

REVIEW ARTICLE

Phytogenic Strategies at the Cold-Humid Environment-Bacterial Disease Interface in Chickens: Translational Evidence and One Health Implications

Phan Vu Hai*

Faculty of Animal Sciences and Veterinary Medicine, University of Agriculture and Forestry, Hue University, Hue, Vietnam

Abstract

Background: Cold-humid poultry environments combine low effective temperature with direct moisture loading, such as a defined elevated-humidity exposure, condensation, or wet litter. Restricted ventilation and ammonia can accompany moisture retention as related co-exposures but are not direct humidity measures. This compound exposure can increase thermoregulatory and oxidative demand while weakening respiratory and intestinal barriers and altering bacterial ecology. The review asked whether phytogenic interventions can interrupt progression from environmental stress to bacterial disease and reduce antimicrobial exposure without compromising welfare.

Methods: A critical structured narrative review of peer-reviewed literature available through 12 September 2026 was undertaken. PubMed was the primary bibliographic database; targeted publisher/journal retrieval and backward/forward citation chaining supplemented record identification and source verification. Searches were iterative and last updated on 12 September 2026. Exact retrieval, deduplication, screening, and exclusion counts were not prospectively logged and therefore are not reconstructed retrospectively. For evidence classification, 'cold-humid' required low effective temperature plus at least one direct moisture indicator: relative humidity when explicitly used to define or contrast a humid exposure, condensation, wet/caked litter, quantified litter moisture, or water activity. Restricted ventilation and ammonia were treated as related co-exposures rather than direct humidity measures.

Results: No study provided direct evidence across cold-humid

*Corresponding author(s)

Phan Vu Hai, Faculty of Animal Sciences and Veterinary Medicine, University of Agriculture and Forestry, Hue University, Hue, Vietnam

Email: phanvuhai@hueuni.edu.vn

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exposure, defined bacterial disease, a characterized phytogetic intervention, and a prospectively measured antimicrobial-use endpoint. Existing *Salmonella* and *Escherichia coli* challenge studies under low temperature are informative but were classified as cold-only because no prespecified humid/moisture exposure was incorporated, even where humidity was monitored. Rutin and a commercial phytogetic blend provide proximal in vivo evidence for host responses during cold stress, whereas fermented *Allium* studies provide separate in vivo challenge, product-characterization, and in vitro antibacterial evidence outside cold-humid conditions. The fermented *Allium* evidence is also concentrated within one investigator network and should not be treated as independent replication.

Conclusions: Current evidence supports phytogetics as preventive or adjunctive candidates, not substitutes for clinically necessary treatment. No reviewed study demonstrated antimicrobial sparing or a downstream One Health benefit under a defined cold-humid bacterial-disease model. Product-specific, independently replicated cold-humid x infection x phytogetic trials with prespecified veterinary rescue criteria, standardized product characterization, and multi-farm winter validation are needed before stewardship or One Health claims can be made at those levels.

Introduction

Reducing unnecessary antimicrobial exposure in poultry is now a production, veterinary, and public-health objective. The difficulty is that antimicrobial use is rarely driven by one factor. It emerges from the interaction among flock immunity, pathogen pressure, housing, litter, ventilation, diagnostics, economics, and the threshold at which disease is treated. Winter and monsoon-transition conditions make this interaction especially visible. Birds may be cold at the same time that ventilation is reduced to conserve heat; moisture then accumulates from respiration, excreta, drinkers, and condensation, and the resulting wet litter and ammonia add another layer of biological stress [1-4].

The term cold-humid environment is used here as an operational production category rather than a rigid meteorological label. For this review, classification required low effective temperature at bird level plus at least one direct moisture indicator: relative humidity when the study explicitly defined or contrasted a humid exposure, condensation, quantified litter moisture or water activity, or directly documented wet/caked litter. Restricted ventilation, ammonia accumulation, drinker

leakage, and bioaerosol load were treated as related management or contaminant co-exposures that can accompany moisture retention, but they were not considered direct measures of humidity. Thus, low temperature combined only with reduced ventilation or elevated ammonia was not sufficient for a cold-humid classification. No universal relative-humidity cut-off was imposed because the biological meaning of humidity depends on temperature, air speed, age, floor and radiant temperature, feather cover, stocking density, and house conditions. Studies reporting low temperature without evidence of a defined direct moisture exposure were retained as cold-only or winter/cold-season evidence with incomplete moisture characterization.

Cold can alter host susceptibility through thermogenic demand, endocrine stress, oxidative imbalance, intestinal injury, and immune modulation [7-12]. The environmental arm is different. Wet litter creates spatially variable microbial niches; restricted ventilation increases ammonia and particulate exposure; and contaminated litter can act as a reservoir from which enteric pathogens circulate between birds and the house environment [1-4]. Older challenge studies remain informative

because they show that low temperature can increase *Salmonella* shedding or severity [5,6]. A combined cold-stress and *Escherichia coli* model also demonstrates that thermal stress can meaningfully alter production losses during bacterial challenge [9]. These studies establish temperature-infection interactions, but because humidity-related exposure was not adequately characterized, they are treated here as cold-only rather than direct cold-humid evidence.

Phytogenic products are attractive in this setting because they are biologically multi-target. Plant phenolics, flavonoids, terpenes, organosulfur compounds, and related metabolites can influence redox balance, inflammatory signaling, epithelial integrity, microbial membranes, adhesion, digestive function, and microbiota composition [21-23]. Fermentation can further modify plant chemistry while adding organic acids, microbial metabolites, or viable beneficial organisms. Yet the breadth of these effects creates a risk of over-interpretation. An inhibition zone is not disease control, a better feed conversion ratio is not antimicrobial sparing, and a lower cecal count is not automatically a One Health benefit.

This review therefore applies endpoint-matched reasoning. The central question is not whether phytochemicals can improve some aspect of chicken physiology; that evidence is already substantial. The question is whether a defined product can reduce bacterial disease and clinically indicated antimicrobial use under a cold-humid challenge, and whether any downstream effect can be demonstrated across the animal-food-environment interface. The distinction matters for both scientific credibility and practical adoption.

Materials and Methods

Review question and operational definitions

This article is a critical structured narrative

review, not a registered systematic review or meta-analysis. The primary review question was whether phytochemical interventions have been shown to reduce bacterial disease and antimicrobial use in chickens exposed to natural or simulated cold-humid conditions. Secondary questions addressed (i) host and environmental pathways by which low temperature and humidity-related housing conditions may modify bacterial disease, (ii) the strength of evidence for candidate phytochemical mechanisms, (iii) safety and implementation constraints, and (iv) the measurements needed to support antimicrobial-stewardship and One Health claims.

