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Predictive Modeling of Fine Particulate Matter (PM2.5) Related Health and Production Risks in Open-Sided Broilers

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ABSTRACT

Rapid increases in fine particulate matter with an aerodynamic diameter of 2.5 μm or less (PM2.5) can occur around open-sided poultry houses during seasonal haze in tropical regions, particularly when crop-residue burning, biomass combustion, wildfires, transboundary smoke transport, traffic emissions, and stagnant meteorological conditions coincide. However, for farm-level decision making, outdoor air quality data are rarely translated into short-term production and health risk estimates for broiler flocks. An empirical, scenario-based Monte Carlo model was developed to estimate the effects of a three-day, acute PM2.5-exposure episode in open-sided broiler houses. The model estimated indoor PM2.5 from daily outdoor PM2.5 using an indoor/outdoor infiltration ratio for naturally-ventilated housing and then linked indoor exposure to endpoint-specific modules for average daily gain, feed conversion ratio, live weight loss, hemoglobin and hematocrit change, exploratory post-vaccination immune retention, general health impairment, and respiratory pathology. Exposure-response assumptions were parameterized from peer-reviewed studies published in 2016–2026 and indexed in PubMed, Scopus, or Web of Science; controlled and longer-duration studies were used to define response direction and conservative bounds, not to infer direct three-day causality. In a moderate three-day regional haze scenario (outdoor PM2.5:150, 150, and 150 μg/m³), the median indoor PM2.5 was 135.0 μg/m³, with predicted average daily gain reduction of 5.2%, feed conversion ratio increase of 2.8%, liveweight loss of 9.4 g/bird, and 11.0% probability of respiratory pathology. In a severe three-day maize stover/biomass-burning scenario (outdoor PM2.5: 193, 400, and 611 μg/m³), the median indoor PM2.5 concentration was 361.2 μg/m³, with predicted average daily gain reduction of 19.3%, feed conversion ratio increase of 9.8%, liveweight loss of 34.2 g/bird, and 40.2% probability of respiratory pathology. These findings suggest that even short-haze events may be biologically relevant in tropical open-sided broiler systems, although external validation with paired indoor/outdoor PM2.5 and flock-health data is necessary.

Keywords: acute exposure; broiler chickens; Monte Carlo simulation; PM2.5; tropical open-sided housing

INTRODUCTION

Haze in northern Thailand and the surrounding areas during the dry season originates from a combination of sources: forest fires, burning of harvested crops, other open biomass burning, cross-border transport of pollutants, emissions from vehicles, and stagnant atmospheric conditions (Jainontee *et al.*, 2023; Suriyawong *et al.*, 2023; Chantaraprachoom *et al.*, 2024; Inlaung *et al.*, 2024). This is an immediate concern in open-sided broiler houses. These agricultural structures were primarily designed to provide relief from heat with lower construction costs and did not explicitly provide for pollutant exclusion. Low sidewall heights, posts, curtains, partial enclosures, and walls that are not sealed create conditions where ambient air quality is typically similar to indoor air quality (Sans *et al.*, 2021; Jongbo, 2024). Fine particulate matter (PM2.5) is of special concern because its small, aerodynamic diameter enables deep respiratory incorporation and provides a high surface area for the adsorption of toxic or biologically-active constituents. PM2.5 from haze and poultry-house environments may contain carbonaceous material, inorganic ions, metals, secondary aerosols, polycyclic aromatic compounds, microbial fragments, endotoxins, feed particles, feather debris, litter dust, and other organic compounds. Recent work in poultry houses has shown that particulate matter can adsorb ammonia, heavy metals, pathogenic microorganisms, and organic pollutants and can promote respiratory irritation, oxidative stress, microbial dysbiosis, inflammation, and reduced immune resistance (Wang *et al.*, 2023a). Environmental control of PM2.5 changes the bacterial community structure of broiler-house aerosols, indicating that airborne biological material is an important component of the exposure mixture (Wang *et al.*, 2023b).

There is biological evidence of harm from exposure to PM2.5 in chickens using research models; however, studies have not been consistent in timing (i.e., duration of study) and the particle source. Broilers exposed to total suspended particulates (TSPs) from broiler housing for 7–14 d develop lung injuries, systemic inflammation, and abnormalities in lipid metabolism (Shen *et al.*, 2023). Conversely, broilers exposed to PM2.5 for 21 d display an alteration in their pulmonary microbiota (Zhou *et al.*, 2023a) and modification of the microbiota may reduce the pulmonary inflammatory response induced by PM2.5 (Zhou *et al.*, 2023b). The inhalation of PM2.5 causes lung inflammation and an alteration of gut microbiota with the production of increased levels of gut-derived lipopolysaccharides and impairment of intestinal barrier markers (Zhou *et al.*, 2024). Long-term PM2.5 exposure has been associated with reductions in growth rate and disruption of serum metabolomic patterns (Chen *et al.*, 2024), whereas a more recent protein-related study determined that particulate matter from poultry housing, when used in a short, controlled manner, produced dysregulation of pulmonary protein pathways (Shen *et al.*, 2025). Although these studies have substantiated a biological basis for injury, the exposure duration in all examples was much greater than 3 d; thus, the duration of the studies will preclude them from being viewed as direct effect estimates for 3-d exposure.

