional strategy for promoting flock health in challenging environments.

Vitamin C as an antioxidant and photoprotectant

Ascorbic acid (AA) serves as a primary, water-soluble antioxidant within the physiological system, playing a multifaceted role in cellular defense and structural integrity. Its fundamental mechanism involves the direct scavenging of reactive oxygen species (ROS) and free radicals, thereby preventing oxidative damage to vital cellular components such as lipids, proteins, and DNA. A critical synergistic function of AA is its ability to regenerate vitamin E (α-tocopherol) from its oxidized form. Vitamin E, a lipid-soluble antioxidant, neutralizes free radicals within cell membranes. In the process, it becomes oxidized, and ascorbic acid efficiently reduces it back to its active state, thus perpetuating the antioxidant cycle and enhancing the overall protective capacity of the cellular antioxidant system [17].

Beyond its systemic antioxidant role, AA is an essential cofactor for the biosynthesis of collagen, the primary structural protein in connective tissues, including the skin. It is required for the hydroxylation of proline and lysine residues, a post-translational modification critical for the stabilization of the collagen triple helix. This function is paramount for maintaining dermal strength and elasticity. By promoting robust collagen synthesis, ascorbic acid significantly accelerates wound healing processes, facilitating tissue repair and regeneration. Furthermore, it contributes to the development and maintenance of a competent skin barrier function, which is the first line of defense against environmental pathogens and stressors [18].

The vitamin's antioxidant properties also provide a direct protective effect against photodamage. Exposure to ultraviolet (UV) radiation generates an excess of ROS in the skin, leading to oxidative stress, inflammation, and tissue degradation. Topical or dietary ascorbic acid helps mitigate UV-induced skin damage by neutralizing these free radicals, reducing inflammatory markers, and supporting the repair of damaged tissues, thereby preserving skin health [17].

Pro-oxidant effects and iron absorption

The biochemical role of ascorbic acid (AA) is complex and context-dependent. In vitro, under specific conditions of high concentration and the presence of free transition metals like iron, AA can exhibit pro-oxidant activity by reducing ferric iron (Fe^{3+}) to ferrous iron (Fe^{2+}). This reduction can potentially catalyze the Fenton reaction, generating highly reactive free radicals. However, within the in vivo environment of the gastrointestinal tract, this same reductive mechanism is highly beneficial and is not considered a significant source of systemic oxidative stress. Here, ascorbic acid acts as a potent enhancer of non-heme iron absorption from plant-based feed ingredients. By reducing dietary iron to its more soluble and bioavailable ferrous state (Fe^{2+}) and forming an absorbable iron-ascorbate chelate, it overcomes the inhibitory effects of phytates and polyphenols. This elevated iron status is crucial for optimal immune function, as iron is a vital cofactor for enzymes involved in lymphocyte proliferation and the respiratory burst activity of macrophages. Consequently, by improving iron bioavailability, AA supplementation indirectly strengthens the avian immune system, enhancing resistance to a spectrum of bacterial infections [18,19,20].

It is critical to differentiate the in vitro pro-oxidant potential from the established in vivo antioxidant and immunomodulatory effects. The biological system is replete with antioxidants and metal-binding proteins that tightly regulate metal ion availability, effectively preventing the widespread pro-oxidant effects observed in cell-free systems. Therefore, the net in vivo effect of dietary ascorbic acid, particularly at standard supplementation levels, remains overwhelmingly that of a crucial antioxidant and a vital nutrient for bolstering disease resistance.

Reproductive benefits

Dietary supplementation with ascorbic acid (AA) has been demonstrated to exert significant positive effects on male reproductive performance in broiler breeders, with optimal dosages typically ranging from 125 to 500 parts per million (ppm) in the diet. Research indicates that this nutritional intervention effectively enhances key semen quality parameters, notably leading to a marked increase in sperm cell concentration and a significant improvement in overall sperm motility [21].

The underlying mechanisms for this improvement are multifaceted and are primarily linked to AA's potent antioxidant properties. The spermatogenic process generates substantial levels of reactive oxygen species (ROS), which can induce oxidative damage to sperm lipids, proteins, and DNA, ultimately impairing sperm viability, motility, and membrane integrity. By scavenging these ROS, ascorbic acid protects developing and stored spermatozoa from peroxidative damage. Furthermore, AA is involved in the synthesis of steroid hormones, potentially supporting optimal testosterone production, and contributes to the maintenance of the structural health of the testes and epididymis. This combination of antioxidant protection and endocrine support helps mitigate the negative impact of environmental and metabolic stressors on spermatogenesis, resulting in the production of a higher quantity of robust, functionally competent spermatozoa, which is critical for improved fertility rates in breeding operations.

