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Review article

Phytogenic interventions at the heat stress-bacterial infection interface in chickens: a critical One Health review and research agenda for antimicrobials

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Abstract

Heat stress affects chickens by reducing feed intake, disrupting redox balance, weakening immune function, and damaging intestinal barriers. These effects increase the risk of enteric infections, complicate treatment decisions, and encourage empirical antimicrobial use. This review examines phytogenic solutions at the intersection of heat stress and infection, assessing whether their advantages support antimicrobial stewardship and One Health goals. Evidence is categorized as direct, proximal, or supportive, with quantitative comparisons extracted when study designs permit. Currently, the strongest evidence supports antioxidant, anti-inflammatory, barrier-protective, and pathogen-reducing effects. Evidence is considerably weaker for reduced antimicrobial use, lower abundance of resistance genes, reduced food-chain contamination, or limited environmental dissemination. Fermented *Allium* preparations are presented as an illustrative case from hot-climate production systems, not as proof of One Health benefit. We propose a staged evaluation framework: phytogenics should (i) demonstrate animal health and performance benefits; (ii) demonstrate reduced antimicrobial use under defined treatment protocols; (iii) quantify effects on resistance, food safety, and environmental burden; (iv) confirm these effects at commercial scale using standardized outcome reporting. Phytogenics are best positioned as preventive, adjunctive tools alongside climate management, diagnostics, vaccination, biosecurity, veterinary care, and necessary antimicrobial treatment. The objective is not to eliminate antibiotics but to remove unnecessary treatment without compromising bird welfare or clinical outcomes.

Keywords: Antimicrobial resistance, Bacterial infection, Heat stress, Phytogenic feed additives, Poultry, One Health

Introduction

The One Health framework recognizes the interconnection among human, animal, plant, and environmental health. The “Quadripartite One Health Joint Plan of Action” places zoonoses, food safety, antimicrobial resistance (AMR), and environmental integration within the same operational agenda [1]. This framing aligns with the World Health Organization Global Action Plan on AMR, which emphasizes infection prevention, surveillance, and optimized antimicrobial use, as well as with Codex foodborne-AMR guidance for the food chain [2, 3]. In poultry, these links are concrete: flocks can carry zoonotic bacteria; meat and eggs connect farms to consumers; workers and service providers connect farms; and litter, dust, wastewater, insects, and wildlife can disseminate microorganisms and resistance determinants.

Increasingly hot and humid climates also affect poultry production. Climate change and episodic heat loads can alter host physiology, pathogen ecology, water consumption, litter conditions, and environmental control system performance. Antimicrobials remain important for treating bacterial disease, but repeated or poorly targeted exposure can select for resistant populations [4, 5]. The stewardship objective is therefore to prevent avoidable disease and improve treatment precision, not to withhold clinically indicated therapy. This objective cannot be achieved by simply replacing one growth-promoting input with another. Phytogenic products are relevant because chemically complex plant preparations can exert antimicrobial, antioxidant, anti-inflammatory, barrier-protective, and microbiome-modulating effects. Fermentation may introduce organic acids, microbial metabolites, and viable or non-viable microbial fractions. These properties suggest plausible preventive pathways, but they do not, by themselves, establish reduced antimicrobial demand or reduced AMR.

Recent reviews have already summarized phytogenic feed additives, their mechanisms, and their nutritional or production effects in poultry and other non-ruminants [6, 7, 8, 9, 10]. The distinct gap addressed here is narrower and more demanding: the dual-stress chain linking heat exposure to barrier dysfunction, bacterial infection, antimicrobial use, and downstream One Health AMR outcomes. The review therefore evaluates herbs, essential oils, defined phytochemicals, and fermented *Allium* products against a causal sequence spanning thermal exposure, host or pathogen response, treatment demand, resistance, food safety, environmental dissemination, and farm feasibility.

The article is intentionally framed as a critical narrative review and research agenda, because no current evidence base establishes this entire chain as a validated One Health intervention strategy.

Review approach and evidentiary boundaries

A structured search was conducted for peer-reviewed studies and authoritative reports available through July 2026. The search terms included chicken, broiler, layer, heat stress, temperature, humidity, temperature-humidity index, *Salmonella* (*S.*), *Escherichia coli (E. coli), Clostridium perfringens* (*C. perfringens*), *Campylobacter,* phytogenic, herb, essential oil, *Allium,* fermentation, antimicrobial resistance, resistome, mobile genetic element, food safety, litter, manure, and One Health. Recent research was prioritized, but foundational studies were kept if they provided insights into heat-infection mechanisms, phytogenic principles, or antimicrobial resistance stewardship.

Evidence was classified as direct when the same study included a defined thermal challenge, a defined bacterial challenge, and a phytogenic intervention. Evidence was classified as proximal when a phytogenic intervention was tested under either heat stress or a bacterial challenge and included relevant mechanistic or clinical endpoints such as barrier integrity, bacterial load, inflammation, or oxidative status. *In vitro* antibacterial tests and non-challenge performance or product-development studies were categorized as supportive. This hierarchy prevents culture inhibition or growth improvement from being mistaken for evidence of reduced antimicrobial use or One Health benefit.

This review is not a meta-analysis. Factors such as botanical identity, active-constituent composition, formulation, challenge model, thermal exposure, sampling site, age, genotype, and outcome definitions varied too widely to allow reliable pooled estimates. To enhance the quantitative analysis without introducing misleading meta-analytic effects, we extracted directly reported within-study contrasts from selected challenge studies. No pooled effect size across studies was computed. The synthesis asks the same question of each study: does the intervention provide evidence along the sequence exposure → intervention → host or pathogen effect → measured antimicrobial sparing → downstream One Health outcome?