Evidence was classified at three levels. Direct evidence required all four elements in the same study: a defined cold-humid exposure meeting the direct-moisture criteria above, a defined bacterial challenge or confirmed bacterial disease, a characterized phytochemical intervention, and a measured antimicrobial-use or veterinary rescue-treatment endpoint. Proximal evidence included either cold or cold-humid exposure paired with a phytochemical intervention, or bacterial challenge paired with a phytochemical intervention, when clinically relevant outcomes were measured. Supportive evidence comprised mechanistic, *in vitro*, non-challenge, non-phytochemical, or non-cold studies that clarified one component of the pathway. *In vitro* antibacterial activity was used only for candidate selection and mechanistic support; it was not counted as evidence of disease control in chickens.

Literature identification and synthesis

Literature searches were conducted iteratively during manuscript development and were last updated on 12 September 2026. PubMed was the primary bibliographic database. Publisher and journal websites were used for targeted retrieval and source verification of records identified by title, DOI, or citation chaining rather



Table 1: Mechanistic pathways linking low-temperature/direct-moisture exposure and related housing co-exposures to bacterial disease and candidate phytogetic targets, with observed evidence separated from proposed intervention pathways.

Exposure or related housing factor	Host/environmental consequence	Disease or One Health implication	Candidate phytogetic target and evidence status (observed vs proposed)
Low effective temperature	Higher thermogenic demand; endocrine and oxidative stress; altered intestinal immune signaling	Reduced resilience and greater severity after bacterial exposure in susceptible birds	Observed host-target evidence: cold studies support oxidative, inflammatory, and barrier modulation by selected phytochemicals, but disease-treatment effects are not demonstrated [7,8,10-12].
Restricted ventilation (related co-exposure)	Reduced removal of water vapor, ammonia, dust, and bioaerosols; respiratory irritation. Ventilation can promote moisture retention but is not a direct humidity measure.	Greater respiratory disease pressure, including APEC-associated disease; occupational exposure	Proposed adjunctive target: mucosal or anti-inflammatory support may be useful, but no reviewed phytogetic trial demonstrated control of a cold-humid respiratory disease pathway; ventilation correction remains primary.
Condensation and wet litter (direct moisture evidence)	Caking, high water activity, footpad damage, patchy microbial hot spots	Persistence and shedding of <i>Salmonella</i> ; greater environmental contamination	Proposed intervention pathway: reduced intestinal shedding is biologically plausible, but no reviewed study showed that a phytogetic reduced environmental <i>Salmonella</i> persistence under cold-humid housing [1-4].
Barrier dysfunction and dysbiosis	Lower colonization resistance; altered permeability and mucosal immunity	Opportunity for <i>Salmonella</i> , APEC, or <i>C. perfringens</i> under predisposing conditions	Observed host-target evidence exists for barrier and redox modulation; translation to bacterial disease control under cold-humid exposure remains untested [9-12,21-24].
Diagnostic uncertainty	Overlapping signs from cold stress, ammonia exposure, coccidiosis, viral disease, and bacterial infection	Empirical or repeated antimicrobial treatment	Stewardship hypothesis only: preventive support may reduce disease pressure, but no reviewed cold-humid phytogetic study measured clinically indicated rescue treatment.
Litter/manure export	Movement of pathogens, residues, resistant bacteria and resistance genes to soil and water	Food, environmental, wildlife, and worker exposure	Proposed One Health pathway: lower shedding plus safe manure management could reduce downstream exposure, but linked animal-food-environment surveillance is required before claiming benefit [18-20].

than treated as independent comprehensive databases. Backward and forward citation chaining was applied to eligible records. Search concepts were combined in several blocks rather than one universal string: (chicken OR broiler) with (cold stress OR low temperature OR winter) and, where relevant, (humidity OR wet litter OR litter moisture OR water activity OR ammonia OR ventilation); bacterial terms including *Salmonella*, avian pathogenic *E. coli*, *E. coli*, and *Clostridium perfringens*; phytogetic terms including phytogetic, essential oil,

flavonoid, polyphenol, *Allium*, fermented botanical, and plant extract; and translational terms including antimicrobial use, treatment, antimicrobial resistance, manure, food safety, and One Health. Foundational older studies were retained when they established environment-infection relationships still relevant to modern production.

Study selection, screening and evidence ing

Records were screened in two stages by the

**Table 2:** Study-level characteristics, evidence type, and key limitations at the low-temperature/moisture-bacterial disease-phytogenic interface.

Evidence/model	Study design and animals	Intervention / dose / formulation	Environment and bacterial challenge	Major outcomes and interpretation / limitation
Cold + <i>S. Typhimurium</i> [5]	Comparative experiments in young chickens; acute-rechallenge subset included 60 previously challenged birds	No phytogenic intervention	Cold-only: 18-22°C vs 32-36°C; separate 10°C/24 h exposure; <i>S. Typhimurium</i> challenge	Low temperature increased susceptibility and shedding. Direct moisture conditions and antimicrobial-use outcomes were not characterized.
Cold + <i>S. Heidelberg</i> [6]	100 day-old SPF White Leghorn chicks; four groups	No phytogenic intervention	Cold-only: 19 +/- 1°C for 6 h/day on days 1-7; <i>S. Heidelberg</i> infection. Temperature and humidity were monitored, but no prespecified humid/moisture exposure was imposed.	Demonstrated a temperature-infection interaction affecting bacterial load and immunity. No phytogenic or antimicrobial-use endpoint; humidity monitoring alone did not meet the review's direct-moisture criterion.
Cold + <i>E. coli</i> model [9]	Three 3-week experiments; 430 day-old broiler chicks in total	Resistant-starch prebiotics (15% first week) and a commercial probiotic in feed/water	Cold-only stress plus oral <i>E. coli</i> challenge; direct moisture exposure was not a defining component of the design.	Shows feasibility of concurrent thermal stress and infection testing, but effects were variable and the probiotic increased mortality in one challenged experiment; not phytogenic.
Rutin [11]	360 one-day-old male Arbor Acres broilers; 4 groups; 6 replicates x 15 birds	Rutin 500 or 1,000 mg/kg diet	Cold-only: cold-stress regimen initiated after the first week with a 12°C target; no bacterial challenge; humidity was not a defined humid/moisture stressor.	Improved growth, redox, barrier, immune and microbiome-associated endpoints. No disease or antimicrobial-treatment endpoint; microbiome changes are supportive rather than stand-alone evidence.
Commercial phytogenic blend [24]	480 one-day-old Ross 308 broilers; 4 groups; 4 replicates x 30 birds	Herb-ALL RESP, 1 kg/tonne feed	Winter low-temperature condition; no defined bacterial challenge; direct moisture exposure incompletely characterized.	Improved performance, antibody response, Nrf2 and IL-1beta-related outcomes. Limited chemical definition and no antimicrobial-use endpoint.
Fermented chive + <i>E. coli</i> [27]	250 male J-Dabaco broilers; 5 groups	<i>L. plantarum</i> 1582-fermented chive at 1% or 3% of feed	Non-cold; <i>E. coli</i> ExPEC_A338 challenge on day 8	At 3%, improved growth/survival and several immune, barrier and pathogen-related outcomes. In vivo disease-mitigation evidence, but no cold-humid exposure or prospectively measured rescue treatment.



