



Article

The Study of Allergic Reactions in Mice Induced by Particulate Matter from Duck Houses

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Simple Summary

Particulate matter (PM) in poultry houses is a potential health concern, yet its ability to induce allergic reactions remains uncertain. This study investigated whether PM collected from duck houses could trigger allergic responses in mice. Mice were exposed to ambient and high concentrations of duck house PM, as well as to the dominant fungi isolated from the same environment. The results showed that exposed mice developed allergic symptoms, including sneezing and coughing. Their serum contained elevated levels of allergy markers such as IgE, histamines, and leukotrienes, and their lung tissues showed microscopic lesions with inflammatory cell infiltration. Allergic reactions were more severe with high-concentration PM and fungal exposure. Furthermore, metabolomics analysis revealed distinct metabolic disturbances in the lungs of exposed mice. In conclusion, duck house PM can induce allergic reactions and pulmonary inflammation, with fungal components playing a significant role. These findings highlight potential health risks for individuals exposed to poultry farming environments and support the need for protective measures.

Abstract

Although particulate matter (PM) is strongly associated with allergic reactions, the potential risk of the ability of PM derived from poultry houses to induce allergic reactions remains unclear. This study investigated the effects of duck housing PM on allergic reactions in mice. PM samples and fungi were collected from a duck farm. Ovalbumin (OVA) was used as a positive control, with ambient-level concentrations of PM, high-concentration PM (HPM), and fungal experimental groups. Aerosol exposure was performed on the mice. Serum IgE, allergic mediators (histamines and leukotrienes), cytokines, and pulmonary histopathology were analyzed. Furthermore, HPM-induced metabolic profiles in bronchoalveolar lavage fluid were measured. The results revealed that all the treatment groups of mice presented allergic symptoms, including sneezing and coughing; higher concentrations of IgE, His, and LTs in the serum; upregulation of allergic reaction-related cytokines, such as *IL4*, *IL5*, and *IL33*; and microscopic lesions of the lungs characterized by inflammatory cell infiltration were observed in all the treatment groups, indicating that PM and fungi can cause allergic reactions. Notably, allergic reactions were more pronounced in the HPM and fungal groups



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than in the PM group. In addition, metabolomics analyses revealed that HPM exposure caused metabolic disorders in mouse lungs. The key pathway with the highest correlation to metabolite differences was pyrimidine metabolism, which is associated with allergic reactions. In conclusion, this study demonstrated that exposure to PM in duck houses can cause allergic reactions in mice and significant metabolomic changes in the lungs, especially HPM. Moreover, the contribution of fungal components in the PM cannot be ignored. These findings highlight the potential health risks associated with PM from the poultry industry.

Keywords: duck; particulate matter; allergic reactions; cytokines; lung metabolic profiles

1. Introduction

Air pollution poses a major threat to public health. Particulate matter (PM), as the most harmful component of airborne pollution, is mainly categorized into PM₁₀ (particles with an aerodynamic diameter less than 10 µm) and PM_{2.5} (aerodynamic diameter less than 2.5 µm) [1]. Aerodynamic classification determines how particles move through the air and how they deposit in the respiratory tract. PM₁₀ tends to accumulate in the upper respiratory tract, whereas PM_{2.5} can penetrate deep into the ends of small bronchial tubes and alveoli [2]. It is well known that exposure to high-concentration PM increases the risk of developing and exacerbating respiratory-related diseases. Reference [3] reported a positive correlation between the PM_{2.5} concentration and hospital admissions for respiratory diseases. PM_{2.5} in urban areas contributes to the etiology of chronic obstructive pulmonary disease, which is characterized by small airway remodeling, mucus hypersecretion, and eosinophilic inflammation [4]. In addition, PM exposure can also trigger diseases related to the cardiovascular, immune, and even reproductive systems [5–7]. According to reports from the World Health Organization (WHO), the combined effect of ambient and household PM is associated with 7 million premature deaths annually [8]. Particulate matter (PM) generated in poultry production poses health risks to both animals and workers [9]. Specifically, long-term exposure to such PM has been shown to contribute to increased mortality in poultry and a higher incidence of respiratory diseases among livestock and poultry production workers [10,11].