The heat stress-infection-antimicrobial demand nexus

Thermal load and systemic adaptation

Modern broilers have limited capacity to dissipate metabolic heat because they lack sweat glands and are insulated by feathers. When environmental heat and humidity exceed the capacity for sensible and evaporative heat loss, panting, peripheral vasodilation, reduced feed intake, and endocrine and acid-base disturbances develop. Acute and chronic heat stress reduce growth, feed efficiency, product quality, welfare, and immune competence [11, 12, 13]. The temperature-humidity index (THI) can support exposure classification, but it should not be treated as a universal biological threshold. In a recent broiler study, high THI was associated with respiratory rates above 200 breaths/min and rectal temperatures above 42 °C [14]. In commercial laying hens, panting-based thresholds were approximately THI 80 for alert and 83.3 for danger [15]. These values illustrate why species, genotype, age, acclimation, air speed, and housing must accompany any THI value.

Reduced splanchnic blood flow contributes to intestinal hypoxia and oxidative stress. Reactive oxygen species, corticosterone, inflammatory signaling, and altered nutrient supply can disrupt tight-junction organization and epithelial turnover. Heat stress is therefore linked to increased intestinal permeability, immune dysregulation, microbiome disruption, and impaired villus architecture [16]. These host changes provide a plausible biological link between thermal load and increased susceptibility to enteric infection.

Barrier dysfunction, dysbiosis, and bacterial invasion

The gastrointestinal barrier serves as both a physical interface and an ecological habitat. Heat and feed-intake disruption can alter microbial substrates, fermentation products, epithelial integrity, and colonization resistance. Feed withdrawal and heat exposure have been linked to changes in ileal morphology and microbiota, as well as increased susceptibility to *S. enteritidis* colonization [17]. When heat stress and *S. enteritidis* infection were combined, intestinal inflammation increased and growth declined, indicating an interaction rather than a simple additive effect of the two stressors [18].

Direct measurements of intestinal permeability, inflammatory markers, and *Salmonella* translocation support the conclusion that acute or chronic heat can weaken the intestinal barrier and increase systemic or tissue exposure [19]. A compromised barrier may also create ecological opportunities for pathogenic *E. coli* or *C. perfringens*. The gut microbiota contributes to colonization resistance and immune maturation and is itself shaped by antimicrobials, environment, diet, and age [20, 21].

The practical consequence is diagnostic ambiguity. Decreased intake, uneven growth, wet litter, mortality, and inflammatory lesions may reflect heat stress, equipment failure, enteric infection, respiratory disease, or multiple concurrent causes. When laboratory access is limited, this ambiguity can prompt empirical medication. Thus, heat mitigation, pathogen prevention, diagnostics, and antimicrobial stewardship should be integrated into a single system rather than maintained as independent programs.

Campylobacter as a One Health food-safety sentinel

Campylobacter jejuni deserves explicit treatment because its importance is primarily a food-safety rather than a flock-clinical problem. Broilers may carry high intestinal loads with little overt disease, yet contaminated poultry meat is a major route of human campylobacteriosis. Phytogenic strategies based on essential oils, including thymol, carvacrol, and cinnamaldehyde, have been investigated as pre- and post-harvest control measures [22]. For this pathogen, cecal burden, carcass contamination, antimicrobial susceptibility, and processing-stage persistence are more informative One Health endpoints than growth performance alone. Heat stress may still matter by modifying intestinal ecology and litter conditions, but direct evidence for phytogenic control under simultaneous heat and *Campylobacter* exposure remains sparse (Table 1; Figure 1).

Table 1: Mechanistic pathways linking heat stress to bacterial disease and antimicrobial demand.

Heat-associated change	Microbiological or host consequence	Clinical/farm manifestation	One Health implication	Candidate phytogenic target
Reduced feed intake and altered digestion	Changed substrates and fermentation; reduced nutrient availability	Poor growth, uneven flocks, greater disease susceptibility	Lower efficiency and increased treatment pressure	Digestive stimulation; microbiome support
Splanchnic hypoperfusion and oxidative stress	Epithelial injury and impaired tight junctions	Enteritis, endotoxemia, bacterial translocation	Higher carcass contamination and pathogen shedding	Polyphenol antioxidant and Nrf2-associated responses
Corticosterone and immune dysregulation	Reduced innate and mucosal defense	Delayed clearance and more severe infection	Longer infectious period and greater antimicrobial exposure	Anti-inflammatory and immunomodulatory compounds
Dysbiosis and loss of colonization resistance	Expansion of *E. coli*, *Salmonella,* or *C. perfringens* niches	Diarrhea, necrotic lesions, mortality	Pathogen amplification and dissemination	Essential oils, fermented botanicals, symbiotic effects
More water consumption and litter moisture	Environmental persistence and horizontal transmission	Wet litter, foot lesions, recurring flock challenge	Litter/manure resistome and environmental spread	Lower pathogen shedding; improved gut function
Diagnostic uncertainty during heat events	Empirical or repeated treatment	Inconsistent response and treatment failure	Selection of resistant bacteria and residues	Prevention plus veterinary decision thresholds
Campylobacter colonization under production stress	High cecal carriage with limited clinical signs	Food-chain contamination rather than overt flock disease	Human campylobacteriosis; antimicrobial-resistant foodborne isolates	Essential oils/ phytochemicals; cecal and carcass endpoints [22]