Fermented onion/chive vs MDR isolates [28]	Poultry-associated MDR <i>E. coli</i> and <i>Salmonella</i> isolates; pathogenicity component in chickens	<i>L. plantarum</i> / <i>B. subtilis</i> -fermented purple onion and chive extracts; antibacterial testing by inhibition/MIC/MBC methods	Non-cold; evidence predominantly in vitro	Supports candidate selection and antibacterial spectrum. In vitro activity is not equivalent to treatment efficacy in infected birds.
Fermented shallot + <i>S. Pullorum</i> [31]	300 one-day-old male broilers; 6 groups	<i>L. plantarum</i> 1582-fermented shallot in drinking water at 1%, 2%, or 3%	Non-cold; <i>S. Pullorum</i> NCTC10705 challenge	2-3% improved survival, pathogen burden, immune and barrier endpoints. Proximal in vivo disease-mitigation evidence, but no cold-humid exposure or direct antimicrobial-sparing endpoint.
Fermented red onion under heat stress [32]	300 one-day-old male Rilai chickens; 4 treatments; 5 replicate pens x 15 birds; 70 days	<i>L. plantarum</i> 1582-fermented red onion in drinking water at 25, 50, or 100 mg/L	Natural chronic heat stress; THI predominantly 28.9-31.1; no bacterial challenge	Improved feed efficiency at higher dose, selective cecal microbial shifts, and lower excreta ammonia. Useful for delivery/environmental feasibility only; opposite thermal stress and no bacterial-disease endpoint.

author: title/abstract screening for topical relevance followed by full-text assessment of studies used for study-level claims or tables. Duplicate records were removed manually. Because this was a structured narrative review rather than a registered systematic review, a prospective PRISMA-style search log was not maintained; exact numbers of records retrieved, deduplicated, screened, and excluded therefore cannot be reconstructed reliably and are not reported retrospectively. The final review cites 32 sources, while table 2 summarizes nine primary study records selected for study-level comparison at the environment-infection-phytogenic interface. Primary poultry studies were prioritized for evidence grading; reviews and non-poultry studies were used only for background or mechanism when a poultry primary study was not the relevant source. Exclusion at the full-text stage occurred when the intervention or exposure could not be characterized sufficiently, when the study did not concern poultry for a poultry efficacy claim, or when only a secondary citation was available for a primary-study statement. No PRISMA flow diagram or formal risk-of-bias score was

generated; that methodological limitation is acknowledged below.

Each included study used in the evidence synthesis was coded for environmental category (cold-humid based on direct moisture evidence, cold-only, winter/cold-season with incomplete direct moisture characterization, or non-cold), evidence setting (*in vitro*, *in vivo* challenge, *in vivo* non-challenge, or field), product identity and formulation, dose and route, animal characteristics, pathogen/challenge details where applicable, and whether antimicrobial treatment was actually measured. Restricted ventilation and ammonia were coded as related housing co-exposures rather than as direct humidity measures. Microbiome changes were not labeled beneficial on taxonomic direction alone; they were interpreted as supportive only when accompanied by a relevant health, pathogen, barrier, or production endpoint.

Quantitative pooling was not attempted. Environmental protocols differed in temperature, duration, bird age, acclimation, room replication, humidity reporting, ventilation, and litter conditions. Phytogenic preparations



differed even more substantially in plant species, plant part, extraction or fermentation process, chemical characterization, microbial strain, dose, carrier, route, and storage. Bacterial challenge studies also varied in strain, inoculum, route, age at challenge, and endpoints. A pooled effect estimate would therefore imply treatment and exposure equivalence that is not supported by the source studies. Cross-study comparisons in this review are qualitative and evidence-level based.

Results of the Evidence Synthesis

Interpretive convention. Throughout the Results, 'demonstrated' refers to an outcome measured in the cited experiment. Terms such as 'proposed,' 'plausible,' or 'candidate' identify mechanisms or cross-domain pathways that were not tested as an integrated outcome. Likewise, microbiome shifts are described neutrally unless they co-occur with a relevant disease, pathogen, barrier, welfare, or production endpoint.

Cold-humid exposure as a coupled host-environment stressor

Cold exposure increases metabolic heat production and redirects dietary energy toward thermoregulation. The endocrine response may include altered adrenal and thyroid activity, and the downstream effect depends on age, genotype, acclimation, duration, and severity. At the intestinal level, cold exposure has been associated with altered immunoglobulin and cytokine expression, villus morphology, heat-shock signaling, redox imbalance, and disruption of inflammatory control [7,8]. More recent work shows that intermittent mild acclimation can partially protect the intestine against a subsequent acute cold episode through modulation of TLR4/MyD88/NF- κ B signaling, antioxidant defenses, and mucosal immune function [10].

These findings are important because

they shift the discussion away from growth alone. A bird can maintain body weight while experiencing a biologically meaningful change in barrier function or inflammatory tone. Conversely, a molecular change is not necessarily clinically important unless it maps onto disease, lesions, pathogen burden, or welfare. The most useful cold-stress studies therefore combine physiological markers with outcomes that matter to flock health.

Moisture, litter, ventilation and pathogen ecology

Humidity is often treated as an independent stressor, but in poultry houses its main importance is operational. During cold weather, reduced ventilation conserves sensible heat but can also reduce removal of water vapor and contaminants. Reduced ventilation is therefore a management co-exposure that can promote moisture retention; it is not itself a direct humidity measure. Wet or caked litter increases bird contact with excreta and can sustain localized microbial activity, while ammonia is a contaminant co-exposure that irritates respiratory tissues and can amplify the consequences of other infectious or environmental insults [1].