To establish evidence of open-environment exposure to airborne particulates in field research, Mahmoud *et al.* (2025) evaluated broiler chickens in an agricultural maize stover burn site and showed elevated concentrations of both carbon monoxide and PM2.5, and increased systemic blood parameters, resulting in decreased lung function and lung tissue pathology (pulmonary fibrosis and metaplasia). Maize stover is an agricultural residue that is included in a wider category of biomass burning of organic materials, which is defined as the process of burning any biological material that is either alive or dead, such as crop residues, forest fuels, grassland fuels, and other plant biomass. Agricultural burning refers only to the intentional burning of crop residues or other agricultural wastes in the field. Transboundary haze refers to pollution created by haze dispersed through the air across an administrative boundary or an international border, which subsequently affects an area that is different from the original area. The definitions of each of these areas become important when evaluating the difference between the chemical composition and co-pollutants because the same PM2.5 metric may have been used to calculate the area. Therefore, the chemical composition of an area and its respective co-pollutants can differ despite the use of the same PM2.5 metric (Suriyawong *et al.*, 2023; Inlaung *et al.*, 2024; Chansuebsri *et al.*, 2024; Yabueng *et al.*, 2025).

Despite this growing evidence, poultry veterinarians and farm managers still have limited support for translating measured outdoor PM2.5 into short-term flock risk. This study addresses this gap by developing a transparent, field-oriented, scenario-based predictive model that links outdoor PM2.5, indoor exposure in open-sided housing, uncertainty propagation, and multiple veterinary endpoints within a single framework. The objective of this study was to estimate indoor PM2.5 and exposure during a 3-d, acute haze episode to predict short-term production and health outcomes relevant to open-sided broilers, including responses in average daily gain (ADG), feed conversion ratio (FCR), live weight (LW) loss, hematological changes, exploratory immune retention, general health impairment, and respiratory pathology.

MATERIALS AND METHODS

Evidence Review and Study Selection

A targeted, systematic evidence review was conducted in accordance with the reporting logic of PRISMA 2020 (Page *et al.*, 2021) to identify peer-reviewed studies relevant to PM2.5, particulate exposure, haze, smoke, biomass burning, respiratory injury, growth performance, hematology, immune response, microbiota, and indoor/outdoor exposure translation in broilers or closely-related avian systems. PubMed, Scopus, and the Web of Science were searched for articles published between January 2016 and March 2026. The search period was selected to comply with the journal's request for up-to-date references and avoid reliance on outdated exposure-response assumptions. The search terms included combinations of broiler, chicken, poultry, PM2.5, fine particulate matter, particulate matter, haze, smoke, biomass burning, agricultural burning, maize stover burning, transboundary haze, tropical, open-sided housing, naturally-ventilated housing, respiratory pathology, lung injury, haematology, immune response, vaccination, microbiota, infiltration factor, indoor/outdoor ratio, acute exposure, and short-term exposure.

This review was designed to parameterize a predictive model rather than estimate the pooled effect size. A formal meta-analysis was not conducted because the eligible evidence differed substantially in terms of bird genotype, age, management, geography, particle source, PM fraction, co-pollutant mixture, exposure duration, outcome definition, and experimental design. The full-text screening retained direct broiler field evidence, controlled broiler exposure studies, tropical or open-sided housing evidence, and regional PM2.5 source-context studies. Eligibility criteria were applied to the records for full-text assessment, and the final model context set contained 18 peer-reviewed publications. Ten publications were used as primary model anchors and eight were used for context, mechanistic interpretation, or methodological support (Figure 1).

Eligibility Criteria and Data Extraction

Eligible studies were original peer-reviewed publications in English that reported PM2.5, poultry-house particulate matter, smoke, haze, biomass-burning particulate exposure, or directly-relevant housing/exposure data linked to at least one veterinary endpoint,

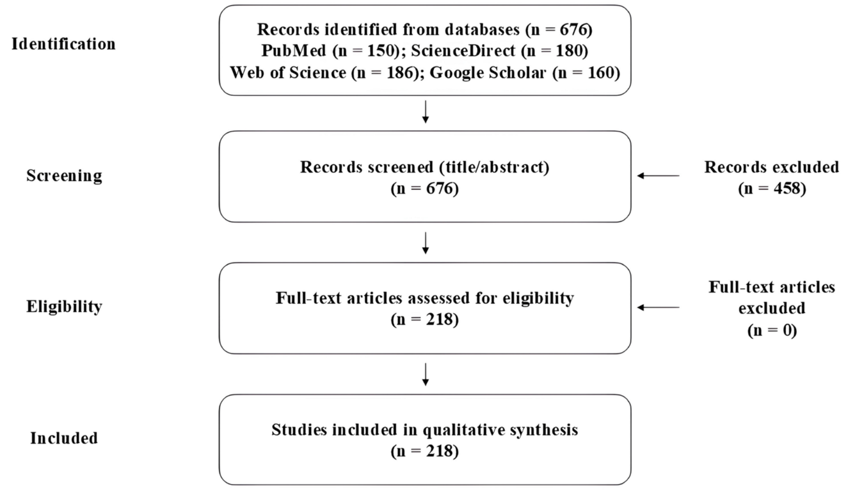

Figure 1. PRISMA 2020 flow diagram of study identification, screening, eligibility assessment, and inclusion for parameterizing the predictive model of PM2.5-related health and production risks in open-sided broilers.