Allergic reactions are abnormal adaptive immune responses directed against non-infectious environmental substances (allergens) [12]. They can be classified into type I (immediate or IgE-mediated), type II (cytotoxic or IgG/IgM-mediated), type III (immune complex-mediated), and type IV (delayed-type or T-cell-mediated). Among the four types of allergic reactions, type I reactions are the most prevalent [13,14], such as anaphylaxis, allergic rhinitis, some food allergies, and allergic asthma, which are characterized by the involvement of allergen-specific IgE [15]. Epidemiological studies have shown that PM is associated with an increase in visits for asthma and allergic rhinitis [16–18]. Fungi, as important contributors to these biological components and major allergens in PM, play important roles in the development of allergic reactions [19,20]. As one of the earliest identified allergens, indoor and outdoor exposure to fungal spores can trigger respiratory allergies [21]. The common fungi that cause allergies include *Aspergillus fumigatus*, *Alternaria alternata*, and *Penicillium notatum*, which belong to the genera *Aspergillus*, *Alternaria*, and *Penicillium*, respectively [22,23].

The poultry industry has expanded considerably in recent years. During production, high levels of PM from feed, feces, feathers, and dander pose significant threats to human and animal health [24]. Many studies have documented the impact of PM, especially PM_{2.5}, on public health, and exposure to urban PM has been shown to promote the development

of allergic reactions. However, the composition of PM varies across different environments [25]. Atmospheric PM is a complex mixture of inorganic materials, dust, smoke, metal elements, and various liquid and solid substances. In contrast, PM in poultry houses contains more complex biological components, including bacteria, fungi, and viruses [26]. Therefore, the differences in the composition of PM may result in variations in the intensity and manifestation of allergic reactions.

Although there is much research on the harmful effects of poultry house PM on the host's respiratory and immune systems, its potentially harmful effects on allergic reactions remain unclear. In a previous study, we collected PM from duck houses, and a component analysis revealed that it contains a significant number of microorganisms, among which fungi closely associated with allergic reactions account for approximately 15.9%. The predominant fungal species were isolated, including *Alternaria*, *Aspergillus*, and *Fusarium*, which constitute the majority of the fungal composition in PM [27]. Therefore, in this study, PM and the dominant fungi were exposed to mice via inhalation to analyze whether they could induce allergic reactions and the potential mechanisms involved. These results provide scientific evidence for assessing the impact of avian house PM on allergic responses and lay the foundation for further exploration of the underlying mechanisms involved.

2. Materials and Methods

2.1. Ethics Approval and Consent to Participate

The animal experiments were approved by the Animal Care and Use Committee of Shandong Agricultural University and performed according to the committee's guidelines (SDAUA-2023-012).

2.2. Experimental Animals

BALB/c mice were purchased from Ji'nan Pengyue Laboratory Animal Breeding Co., Ltd. (Jinan, China). They were maintained on a 12 h light–dark cycle with controlled temperature (23–25 °C) and humidity (40–60%). The animals were allowed free access to tap water and food. At the beginning of the experiments, the mice were 4 weeks old and weighed approximately 13 g.

2.3. PM Collection and Processing

The PM was collected on glass fiber filter membranes following a previously described method [27]. The membranes were carefully cut into 1 cm² sections via sterile scissors and submerged in ultrapure water. The fragments were then disrupted using a JY92-II ultrasonic oscillator (Scientz Biotechnology Co., Ningbo, China) under pulsed conditions of 3 s on and 3 s off for a total duration of 2–3 min. The resulting suspension was filtered through an 800-mesh sterile sieve, and the filtrate was centrifuged at 12,000 rpm for 15 min to collect the precipitate. The pellet was subsequently lyophilized for 12 h at 0.0110 mbar and −50 °C in a freeze dryer (Alpha-1-4-LSCplus, Christ, Hagen, Germany). The dried sediment was weighed and quantitatively resuspended in ultrapure water for subsequent animal exposure experiments.

2.4. The Collection and Isolation of Airborne Fungi

Airborne microorganisms were collected via an international standard ANDERSEN-6 stage impactor [28] at an airflow rate of 28.3 L/min. On the basis of the results of metagenomic sequencing in the duck house [27], we selected *Alternaria*, *Aspergillus*, and *Fusarium* as the target fungi for this experiment. Potato dextrose agar (PDA) was used as the sampling medium for isolating these three fungal species [29]. The impactor was placed at the center of the duck house, with the sampling height set between 0.5 m and 0.7 m,

and the sampling duration was 5 min. The collected samples were cultured at 28 °C under aerobic conditions for 3 to 7 days. Colony growth on PDA medium was monitored, and the total number of airborne fungi was recorded. On the basis of the morphological criteria described in Koneman's diagnostic atlas [30], colonies with the desired morphology were selected and subcultured for further purification. After incubation, fungal colonies were stained with lactic acid phenol cotton blue, mounted on glass slides, and examined under an optical microscope for species identification based on colony and spore characteristics [31].