The relationship between litter moisture and *Salmonella* is not uniform. Commercial-house studies showed associations among litter water activity, construction, watering devices, and *Salmonella* prevalence, while later work emphasized that contamination is spatially heterogeneous [2,3]. Recent genomic-ecological evidence adds another layer: moisture and trace metals may contribute to persistence of *Salmonella* strains carrying large plasmids and siderophore systems [4]. These are environmental-association data, not phytogenic intervention trials. The practical implication is that one litter sample and one humidity sensor are insufficient to characterize pathogen-relevant microenvironments.

For future direct cold-humid experiments, low effective temperature should therefore be reported together with direct moisture evidence at bird level, ideally including relative humidity in a defined humid condition, condensation, litter moisture or water activity, and wet/caked-litter scoring. Contextual co-exposures such as air speed, ventilation rate, and ammonia should be reported separately. Studies that report cold temperature alone, or only reduced ventilation/ammonia without direct moisture evidence, can still inform host susceptibility but should remain explicitly classified as cold-only evidence (Table 1).

Evidence-status note: the environmental and host relationships in the first three columns are supported to varying degrees by the cited literature; the phytogetic intervention points in the final column are proposed unless explicitly described as observed. Direct moisture indicators (defined humid exposure/relative humidity, condensation, wet/caked litter, litter moisture, or water activity) are distinguished from related factors such as restricted

ventilation and ammonia; the latter can intensify risk but did not by themselves qualify a study as cold-humid. No row constitutes proof of an integrated antimicrobial-sparing or One Health outcome (Figure 1).

Bacterial disease at the cold-humid rface

Salmonella: cold-only evidence that low temperature can amplify infection: The strongest temperature-infection evidence comes from *Salmonella* challenge models, but these studies are cold-only rather than cold-humid under the definition used here. In young chickens, cold housing at 18–22°C increased susceptibility to low and moderate *S. Typhimurium* challenge, and a separate acute 10°C exposure increased shedding [5]. In 100 SPF White Leghorn chicks, early-life cold stress at 19 +/- 1°C for 6 h/day interacted with *S. Heidelberg* infection at the level of bacterial load and immunity [6]. Neither study incorporated a prespecified humid/moisture exposure. In the *S. Heidelberg* study, humidity was monitored, but it was not the experimental humid condition and no direct litter-moisture

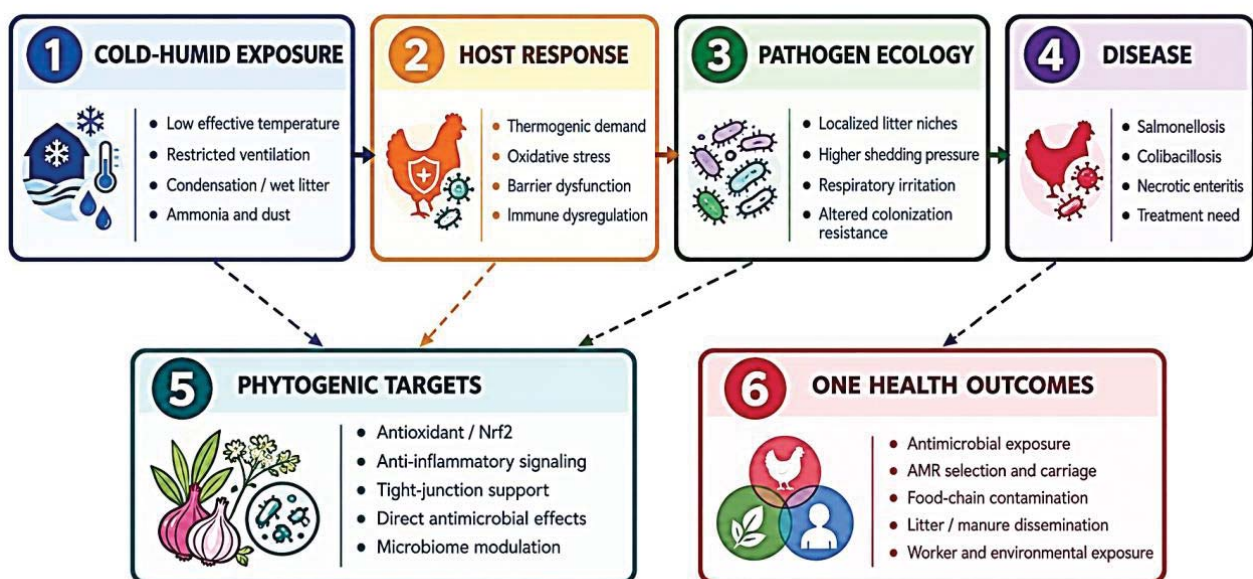


Figure 1 Causal framework for the cold-humid environment-bacterial disease interface. Dashed arrows indicate proposed phytogetic intervention points; environmental management remains the foundation of control. The figure is conceptual and should not be read as evidence that the full pathway has been demonstrated in one study.

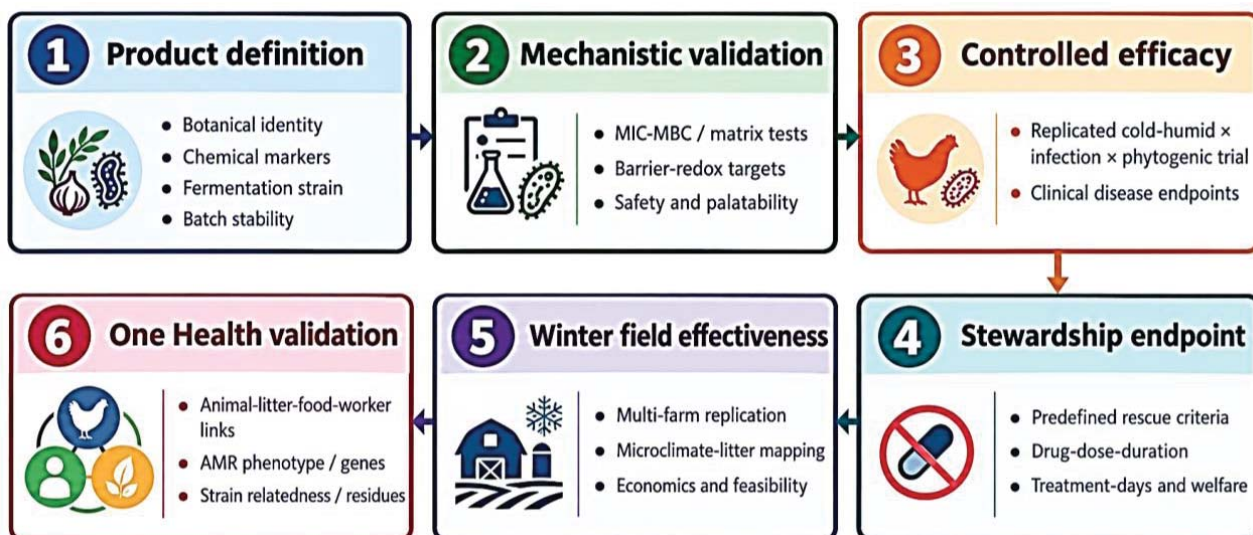


Figure 2 Staged translational pathway. Advancement from physiological support to antimicrobial-sparing and One Health claims should occur only when the corresponding endpoints are measured directly. The later stages are proposed validation targets, not established outcomes of the current evidence base.

or condensation criterion was integrated; the study therefore remains cold-only under the operational definition used here. Both studies demonstrate that low temperature can modify the host-pathogen relationship rather than merely depress growth.