Table 1. Evidence eligibility criteria used for model parameterization

Component	Inclusion criteria	Exclusion criteria
Population/context	Broiler chickens reared in open-sided, naturally ventilated, tropical, subtropical, or field-relevant poultry systems; closely related avian studies retained only when mechanistically transferable.	Non-broiler or non-avian systems without clear translational value; studies whose housing or exposure context could not inform broiler risk.
Exposure	PM2.5, fine particulate matter, poultry-house particulate matter, smoke exposure, regional haze, biomass-burning-associated PM2.5, agricultural burning, maize stover burning, or transboundary haze.	Studies limited only to gaseous pollutants without particulate exposure data; PM10/TSP-only studies were excluded from quantitative PM2.5 modules unless a PM2.5-equivalent fraction was reported.
Duration	Acute or short-term exposure studies were prioritized. Longer studies were retained to define direction, biological plausibility, and conservative response bounds, not to generate direct three-day coefficients.	Studies without exposure duration or without extractable endpoint direction.
Outcomes	Growth performance, feed efficiency, hematology, respiratory pathology, immune response, microbiota, pulmonary function, or clinically relevant health indicators.	Air-quality monitoring studies without biological or veterinary endpoints, unless used only for exposure or regional context.
Study type	Peer-reviewed journal articles published from 2016 to 2026 and indexed in PubMed, Scopus, or Web of Science.	Conference abstracts, theses, dissertations, grey literature, non-peer-reviewed reports, or non-verifiable references.

including growth performance, feed efficiency, hematology, respiratory function, respiratory pathology, microbiota, immune response, and clinically-relevant health indicators. These reviews were only used for mechanistic support or in regional contexts. Conference abstracts, theses, dissertations, grey literature, and non-peer-reviewed reports were excluded. Studies reporting only gaseous pollutants were excluded unless particulate data were available. Only studies with PM2.5 measurements were used quantitatively in the PM2.5 exposure modules. Studies reporting PM10, total suspended particles (TSP), or other proxies were not converted to PM2.5 unless the original publication provided a PM2.5-equivalent fraction or size-specific relationship. When no defensible conversion was available, those studies were retained only as qualitative or mechanistic evidence of the response direction and endpoint plausibility. This rule was intended to avoid unvalidated PM10/TSP-to-PM2.5 conversions and to prevent hidden uncertainty from being transferred into the Monte Carlo model.

The extracted data included bird type, age, housing system, climate or geographic context, particle source, PM metric, co-pollutants, exposure duration, daily or cumulative exposure profile, biological endpoint, direction of effect, approximate magnitude of effect, and whether the evidence was field-based, controlled exposure, or mechanistic. The exposure duration was explicitly recorded because the present model estimates a 3-d, acute PM2.5. Longer studies were not mathematically converted into a 3-d effect size. Instead, they were used conservatively to define directionality, plausible upper bounds, and biological pathways, whereas the 3-d scenario was generated from daily outdoor PM2.5, as well as the indoor infiltration model.

Evidence Source Classification

For clarity, evidence was classified by the source and modeling role of PM2.5. Poultry-house PM/PM2.5 studies were used to support respiratory, inflammatory, microbiota, metabolic, and production-response directions. Maize stover and agricultural residue burning were used as the closest direct field analogs for smoke-associated hematological and respiratory pathology. Tropical/regional haze studies from Thailand and mainland southeast Asia were used to justify the environmental context, source categories, and transboundary haze relevance, but not to infer direct broiler dose–response coefficients. Open-sided housing studies support the assumption that naturally-ventilated broiler houses have strong indoor/outdoor environmental coupling.

Predictive Modeling Framework

A field-oriented, empirical model was developed to estimate the impact of a 3-d, acute PM2.5 exposure episode on production and health outcomes in open-sided broilers. Daily indoor PM2.5 was first estimated from daily outdoor PM2.5 using a simplified indoor/outdoor ratio approach adapted from established indoor particle infiltration and mass-balance frameworks (Bekierski & Kostyrko, 2021; Wallace & Ott, 2023) and from natural-ventilation studies using paired indoor/outdoor PM2.5 monitoring (Luo *et al.*, 2021). The equation is not proposed as a new mechanistic airflow model; rather, it serves as a field-applied, exposure-translation equation for situations in which outdoor PM2.5 data are available but direct indoor monitoring is absent. The wind direction, distance from the burning source, curtain position, fan operation, and particle deposition were not explicitly simulated, and their effects were represented by uncertainty in the effective indoor/outdoor ratio.

$$C_{\mathrm{in},t} = r_{\mathrm{IO}} \times C_{\mathrm{out},t} \quad (1)$$

where $C_{\mathrm{in},t}$ is the estimated indoor PM2.5 concentration on day t, $C_{\mathrm{out},t}$ is the 24-h mean outdoor PM2.5 concentration on day t, and r_{IO} is the effective indoor/outdoor ratio. In this study, r_{IO} was interpreted as an aggregate field coefficient that combines outdoor PM2.5 infiltration with potential indoor contribution from litter dust, feed dust, feather debris, bird activity, manure-derived particles, and the resuspension of settled particles. Therefore, r_{IO} should not be interpreted as a purely physical penetration coefficient.

The r_{IO} was modeled as r_{IO} ~ Uniform (0.6, 1.2) to obtain an effective indoor/outdoor ratio. Values of $r_{\mathrm{IO}} < 1.0$ represent moderate attenuation from dilution, deposition, or short-term shielding within the home; values of $r_{\mathrm{IO}} > 1.0$ indicate potential amplification of PM2.5 indoors from disturbances caused by litter, birds, feed dust, feather debris, manure-based particles, and resuspension of settled dust. The lower (0.6) and upper bounds (1.2) were selected based on the pragmatic

Table 2. Evidence source classification used for model development

Evidence category	Operational definition	Use in the model	Final reference records used
Poultry-house PM/PM2.5	Particles are generated inside poultry houses from litter, feed, feather debris, manure, bioaerosols, and resuspension.	Production, respiratory, microbiota, and mechanistic response direction.	8
Agricultural burning/maize stover burning	Combustion of crop residues or field wastes; maize stover burning was treated as the closest field analog for broilers.	Hematology and respiratory pathology anchors.	2
Biomass burning	Broader combustion of plant biomass, including crop residues, forest fuels, and vegetation fires.	Regional source context and severe haze plausibility.	3
Transboundary haze	Pollution is transported across administrative or national borders before affecting the farm region.	Tropical/regional PM2.5 context; not used as a direct broiler coefficient.	3
Open-sided/tropical housing	Naturally ventilated broiler houses with low sidewalls, curtains, or partial enclosures and no sealed filtration barrier.	Indoor/outdoor coupling and model applicability.	2
Methodological support	PRISMA-style reporting and probabilistic uncertainty propagation.	Review transparency and Monte Carlo uncertainty propagation.	2

