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Review Article

Dietary Trace Minerals, Organic Compounds and Phytochemical Bioactives as Nutritional Strategies to Mitigate Heat Stress in Broiler Chickens: A Review

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Abstract

This narrative review examines dietary strategies that have been investigated for mitigating heat stress (HS) in broiler chickens, with particular emphasis on trace minerals (chromium, copper, zinc and selenium) and bioactive compounds including yeast-derived products, polyphenols, essential oils and gamma-aminobutyric acid (GABA). Rather than treating reported optimum doses as directly comparable across studies, the review considers how supplement source, dose, bird genotype, housing and HS conditions may influence the observed response; a dose-response meta-analysis is discussed specifically for chromium, where such evidence is available. Heat stress depresses feed intake and growth and can alter endocrine, immune, oxidative and intestinal responses, with important consequences for poultry production. Nutritional interventions are therefore considered as complementary measures alongside environmental, genetic and feeding-management approaches. The literature reviewed suggests that organic or chelated trace-mineral sources can improve selected performance, mineral-retention or antioxidant outcomes in some heat-stress models, while yeast products, plant-derived bioactives and GABA have also shown beneficial responses in individual studies. However, the strength and consistency of evidence differ among compounds, and mechanisms proposed for these effects are not equally well demonstrated. Reported dose-response relationships are not uniform across supplement classes, and responses may vary with housing conditions, sex, genetic background and the nature of the heat challenge. The review therefore emphasizes evidence-based interpretation, the need for source- and condition-specific dose evaluation, and the importance of validating combination strategies and practical recommendations under commercial conditions.

Keywords: Heat Stress; Broiler Chickens; Trace Minerals; Chromium; Copper; Phytochemicals; Gamma-Aminobutyric Acid; Oxidative Stress; Feed Additives; Poultry Management

Abbreviations

HS: Heat Stress; Cr: Chromium; Cu: Copper; Zn: Zinc; Se: Selenium; GABA: Gamma-Aminobutyric Acid; SOD: Superoxide Dismutase; GP_x: Glutathione Peroxidase; MDA: Malondialdehyde; Nrf2: Nuclear Factor Erythroid 2-Related Factor 2; IL: Interleukin; TNF-α: Tumour Necrosis Factor-A; HPA: Hypothalamic-Pituitary-Adrenal; YPC: Yeast Protein Concentrate; Cu-P: Copper Proteinate; ROS: Reactive Oxygen Species; FCR: Feed Conversion Ratio; BW: Body Weight; HSP70: Heat Shock Protein 70; NF-kB: Nuclear Factor Kappa B; H:L ratio: Heterophil to Lymphocyte Ratio; THI: Temperature-Humidity Index; Na: Naked Neck Gene; F: Frizzle Gene; dw: Dwarf Gene.

Introduction

Global demand for poultry meat and eggs has grown rapidly alongside genetic improvements in broiler productivity, with global consumption of poultry products roughly doubling over the past decade and projected to double again by 2050 [1]. This expansion is occurring against a backdrop of measurable climate warming and an increasing frequency and severity of heatwave events, placing an increasing share of global broiler production at risk of heat stress (HS) even as the industry intensifies to meet rising demand.

Ambient temperature is among the most consequential environmental variables affecting the productivity of broiler chickens. Modern broiler genotypes, selected intensively for rapid growth and a correspondingly high metabolic rate, possess a narrow thermoneutral zone and a limited capacity for evaporative heat loss, since chickens lack sweat glands and rely principally on panting to dissipate excess body heat [1,2]. The normal core body temperature of a chicken is approximately 41-42°C, and the thermoneutral zone that maximizes growth lies between about 18-24°C for adult birds, with chicks requiring temperatures near 32°C during the first week of life [1]. Ambient temperatures above roughly 25-32°C are consistently reported to elicit heat stress (HS) and depress feed intake, while a core body temperature exceeding approximately 45-47°C, the so-called upper lethal temperature, can result in death from heat prostration [1]. When ambient temperature and humidity exceed the upper critical limit of the thermal comfort zone, birds experience HS, a state in which the physiological burden of thermoregulation exceeds homeostatic capacity. Acute HS, a short and rapid rise in temperature, and chronic HS, extended exposure to moderately elevated temperature (sometimes further distinguished as cyclic or constant chronic HS), both compromise feed intake, growth rate, feed conversion, carcass yield and meat quality, and increase susceptibility to enteric and systemic disease [1-3].

The economic scale of the problem is considerable. HS-related losses to the United States poultry industry alone were estimated at $128 to $165 million annually since early 2000s [4], a figure that almost certainly understates current losses given the continued expansion of the industry and the trajectory of global warming [1,2]. The problem is most acute in tropical and subtropical regions, including South Asia and sub-Saharan Africa, where high ambient temperature frequently coincides with high relative humidity, further impairing evaporative cooling, and where open-sided housing without environmental control remains common [2]. Broiler chickens are disproportionately affected relative to laying hens because of their insulating feather cover, absence of sweat glands, and high mass-to-surface-area ratio combined with a faster growth rate and higher metabolic heat output.

Environmental control, comprising fans, evaporative cooling pads and insulated housing, remains the most direct means of managing HS, but is capital-intensive and not universally accessible, particularly to smallholder and open-house operations typical of much of the developing world [2]. Genetic approaches, including selection for the naked neck (Na), frizzle (F) and dwarf (dw) genes and identification of thermotolerance-associated single nucleotide polymorphisms, offer a longer-term route to heat-resilient stock but require sustained breeding investment [2,3]. Nutritional intervention, by contrast, is comparatively low-cost, scalable and compatible with existing infrastructure, and has therefore attracted sustained research interest as a complementary or alternative HS-mitigation strategy that can be deployed immediately within existing flocks.