For translation, serovar and strain matter. *Salmonella* control is a production-continuum problem involving breeder flocks, hatcheries, farm biosecurity, feed, litter, transport, slaughter, and processing [13,19]. A phytogenic that reduces cecal shedding under one experimental condition cannot be assumed to reduce carcass contamination unless that effect persists across later stages. The cold-only challenge evidence therefore supports biological plausibility for winter risk, not a demonstrated cold-humid phytogenic effect.

APEC: respiratory and systemic disease in a stressed host: APEC is relevant to cold-humid production because respiratory irritation, viral co-infection, immune suppression, and ventilation failure can converge, but the available combined challenge evidence is again cold-only. In three experiments involving 430 day-old broiler chicks, cold stress and oral *E. coli*

challenge were combined with resistant-starch prebiotics or a commercial probiotic [9]. The nutritional responses were inconsistent across experiments and the intervention was not phytogenic. The study is valuable mainly because it demonstrates how temperature stress and bacterial challenge can be tested concurrently; it does not establish a cold-humid phytogenic or antimicrobial-sparing effect.

Contemporary APEC isolates frequently carry multiple virulence determinants and antimicrobial resistance phenotypes [14]. Future phytogenic trials should therefore characterize challenge strains beyond species identification. Virulence profile, susceptibility phenotype, inoculum, route, and recovery from lesions or target tissues are needed to interpret whether an intervention is reducing colonization, invasion, systemic disease, or only performance loss.

***Clostridium perfringens* and the importance of predisposing conditions:** Necrotic enteritis illustrates why bacterial abundance alone is an inadequate disease endpoint. *C. perfringens* is common in the chicken intestine, whereas clinical disease reflects toxin genotype, mucosal injury, coccidiosis, diet, microbiota, and host



susceptibility [15]. Wet litter may increase coccidial cycling and worsen management conditions that predispose to enteric injury. A convincing study should therefore include lesion scoring, coccidial assessment, toxin-relevant pathogen characterization, and flock performance rather than simply report a change in *C. perfringens* counts.

The broader microbiome remains relevant because commensal communities shape mucosal maturation, competitive exclusion, and immune calibration [16]. However, a taxonomic shift is not intrinsically beneficial. Relative-abundance changes should be interpreted together with absolute pathogen counts, barrier measures, lesions, clinical disease, or production outcomes. In this review, microbiome findings are therefore treated as supportive when they accompany a relevant host or pathogen endpoint, not as stand-alone proof of improved health.

Phytogenic strategies: mechanism, evidence, and limits

Why multi-target phytogenics are attractive under compound stress: Phytogenic feed additives include plant powders, essential oils, oleoresins, extracts, purified compounds, and fermented botanical preparations. Their appeal under cold-humid stress comes from mechanistic breadth. Oxidative injury, inflammatory signaling, barrier integrity, pathogen pressure, and microbiome instability can occur together, so a multi-target intervention may be more useful than a compound that acts on only one pathway [21–23].

That breadth should not be confused with reproducibility. Botanical identity, cultivar, plant part, soil and climate, harvest maturity, drying, extraction solvent, storage, encapsulation, feed matrix, and dose can materially change exposure. Fermented products add another layer: strain identity, inoculum, fermentation time, pH, viable count, transformed phytochemicals,

organic acids, and newly formed metabolites all contribute to the final intervention. The term phytogenic is therefore too broad to function as a treatment description.

Safety deserves the same attention as efficacy. High concentrations of essential oils can reduce palatability or irritate mucosa; polyphenols can bind feed components; fermented products require strain-level microbial safety and contaminant control; and drinking-water products can interact with biofilms and line sanitation [21–23]. Product safety is dose-, formulation-, route-, and duration-specific. A preparation intended for repeated winter use should therefore have batch chemical markers, microbial specifications, contaminant limits, stability data, palatability observations, and a realistic delivery protocol rather than relying on a generic assumption that botanical origin implies safety.

Proximal evidence during cold stress: Rutin provides relatively clear proximal evidence because the intervention is chemically defined. In 360 one-day-old male Arbor Acres broilers, dietary rutin at 500 or 1,000 mg/kg was tested during a cold-stress regimen initiated after the first week, with a 12°C cold-stress target, and improved growth, redox indices, intestinal barrier measures, inflammatory signaling, and cecal community structure [11]. Humidity was not used to define an experimental humid/moisture stressor, so this study is classified as cold-only. The microbiome shifts are interpreted here as secondary/supportive because they occurred alongside barrier and performance outcomes. The experiment did not include bacterial challenge or antimicrobial treatment, so it supports cold-responsive host modulation rather than disease control or antimicrobial sparing.

A commercial phytogenic blend evaluated during winter cold stress provides another *in vivo* example. In 480 Ross 308 broilers, 1 kg/



tonne of feed improved performance, antibody response, Nrf2 signaling, and inflammatory indicators under the study's low-temperature winter condition [24]. The whole-product evidence is practically relevant, but chemical definition was limited, direct moisture exposure was not sufficiently characterized for a cold-humid classification, and there was no bacterial challenge or antimicrobial-use endpoint.

Fermented *Allium* products as a staged product-development case study: The fermented *Allium* literature should be interpreted as a staged product-development program rather than as eight independent confirmations. Most studies [25–32] involve overlapping investigators, related plant substrates, and a shared *Lactobacillus*/*Lactiplantibacillus* platform. This continuity is useful for product development, but repeated positive findings from the same network do not provide the same evidentiary weight as independent replication. Accordingly, the studies are used here to illustrate staged development and biological plausibility, not to establish general efficacy across laboratories, climates, or production systems.