E. coli = Escherichia coli, C. perfringens = Clostridium perfringens

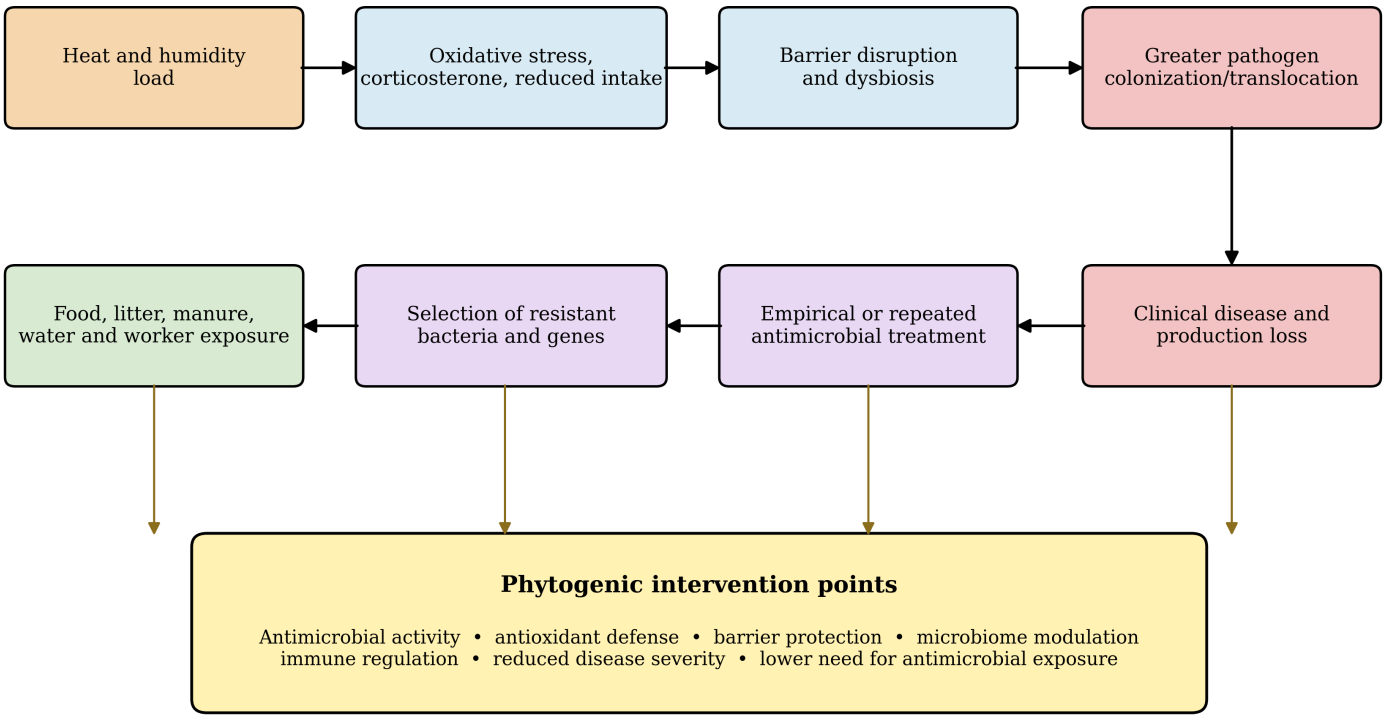

Figure 1: Heat stress-infection-antimicrobial-resistance continuum and specific phytogenic intervention points. Labels link major compound classes to plausible pathway nodes; the clinical-disease stage is explicitly connected to the intervention layer. Arrows indicate mechanistic hypotheses and prevention opportunities, not proof that every product affects every node.

Phytogenic interventions: mechanisms and formulation-dependent effects

Direct antimicrobial effects are necessary but insufficient

Phytogenic feed additives comprise whole herbs, powders, extracts, essential oils, oleoresins, purified phytochemicals, and fermented products. They contain compounds such as phenolics, flavonoids, terpenes, organosulfur compounds, alkaloids, tannins, and saponins. Their antibacterial mechanisms include disrupting membranes, causing leakage of cell contents, interfering with energy metabolism, chelating metals, inhibiting enzymes and efflux systems, and suppressing quorum sensing or virulence factors. Nonetheless, the effective concentrations observed *in vitro* may not be attainable in the intestine, as these compounds are absorbed, metabolized, bound to feed, or diluted [23].

Product chemistry is a key source of variability. Factors such as cultivar, growing conditions, plant part, harvest timing, drying temperature, extraction solvent, storage, and encapsulation can significantly alter the active profile. Reviews of phytogenics and essential oils consistently highlight antimicrobial, antioxidant, digestive, and immune benefits, yet these effects are highly context-dependent [6, 7, 8, 9, 10, 24, 25]. Therefore, to ensure reproducible claims, it is essential to verify botanical identity, conduct batch-specific marker analysis, provide formulation details, and justify dosages, rather than assuming products with the same plant name are equivalent.

Antioxidant and barrier-protective actions under heat stress

Heat stress creates a context in which antioxidant and anti-inflammatory effects are as crucial as directly killing bacteria. Polyphenols help by scavenging radicals, boosting natural antioxidant defenses, and regulating responses mediated by nuclear factor kappa B (NF-κB), nuclear factor erythroid 2-related factor 2 (Nrf2), mitogen-activated protein kinase (MAPK), and heat-shock proteins. Polyphenol-rich supplements administered to heat-stressed broilers improve redox balance, gut integrity, and overall performance, though results vary by product and dosage [26].

Controlled studies provide proximal evidence. A standardized blend of plant-derived isoquinoline alkaloids altered intestinal barrier function and inflammatory responses in heat-stressed broilers, though it did not fully restore growth across all periods [27]. Chlorogenic acid improved antioxidant, inflammatory, heat-shock, and tight-junction indicators under chronic heat stress, with greater effects at the higher tested dose [28]. A multi-herb Chinese veterinary formulation similarly reduced Toll-like receptor 4 (TLR4)- related inflammatory signaling and increased tight-junction gene expression in heat-stressed broilers [29]. These studies support biological plausibility but did not simultaneously measure bacterial challenge, antimicrobial treatment, and AMR outcomes.