Existing reviews of poultry HS mitigation commonly address genetics, housing and broad nutritional strategies, whereas fewer reviews compare trace minerals and bioactive compounds in a single framework while also considering differences in dose, source and experimental conditions. GABA has been examined in several experimental studies, but its evidence base is smaller and less consistent than that for some established nutritional interventions. This review brings together experimental work on chromium, copper, yeast and yeast protein derivatives, GABA and selected phytochemical, vitamin and osmolyte strategies, while placing these findings alongside the wider genetic, environmental and feeding-management literature. The aim is to compare the evidence without assuming that similar performance or biomarker responses necessarily arise from a common mechanism. The review is organized around the physiological and economic basis of HS, nutritional interventions, complementary genetic and management strategies, emerging molecular approaches and practical limitations.

Literature Search Strategy

This narrative review is based on the peer-reviewed original articles published between 2000 and 2025, which were identified through searches of PubMed, Scopus, Web of Science and Google Scholar using combinations of terms relating to broilers, heat stress, chromium, copper, zinc, selenium, phytochemicals, ginger, curcumin, essential oils, yeast, GABA, gut microbiota and antioxidant responses. Reference lists of relevant reviews, systematic reviews and meta-analyses were also checked to identify additional primary studies.

Studies were prioritized for inclusion when they investigated broiler chickens exposed to an experimentally defined or naturally occurring heat-stress condition and evaluated a dietary nutritional intervention, clearly described supplementation levels and reported production performance along with any of the physiological, antioxidant, immune, endocrine, gut-microbial, intestinal, or molecular responses. Relevant reviews and meta-analyses were also considered to provide broader context. Where direct evidence in heat-stressed broilers was limited, studies in other poultry categories were considered for contextual purposes and are identified as such in the text. Few studies from the authors' research programme were included where directly relevant, but their findings were considered alongside various independent international studies and were not used alone to establish general recommendations. Geographical origin was not used as an exclusion criterion; relevant studies from different geographical and production settings were considered according to their relevance to the objectives and nutritional interventions addressed in the review. This review retains a broad, international scope rather than a regional focus, and this choice has a direct bearing on how completeness and geographical representativeness should be read. Of the 34 studies and reviews cited, 5 (15%) originate from the authors' own research programme; the remaining 29 report independent findings from more than 25 separate research groups of different global origin (Asia, the Middle East, North Africa, sub-Saharan Africa, Europe and North America). The conclusions and practical recommendations presented in this review are therefore drawn from, and should be interpreted as reflecting, this internationally distributed independent evidence base rather than from a single research group or geographical setting.

Studies were not retained when they did not address poultry heat stress or the nutritional interventions covered by this review, when the report did not provide sufficient information to interpret the intervention and outcome, or when it was duplicative of another report.

The literature was selected to support a narrative synthesis of the major internationally tested nutritional approaches discussed in this review rather than to provide a comprehensive enumeration of all publications on heat stress in poultry. Consequently, the evidence presented reflects the selected body of studies considered most relevant to the objectives of the review, and the conclusions should be interpreted in light of this selective literature coverage.

Physiological and economic basis of heat stress in broilers

When core body temperature rises, birds first attempt behavioural thermoregulation, reducing feed intake, increasing water intake, spreading their wings, reducing activity and, above approximately 32-35°C, panting to increase evaporative heat loss from the respiratory tract [1]. Because panting excretes CO_2 faster than it is produced metabolically, it disrupts the blood bicarbonate buffer system: carbonic acid and hydrogen ion concentrations fall while bicarbonate rises, raising blood pH towards alkalosis. To compensate, the kidney excretes more bicarbonate and retains hydrogen ions, which in turn produces a secondary metabolic acidosis; this combined respiratory alkalosis-metabolic acidosis state is consistently associated with depressed growth and feed efficiency in the reviewed literature [1,2].

In parallel, the hypothalamic-pituitary-adrenal (HPA) axis is activated: the hypothalamus releases corticotrophin-releasing hormone, which stimulates pituitary adrenocorticotrophic hormone and, in turn, adrenal corticosterone, the principal avian glucocorticoid, often referred to as cortisol in the applied literature [3,5]. Elevated corticosterone is catabolic, mobilizing amino acids and glucose at the expense of lean tissue accretion, and is immunosuppressive, blunting both humoral and cell-mediated immune responses [3,5]. Circulating triiodothyronine (T3) typically falls under HS, reducing basal metabolic rate as a further compensatory response, while thyroxine (T4) responses are less consistent across studies [1].

HS also drives overproduction of reactive oxygen species (ROS), in part through compromised splanchnic blood flow and disrupted mitochondrial electron transport during panting, overwhelming the bird's endogenous antioxidant defences (superoxide dismutase, SOD; glutathione peroxidase, GP_x; and catalase) and elevating lipid peroxidation products such as malondialdehyde (MDA) [1]. The transcription factor Nrf2 is the principal upstream regulator of this antioxidant defence system, and its expression is frequently used as a biomarker of oxidative stress in nutritional trials [1]. Heat shock proteins, particularly HSP70 and HSP90, are induced as a complementary cytoprotective mechanism, acting as molecular chaperones that stabilize protein folding and limit apoptosis during thermal insult [1].