Figure 2. Schematic illustration of haze PM2.5 infiltration into an open-sided broiler house under naturally ventilated tropical conditions. Outdoor haze PM2.5 and indoor litter/feed dust jointly contribute to the estimated indoor PM2.5.

uncertainty range for commercial open-sided poultry housing, which does not have a fully-sealed, mechanical filtration system because of the low sidewall/curtain height. The selected bounds were supported by information indicating that there could be both indoor and outdoor sources of PM2.5 exposure to birds in the home (Wallace & Ott, 2023); that indoor/outdoor ratios and penetration processes are widely used to estimate indoor exposure due to outdoor-generated PM2.5 (Bekierski & Kostyrko, 2021) and that passive, natural ventilation creates a high degree of coupling between outdoor and in-home concentrations of PM2.5 (Luo *et al.*, 2021). Additionally, evidence from poultry-house studies indicates that bird behavior in these environments and how they disturb and resuspend the litter can contribute substantially to the generation of fine particulate matter (Sans *et al.*, 2021; Wang *et al.*, 2023a; Jongbo, 2024).

Response Modules and Diagnostic Definitions

The model included endpoint-specific modules for production performance, hematology, exploratory post-vaccination immune retention, general health impairment, and respiratory pathology. Production performance was expressed as a percentage reduction in ADG, percentage increase in FCR, and LW loss per bird during a 3-d episode. Because available broiler PM2.5 studies do not yet provide stable non-linear estimates for 3-d events, production responses were modeled as bounded linear functions above an indoor PM2.5 threshold of 50 μg/m^3. The literature search identified 676 records; after title and abstract screening, 458 records were excluded and 218 full-text articles were assessed for eligibility. No full-text articles were excluded. Therefore, 218 studies were retained in the final qualitative synthesis and used as the evidence base for model parameterization.

Hematological responses were expressed as bounded percentage changes in hemoglobin and hematocrit because these were the most transferable physiological markers reported during field exposure to maize stover burning (Mahmoud *et al.*, 2025). The postvaccination module was retained only as an exploratory retention function. Here, immune retention refers to the percentage of baseline post-vaccination immune responses assumed to remain during a PM2.5 episode; it does not predict antibody titer, protective threshold, or vaccine failure. Because no episode-matched field study has quantified short-term PM2.5-related vaccine titer decline in broilers, the β_{Vac} coefficient was assigned conservatively based on the mechanistic plausibility regarding PM-induced inflammation, oxidative stress, and immune perturbation. Therefore, the output should be interpreted as a trigger for enhanced serological monitoring, rather than as a diagnostic prediction.

General health impairment and respiratory pathology were modeled using logistic probability functions:

$$P = \frac{1}{1+exp\left[-\left(\alpha+\delta\times\frac{\overline{C}_{in}}{100}\right)\right]} \quad (2)$$

where $\overline{C}_{in}$ is the 3-d mean indoor PM2.5. The event definition for clinically-significant general health impairment was defined as a PM2.5-associated response that justified veterinary attention at the flock level, such as measurable growth loss, abnormal hematological shift, respiratory signs, or systemic pathology. The event definition for clinically-significant respiratory pathology was defined as the presence of lung injury reported in the source studies, including airway obstruction, epithelial injury, pulmonary inflammation, congestion, fibrosis, metaplasia, or histopathological lesions. As the source studies did not use a common lesion grading system, the model was not calibrated to a universal lesion grade. Instead, the logistic modules estimated the probability of a clinically-meaningful pathological event, and the outputs should be interpreted as decision support probabilities rather than formal diagnostic criteria.

Monte Carlo Simulation, Validation, and Sensitivity Analysis

A Monte Carlo simulation was used to propagate uncertainty through the indoor exposure and response modules. Simulations were conducted in Python 3.11 using *NumPy* for random sampling and numerical operations, *pandas* for data handling, *SciPy* for probability functions and rank-correlation analysis, and *Matplotlib* for figure production. A reference run of 1,000,000 iterations was compared with smaller candidate runs. The final analysis used 150,000 iterations because this was the smallest run size that maintained the maximum relative error of the scenario summary statistics below 0.5%. Monte Carlo simulation was selected because it permits uncertainty in exposure, infiltration, and biological response coefficients to be carried through to endpoint predictions, rather than being represented by a single deterministic value (Jiang *et al.*, 2021).

Two 3-d scenarios were evaluated. The moderate, regional haze scenario used outdoor PM2.5 values of 150, 150, and 150 μg/m^3 as 24-h means for days 1–3. The severe maize stover/biomass-burning scenario used outdoor PM2.5 values of 193, 400, and 611 μg/m^3 as 24-h means for days 1–3, reflecting the field range reported during maize stover burning by Mahmoud *et al.* (2025). These inputs define the environmental haze intensity; the biological exposure dose was the modeled indoor PM2.5, concentration after applying r_{IO}. The distance from the burning source was not explicitly included because the model assumes that the outdoor PM2.5, measurement is farm-relevant; therefore, the source distance is implicitly reflected in the measured or assumed outdoor concentration.