A phytogenic product intended to address heat-associated bacterial disease should therefore be evaluated on multiple levels: preservation of feed and water intake; reduction of oxidative injury; maintenance of villus and tight-junction function; effects on the microbiome; reduction of pathogen burden or lesions; and ultimately, reduced frequency or duration of antimicrobial treatment. Improvement in growth alone does not establish antimicrobial-sparing activity.

Evidence from bacterial challenge models

Challenge models can demonstrate whether a product alters disease severity under controlled exposure conditions. Earlier *S. typhimurium* studies showed that phytogenic or essential oil treatments could influence performance and antioxidant responses under infection pressure [30]. More recent studies provide stronger contemporary comparators (Table 2): a water-soluble phytogenic blend reduced cecal *S. enteritidis* at 5- and 11-days post-infection in one trial [31], and oregano essential oil reduced *S. enteritidis* prevalence from 49% to 34% and cecal load by approximately 1.15 $\log_{10}$ CFU/g in market-age broilers [32]. These findings strengthen the challenge-model evidence base, but neither study combined bacterial challenge with concurrent heat stress and measured antimicrobial-use or resistome outcomes.

Necrotic enteritis is another relevant model because *C. perfringens* disease often emerges from interactions among coccidial damage, diet, microbiota, and environmental stress. A 2026 phytogenic-blend study reported improved intestinal morphology, liveability, inflammatory biomarkers, and performance in challenged broilers [33]. Its absence of a non-challenged phytogenic group nevertheless illustrates a recurring design limitation: without a complete factorial structure, general growth effects cannot be cleanly separated from disease-specific protection.

Fermented *Allium* products as a tropical case study

Tropical and subtropical poultry systems can leverage locally available Allium species because of their flavonoid and organosulfur chemistry and compatibility with lactic fermentation. Fermentation may enhance the extractability of selected compounds, generate organic acids, and yield viable or inactivated microbial fractions. The resulting intervention should therefore be classified as a strain-substrate-process product (Figure 2) rather than simply as an herb.

Earlier work in central Vietnam reported antibacterial activity of chive and ginger extracts against chicken-

derived *E. coli* and *Salmonella* isolates [34]. Subsequent feeding studies using chive essential oil or ethanol extracts examined performance and health indicators [35, 36]. These studies established local feasibility but provided limited standardization of active markers and did not measure reductions in antimicrobial use.

A subsequent line of work used chicken-derived *Lactobacillus plantarum* (*L. plantarum*) for purple-onion fermentation and reported antibacterial activity against *Salmonella*, product safety, and transient intestinal persistence [37]. Fermented chive prepared with *L. plantarum* was then tested in *E. coli*-challenged broilers, showing improvements in survival, mucosal immunity, barrier-related endpoints, microbiota, and performance [38]. Fermented onion and chive extracts were also tested against multidrug-resistant toxin-associated *E. coli* and *Salmonella* isolates [39].

Two more recent studies address the review question more directly. A purple onion-*L. plantarum* synbiotic delivered in drinking water improved feed conversion, intestinal morphology, microbial profiles, antioxidant status, and resilience in yellow-feather chickens exposed to natural heat stress [40]. Separately, *L. plantarum*-fermented shallot delivered in drinking water reduced *S. pullorum* and *E. coli* burdens and improved survival, inflammatory, and barrier-related outcomes in challenged broilers [41]. Together, these studies (Table 3) provide complementary evidence on heat stress and infection but do not directly test the same product under simultaneous heat stress and bacterial challenge with measured antimicrobial use and AMR outcomes.

These case studies also highlight naming and reproducibility issues. Shallot, chive, garlic chive, and local onion are common names that may refer to different taxa or cultivars. Studies should report the accepted botanical name, plant part, voucher or traceability record, and genetic confirmation when taxonomy is uncertain. Microbial nomenclature should follow current classification; in the main text, *L. plantarum* is used consistently. Batch-level measurements should include viable counts, pH, key organic acids, thiosulfinate or sulfur-marker profiles, flavonoids or other justified markers, water activity where relevant, and storage stability.

Table 2: Representative phytogenic evidence relevant to heat stress, bacterial infection, and antimicrobial-sparing strategies in chickens.