HS further compromises intestinal barrier integrity, reducing jejunal villus height, down-regulating tight-junction proteins such as occludin and claudin, and permitting paracellular translocation of endotoxin and pathogens; it also atrophies primary and secondary lymphoid tissue (bursa, thymus, spleen) and elevates the expression of pro-inflammatory cytokines including interleukin-6 (IL-6), interleukin-1β (IL-1β), interleukin-8 (IL-8) and tumour necrosis factor-α (TNF-α) [1,3]. Together, these endocrine, oxidative, acid-base and immune disturbances translate into reduced feed intake, poorer feed conversion, lower body weight and carcass yield, degraded meat and egg quality, and heightened morbidity from opportunistic pathogens such as *Salmonella* and *Escherichia coli* [1-3] (Figure 1).

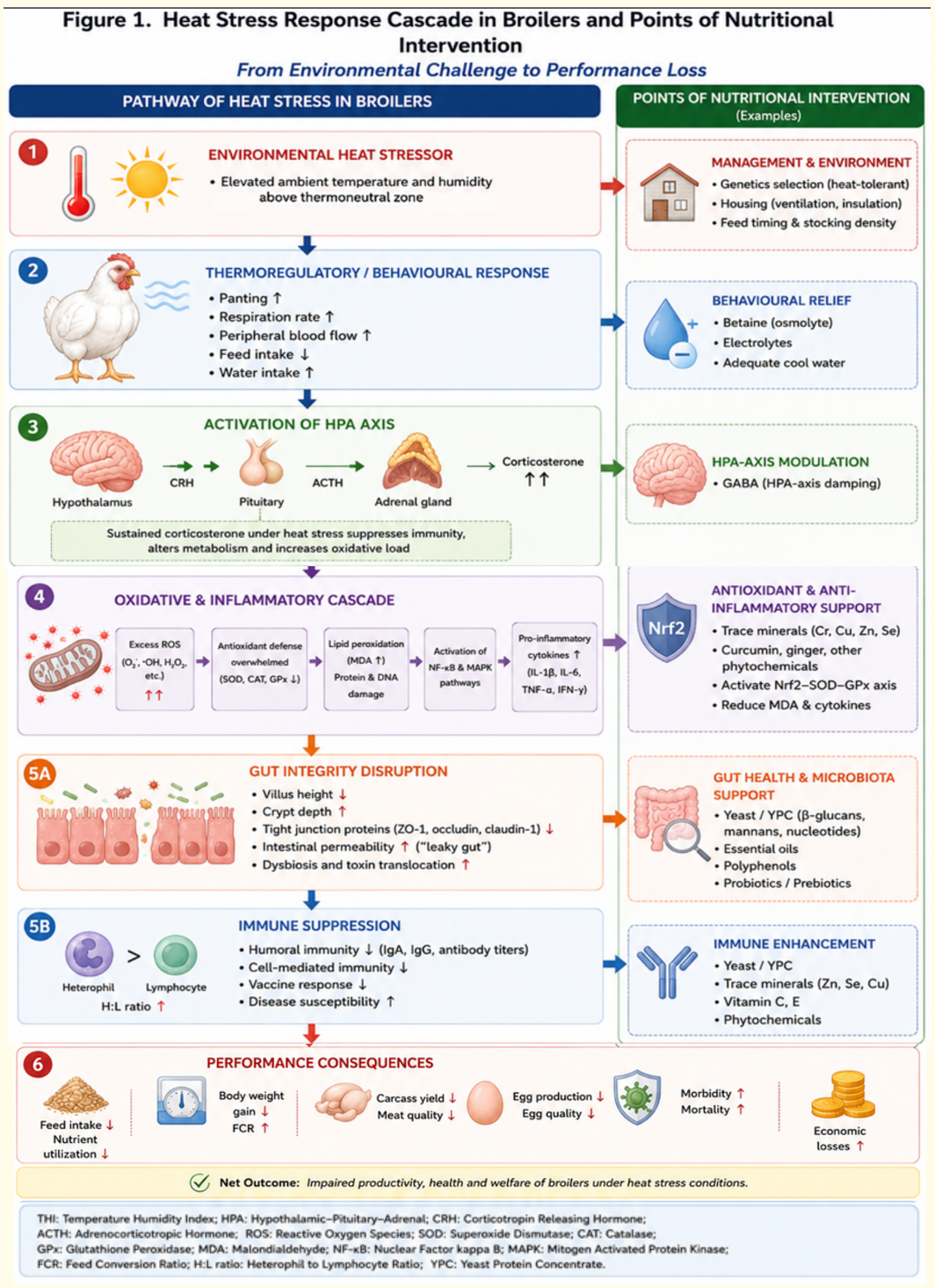

Figure 1: Heat-stress response cascade in broilers, from behavioural and neuroendocrine onset through to performance loss, with the corresponding nutritional intervention points annotated at right.

Before turning to the individual supplement classes, Table 1 and Table 2 summarize the biomarker targets and the practical, ready-to-apply dose ranges reported across the trace mineral, yeast, phytochemical and GABA literature reviewed in their respective sections. Read together, they are meant to be referred back to rather than restated: the narrative sections that follow concentrate on why the doses differ across trials, which chemical forms outperform others, and where the mechanistic reasoning is still incomplete.