A fixed random seed (20260308) was established prior to sampling to ensure reproducibility. This number was a user-defined reproducibility identifier corresponding to the simulation date format and was

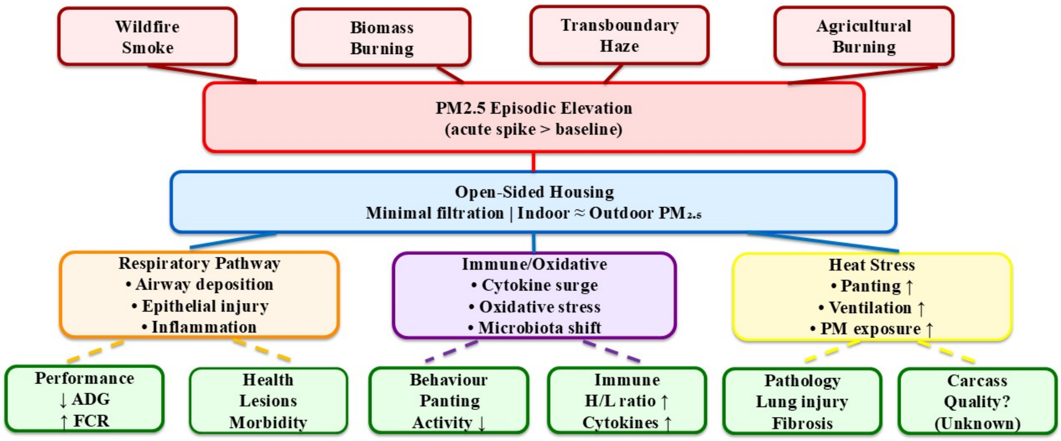

Figure 3. Conceptual framework of haze-related PM2.5 exposure in open-sided broiler housing, integrating outdoor emission sources, indoor infiltration, evidence synthesis, model parameterization, Monte Carlo uncertainty propagation, and field-level endpoint prediction. Color coding denotes exposure sources (red), housing context (blue), mechanistic pathways (orange, purple, and yellow), and outcomes (green).

not generated from the data. Model validation was limited to internal verification because no independent commercial broiler dataset with paired outdoor PM2.5, indoor PM2.5, production records, hematology, vaccine response, or lesion scoring was available. Internal verification included a convergence assessment, range checks against biological anchors, and Spearman's rank correlation sensitivity analysis to identify the most influential uncertain parameters. External validation is explicitly required before the model can be used for regulatory or definitive diagnostic purposes.

RESULTS

Evidence Base for Model Parameterization

The final model context evidence set consisted of 18 peer-reviewed publications published between 2016 and 2026. The set included poultry-house PM/PM2.5 studies addressing respiratory, inflammatory, microbiota, metabolic, and production endpoints (Shen *et al.*, 2023; Wang *et al.*, 2023a; Wang *et al.*, 2023b; Zhou *et al.*, 2023a; Zhou *et al.*, 2023b; Zhou *et al.*, 2024; Chen *et al.*, 2024; Shen *et al.*, 2025), as well as direct field evidence from maize stover burning in broilers (Mahmoud *et al.*, 2025). Tropical and regional source context studies supported the interpretation of biomass burning, agricultural burning, transboundary haze, PM2.5, chemical composition, and microbial components in Northern Thailand and mainland Southeast Asia (Jainontee *et al.*, 2023; Suriyawong *et al.*, 2023; Chantaraprachoom *et al.*, 2024; Inlaung *et al.*, 2024; Chansuebsri *et al.*, 2024; Yabueng *et al.*, 2025). Additional studies support open-sided or naturally-ventilated housing context (Sans *et al.*, 2021; Jongbo, 2024), PRISMA-style reporting, and probabilistic uncertainty propagation (Jiang *et al.*, 2021; Page *et al.*, 2021).

Exposure durations in biological studies ranged from controlled 7–14-d poultry-house particle exposure and 21-d PM2.5 exposure to longer-term PM2.5 inoculation or seasonal field exposure. These durations were longer than the 3-d haze episodes modeled in the present study. Consequently, long-duration studies were not converted into direct 3-d coefficients. Instead, they were used to define directionality, affected endpoint domains, and conservative bounds, whereas the 3-d predictions were generated from daily PM2.5 scenario inputs and bounded response functions.

Production and Physiological Evidence

Controlled broiler trials that were conducted to support this approach have examined how ADG, FCR, LW, and multiple systemic physiological modules may be integrated into the overall model of poultry health. Credible studies (Shen *et al.*, 2023) have shown that exposure of poultry to particulates suspended in the air has negative effects on growth performance because of pulmonary damage, and many have found similar results with metabolic disease. Chen *et al.* (2024) demonstrated that exposing chickens to long-term levels of PM2.5 lowered their growth performance and changed the sero-metabolomic pathway. Although these studies were helpful in establishing the direction and maximum limit of a response, they were not used to conclude that there would necessarily be the same magnitude of effect based on an exact timeframe of 3 d.

Respiratory and Pathological Evidence

Exposure to PM2.5 consistently supports respiratory injury as a biologically plausible outcome. PM2.5, alters the pulmonary microbiota and increases inflammatory signaling in broilers (Zhou *et al.*, 2023a). Microbiota intervention reduced PM2.5-induced lung inflammation, supporting the causal contribution of dysbiosis to respiratory injury (Zhou *et al.*, 2023b). Inhaled PM2.5 also caused disrupted gut microbiota, increased gut-derived lipopolysaccharides, and impaired intestinal barrier markers (Zhou *et al.*, 2024). A recent proteomic study identified dysregulated pulmonary proteins and potential injury pathways after exposure to poultry house particulates (Shen *et al.*, 2025).