2.5. Animal Experiments

According to the previous studies [32–34], the commonly used model antigen ovalbumin (OVA) was dissolved in physiological saline (0.9% NaCl) and prepared in a 1% OVA solution as a positive control. Based on the results of metagenomic sequencing and the total fungal concentration in the duck house [27], the target fungi were prepared as a suspension at a final concentration of 10^3 CFU/mL according to the indicated ratio (*Fusarium*: *Aspergillus*: *Alternaria* = 1:4:20). Sixty female BALB/c mice were randomly divided into five groups (n = 12 per group): a mock group, a positive control group, a fungal group, a high-concentration PM (HPM) group, and an ambient-level concentration PM (PM) group.

The mice were placed in an aerosol chamber connected to an aerosol generator TK-3 (Kangjie, Liaoyang, China). This generator is capable of producing aerosols with a diameter of 3.2 μ m at a flow rate of 0.3 mL/min. To simulate the ambient-level concentration of PM in the duck houses ($2.31 \pm 0.87 \mu\text{g}/\text{m}^3$), we set the exposure dose for the PM group to 0.05 mg/mL [27], whereas that for the HPM group was determined to be 7.5 mg/mL. The real-time concentrations of PM and HPM in the exposure chamber were measured via a Lighthouse handheld 3016 portable dust particle counter (North River Instruments Co., Beijing, China), and they were found to be 0.23 ± 0.07 and $37 \pm 2.14 \text{ mg}/\text{m}^3$, respectively. The exposure doses for the OVA group and the fungal group were 10 mg/mL and 1×10^3 CFU/mL, respectively [35].

As shown in Figure 1, sensitization was performed three times on days 3, 6, and 9 of the experiment, followed by four consecutive challenges on days 16, 17, 18, and 19, with each inhalation lasting 60 min. Mice were monitored daily for clinical symptoms after the challenge. According to the evaluation criteria (Table 1) for systemic allergies issued by the China Food and Drug Administration, the symptoms of allergic reactions were observed for 30 min. All the mice were euthanized via intraperitoneal injection of pentobarbital sodium (10 mg/100 g) on day 20. Blood was collected from all the mice prior to euthanasia. The left lungs of six mice in each group were fixed in 4% paraformaldehyde solution for histopathological analysis, and the right lungs were sampled for detection of inflammatory cytokines. In the mock, OVA, and HPM groups, another six mice were euthanized, and bronchoalveolar lavage fluid (BALF) was collected for metabolomics analysis.

2.6. Enzyme-Linked Immunosorbent Assay

The blood samples were centrifuged at 1000 rpm for 5 min to isolate the serum. Then, IgE, histamine (His), and leukotriene (LT) production were detected using the enzyme-linked immunosorbent assay (ELISA) method according to the kits' instructions (Mlbio, Shanghai, China).

2.7. Quantitative Real-Time PCR

Total RNA was extracted from the tissues via an RNAiso Plus kit (Takara, Dalian, China) according to the manufacturer's instructions. The concentration of RNA was measured via an ultraviolet spectrophotometer, and a total of 1 μ g of RNA was reverse transcribed into cDNA via the HiScript II 1st Strand cDNA Synthesis Kit (+gDNA wiper) (Vazyme, Nanjing, China). The relative expression levels of *IL-4*, *IL-5*, *IL-10*, *IL-13*, *IL-33*,

and *IFN- γ* were determined using the quantitative real-time PCR (qPCR) method using AceQ qPCR SYBR Green Master Mix (Vazyme, Nanjing, China) on LightCycler 96 (Roche, Basel, Switzerland). The primers used are shown in Table 2. The reaction volume was 20 μ L, including 10 μ L of SYBR Green mix, 1 μ L of cDNA, 0.4 μ L of each forward (10 μ M) and reverse (10 μ M) primer, and 8.2 μ L of sterile water. The reaction conditions were predenaturation at 95 °C for 5 min, followed by 40 cycles at 95 °C for 10 s and 60 °C for 30 s, and dissociation curve analysis was performed. Each sample was analyzed three times. The relative fold changes in the detected genes were analyzed using the $2^{-\Delta\Delta CT}$ method. *GAPDH* functions as a housekeeping gene to normalize the mRNA expression of targeted genes.