Intervention/model	Exposure	Main outcomes	Relevance	Critical limitation
Polyphenol-rich botanicals [26]	Heat stress; review evidence	Redox, inflammatory and barrier responses	Mechanistic support for thermal resilience	Large heterogeneity among products
Isoquinoline alkaloids [27]	Controlled heat stress	Barrier and inflammatory modulation	Standardized active compounds	Incomplete recovery of performance
Chlorogenic acid [28]	Chronic heat stress	Antioxidant enzymes, cytokines, tight junctions	Dose-responsive proximal evidence	No bacterial challenge or antimicrobial-use endpoint
Multi-herb BLZJS [29]	Chronic cyclic heat stress	Reduced TLR4 signaling; improved barrier genes	Multi-target phytotherapy	Complex formula limits attribution
Phytogenic/essential-oil products [30]	*S. typhimurium* challenge	Performance and antioxidant responses	Direct bacterial-challenge evidence	No concurrent heat stress
Chive/ginger extracts [34, 35, 36]	*In vitro* isolates and broiler feeding	Antibacterial activity and health/productivity indicators	Locally available tropical herbs	Limited chemical standardization
Fermented purple onion [37]	*Salmonella* screening and chicken safety	Selected *L. plantarum* strain; antibacterial activity	Foundation for synbiotic product	Transient colonization and early-stage validation
Fermented chive [38]	APEC *E. coli* challenge	Survival, immunity, barrier, morphology, microbiota	Strong disease-challenge evidence	Single experimental setting
Fermented onion/chive [39]	MDR toxin-carrying bacteria	*In vitro* inhibition and pathogenicity assessment	Targets resistant poultry isolates	Commercial efficacy not established
Purple onion synbiotic [40]	Natural heat stress	Antioxidant status, morphology, lower *E. coli*	Direct heat-stress evidence	No defined bacterial challenge
Fermented shallot [41]	*S. pullorum* challenge	Lower pathogen load, inflammation; higher survival	Water-delivered disease control	No concurrent heat challenge
Thymol-containing blend [33]	Necrotic enteritis challenge	Liveability, gut damage, biomarkers, performance	Recent bacterial-disease evidence	No non-challenged phytogenic group
Essential oils against *Campylobacter* [22]	Pre-/post-harvest poultry evidence	Potential reduction in *Campylobacter* concentrations	Direct food-safety relevance	Effects vary by compound, dose and application stage
Water-soluble phytogenic blend [31]	*Salmonella* challenge	Lower cecal *S. Enteritidis* at 5 and 11 dpi in one trial	Recent bacterial-challenge evidence	No concurrent heat stress; mixed time-point effects
Oregano essential oil [32]	*S. Enteritidis* challenge to market age	Prevalence 49%→34%; cecal load reduced ~1.15 log10 CFU/g	Quantified pre-harvest pathogen reduction	No antimicrobial-use or resistome endpoint

APEC = avian pathogenic *Escherichia coli*, MDR = multidrug-resistant, CFU = colony-forming unit, *L. plantarum* = *Lactobacillus plantarum*, S. *pullorum* = *Salmonella pullorum,* TLR4 = Toll-like receptor 4, *E. coli* = *Escherichia coli*, *S. typhimurium* = *Salmonella typhimurium*, dpi = days post-infection.

Table 3: Selected within-study quantitative signals from phytogenic challenge studies. Values are reported as contrasts from individual studies and are not pooled effect estimates.

Study/intervention	Model	Comparator	Reported quantitative signal	Interpretation
Fermented chive, 3% [38]	*E. coli* challenge	Positive challenged control	Body-weight gain +11.2%; challenged-control *E. coli* 7.1 $\log_{10}$ CFU/g vs 5.5-6.2 $\log_{10}$ CFU/g across treated groups	Meaningful disease-model signal; exact causal contribution of fermentation components unresolved
Fermented shallot [41]	*S. pullorum* challenge	Positive challenged control	2-3% inclusion restored survival to 90.0-91.8%, comparable with unchallenged/antibiotic groups	Clinically relevant proximal evidence; antimicrobial-use reduction not measured
Water-soluble phytogenic blend [31]	*S. Enteritidis* challenge	Infected control	Significantly lower cecal burden at 5 and 11 dpi; no consistent difference at all time points	Illustrates time-dependent effect and need for longitudinal sampling
Oregano essential oil [32]	*S. Enteritidis* challenge	Infected control	Prevalence 49%→34% (−15 percentage points); 2.7×10^3→1.9×10^2 CFU/g (~1.15 $\log_{10}$ reduction)	Strong recent quantitative pathogen endpoint; no heat-stress or AMR endpoint

E. coli = *Escherichia coli*, *S. pullorum* = *Salmonella pullorum*, S. *Enteritidis* = *Salmonella Enteritidis,* CFU = colony forming unit, dpi = days post infection