Table 3. Parameterization of the multi-endpoint Monte Carlo model

Module	Response expression	Uncertain parameter(s)	Primary evidence anchor
Indoor exposure	$C_{in,t} = r_{IO} \times C_{out,t}$, applied to each 24-h outdoor PM2.5 value during the three-day episode.	r_{IO} ~ Uniform(0.6, 1.2).	Open-sided housing evidence and poultry-house PM2.5 context (Sans *et al*., 2021; Wang *et al*., 2023a; Jongbo, 2024).
Production performance	Bounded reduction in ADG and bounded increase in FCR above 50 µg/m3 indoor PM2.5.	β_{ADG} and λ_{FCR} sampled from uniform ranges.	Shen *et al*. (2023); Chen *et al*. (2024).
Hematology	Bounded percentage change in hemoglobin and hematocrit.	γ_{Hb} and γ_{Hct} sampled from uniform ranges.	Mahmoud *et al*. (2025).
Post-vaccination immunity	Exploratory immune-retention function relative to baseline response; not a titer-prediction model.	β_{Vac} sampled conservatively.	Mechanistic immunotoxic plausibility from PM-induced inflammation and immune disturbance; no direct field coefficient.
General health impairment	Logistic probability of three-day indoor PM2.5 means.	α_H and δ_H sampled from normal ranges.	Integrated clinical interpretation of recent evidence.
Respiratory pathology	Logistic probability of three-day indoor PM2.5 means.	α_R and δ_R sampled from normal ranges.	Mahmoud *et al*. (2025); Zhou *et al*. (2023a, 2023b, 2024); Shen *et al*. (2025).

Table 4. Scenario inputs used in the Monte Carlo simulation

Scenario	Outdoor PM2.5 profile (µg/m³, 24-h mean for day 1, day 2, and day 3)	Interpretation
Moderate three-day regional haze episode	150, 150, 150	Persistent but non-extreme haze event; environmental haze is translated into biological exposure through the indoor/outdoor ratio.
Severe three-day maize stover/ biomass-burning episode	193, 400, 611	Short severe episode reflecting field maize-stover/biomass-burning PM2.5 range reported by Mahmoud *et al*. (2025).

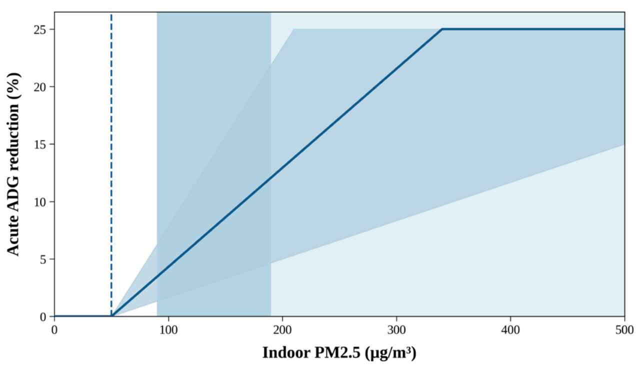

Figure 4. Dose–response relationship between indoor $PM_{2.5}$ concentration and predicted acute reduction in average daily gain (ADG). The solid blue line represents the median prediction, the blue shaded band indicates the 95% uncertainty interval, the darker vertical band denotes a moderate indoor $PM_{2.5}$ episode, and the lighter vertical band denotes an indoor biomass-burning episode

Field Evidence Supporting Clinical Translation

Mahmoud *et al.* (2025) provided the most direct field anchor for clinical translation because broilers were evaluated during the burning of maize stover, a specific form of agricultural residue. The study reported a substantial elevation in PM2.5 carbon monoxide during the burning season, increased hemoglobin and hematocrit levels, decreased pulmonary function, pulmonary fibrosis, pulmonary and hepatic vascular embolism, and other systemic pathological changes. On this basis, the study was used as the principal field anchor for the hematological direction and respiratory pathology probability.

Monte Carlo Predictions

The indoor PM2.5 concentration was 135.0 µg/m³ under a moderate 3-d regional haze scenario with 95% prediction interval of 92.2–177.8 µg/m³. As reported by the model, the median ADG reduction was 5.2%, FCR increased by 2.8%, and LW loss was 9.4 g/bird/episode. Hemoglobin increased by 3.1%, and hematocrit increased by 4.4 %. The immunity measure retained 97.5% of baseline; there was an overall 17.1% probability of health-related impairments and 11.0% probability of respiratory pathologies occurring as a result of exposure. The median response curves and corresponding uncertainty bands constructed for the indoor PM2.5 concentrations are shown in Figure 5. The summary predictions for both moderate and severe scenarios are reported in Table 5.

The maize stover and biomass burning construction within a 3-d severity scenario yielded an indoor PM2.5 concentration of 361.2 µg/m³ with a 95% prediction interval of 246.8–475.5 µg/m³. The median ADG would be reduced by 19.3%, FCR would increase by 9.8%, and LW loss would reach 34.2 g/bird/episode. The median changes in hemoglobin and hematocrit were 11.8% and 16.0%, respectively. Exploratory immune retention decreased to 90.4% of the baseline,