Mechanistic Target	Trace Minerals (Cr, Cu, Zn, Se)	Yeast/YPC	Phytochemicals	GABA
Corticosterone/cortisol reduction	Yes in selected studies [5,6]	Yes [12]	Yes [14,17]	Yes [24,28]
Nrf2-SOD-GP_x antioxidant axis	Yes [9]	Yes [13]	Yes [14-18]	Yes [24,28]
Pro-inflammatory cytokine modulation	Yes (Cu; IL-6, TNF-α) [9]	Yes [12,13]	Yes (HSP70/NF-kB) [14,17,19]	Yes (IL-1β, IL-8, TNF-α) [24]
Gut microbiota shift	Yes (Cu) [9]	Yes (mannan/β-glucan) [12]	Not directly studied	Yes (caecal metagenome) [24]
Nutrient/mineral retention	Yes, central mechanism [5]	Indirect (trophic) [12]	Limited direct data	Not primary mechanism

Table 1: Reported mechanistic targets of dietary heat-stress mitigation strategies in broiler chickens.

Supplement/Source	Practical Dose Range	Principal Outcomes	Reference
Chromium (K-chromate, $CrCl_3$, Cr-yeast)	0.2 mg Cr/kg diet	Improved nutrient metabolizability and trace element retention; Cr-yeast most effective source	[5]
Chromium picolinate	0.5 mg Cr3+/kg diet (study-specific; 1 mg/kg no better)	Improved BW and FCR in a quadratic dose-response model; cortisol was reduced	[6]
Copper (yeast-protein chelate, Cu-P)	112.5 mg Cu/kg diet (study-specific plateau)	Linear BW/FCR gain to 112.5 mg/kg; increased SOD, GP_x; decreased *E. coli*, *Salmonella*	[9]
Yeast, YPC, YPC-pellets, Hydrolysed Yeast	1 g/kg diet, hydrolysed yeast as 400, 800, or 1,200 mg/kg	Improved BW, FCR, Newcastle disease antibody titre; reduced *Salmonella*/*E. coli* shedding	[12-13]
Zinc methionine + selenium-yeast	~40-80 mg Zn + 0.15-0.3 mg Se/kg diet	Restored villus architecture; improved antioxidant and immune status under HS	[10,11]
Zinc-chromium mixture + organic Se	100 mg Zn + 1.5 mg Cr + 0.6-0.9 mg Se/kg	Improved carcass traits and blood profile under HS	[8]
Betaine	1-2 g/kg diet	Improved feed intake, BW gain, breast muscle, FCR; lower H:L ratio	[19,23]
Vitamin C (ascorbic acid)	200-250 mg/kg diet (including broiler and layer trials)	Improved performance and antioxidant status in broilers; laying-hen studies reported effects on production and oxidative/stress measures	[19-21]
Curcumin/turmeric	50-200 mg/kg diet (better results in 20-100mg/kg groups), other studies report 0.4%, 0.5%, 0.8% of diet	Reported antioxidant and performance benefits under HS; effects vary with preparation and study design	[14,15]

Ginger root powder/oil	Variable Feed inclusion, 7.5 or 15 g/kg of root powder, 75 or 150 mg/kg of ginger essential oil	Improved feed digestion, growth and antioxidant status (raised TAC, TSOD; lowered MDA)	[18]
GABA	50-120 mg/kg diet	Improved performance and/or selected anti-oxidant, intestinal, endocrine or feed-intake outcomes in individual studies	[24-31]

Table 2: Summary of key trace mineral, yeast-derived, phytochemical and neuromodulatory trials in heat-stressed broilers, with dose ranges as reported.

Trace mineral supplementation strategies

Chromium

A dose-response trial using chromium picolinate (0, 0.5 and 1 mg Cr^{3+}/kg diet) in heat-stressed, feed-restricted Cobb broilers found that live weight, feed efficiency and protein-conversion efficiency all improved in a quadratic rather than linear fashion, with the 0.5 mg/kg dose consistently outperforming the higher 1 mg/kg dose [6]. Individual trials report different apparent optimum chromium doses. A 2022 systematic review and dose-response meta-analysis of 39 broiler trials provides a useful quantitative context for these differences [7]. The meta-analysis reported non-linear associations for several outcomes and identified an estimated supplemental level near 1.1 mg Cr/kg under the conditions represented in the pooled studies, while also indicating that responses varied with chromium source and heat-stress conditions [7]. The lower doses reported in the two trials above may therefore represent different positions on a dose-response curve, but this should be regarded as an interpretation rather than evidence that all chromium studies follow a single common nonlinear relationship. Differences in bird strain, heat-stress severity, basal diet, chromium source and experimental design may also contribute to the apparently different responses.

Complementary work combining a zinc-chromium mixture with organic selenium in heat-stressed broilers reported improved haematological, biochemical and productive outcomes in some treatment groups [8]. Taken together, the available studies support chromium as a potentially useful nutritional intervention under heat stress, but they do not establish one universal dose or a single mechanism of action. Organic or chelated chromium sources may perform differently from inorganic sources, and the dose-response pattern should be interpreted in relation to chromium form, bird genotype and the intensity and duration of the heat challenge rather than described as an established quadratic or saturating relationship (Figure 2).