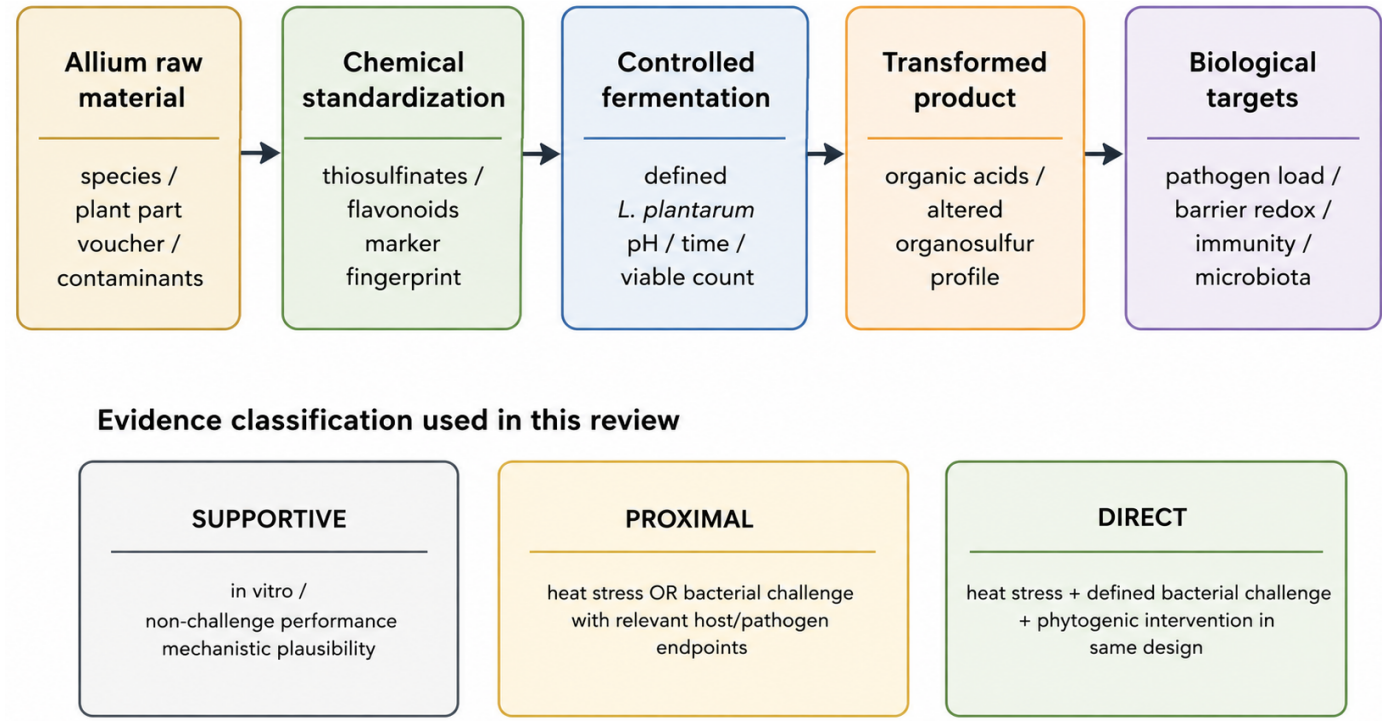

Figure 2: Fermented *Allium* development pipeline and evidence classification used in this review. Product identity progresses from botanical and chemical standardization through controlled fermentation to defined biological targets; evidence is classified as supportive, proximal, or direct according to study design. Antimicrobial-sparing and One Health claims require additional measured treatment and downstream AMR outcomes.

One Health pathways for reducing antimicrobial demand

Animal health and antimicrobial stewardship

The primary stewardship endpoint is not the absence of antimicrobial use; it is appropriate use. This principle aligns with WHO and Codex AMR frameworks, which link infection prevention with responsible antimicrobial use and food-chain risk management [2, 3]. A phytogenic program should reduce preventable disease and unnecessary treatment while preserving rapid access to effective therapy for birds with confirmed or strongly suspected bacterial disease. Veterinary oversight should guide treatment decisions and incorporate flock history, clinical examination, necropsy, bacteriology, susceptibility testing, and predefined rescue thresholds [42].

Antimicrobial-sparing claims require direct measurement. Trials should report the proportion of pens or flocks treated, the indication, antimicrobial class, dose, duration, treatment incidence or defined daily dose metrics, retreatment, mortality, and reasons for rescue therapy. A phytogenic group should not be considered successful if lower antimicrobial use is achieved at the cost of greater suffering, delayed treatment, or mortality. Conversely, improved growth without reduced disease or treatment does not demonstrate stewardship.

Food safety and resistant bacteria

Reducing intestinal pathogen burden may lower contamination entering slaughter, but farm-level reductions do not necessarily persist through transport and processing. *Campylobacter* is particularly important because birds can carry high cecal loads with limited clinical disease, and contaminated carcasses remain a major human health pathway. Essential oils such as thymol, carvacrol, and cinnamaldehyde have been evaluated as pre- and post-harvest interventions against *Campylobacter* [22]. Consequently, One Health trials should align endpoints with pathogen biology: cecal and carcass *Campylobacter* loads, *Salmonella* strain relatedness across farm and processing, antimicrobial susceptibility, residues, and food-chain exposure.

Reducing antimicrobial exposure can lessen selection pressure, but phytogenics may also impose ecological selection on microbial communities. Subinhibitory plant compounds could favor tolerant populations, and resistance determinants can be linked to mobile genetic elements (MGEs) that carry traits selected by unrelated pressures. Chicken cloaca and litter harbor dynamic resistomes even without antimicrobial treatment, and antimicrobial exposure can shift specific resistance genes and MGEs [43]. Poultry litter used as fertilizer can also contribute antimicrobial-resistance genes (ARGs) and integrase-associated resistance signals to agricultural soils [44]. Therefore, studies should compare resistant organisms, ARG abundance, MGE context, and environmental dissemination rather than assuming that a natural product is resistance-neutral.

Environment and occupational interfaces

Poultry litter and wastewater contain intestinal bacteria, ARGs, residual antimicrobials, plant metabolites, nutrients, and MGEs. Reuse of litter or manure can extend exposure beyond the poultry house. A One Health trial should therefore measure litter moisture and pathogen persistence, along with selected ARGs or, where justified, resistome and mobilome profiles, before and after manure treatment or storage. Worker exposure through dust, direct contact, equipment, and domestic transmission should be addressed through risk-focused sampling and hygiene measures in longitudinal studies [43, 44].

Phytogenic production also has an environmental footprint. Land, water, solvents, drying or extraction energy, packaging, transport, and product losses should be considered. Locally sourced plants or by-products may improve circularity, but pesticide residues, mycotoxins, heavy metals, and pathogenic microorganisms can create new hazards. Microbiome responses are context-dependent [45, 46], and global antimicrobial-use projections reinforce the need to document actual exposure reduction rather than infer stewardship from performance alone [47, 48]. Cross-domain outcomes should be measured explicitly because AMR can move among animals, food, people, and the environment [43, 44, 49].

A gated One Health claim framework

To operationalize the One Health component, we propose a four-gate claim framework (Figure 3). Gate 1 is animal health validity: morbidity, mortality, welfare, and clinically relevant disease outcomes must be no worse than those of the appropriate comparator. Gate 2 is measured antimicrobial sparing: treatment incidence, treatment days, or dose must be lower under predefined veterinary rescue criteria. Gate 3 is downstream AMR or exposure benefit: resistant organisms, ARGs, residues, or pathogen contamination should decline in at least one food-chain, farm-environment, or occupational compartment. Gate 4 is implementation validity: the product must be standardized, safe, economically plausible, and reproducible across batches or farms. The highest claim justified is determined by the highest gate actually crossed, not by the number of mechanistic biomarkers improved.