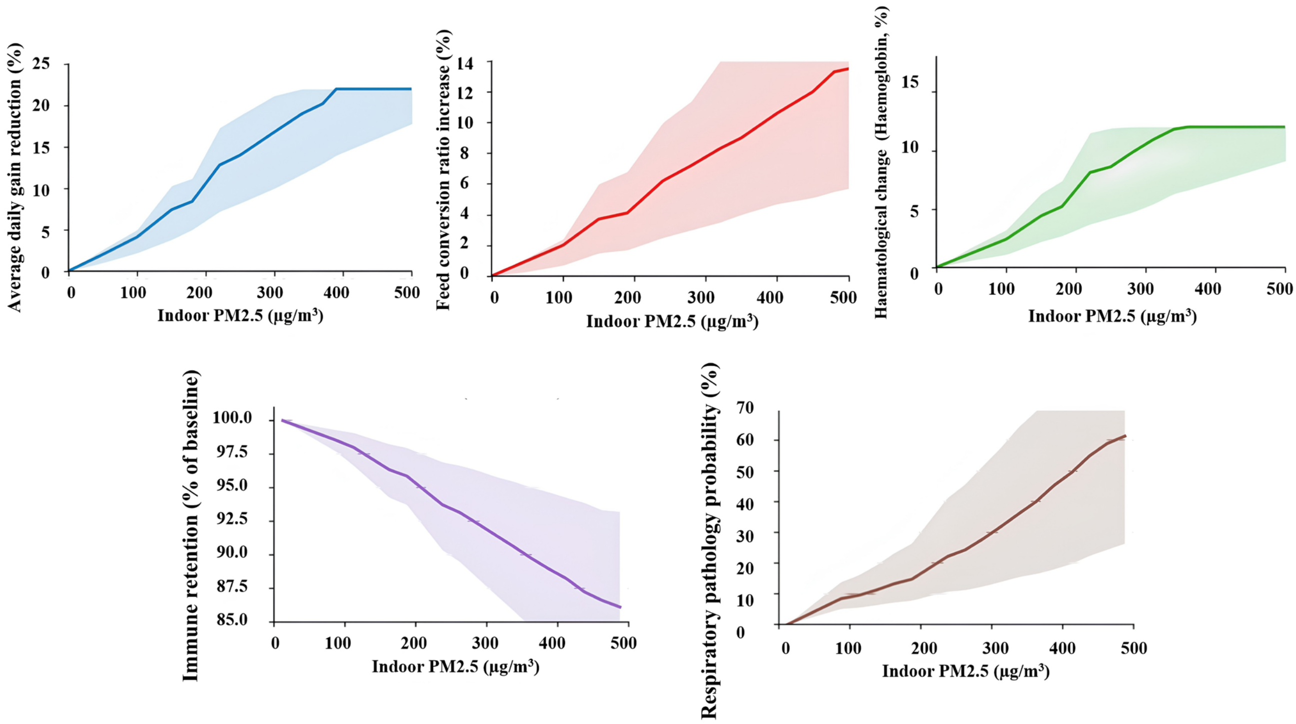

Figure 5. Multi-endpoint response curves showing median and 95% uncertainty bands for average daily gain reduction, feed conversion ratio increase, hematological change, exploratory immune retention, and respiratory pathology probability across indoor PM2.5 concentrations.

Table 5. Multi-endpoint Monte Carlo predictions for three-day PM2.5 episodes

Outcome	Moderate three-day regional haze episode	Severe three-day maize stover/ biomass-burning episode
Indoor PM2.5 mean (μg/m3)	135.0 (92.2-177.8)	361.2 (246.8-475.5)
ADG reduction (%)	5.2 (2.2-10.1)	19.3 (9.9-22.0)
FCR increase (%)	2.8 (1.1-6.2)	9.8 (4.7-15.9)
Liveweight loss (g/bird/episode)	9.4 (4.0-18.6)	34.2 (17.4-44.6)
Hemoglobin change (%)	3.1 (1.2-6.6)	11.8 (5.2-12.0)
Hematocrit change (%)	4.4 (1.8-8.9)	16.0 (7.5-16.1)
Exploratory post-vaccination immune retention (%)	97.5 (94.6-99.1)	90.4 (84.1-94.8)
General health impairment probability (%)	17.1 (8.4-29.7)	46.2 (28.5-64.0)
Respiratory pathology probability (%)	11.0 (5.0-20.4)	40.2 (23.2-57.3)

whereas the probabilities of general health impairment and respiratory pathology increased to 46.2% and 40.2%, respectively. Sensitivity analysis showed that the indoor/outdoor ratio and production-response coefficients contributed most strongly to growth-output uncertainty, whereas the pathology intercept and slope parameters dominated lesion-risk uncertainty. The median predictions and 95% prediction intervals are compared in Figure 6.

DISCUSSION

The present work is intended to be a predictive modeling study rather than a narrative review. By separating the environmental haze event from the biological PM2.5 exposure dose, the model provides a practical way to translate daily outdoor PM2.5 into indoor exposure estimates and then into flock-level production and health risk indicators. These results suggest that a 3-d haze episode may not be biologically-neutral for open-sided broilers. Even the moderate scenario was associated with measurable, predicted reductions in ADG and feed efficiency, whereas the severe maize stover/biomass-burning scenario was associated with clinically-relevant probabilities of general health impairment and respiratory pathology. The results obtained from the present study support those of more recent studies showing particulate matter exposure in broilers can cause lung injury, alter the pulmonary microbiota, induce systemic inflammation, result in dyslipidemia, disrupt the gut-lung axis, and cause structural changes in tissues (Shen *et al.*, 2023; Zhou *et al.*, 2023a; Zhou *et al.*, 2023b; Zhou *et al.*, 2024; Shen *et al.*, 2025).