Sensitization

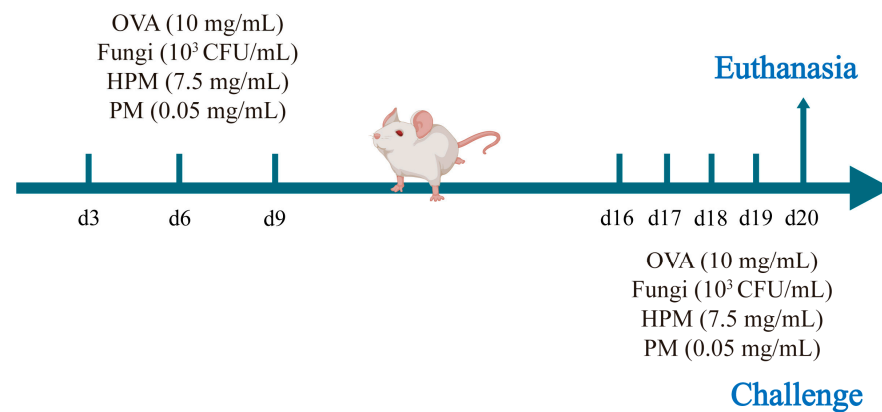


Figure 1. Diagram of the animal experimental design. TK-3 was used to produce aerosols. The exposure dose was 9 mL of suspension for the fungi and OVA groups and 4.5 mL of suspension for the PM and HPM groups. Sensitization was performed on days 3, 6, 9, 16–20 of the experiment.

Table 1. The evaluation criteria for systemic allergy.

Symptoms	Evaluation Criterion
Normal	Negative allergic reaction (–)
Agitation, Piloerection, Trembling, Nasal pruritus	Weakly positive allergic reaction (+)
Sneeze, Cough, Dyspnea, Urination, Defecation, Tearing	Positive allergic reaction (++)
Dyspnea, Wheezing, Purpura, Ataxia, Jumping, Wheezing, Rotation, Cheyne-Stokes respiration	Strongly positive allergic reaction (+++)
Death	Highly positive allergic reaction (++++)

Table 2. The information of qPCR primers used in the study.

Genes	Primer Sequences (5′-3′)	Sizes (bp)
<i>IL-4</i>	F: ACCCAAGACACCCTCAAAC R: CAACAGCACACTCACTCACCT	131
<i>IL-5</i>	F: GGCTTCCTGCTCCTATCTAAC R: CAACCTTCTCTCTCCCAA	120
<i>IL-10</i>	F: TGAAAATAAGAGCAAGGCAGT R: GTCCAGCAGACTCAATACACA	174
<i>IL-13</i>	F: GAGCAACATCACACAAGACC R: AATCCAGGGCTACACAGAAC	138
<i>IL-33</i>	F: CCTTCTTCGTCCTTCACAA R: GCTCTCATCTTTCTCTCCA	122
<i>IFN-γ</i>	F: AGGTCAACAACCCACAGGT R: AATCAGCAGCGACTCCTTT	112
<i>GAPDH</i>	F: AGGTCGGTGTGAACGGATTG R: TGTAGACCATGTAGTTGAGGTCA	129

2.8. Histopathological Examination

The fixed tissues were embedded in paraffin, and 5 µm thick sections were prepared and stained with hematoxylin–eosin (HE). The pathological changes in the lungs were observed under an optical microscope (Eclipse, Nikon, Tokyo, Japan). Lung injury was scored according to the degree of inflammatory cell infiltration, alveolar interstitial thickening, and hemorrhage of the lungs. The scores ranged from 0–4: 0 for normal lungs; 1 for mild lung injury (i.e., less than 25% injury size); 2 for moderate lung injury (i.e., 25–50% injury); 3 for severe lung injury (i.e., 50–75% injury); and 4 for extensive lung injury (i.e., more than 75% injury). The individual scores for each criterion were calculated to determine the final score of the lung lesions.