The framework includes hard gates. A high composite score cannot compensate for missing animal-welfare safeguards, the absence of measured antimicrobial-use data when an antibiotic-sparing claim is made, or a lack of product safety. Likewise, lower pathogen abundance does not equal lower resistome risk. If the claim concerns AMR, isolate susceptibility, ARG abundance, or preferably ARG-host-MGE context should be measured at the ecological level relevant to the claim. This prevents promoting a mechanistic endpoint as a public-health outcome.

For translation, the framework should be applied prospectively. Investigators should define the intended claim before trial initiation, identify the minimum gate-specific endpoints, and prespecify treatment thresholds and analytical methods. Table 4 provides a simple 0-2 scoring rubric across five domains. The score is intended for study planning and transparent interpretation, not regulatory qualification; the hard-gate rules remain primary.

Product standardization, safety, and implementation in tropical systems

Tropical poultry systems range from fully climate-controlled integrated farms to open-sided houses with unreliable power, water, and diagnostic access. Delivery systems should be tailored to the production context. Feed inclusion enables continuous dosing but responds slowly to acute events and may expose phytochemicals to pelleting heat. Drinking-water delivery enables rapid flock-level dosing but requires validation of solubility, line hygiene, biofilm, palatability, and dose stability.

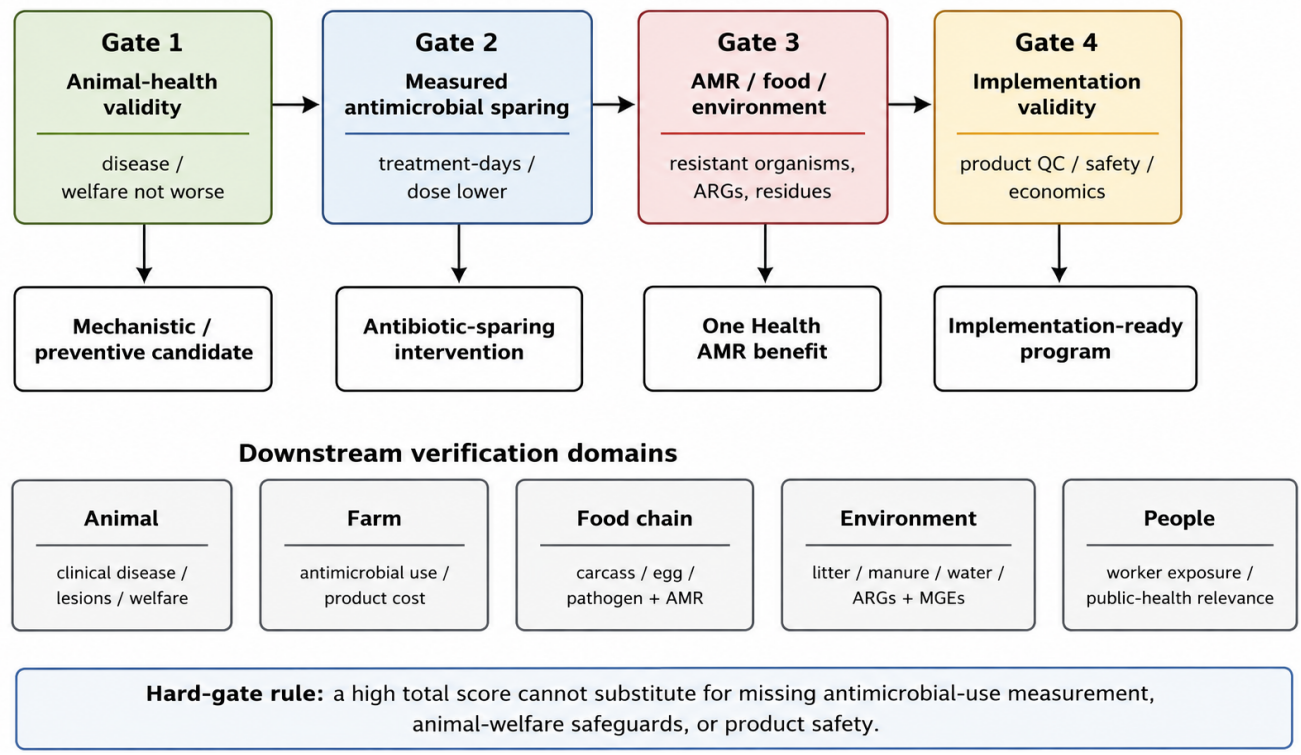

Figure 3: Gated One Health framework for phytogenic claims in poultry. Evidence progresses from animal-health validity to measured antimicrobial sparing, downstream AMR/food/environment benefit, and implementation validity. Hard-gate requirements prevent high mechanistic scores from substituting for welfare, antimicrobial-use, or safety evidence.

Encapsulation may be needed for volatile oils, whereas fermented liquids require controlled pH, microbial quality, and shelf-life specifications.

Safety assessment should extend beyond acute lethality. Organosulfur compounds, tannins, essential oils, and alkaloids can reduce feed intake, irritate the gut, or alter drug-metabolizing pathways at inappropriate exposure levels. Studies should assess clinically relevant hematology and biochemistry, target-organ histopathology when indicated, palatability, residue or withdrawal implications, and dose margins. Potential interactions with vaccines, coccidiostats, and antimicrobial agents should be evaluated separately rather than included in a single run-on checklist. Live microbial components also require strain-level assessment of virulence, acquired AMR, and manufacturing quality.