ABSORPTION, TRANSPORT, AND METABOLISM OF VITAMIN C IN POULTRY

The intestinal absorption and cellular uptake of vitamin C (L-ascorbic acid, AA) in poultry are governed by specialized and complementary transport systems, ensuring its distribution to vital tissues. The primary mechanism for the active transport of reduced AA is mediated by sodium-dependent vitamin C transporters (SVCTs), specifically the isoforms SVCT1 and SVCT2. SVCT1 is predominantly responsible for bulk absorption across the intestinal epithelium. In contrast, the SVCT2 isoform exhibits nearly ubiquitous expression throughout the body, facilitating the uptake of AA into most tissues and organs to maintain critical physiological concentrations. An exception is mature erythrocytes (red blood cells), which lose their SVCT proteins during cellular maturation and therefore cannot accumulate AA [18,22].

A secondary, parallel pathway exists for the oxidized form of vitamin C, dehydroascorbic acid (DHA). Due to its structural similarity to glucose, DHA is absorbed via facilitated diffusion through sodium-independent glucose transporters, primarily GLUT1 and GLUT3 [20]. Following cellular uptake, DHA is rapidly reduced back to its functionally active form, AA, by enzymatic systems utilizing glutathione and NADPH. This efficient intracellular recycling mechanism ensures that plasma levels of DHA remain exceptionally low under normal physiological conditions [15].

The efficiency of this absorptive machinery is highly dependent on dosage. At standard dietary levels, poultry absorb AA with high efficiency, typically ranging from 70% to 95%. However, this efficiency exhibits a dose-dependent decrease. As dietary intake escalates, the finite number of SVCT and GLUT transporter proteins becomes saturated, leading to a progressive decline in the fractional absorption rate. This pharmacokinetic principle is crucial for determining optimal supplementation strategies to achieve desired plasma and tissue concentrations without wastage.

TISSUE DISTRIBUTION AND STORAGE

IVitamin C (L-ascorbic acid, AA) does not distribute uniformly throughout the body but instead exhibits a pattern of selective tissue accumulation, reflecting the specific metabolic and protective demands of different organs. This targeted distribution is primarily governed by the regulated expression of sodium-dependent vitamin C transporters (SVCT2), which actively concentrate AA against a gradient from the plasma into cells.

The highest concentrations of ascorbic acid, reaching up to 100 times that of plasma levels, are found in tissues with exceptionally high oxidative metabolic activity, endocrine function, or roles in immune cell maturation. These tissues include the adrenal glands (where it is crucial for catecholamine and steroid hormone synthesis), the pituitary gland, the retina (for protection against photoxidative stress), the corpus luteum, and the thymus (supporting lymphocyte development and function) [5].

A second tier of significant accumulation, ranging from 10 to 50 times plasma levels, is observed in organs with substantial biosynthetic, detoxification, or filtering roles. This group includes the brain, where AA acts as a key neuromodulator and antioxidant protector of neural tissue; the liver, a central hub for metabolism and xenobiotic processing; the spleen and leukocytes, which are central to immune defense; and the testes and kidneys. The kidney's high concentration is particularly critical as it is the primary site of endogenous AA synthesis in poultry.

This stratified distribution underscores the physiological priority of allocating ascorbic acid to organs that are most vulnerable to oxidative insult or are essential for systemic stress response and homeostasis.