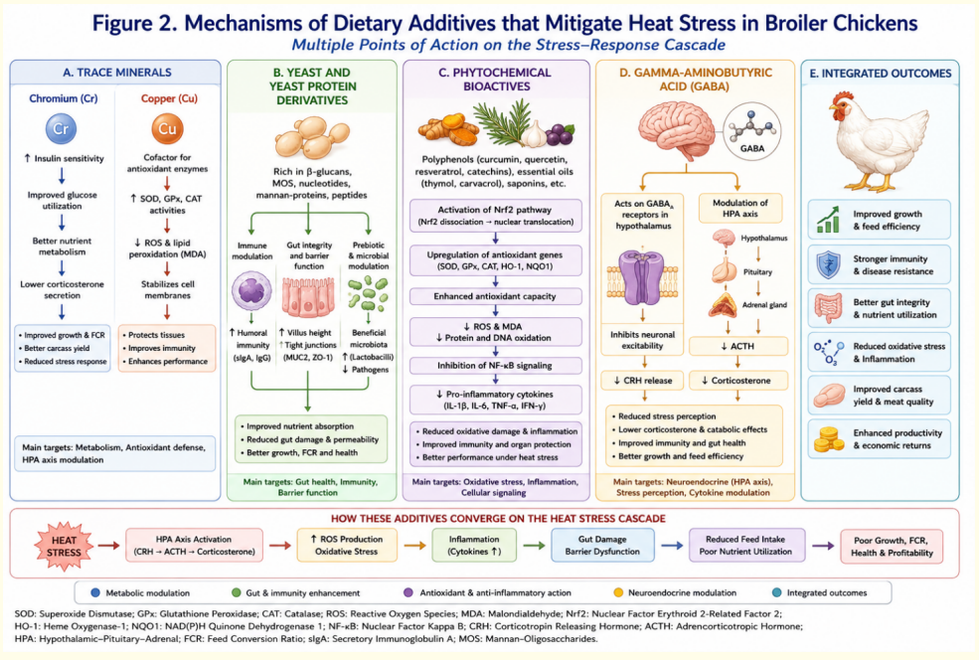

Figure 2: Principal reported mechanistic route by which each nutritional supplement class discussed in this review is proposed to mitigate heat stress in broilers.

Copper

Copper (Cu) is a cofactor of cytochrome oxidase, lysyl oxidase, ceruloplasmin and Cu-Zn superoxide dismutase, giving it a central role in cellular redox balance, and copper super-dosing, inclusion well above the approximately 8 mg/kg National Research Council recommendation, has been explored as a growth-promoting and, more recently, an HS-mitigating strategy [9]. In broiler chickens exposed to naturally fluctuating cyclic heat stress over 35 days, graded Cu supplementation as a yeast-protein chelate at 37.5 to 150 mg/kg diet produced a linear improvement in body weight, average daily gain and feed conversion ratio relative to a copper-deficient negative control, with body weight and feed conversion plateauing beyond 112.5 mg/kg [9].

The copper findings also illustrate why dose cannot be interpreted independently of chemical form. In the cited heat-stress study, increasing copper as a yeast-protein chelate improved several performance and antioxidant outcomes, while hepatic copper increased with dose and inflammatory-gene expression also changed [9] (Table 1). Notably, Cu super-dosing also reduced ileal *Escherichia coli* and *Salmonella* counts while increasing *Lactobacillus*, and up-regulated hepatic IL-6 and TNF-α gene expression -- an ambiguous signal that the authors interpret, on balance of the accompanying haematological evidence, as a ceruloplasmin-mediated, largely adaptive response rather than overt Cu toxicity, though a subtle toxic contribution beyond 112.5 mg/kg cannot be excluded [9]. These findings support the importance of source and dose, but they do not establish a universal toxicity threshold for different copper salts. More broadly, differences among chromium and copper studies may reflect chemical form, bioavailability, basal-diet composition and the severity of the heat challenge. Thus, source-specific evidence is preferable to a general rule that elemental dose alone determines efficacy or toxicity [7,9] (Figure 2).

Zinc and selenium

Zinc and selenium are frequently investigated alongside chromium and copper in HS-mitigation studies. In broilers reared under tropical summer conditions, organic forms of zinc, selenium and chromium improved selected performance and antioxidant responses in some treatment groups [10]. Selenium supplementation has also been associated with improved antioxidant and production outcomes, although the magnitude of response depends on source and experimental conditions [11]. The available evidence therefore supports consideration of mineral source and bioavailability, but the statement that organic or chelated minerals consistently outperform inorganic salts at the same elemental inclusion should be treated as a general tendency rather than a universal finding (Figure 2).

Yeast and yeast-derived organic carriers

Beyond their role as mineral chelation vehicles, yeast (*Saccharomyces cerevisiae*) and yeast-derived products have been investigated for independent effects on broiler performance and intestinal responses under stress. In a 35-day trial in Cobb broilers reared under sustained heat stress and challenged at 21 days with *Salmonella enteritidis*, dietary supplementation at 1 g/kg with whole yeast, yeast protein concentrate (YPC) or pelleted YPC improved selected performance and immune outcomes, while YPC products reduced *Salmonella* and *E. coli* shedding [12]. More recent work with hydrolyzed yeast in Ross 308 broilers exposed to cyclic heat stress reported effects on mortality and intestinal redox-related markers, although the response of growth performance was limited and dose-dependent [13]. These studies suggest that yeast-derived products may influence gut and stress responses, but differences in product composition and experimental design make it inappropriate to assume that all yeast preparations have equivalent effects. Proposed contributions of nucleotides present in crude protein fraction of yeast extracts, and β-glucans and mannans of yeast cell wall therefore remain plausible mechanisms rather than universal explanations for the observed responses. The extent to which each component contributes to the heat-stress response, however, depends on the composition of the product and was not directly established in all of the studies reviewed. The chromium-yeast and copper-yeast-protein preparations discussed elsewhere should therefore be interpreted as specific formulations rather than evidence that the yeast matrix necessarily acts synergistically with every trace mineral.