The evidence level also differs markedly within that program. Dietary chive essential oil was evaluated *in vivo* in broilers without a defined infection or cold-humid model [25]. Selection of chicken-derived *Lactobacillus* strains for purple-onion fermentation primarily addressed probiotic screening, strain behavior, and antibacterial activity against poultry-associated *Salmonella* [26]. In contrast, fermented chive was tested *in vivo* in 250 male J-Dabaco broilers challenged with *E. coli*, using 1% and 3% dietary inclusion; growth, survival, immune, barrier, and pathogen-related outcomes improved most clearly at 3% [27]. That study supports disease-mitigation potential for a defined challenge model, but not efficacy during cold-humid exposure or reduced clinically indicated antimicrobial treatment.

Other studies provide different, non-equivalent forms of evidence. Fermented purple onion and chive extracts showed activity against toxin-marker-positive multidrug-resistant *E. coli* and *Salmonella* isolates, but the antibacterial component was predominantly *in vitro* and should remain a candidate-selection result rather than a field disease-control claim [28]. The *E. acervulina* challenge study with fermented chive was *in vivo*, yet it addressed a parasitic predisposing condition rather than bacterial disease [29]. Fermentation of purple onion altered flavonoid, sulfur-compound, and antioxidant profiles, supporting product characterization but not therapeutic efficacy [30]. These studies are mechanistically complementary, not interchangeable efficacy trials.

A later bacterial challenge study in this series involved 300 one-day-old male broilers given *L. plantarum*-fermented shallot in drinking water at 1–3% and challenged with *S. Pullorum*; the 2–3% levels improved survival, pathogen burden, inflammatory responses, and intestinal barrier-related outcomes [31]. The study did not include cold-humid exposure or an antimicrobial-use endpoint. A separate trial in 300 male Rilai chickens used 25, 50, or 100 mg/L lactic-fermented red onion in drinking water during chronic natural heat stress and reported improved feed efficiency, selective cecal microbial shifts, and lower ammonia emission at the higher dose [32]. Because the thermal exposure was heat rather than cold, those results support delivery feasibility and selected environmental endpoints only. Across [25–32], plant species, preparation, dose, route, and outcome definitions vary substantially; cross-study comparisons are therefore qualitative. Independent replication remains a major evidence gap.

From *in vitro* activity to reproducible *in vivo* exposure: *In vitro* antibacterial activity should be treated as a screening endpoint. Agar



diffusion reflects diffusion as well as potency, and concentrations that suppress bacteria in broth may never be reached in feed, drinking water, mucus, or intestinal contents. Candidate products should progress through minimum inhibitory and bactericidal concentrations, time-kill studies, feed- or water-matrix testing, adhesion or invasion assays, and dose-recovery measurements before challenge trials. An *in vitro* signal becomes relevant to practical disease control only after *in vivo* exposure, tolerability, and clinically meaningful outcomes are demonstrated.

Host-directed effects may be at least as important as direct killing. Nrf2-associated antioxidant defenses, epithelial junctions, mucosal immunoglobulins, and inflammatory control are plausible targets during cold stress [10-12,24]. Cross-study comparison nevertheless requires caution: powders, essential oils, purified flavonoids, aqueous or alcoholic extracts, and fermented preparations are different interventions. Fermentation further changes viable counts, organic acids, transformed phytochemicals, and storage behavior. Dose cannot be compared meaningfully without route and formulation; for example, mg/kg feed, percent feed inclusion, and percent or mg/L drinking-water dosing generate different exposures. The claim should therefore follow the characterized product and endpoint actually tested rather than the plant name alone.

Safety and tolerability: evidence remains product-specific: The reviewed *in vivo* studies did not reveal an obvious common toxicity signal at the tested doses, but most were efficacy studies rather than dedicated safety studies. Rutin at 500-1,000 mg/kg and the commercial phytogenic blend at 1 kg/tonne did not produce a reported performance penalty during their short cold-stress experiments [11,24]. Chive essential oil at 100-500 mg/kg, fermented chive at 1-3% of feed, and fermented shallot at 1-3% in drinking water were also tolerated within their

respective experimental windows [25,27,31]. These observations support short-term tolerability only; they do not establish chronic safety, reproductive safety, residue absence, or safety at higher concentrations.

Future product dossiers should therefore include feed or water intake, palatability, clinical observations, mortality, hematology and serum biochemistry where relevant, target-organ assessment, microbial purity, antimicrobial-resistance genes in live fermentation strains, chemical contaminants, shelf-life change, and interactions with water-line sanitation. Safety conclusions should remain tied to a specific formulation, dose, route, bird age, and duration.

Table 2 deliberately distinguishes cold-only from cold-humid exposure, direct moisture evidence from related housing co-exposures, and *in vitro* from *in vivo* evidence. None of the listed phytogenic studies measured a reduction in clinically indicated antimicrobial treatment under a defined cold-humid bacterial challenge.

Discussion

Translational and one health implications

In this review, antimicrobial stewardship and One Health are used as frameworks for endpoint selection, not as outcomes already demonstrated by phytogenic studies. The current evidence is strongest at the animal and mechanistic levels. Downstream food-safety, resistance, environmental, worker, and antimicrobial-use effects remain proposed pathways unless they were measured directly.

A claim ladder: from physiology to antimicrobial sparing: Four claims are often compressed into the phrase antibiotic alternative, but they are not equivalent. A product may improve performance under stress; modify a host pathway associated with resilience; reduce a pathogen or disease endpoint in a challenge model; or reduce clinically indicated antimicrobial treatment without worsening



welfare. Each step requires a stronger design. None of the reviewed phytogenic studies reached the fourth step under a defined cold-humid bacterial challenge. A fifth claim - reduced antimicrobial resistance or a broader One Health benefit - requires linked cross-domain evidence and should not be inferred from animal performance or cecal microbiota alone.

This hierarchy should change how trials are planned. If antimicrobial sparing is the claim, veterinary rescue criteria should be written before the experiment. Investigators should report the proportion of pens or flocks treated, clinical indication, antimicrobial class, dose, duration, retreatment, mortality, and reasons for intervention. Withholding effective therapy from clinically ill birds is ethically unacceptable. The relevant question is whether fewer animals or pens reach a predefined treatment threshold while welfare and disease outcomes are maintained or improved.

The decisive controlled experiment: A key next-step experiment is an integrated controlled design that tests environmental condition (thermoneutral/dry versus cold-humid), infection status (unchallenged versus a defined bacterial challenge), and phytogenic treatment (absent versus present), generating a 2 x 2 x 2 factorial structure. If low temperature and direct moisture exposure are manipulated independently, the design expands accordingly. Antibiotic comparators and dose groups can be added, but they should not replace the core interaction structure.