INTERACTIONS WITH OTHER NUTRIENTS

1. **Folate Metabolism:** Ascorbic acid is a crucial facilitator of one-carbon metabolism. It directly participates in the enzymatic reduction of folic acid to its metabolically active form, tetrahydrofolate (THF), and helps maintain the folate pool in its reduced state. A deficiency in AA can lead to functional folate deficiency even with adequate dietary intake, as the impaired conversion disrupts nucleotide synthesis and amino acid metabolism, processes vital for cell division and growth [23].
2. **Synergism with Vitamin E:** AA exhibits a powerful synergistic relationship with the lipid-soluble antioxidant vitamin E (α-tocopherol). Vitamin E neutralizes free radicals within cell membranes, becoming oxidized to the tocopheroxyl radical in the process. Ascorbic acid, residing in the aqueous cellular compartment, efficiently regenerates vitamin E from this radical form, restoring its antioxidant capacity. This recycling mechanism profoundly enhances the overall lipid antioxidant defense system, protecting cellular membranes from peroxidative damage more effectively than either vitamin could alone [24].
3. **Vitamin D Activation and Calcium Homeostasis:** Ascorbic acid acts as a cofactor for the 25-hydroxyvitamin D_3 1-α-hydroxylase enzyme, which catalyzes the final, rate-limiting step in the activation of vitamin D to its hormonal form, 1,25-dihydroxyvitamin D_3 (calcitriol). This is not merely a biochemical role; it has direct physiological consequences. In poultry, supplementation at levels such as 100 mg/kg diet has been shown to upregulate the expression of calcium-binding proteins (e.g., calbindin-D28k) in the intestinal mucosa. This enhancement significantly improves intestinal calcium absorption, which is directly translated into improved bone mineralization, enhanced skeletal strength, and superior eggshell quality in laying hens [25].
4. **Iron Absorption and Bioavailability:** Within the gastrointestinal tract, ascorbic acid powerfully enhances the absorption of dietary non-heme iron (Fe^{3+}). It acts by reducing ferric iron to the more soluble ferrous (Fe^{2+}) form and forms a stable, absorbable iron-ascorbate chelate. This action effectively overcomes the inhibitory

effects of common dietary compounds like phytates and polyphenols, which can bind iron and render it insoluble. By significantly improving iron bioavailability, AA supplementation supports critical iron-dependent functions such as hemoglobin synthesis and immune competence [26].

DEFICIENCY AND ASSOCIATED PATHOLOGIES

Clinical signs of hypovitaminosis C

Scurvy, the clinical syndrome of profound ascorbic acid (AA) deficiency, manifests when plasma concentrations fall below a critical threshold of approximately 10 μM [24]. This state precipitates a cascade of physiological failures primarily stemming from the vitamin's role as an essential cofactor for numerous enzymes.

The most clinically evident pathology arises from the catastrophic impairment of collagen synthesis. Ascorbic acid is an indispensable cofactor for prolyl and lysyl hydroxylase, enzymes required for the post-translational modification and stabilization of the collagen triple helix. Without adequate AA, the synthesis of mature, functional collagen is severely disrupted. This failure of collagen production compromises the structural integrity of connective tissues throughout the body, leading to the classic signs of scurvy: skin fragility, perifollicular hemorrhages and petechiae, bleeding gums, loosening of teeth, and profoundly impaired wound healing [27].

Concurrently, a hallmark hematological complication of scurvy is the development of anemia. This anemia is multifactorial. A significant contributor is the oxidative degradation of folate. Ascorbic acid normally protects the labile tetrahydrofolate (THF) form from oxidation; in its absence, folate is rapidly destroyed, leading to a functional folate deficiency that impairs DNA synthesis and erythrocyte production (megaloblastic anemia) [24]. Furthermore, AA deficiency can concurrently impair iron metabolism by reducing its absorption from the diet (non-heme iron) and potentially affecting its mobilization, thereby contributing to a microcytic hypochromic anemia. The observed anemia in scurvy is often a complex combination of these processes.

Disease-induced deficiency

Pathogenic challenges, including bacterial infections like fowl typhoid (*Salmonella Gallinarum*) and parasitic infestations such as coccidiosis (*Eimeria* spp.), induce a significant depletion of plasma ascorbic acid (AA) levels in poultry [19]. This depletion occurs as the vitamin is mobilized to support critical immune functions. AA is consumed during the respiratory burst of macrophages for its potent antioxidant activity, which protects immune cells from self-inflicted oxidative damage while they neutralize pathogens. Furthermore, the metabolic stress and inflammatory response associated with infection increase the turnover and metabolic demand for antioxidants, rapidly exhausting circulating AA reserves. This creates a vicious cycle where infection depletes AA, and the resulting deficiency can, in turn, compromise the very immune responses necessary to clear the infection.

Beyond infectious diseases, ascorbic acid supplementation has demonstrated therapeutic efficacy against metabolic disorders like ascites syndrome (pulmonary hypertension syndrome), a leading cause of mortality in fast-growing broilers. The pathophysiology of ascites involves hypoxia, oxidative stress, and pulmonary hypertension. AA supplementation, typically ranging from 100 to 500 mg/kg of feed, helps mitigate this condition through several mechanisms: it improves oxygen utilization, reduces capillary permeability through enhanced collagen synthesis, and acts as a powerful antioxidant to combat the associated oxidative stress. Research has shown that this intervention can lead to a significant reduction in mortality rates attributable to ascites, often by 30-50% [28]. Crucially, these benefits are achieved without any adverse effects on key performance metrics such as body weight gain or feed conversion efficiency, making it a viable nutritional strategy for improving flock health and economic outcomes in high-risk populations.