Economic sustainability depends more on disease prevention than on feed conversion alone. The assessment should consider raw-material variability, production, processing, quality control, spoilage, dosing facilities, labor, veterinary expenses, mortality, and carcass downgrading. Local formulations may benefit smallholder poultry systems, but decentralized production systems pose serious quality assurance problems. Shared laboratories, cooperatives, planned extension facilities, and controlled batch processes may be more beneficial than unregulated on-farm production.

Research priorities

The central evidence gap is the simultaneous experimental combination of thermal and bacterial stress while measuring antimicrobial use and One Health outcomes. At a minimum, factorial designs should separate thermoneutral from heat-stressed conditions and unchallenged from bacterially challenged conditions, with phytogenic and clinically relevant comparator groups. Heat exposure must be described by air temperature, relative humidity, air speed, duration, diurnal recovery, THI where used, and bird-level responses such as respiratory rate and body temperature. Age, genotype, acclimation, stocking density, and housing should also be reported because the same nominal THI can have different biological consequences [14, 15]. Bacterial strains should be characterized for identity, virulence, and antimicrobial susceptibility.

Independent biological replication is essential. Experimental units for feed and environmental interventions are typically pens or housing units, not individual birds or repeated technical measurements. Multi-farm validation should span relevant seasons, genotypes, pathogen pressures, diets, and management systems. Rescue-treatment criteria must be prespecified to protect welfare and enable valid comparisons of antimicrobial use. Table 5 summarizes the minimum evidence domains, while Table 4 provides a translational scoring rubric that complements the decision pathway in Figure 3.

Table 4: Translational readiness scoring rubric for phytogenic One Health studies (0-10 points, with hard-gate rules).

Domain	0 points	1 point	2 points
Product identity and safety	Poorly defined product; no safety characterization	Basic botanical/microbial identity and short-term safety	Batch-resolved chemistry/strain identity, contaminants, stability, dose safety
Dual-stress biological relevance	No heat or bacterial challenge	Heat stress or bacterial challenge with relevant endpoints	Factorial heat + bacterial challenge with clinically relevant endpoints
Antimicrobial-use measurement	Not measured	Rescue treatment described retrospectively	Prospective treatment incidence/dose/duration under predefined veterinary thresholds
AMR / food / environment evidence	No downstream endpoint	Phenotype, ARG, residue or contamination measured in one compartment	Longitudinal cross-compartment AMR/food/environment evidence with organism or gene context
External validity and feasibility	Single batch/facility; no economics	Independent replication or partial budget	Multi-batch/multi-farm validation plus safety, economics and implementation analysis

Interpretation: 0-3 points = mechanistic/supportive candidate; 4-6 = promising translational candidate; 7-8 = potentially antibiotic-sparing if Gate 2 is satisfied; 9-10 = One Health translation-ready only if Gates 1-4 are all satisfied. Scores cannot override the hard-gate requirements.

Table 5: Minimum evidence for translating phytogenic interventions into One Health antimicrobial-sparing practice.

Domain	Minimum measurements	Preferred design	Claim supported
Botanical/product identity	Accepted species name, plant part, voucher or traceability, active markers, contaminants, stability	Validated analytical methods and batch-to-batch specifications	Reproducible product
Heat exposure	Temperature, humidity, air speed, duration, recovery, bird responses	Controlled and field heat models	Thermal relevance
Bacterial disease	Strain, dose, route, virulence, susceptibility, pathogen load, lesions, mortality	Defined challenge plus commercial validation	Disease prevention or mitigation
Antimicrobial use	Indication, treatment incidence, dose, duration, rescue treatment, clinical outcome	Prospective blinded thresholds and veterinary oversight	Antimicrobial-sparing effect
Microbiome and AMR	Absolute counts, susceptibility, resistance genes, mobile elements, microbiome/metabolites	Longitudinal animal-litter-food sampling	Resistance and transmission effect
Food safety	Cecal/cloacal carriage, carcass contamination, residues, strain relatedness	Linked farm-processing study	Consumer-health relevance
Environment and workers	Litter, manure, water, dust, exposure practices	One Health cohort or sentinel surveillance	Cross-sector impact
Economics and feasibility	Full product cost, avoided loss, labor, infrastructure, sensitivity analysis	Multi-farm partial budget and adoption study	Practical scalability
Safety	Palatability, hematology, liver/kidney, histology, interactions, microbial safety	Dose-range and long-duration assessment	Responsible use

Conclusions

Heat stress can exacerbate the consequences of microbial exposure by weakening intestinal barriers, altering the microbiota, and impairing host defenses. These effects can increase disease severity, diagnostic uncertainty, and pressure to use antimicrobials. The pathway is biologically plausible, but the current literature does not establish a complete causal chain from phytogenic intervention to reduced antimicrobial use and, ultimately, reduced AMR across animal, food, human, and environmental compartments.

Phytogenic interventions can plausibly interrupt several points in this continuum. Evidence is strongest for antioxidant and barrier support under heat stress and for reduced disease severity or pathogen burden in selected challenge models. Fermented *Allium* products provide an informative tropical case study because heat-stress and infection benefits have been demonstrated in separate experiments. However, direct evidence that these products reduce clinically indicated antimicrobial use during concurrent heat and bacterial stress remains insufficient, and evidence for resistome or environmental benefit is still more limited.

Accordingly, phytogenics should be developed within an integrated prevention system that includes climate mitigation, safe water, vaccination, biosecurity, nutrition, diagnostics, veterinary management, and environmental stewardship. The gated framework proposed here requires animal-health validity before antimicrobial-sparing claims, measured reductions in antimicrobial use before

One Health AMR claims, and downstream evidence before asserting a public-health benefit. The goal is not antibiotic elimination at any cost; it is healthier flocks that require fewer avoidable treatments while preserving effective therapy when disease occurs.

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