Phytochemical and plant-derived bioactive compounds

Polyphenols, curcumin and turmeric

Plant polyphenols, including the curcuminoids of turmeric (*Curcuma longa*), are among the most extensively investigated phytochemical HS-mitigators in broilers. Dietary curcumin at approximately 50-200 mg/kg feed simultaneously activates the Nrf2-mediated antioxidant pathway and improves final body weight

and egg quality in heat-stressed birds (Table 1) [14]. Turmeric powder, rich in curcumin, tetrahydrocurcuminoids, polyphenols and essential oils, has similarly been associated with improved antioxidant enzyme status, immune response and productive performance under HS [15]. Polyphenol-rich supplements derived from other woody or bark sources, notably willow (*Salix* spp.) bark, have likewise been shown to blunt oxidative stress markers and support performance under HS, with polyphenol content and extraction method strongly influencing efficacy [16] (Figure 2).

Essential Oils and allium/zingiber derivatives

Phytochemical extracts including essential oils extracted from oregano, thyme, rosemary, cinnamon, garlic and ginger contain phenolic, terpene and flavonoid constituents with antibacterial, anti-inflammatory and antioxidant activity, and a growing body of trials has evaluated their use specifically under heat-stress conditions in poultry [17,18]. Dietary ginger, whose active constituents include zingiberene, gingerols, shogaol and zingerone, enhances feed digestion, growth performance and antioxidant status in heat-stressed broilers (Table 1), an effect attributed in part to its polyphenol content and stimulation of digestive enzyme secretion [18]. Rosemary has been reported to pre-induce HSP70 expression ahead of a heat challenge, while ginger and thyme extracts consistently lower lipid peroxidation markers under HS, and garlic and other *Allium*-derived essential oils, rich in organosulfur compounds, show comparable benefits on gut health and performance. Mechanistically, these plant-derived compounds are thought to act principally by scavenging reactive oxygen species directly, by modulating HSP70 and NF-kB signalling, and by favourably shifting gut microbial populations, broadly indicating towards the Nrf2-centred antioxidant mechanism described for curcumin and the trace minerals above [14,17] (Figure 2).

Osmolytes and Vitamins: Betaine and Vitamin C

Betaine, a methyl donor and osmolyte, and ascorbic acid, a water-soluble antioxidant vitamin, are among the more widely studied nutritional approaches to heat stress [19-23]. Betaine (supplemented as 1-2g/kg diet) has been associated with improved performance and osmotic regulation in heat-stressed poultry, including broiler studies [19,22,23]. Vitamin C (supplemented as 200mg/kg diet) has also been evaluated in heat-stressed broilers [20], while the studies by Attia., *et al.* and Sahin., *et al.* cited here provide evidence from laying hens rather than broilers [19,21]. These laying-hen findings (suggesting 200-250mg of Vitamin C /kg diet with or without Vitamin E) are therefore relevant as supporting poultry evidence but should not be used on their own to establish broiler-specific dose recommendations. Neither betaine nor vitamin C is a trace mineral or, strictly, a phytochemical; they are included here to place the principal supplement classes in the context of the broader nutritional HS-mitigation literature (Figure 2).

Gamma-Aminobutyric Acid: A Neuromodulatory nutritional strategy

Gamma-aminobutyric acid (GABA), is the principal inhibitory neurotransmitter of the vertebrate central nervous system. Dietary GABA has been investigated in heat-stressed broilers for effects on performance, body temperature, antioxidant status, intestinal morphology and feed-intake regulation [24-31]. The biological rationale includes a possible influence on neuroendocrine and feeding pathways, but the extent to which dietary GABA directly attenuates the HPA-axis response in heat-stressed broilers remains incompletely established.

Earlier studies provide a broader basis for interpreting the more recent 2025 trial by the author group [24]. Dai., *et al.* reported effects of 100 mg/kg dietary GABA on performance and serum variables in broilers exposed to cyclic heat stress [25]. Chen., *et al.* reported protective effects of GABA on antioxidant function in the intestinal mucosa of heat-stressed chickens [26]. Chand., *et al.* evaluated 25-100 mg/kg GABA in Ross 308 broilers exposed to cyclic summer heat stress and reported improvements in selected performance, antioxidant and immune measures, particularly at 100 mg/kg [27]. Al Wakeel., *et al.* subsequently reported that GABA supplementation attenuated heat-stress-associated changes in body temperature and jejunal morphology including expression of selected nutrient transporters, HSP70 etc. in Ross 308 broilers [28]. El-Naggar., *et al.* found that 100 mg/kg GABA affected feed intake and related hypothalamic gene expression under heat stress, with responses differing between broiler strains [29]. Zhong., *et al.* reported improved growth performance and intestinal morphology with 100 mg/kg GABA in yellow-feathered broilers exposed to high temperature [30]. More recently, Park., *et al.* reported beneficial effects of 50 mg/kg GABA under several environmental stress conditions, including high temperature [31]. Against this background, the 2025 factorial study in Ven Cobb 430Y broilers

reported improved body weight and feed conversion at 60 or 120 mg/kg, together with changes in corticosterone, oxidative markers, inflammatory-gene expression and caecal microbiota [24]. These studies collectively support continued interest in GABA, but the differences in dose, strain, stress model and endpoints make direct comparison difficult.