TOXICITY AND ADVERSE EFFECTS

Ascorbic acid is generally regarded as a safe compound with very low acute toxicity, as evidenced by a high median lethal dose (LD_{50}) of 11.9 g/kg body weight in rodent models. However, chronic consumption of supraphysiological doses can lead to several specific adverse effects, primarily related to its pro-oxidant chemistry and role in metabolic pathways.

The most commonly reported adverse effects are gastrointestinal disturbances. The osmotic effect of a high, unabsorbed concentration of vitamin C in the intestinal lumen can draw fluid into the gut, leading to diarrhea, bloating, and abdominal cramps. Furthermore, the acidity of

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the compound can contribute to nausea and gastrointestinal discomfort, particularly when consumed on an empty stomach.

A more significant pharmacological concern is the potential for iron overload. As a potent reducer of dietary ferric iron (Fe^{3+}) to the more bioavailable ferrous form (Fe^{2+}), excessive ascorbic acid intake can markedly enhance non-heme iron absorption. In susceptible individuals, particularly those with hereditary hemochromatosis or other iron-storage disorders, this can exacerbate iron accumulation in tissues (liver, heart, pancreas), potentially leading to organ damage through iron-induced oxidative stress [29].

Finally, a key metabolic consideration is the potential promotion of renal calculi (kidney stones). A portion of ingested ascorbic acid is metabolized to oxalate, a primary constituent of calcium oxalate stones. High-dose supplementation can significantly increase urinary oxalate excretion (hyperoxaluria), thereby elevating the supersaturation of urine with calcium oxalate and creating a favorable environment for the nucleation and growth of stones, a condition known as oxalate nephropathy. The risk is highest in individuals with a history of renal impairment or calcium oxalate stone formation.

DIETARY SOURCES AND BIOAVAILABILITY

Rich sources: Citrus fruits, peppers, cruciferous vegetables [28].

Stability: Degrades rapidly during feed processing; synthetic AA is more stable than plant-derived forms.

ANALYTICAL METHODS FOR ASSESSMENT

The concentration of AA in plasma or urine is a common and relatively simple measurement, typically conducted using a colorimetric assay based on the reagent 2,6-dichlorophenolindophenol (DCPIP). In this method, the reduction of the blue-colored DCPIP to a colorless state by AA is measured spectrophotometrically. While this assay provides a useful indicator, it primarily reflects *short-term* dietary intake and recent absorption, making it highly variable and less reliable for assessing overall body stores or long-term nutritional status.

For a more robust and definitive evaluation of long-term ascorbic acid status, quantification of tissue reserves is essential. This is most accurately achieved through techniques such as high-performance liquid chromatography (HPLC), often coupled with electrochemical or ultraviolet detection. HPLC allows for the precise separation and measurement of AA within specific tissues, such as the liver, adrenal glands, or leukocytes (white blood cells). Leukocyte AA concentration, in particular, is considered a superior biomarker as it correlates well with tissue saturation levels and is less subject to short-term fluctuations than plasma levels, providing a more integrated measure of the body's functional reserves [29,30].

CONCLUSIONS

Vitamin C (ascorbic acid) is a potent anti-stress agent that is critically important for maintaining homeostasis and enhancing productivity in poultry. Its efficacy stems from a multi-faceted role in physiological regulation. Primarily, it functions as a key component of the antioxidant system, directly scavenging reactive oxygen species and regenerating other vital antioxidants, such as vitamin E and glutathione, thereby restoring redox balance under stressful conditions. Furthermore, it acts as an endocrine modulator, mitigating the stress response by suppressing the secretion of corticotropin-releasing hormone (CRH), adrenocorticotropic hormone (ACTH), and corticosterone.

These fundamental actions translate into significant production benefits. Supplementation, particularly at levels of 100-200 mg/kg of feed, consistently demonstrates enhanced immune competence through improved leukocyte function and antibody production, leading to greater disease resistance. It also improves nutrient utilization by promoting the absorption of dietary iron. In breeding stock, vitamin C is essential for reproductive health, where it enhances semen quality, concentration, and motility in males. Consequently, dietary supplementation at the recommended levels is a strategic nutritional intervention to effectively mitigate the adverse effects of environmental, metabolic, and oxidative stress, thereby reducing mortality and safeguarding overall flock performance and profitability.

DECLARATION

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CONFLICT OF INTEREST

The authors declare that there are no conflicts of interest regarding the publication of this article.

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