Environmental treatment must be replicated at the room or chamber level. Birds sharing one room are not independent replicates for temperature or humidity. Multiple independent rooms per environmental condition are needed to avoid pseudoreplication. Temperature, relative humidity, condensation where relevant, air speed, floor temperature, litter moisture, water activity, exposure duration, and diurnal

recovery should be reported as direct exposure descriptors; ventilation rate and ammonia should be reported separately as related management/contaminant co-exposures. Natural-winter trials should map within-house microclimate rather than rely on a single fixed sensor.

Challenge strains should be characterized for virulence, antimicrobial susceptibility, relevant genes or genome sequence, dose, route, and recovery from target tissues. Primary endpoints should be clinical disease, lesions, pathogen burden, mortality, or treatment need. Molecular measurements are valuable when they explain those outcomes rather than substitute for them.

Food safety, AMR, and environmental dissemination: The following food-safety and AMR pathways are prospective One Health endpoints, not established effects of the phytogenic interventions reviewed here. A reduction in intestinal *Salmonella* can be useful, but a food-safety claim requires continuity from farm to processing. Transport, lairage, slaughter, and cross-contamination can erase farm-level differences [19]. Appropriate outcomes include fecal or cecal concentration, carcass prevalence and concentration, serovar or strain relatedness, susceptibility phenotype, resistance genes, and residues. Farm-level evidence should remain a farm-level claim until later steps in the chain are measured.

The environmental pathway is equally important. Poultry litter can transport resistant bacteria, resistance genes, antimicrobial residues, and other biologically active compounds. Land application links poultry houses to soil, crops, surface water, groundwater, wildlife, and people. Cross-environmental cycling of antimicrobial resistance has been documented in agricultural systems fertilized with poultry litter [20]. A convincing One Health study should therefore connect samples longitudinally and use strain or resistance-gene



relatedness to test transmission hypotheses rather than simply report separate prevalence estimates.

Antimicrobial resistance should also be distinguished from pathogen reduction. A lower pathogen count can improve health even if the proportion resistant among survivors does not change. Conversely, resistance-gene abundance may shift without a large change in total bacterial burden. Susceptibility testing, targeted genes, mobile genetic elements, and strain relatedness provide different pieces of the same problem. This is relevant to plant-derived antimicrobials as well: repeated subinhibitory exposure should not be assumed to be evolutionarily neutral.

Field implementation and economics: In practice, phytogenics cannot compensate for inadequate heating, ventilation, drainage, drinker maintenance, litter management, vaccination, or diagnosis. The first response to cold-humid conditions is to restore an appropriate effective temperature while maintaining minimum ventilation and drying the litter. A phytogenic product can then be layered onto this management foundation as a preventive or adjunctive strategy.

Delivery route should match the risk window and the product specification. Feed inclusion offers consistent preventive exposure, whereas drinking-water delivery can respond quickly at flock level but raises questions about solubility, intake, biofilm control, line sanitation, dosing uniformity, and compatibility with acidifiers or disinfectants. Encapsulation may protect volatile compounds or improve intestinal delivery. Fermented liquids require defined pH, microbial purity, viable count where relevant, shelf life, and water-line compatibility. Commercial scale-up should reproduce the product - botanical identity, chemical marker range, viable count, contaminant limits, and dose recovery - rather than merely reproduce nominal fermentation settings.

Economic evaluations should include more than additive cost per tonne of feed. Manufacturing and quality control, batch rejection, storage, packaging, dosing equipment, labor, feed or water intake, mortality, veterinary treatment, carcass downgrading, and uncertainty all matter. Multi-farm adoption also requires a practical quality system: raw-material traceability, batch release specifications, stability through the claimed shelf life, dosing instructions, worker training, and complaint/adverse-event capture. A product that works in a controlled chamber but cannot maintain composition or dosing uniformity at farm scale would have limited translational relevance (Figure 2)

Boundary conditions, reproducibility, and external validity: The response to a phytogenic intervention is unlikely to be constant across cold-humid production systems. Age, genotype, feather cover, stocking density, floor temperature, air speed, litter reuse, drinker design, ventilation strategy, and the duration of nightly thermal recovery all alter the exposure experienced by the bird. Pathogen factors add another layer: serovar or strain, inoculum, route of exposure, virulence repertoire, antimicrobial susceptibility, and preceding viral or coccidial injury can change the clinical phenotype even when the nominal environmental treatment is similar [1-6,13-16]. For that reason, a single temperature-relative-humidity pair should not be treated as a universal definition of cold-humid stress. Studies need to report the exposure actually experienced at bird level and the environmental heterogeneity within the house.

Product definition creates an equally important boundary condition. Reviews of phytogenic feed additives repeatedly identify botanical identity, dose, processing, stability, feed-matrix interactions, and batch variation as reasons why apparently similar products can produce different biological responses [21-23]. Fermented preparations are more complex



because the final exposure may contain residual plant compounds, transformed metabolites, organic acids, microbial biomass, and viable fermentation organisms. The regional Allium program illustrates this point: different plant species, *Lactobacillus/Lactiplantibacillus* strains, fermentation conditions, and delivery routes generated distinct products and should not be treated as interchangeable interventions [25-32]. At minimum, efficacy trials should define the botanical source and plant part, fermentation strain where applicable, viable count, pH, relevant chemical markers, storage conditions, and dose recovery in feed or drinking water. Without those data, replication becomes a comparison of labels rather than a comparison of treatments.

External validity also depends on the experimental unit. Environmental treatments are imposed at room, chamber, or house level, whereas many outcomes are measured on individual birds. Treating birds from one chamber as independent replicates creates pseudoreplication and can exaggerate precision. Replicated rooms or independent houses are therefore essential when temperature, direct moisture exposure, ventilation, or litter condition constitutes the treatment. The same principle applies to field effectiveness: several farms across more than one winter cycle are preferable to a single-farm demonstration because low-temperature and moisture exposures are inherently variable. A robust validation program should expect some loss of effect size as control decreases from laboratory to farm conditions. That loss is informative; it indicates whether the intervention is resilient enough for routine production rather than whether it can work under idealized conditions.

Implementation in resource-variable poultry systems: The implementation question is particularly important in poultry systems where climate control, diagnostic access, and veterinary support are uneven. Global

analyses show that antimicrobial use and resistance remain major concerns in food-producing animals, especially where production intensification occurs faster than diagnostic and stewardship infrastructure [17,18]. In these settings, a phytogenic product may be attractive because it can be manufactured locally, delivered through feed or water, and integrated into preventive management. Yet low cost at purchase does not guarantee value. Quality-control failure, inconsistent raw material, poor solubility, blocked drinker lines, reduced feed intake, or delayed treatment can erase any biological advantage.