The GABA literature therefore suggests several possible routes of action, including effects on feed-intake regulation, antioxidant status, intestinal morphology and stress-related endocrine responses. The 2025 study provides evidence for changes in corticosterone, Nrf2-related expression, inflammatory markers and caecal microbiota [24]. The response also appears to depend on the experimental environment and bird genotype. GABA may consequently be considered a promising nutritional intervention, while the relative contribution of neuroendocrine, antioxidant, intestinal and microbiome-related mechanisms remains to be resolved (Figure 2).

Complementary non-nutritional strategies: genetics, housing and feeding management

Nutritional supplementation does not operate in isolation, and the wider poultry heat-stress literature makes clear that its benefits are best realized alongside complementary genetic, environmental and feeding-management measures. Genetic strategies centre on major genes that reduce feather insulation or metabolic heat load: the naked-neck (Na) gene reduces neck plumage by 20% (Na/na) to 40% (Na/Na) and is associated with lower heterophil-to-lymphocyte ratio, lower plasma cholesterol and improved growth and egg quality under heat; the frizzle (F) gene curls the feather and increases heat radiation, improving egg production and quality under high temperature relative to normally feathered birds; and the dwarf (dw) gene reduces body size by 30-40%, though its heat-tolerance benefit in layers remains debated [1,2]. Housing and environmental measures include east-west oriented, naturally ventilated open-sided sheds with a roof slope of about 45 degrees, reflective or insulated roofing, adequate sidewall height with adjustable curtains, fans, fogging or cooling-pad systems, and stocking densities generally recommended below about 10-12 birds/m^2 in hot climates to preserve feed and water access and heat dissipation [2]. Feeding-management measures, applicable without any change in diet formulation, include shifting the bulk of feed provision to the cooler early morning and evening hours, coarser particle size or pelleted rather than mash feed (which lowers the metabolic heat cost of prehension and, in layers, improves feed efficiency and water intake), wet or rehydrated feeding to increase dry-matter intake and pre-digestion, dual (protein/energy) feeding regimes timed to the diurnal temperature cycle, and provision of cool (10-25°C), clean drinking water at all times, since water intake can rise several-fold under heat and each additional increment of water consumption meaningfully increases evaporative heat loss per breath [2]. Table 3 summarizes these complementary strategies alongside their principal mechanism and practical constraint, to allow a producer to weigh them against the nutritional strategies detailed in earlier sections.

Category	Representative Measures	Principal Mechanism	Key References
Genetic	Naked-neck (Na), frizzle (F), dwarf (dw) genes; thermotolerance-associated SNP selection	Reduced feather insulation/metabolic heat load; improved heat dissipation	[1,2]
Housing	East-west orientation, 45° roof slope, reflective/insulated roofing, adjustable curtains, fans, fogging/cooling pads, stocking density <=10-12 birds/m2	Lowers ambient heat load and radiant gain; preserves feed/water access	[2]
Feeding time	Bulk feeding in cooler early morning/evening hours; feed withdrawal during peak heat	Reduces dietary heat increment coinciding with peak ambient temperature	[2]
Feed form	Pelleted or coarse-particle feed rather than fine mash	Lowers energy expenditure of prehension; improves FCR and water intake	[2]
Wet/dual feeding	Rehydrated (1:1 feed:water) feeding; protein-rich diet in cool hours, energy-rich diet in warm hours	Faster gut passage and pre-digestion; lowers protein-associated heat increment	[2]
Water management	Cool (10-25°C), clean water available ad libitum; wider/deeper or elevated drinkers	Increases evaporative heat loss per breath; supports intake under HS	[2]

Table 3: Complementary genetic, housing and feeding-management strategies for mitigating heat stress in broilers, for use alongside the nutritional strategies reviewed in this study.

Emerging molecular and omics perspectives

The mechanisms described in earlier sections were established largely through targeted, single-endpoint measurements (serum cortisol, a named antioxidant enzyme, a specific cytokine). Since roughly 2023, the field has begun to move toward untargeted, multi-omics characterization of HS, and this shift is starting to reframe several of the mechanisms discussed above rather than simply adding detail to them.

Gut microbiota profiling is the most mature of these approaches. A 2025 systematic review of eighteen studies applying 16S rRNA and shotgun metagenomic sequencing to the heat-stressed chicken gut found consistent reductions in microbial diversity, depletion of short-chain-fatty-acid-producing genera, and disruption of microbial co-occurrence networks, though the direction and magnitude of specific taxonomic shifts varied considerably with HS duration, severity and gut segment sampled [32]. A complementary 2025 review frames these microbiota shifts within a bidirectional gut-brain-microbiome axis, in which HS-induced dysbiosis both results from and further aggravates HPA-axis activation and intestinal barrier loss [33], extending the corticosterone-oxidative-immune cascade in Figure 1 to include a microbial feedback loop that current interventions (yeast/YPC β-glucan mechanism in particular) already engage empirically, even where the underlying microbial pathway was not yet characterized at the time.

Metabolomic and transcriptomic approaches are adding a second layer of mechanistic resolution, particularly around lipid and iron metabolism. A 2024 study combining serum and muscle metabolomics in heat-stressed broilers supplemented with biogenic selenium nanoparticles linked the meat-quality benefit of selenium to suppression of the ferroptosis pathway, an iron-dependent, lipid-peroxidation-driven form of regulated cell death that mechanistically follows the lipid-peroxidation pathway (malondialdehyde) already used throughout the review as an established oxidative-stress marker [34]. This reframes malondialdehyde not simply as a proxy for "oxidative stress" in the abstract but as a readout of a specific, druggable death pathway, and suggests that antioxidant trace minerals and phytochemicals reviewed above may owe part of their protective effect to ferroptosis suppression specifically, a hypothesis that has not yet been tested directly for chromium, copper or curcumin in broilers.