The hematology module of the model is the most field-driven physiological component because hemoglobin and hematocrit values were directly affected by maize stover exposure during burning

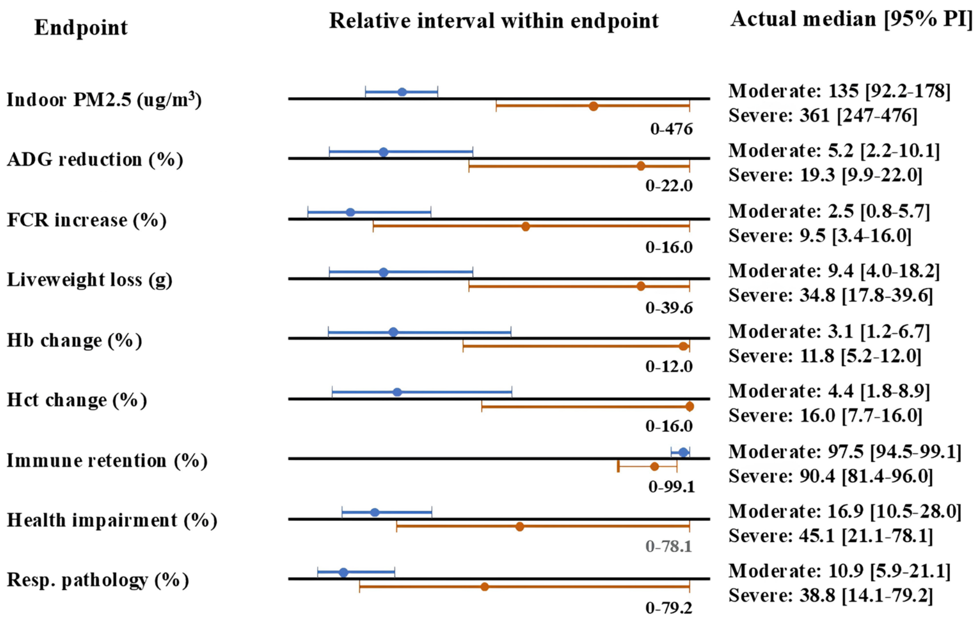

Figure 6. Median values and 95% prediction intervals (PIs) from Monte Carlo simulations for two three-day exposure scenarios across all modeled endpoints: a moderate regional haze episode (blue) and a severe maize stover/biomass-burning episode (red).

(Mahmoud *et al.*, 2025). These variables can function as additional indicators to be interpreted with respiratory signs, production within the flock, hydration, heat load, and concomitant disease during haze events. However, these variables should not be used as standalone diagnostic tools, as hematological values can also be altered by haemocentration, dehydration, thermal strain, and/or systemic diseases.

The immune retention module requires careful interpretation. This was included because particulate matter can promote inflammation, oxidative stress, and immune perturbation, but direct field coefficients linking 3-d PM2.5 episodes to post-vaccination antibody titer decline in broilers are not currently available. Therefore, this module should only be used as a conservative signal for enhanced serological monitoring after vaccination during hazy periods. Therefore, it should not be interpreted as a validated predictor of vaccine failure or protective immunity.

This model is most applicable to tropical or subtropical open-sided broiler systems, in which natural ventilation and partial sidewall structures allow rapid exchange between outdoor and indoor air. Recent housing studies have indicated that sidewall openings, building type, and environmental control can affect broiler performance and indoor climatic conditions (Sans *et al.*, 2021; Jongbo, 2024). Its tropical relevance is supported by regional evidence showing that dry season PM2.5 in northern Thailand and mainland Southeast Asia is strongly influenced by biomass burning, crop residue burning, transboundary haze, and smoke-haze microbial components (Jainontee *et al.*, 2023; Suriyawong *et al.*, 2023; Inlaung *et al.*, 2024; Chansuebsri *et al.*, 2024; Yabueng *et al.*, 2025). However, the model should not be transferred uncritically to sealed, tunnel-ventilated, filtered, or mechanically-controlled houses.

This model had several limitations. The source evidence was heterogeneous with respect to particle source, PM fraction, co-pollutants, bird age, genotype, exposure duration, and endpoint definition. Co-pollutants such as carbon monoxide, ammonia, volatile organic compounds, and microbial components were not modeled separately, although they may modify the biological response. PM10 and TSP studies were not converted to PM2.5, unless an explicit size-fraction relationship was available; this conservative rule reduces conversion bias but also limits the amount of quantitative evidence. The model also estimates only 3-d episodes and does not account for repeated haze episodes over the entire grow-out cycle. External validation using commercial farm data was unavailable. Therefore, the present model should be considered a transparent scenario analysis and veterinary decision-support tool, and not a definitive diagnostic or regulatory model.

Future work should prioritize paired outdoor and indoor PM2.5 monitoring in open-sided broiler farms, farm-level recording of sidewall/curtain status and ventilation conditions, repeated hematological profiling during haze events, standardized respiratory lesion scoring, and post-vaccination serological monitoring. Such datasets would facilitate external validation, recalibration of response coefficients, inclusion of source distances and co-pollutants, and refinement of diagnostic cutoffs for clinically-significant respiratory pathology.

CONCLUSION

The predictive model indicated that a 3-d PM2.5 episode could create biologically relevant production and health risks in open-sided broilers. Moderate regional haze events were predicted to cause modest but measurable reductions in growth and feed efficiency, whereas severe maize stover/biomass-burning events were predicted to produce larger LW losses, greater hematological changes, and substantially higher probabilities of general health impairment and respiratory pathology. In practical terms, the model supports routine outdoor and indoor PM2.5 monitoring during haze periods, closer observation of respiratory signs, temporary mitigation of litter dust and bird disturbance, targeted hematological or serological checks when clinically justified, and early veterinary intervention when PM2.5 concentrations approach severe scenario ranges. This framework is most relevant to tropical open-sided broiler houses. However, external validation is required before farm-specific thresholds are adopted.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

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DECLARATION OF GENERATIVE AI AND AI-ASSISTED TECHNOLOGIES IN THE WRITING PROCESS

The authors used Grammarly (grammarly.com) only for grammar checking and language clarity during manuscript preparation. The authors reviewed and edited the final manuscript and accepted full responsibility for all scientific content, data interpretation, modeling assumptions, and conclusions.

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