A practical field protocol should therefore begin with management correction. Minimum ventilation must be maintained while thermal comfort is restored; wet litter and leaking drinkers require direct correction; vaccination, coccidiosis control, biosecurity, and flock observation remain non-negotiable. Phytogenics can then be placed against a clearly defined risk window, for example before a predictable winter transition or during a period of elevated enteric challenge. Delivery route should match both pharmacological logic and farm capacity. Feed inclusion gives relatively stable preventive exposure, whereas drinking-water delivery can be more responsive but is vulnerable to changes in water intake, pH, solubility, biofilm, and line sanitation. Fermented liquids require additional attention to microbial purity and shelf stability.

The economic endpoint should be specified as carefully as the biological endpoint. Cost per kilogram of product is less informative than cost per bird placed, cost per kilogram live weight, treatment cost avoided, mortality avoided, and any change in carcass quality or condemnation. A product that modestly improves feed conversion but does not alter disease incidence may still be economically useful, but it should not be described as antimicrobial-sparing. Conversely, a product that reduces the number of flock episodes meeting predefined treatment criteria



could be evaluated for stewardship relevance even if growth responses are small. Linking veterinary decisions to economics can also help determine whether a measured reduction in antimicrobial exposure reflects a clinically meaningful management effect rather than simple substitution of one input for another.

Limitations of this review and evidence base

This synthesis has several limitations. First, it is a structured narrative review conducted by a single author rather than a registered systematic review. PubMed was the primary bibliographic database, supplemented by targeted publisher/journal retrieval and citation chaining, and the final search update was 12 September 2026. A prospective retrieval/screening log was not maintained, so exact record counts, formal duplicate-screening documentation, a PRISMA flow diagram, and study-level risk-of-bias scoring are unavailable. Second, the evidence is heterogeneous in temperature protocol, direct moisture characterization, litter conditions, animal age and genotype, pathogen strain, dose, formulation, route, and outcome definition, which precluded quantitative pooling and limits cross-study comparability.

Third, direct evidence is extremely limited: no reviewed study combined a characterized cold-humid exposure, defined bacterial disease, a characterized phytogenic, and a measured antimicrobial-use endpoint. Several key environment-infection studies are old and cold-only, while newer phytogenic studies generally address host physiology without bacterial challenge. Fourth, positive-result publication and selective outcome reporting cannot be excluded. Finally, much of the fermented *Allium* evidence comes from an overlapping investigator network; this concentration raises the possibility of laboratory-, formulation-, or investigator-specific effects and means repeated findings should not be counted as

independent replication. These constraints are the main reason the review frames stewardship and One Health effects as research targets rather than established benefits.

Research priorities

Standardize and replicate the exposure, not just the diet. Cold-humid studies should prespecify the operational exposure and report direct moisture indicators separately from related co-exposures: bird-level thermal context; relative humidity in a defined humid condition; condensation; litter moisture or water activity; wet/caked-litter scoring; plus air speed, ventilation rate, ammonia, sensor location, duration, and recovery periods. Independent rooms or houses should be used as environmental replicates.

Define the product chemically and microbiologically. Botanical traceability, cultivar and plant part, analytical marker range, extraction or fermentation process, microbial strain identity and safety, viable count where relevant, contaminants, batch variation, stability, and dose recovery in feed or drinking water should be documented before efficacy claims are made. At least three independent production batches should be represented in pivotal studies.

Use clinically meaningful primary outcomes and prespecified analysis plans. Pathogen load, lesions, disease incidence, mortality, welfare, or rescue treatment should be primary outcomes; redox, cytokine, barrier, and microbiome measures should explain rather than replace clinical effects. Microbiome hypotheses should specify whether absolute abundance, function, or pathogen displacement is expected.

Measure antimicrobial use prospectively. Treatment thresholds, veterinary decision rules, drug, dose, duration, retreatment, and withdrawal should be prespecified so antimicrobial sparing becomes a measurable



endpoint rather than an inference. Trials should be designed to permit rescue treatment without penalizing animal welfare.

Build independent replication into the evidence base. Candidate products should first be repeated by a laboratory not involved in product development, then tested across multiple farms, genotypes, and at least two winter cycles. The unit of inference should match the treatment level, and field studies should include sufficient independent houses or farms to estimate between-site variability rather than relying on one demonstration flock.

Link animal effects to downstream domains only when the claim requires it. For One Health validation, animal, litter/manure, carcass or food, environmental, and where ethically feasible occupational samples should be collected longitudinally, with susceptibility testing, resistance genes, and strain relatedness used to test transmission hypotheses. Multi-site validation should also report product cost, treatment-days avoided, mortality, carcass outcomes, and feasibility so biological efficacy and implementation value are assessed together.

Conclusion

Cold-humid poultry environments create a coupled host and environmental challenge, but the term should be reserved for studies that document both low effective temperature and direct moisture exposure. Direct moisture evidence includes a defined relative-humidity condition, condensation, wet/caked litter, quantified litter moisture, or water activity; restricted ventilation and ammonia are related co-exposures rather than humidity measures. Low temperature alone can increase thermoregulatory, endocrine, and oxidative demand and can alter susceptibility to *Salmonella* or *E. coli* challenge [5,6,9]. The reviewed infection studies did not combine all components within a defined cold-humid model. Phytogetic strategies remain biologically plausible because

they can influence oxidative stress, inflammatory signaling, epithelial integrity, adhesion, bacterial viability, and microbiota-associated functions. Rutin and a commercial blend provide proximal *in vivo* evidence during cold stress [11,24]. Fermented *Allium* preparations provide separate *in vivo* challenge, *in vitro* antibacterial, product-characterization, and non-cold environmental-stress evidence [25–32]. These studies vary in formulation, dose, route, and endpoints and are concentrated within a limited investigator network, so they should be treated as a staged development program that still requires independent replication rather than as independent confirmation of general efficacy.

The most defensible position is therefore deliberately narrow. Phyto-genics can be developed as preventive or adjunctive tools within climate control, litter management, vaccination, diagnostics, biosecurity, and veterinary stewardship, but current evidence does not demonstrate reduced clinically indicated antimicrobial treatment or a downstream One Health benefit under cold-humid bacterial disease. Those claims should wait for adequately replicated, standardized, independently validated, multi-site studies that measure the corresponding endpoints directly.

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Author Contributions (CRediT)

Phan Vu Hai: Conceptualization; Methodology; Investigation (literature review); Data curation; Critical interpretation; Visualization; Writing - original draft; Writing - review and editing.

Ethics Statement

Not applicable. This review analyzes previously published studies and reports no new research involving humans or animals.

Data Availability Statement

No new dataset was generated or analyzed. All evidence discussed in this review is available in the cited publications.

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