Autophagy and mitochondrial dysfunction, both already implicated qualitatively in the ROS-generation step of Figure 1, are similarly being re-examined with quantitative omics tools, though broiler-specific evidence for these pathways under HS remains sparser and more fragmented than for the microbiome or ferroptosis. Taken together, the emerging omics literature does not yet overturn the mechanistic framework built in earlier sections; rather, it decomposes several of that framework's broad nodes (oxidative stress, gut barrier disruption) into more specific, testable sub-pathways, and future dose-response and combination trials of the supplements reviewed here would benefit from incorporating at least one such omics endpoint alongside the traditional performance and single-marker measurements.

Reported mechanisms and comparative synthesis

The nutritional supplements reviewed above affect several overlapping physiological endpoints, including corticosterone or other stress markers, antioxidant status, inflammatory responses, gut integrity and nutrient utilization. However, the evidence does not show that all supplement classes act through the same pathway. Tables 1 and 2 summarize reported biomarker responses and dose ranges, while the narrative sections emphasize differences among studies. The shared movement of markers such as corticosterone, MDA and antioxidant enzymes should be interpreted cautiously because these endpoints are not mechanistically specific. The omics evidence discussed in this review further illustrates that similar changes in a marker can arise through different biological pathways. The available literature therefore supports the possibility of complementary effects among interventions, but it does not establish that combination regimens are superior to single additives. Direct combination studies are still needed.

Practical considerations, limitations and future directions

Several practical implications follow from this synthesis. First, supplement source and chemical form may influence response as much as, or more than, nominal elemental dose in some trace-mineral studies [5,6,9]. This pattern should not be generalized to every mineral or preparation without direct comparison. Second, housing condition, sex and genetic strain can modify the magnitude of a response, as illustrated by the GABA literatures [24-31]. Dose recommendations obtained under one experimental setting should therefore not be transferred to another without validation. Third, nutritional supplementation should be treated as one component

of a broader HS-management strategy rather than a substitute for appropriate housing, ventilation, stocking density and water management [2]. Fourth, combinations of nutritional interventions are a reasonable area for investigation because different additives may influence different physiological endpoints; however, most combinations discussed here have not been tested directly, so they should not be presented as established practical recommendations.

For producers and nutritionists, the evidence supports a layered approach rather than a single universal supplement programme: (i) maintain appropriate housing, stocking density, feed timing and water management as the foundation of HS control (Table 3); (ii) consider nutritional interventions for which the relevant bird type, dose, source and heat-stress model have been adequately studied; and (iii) treat phytochemical and GABA supplementation as options requiring attention to product composition and experimental context. The doses in Table 2 are reported values from individual studies and should not be interpreted as universal commercial recommendations. In particular, the 0.5 mg Cr/kg and 112.5 mg Cu/kg values represent outcomes from specific experimental studies rather than recommended inclusion rates for all broiler diets. This distinction is important when translating experimental evidence to farm practice.

Further work is also needed on: (i) head-to-head economic evaluation of these strategies at commercial farm scale, particularly in the tropical, open-house conditions of South Asia and sub-Saharan Africa where the burden of HS is greatest and where much of the work reviewed here was conducted [2]; (ii) standardization of phytochemical preparations, whose active-constituent concentration can vary considerably with plant source, extraction method and batch; (iii) longer-term and multi-generational safety evaluation of copper and other trace mineral super-dosing regimens, given the subtle pro-inflammatory gene expression signal noted even in the absence of overt toxicity [9]; and (iv) mechanistic studies directly linking the gut microbiome shifts observed with yeast, copper and GABA supplementation to the systemic antioxidant and immune benefits they appear to share, an area currently supported mainly by correlative rather than causal evidence [12,9,24].

Conclusion

Heat stress remains an important challenge for broiler production, and nutritional interventions may help reduce some of its physiological and performance consequences. The evidence reviewed here supports beneficial responses to several dietary approaches, including selected organic trace-mineral sources, yeast-derived products, plant-derived bioactives and GABA, but the strength of evidence varies among compounds and experimental conditions. The available studies do not demonstrate a single common mechanism or a universally optimal dose. Differences among studies conducted in different geographical and production settings may reflect variation in genotype, bird age, basal diet, supplementation level, housing and management, and the severity and duration of heat exposure; therefore, responses should be interpreted within the context of the experimental conditions under which they were obtained. Nutritional interventions are therefore best considered as complementary to appropriate housing, water supply, feeding management and genetic strategies. Future work should place greater emphasis on independent replication, source-specific dose-response studies, direct comparison of interventions and validation under commercial conditions.

Future research should extend dose-response analyses beyond chromium where sufficient comparable studies are available, while accounting explicitly for mineral source, phytochemical composition, bird genotype and HS severity. Omics approaches may help resolve mechanisms that cannot be distinguished using conventional performance and biomarker measurements alone, but their value will depend on linking molecular changes to reproducible physiological outcomes. Precision livestock technologies also offer a potential route toward more responsive nutritional management. AI-assisted adjustment of supplementation according to environmental and performance data remains a future research concept rather than an established feeding practice; any such system would first require standardized measurements, independent validation and sufficiently large datasets from commercial conditions. These priorities are consistent with the broader need for better evidence on dose, source, economic return and long-term safety.

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