2.9. Metabolic Profiling Analysis

Four hundred microliters of extraction solvent (MeOH:ACN:H₂O, 2:2:1 *v/v*) was added to 100 µL of BALF and mixed thoroughly. The sample was then subjected to ultrasonic treatment in an ice–water bath for 10 min. Afterward, the sample was incubated at −40 °C for 1 h. The sample was centrifuged at 12,000 rpm for 15 min at 4 °C, and the supernatant was collected. LC–MS/MS analyses were performed via a UHPLC system (Vanquish, Thermo Fisher Scientific, Waltham, MA, USA). The quality control (QC) samples were interspersed throughout the analytical runs to ensure data reproducibility.

The SIMCA-P 13.0 software package (Umetrics, Umea, Sweden) was used for principal component analysis (PCA) and orthogonal partial least squares discriminant analysis (OPLS-DA). Metabolites with $p < 0.05$ (Student's *t* test) and variable importance in projection (VIP) > 1 were considered significant. The Kyoto Encyclopedia of Genes and Genomes (<https://www.genome.jp/kegg/>, accessed on 3 December 2025) was utilized to analyze the metabolic pathways.

2.10. Statistical Analysis

Data processing and analysis were performed by one-way ANOVA with Duncan's method using SPSS 27.0 (IBM, Armonk, NY, USA), and visualization was performed via Prism 10.1.2 (GraphPad, San Diego, CA, USA). All the data are presented as the means ± SDs, and * $p < 0.05$ and ** $p < 0.01$ were considered statistically significant and highly significant, respectively.

3. Results

3.1. Analysis of Systemic Allergic Reaction Symptoms

After challenge on day 16, weakly positive allergic reactions, such as nose scratching and agitation, were observed in all the treatment groups. Moreover, all the mice in the fungal group also exhibited positive allergic reactions, such as sneezing and coughing, compared to 83.3%, 66.6%, and 58.3% of the mice in the OVA, HPM, and PM groups, respectively. No allergic reactions were noted in the mock group. Between days 17 and 19 of the experiment, all the mice in the fungal, HPM, and OVA groups exhibited positive reactions. However, in the PM group, two mice remained asymptomatic throughout the experiment, while only three mice exhibited rhinorrhea on day 18, and the remaining mice displayed mild agitation. Compared with the PM group, the HPM and fungal groups exhibited more severe allergic reactions. The above results demonstrated that exposure to certain concentrations of fungi, HPM, and PM could cause systemic allergic reactions in mice.

3.2. The Production of IgE, His, and LTs in the Serum of the Mice

As shown in Table 3, the levels of IgE, His, and LTs were significantly increased in the mice in the OVA group compared with those in the mock group. Similar trends were also

observed in the fungi, HPM, and PM groups. Notably, the fungi group showed a higher level than those in other treatment groups. These results suggest that the exposure of mice to duck house PM induces significant allergic reactions, with fungal components likely identified as key contributors to these reactions.

Table 3. The contents of serum IgE, His, and LTs in mice.

Groups	IgE (ng·mL ⁻¹)	His (ng·mL ⁻¹)	LTs (ng·mL ⁻¹)
Mock	42.81 ± 4.52	5.40 ± 0.35	251.12 ± 8.47
OVA	73.05 ± 1.44 **	11.72 ± 0.29 **	440.24 ± 12.07 **
HPM	61.46 ± 0.61 **	9.24 ± 0.25 **	313.08 ± 16.08 **
PM	57.85 ± 2.29 **	8.75 ± 0.32 **	340.45 ± 12.14 **
Fungi	86.43 ± 4.29 **	15.29 ± 0.92 **	391.95 ± 11.06 **

** ($p < 0.01$) indicates a significant difference between the treatment group and the Mock group.

3.3. Microscopic Lesions

As shown in Figure 2A, there were no obvious microscopic lesions in the mock group. In contrast, the OVA group exhibited a desquamation of ciliated epithelial cells in the bronchi, along with interstitial widening and congestion (Figure 2B). In the fungal group, desquamation of the bronchial ciliated epithelium was observed (Figure 2C). In the PM group, widening of the alveolar interstitium and mild tissue hemorrhage were observed (Figure 2D). As shown in Figure 2E, the HPM group presented similar but more pronounced pathological changes, including widened alveolar interstitium, congestion, and the presence of red-stained inflammatory exudates within the alveolar spaces. As shown in Figure 2F, the histopathological change scores in the OVA, fungal, and HPM groups were significantly higher than those in the Mock group. Although no significant difference was observed between the PM and Mock groups, the lesion score in the PM group was numerically elevated compared with that in the Mock group. These results indicated that duck house PM exposure caused lung damage and inflammatory responses in mice.

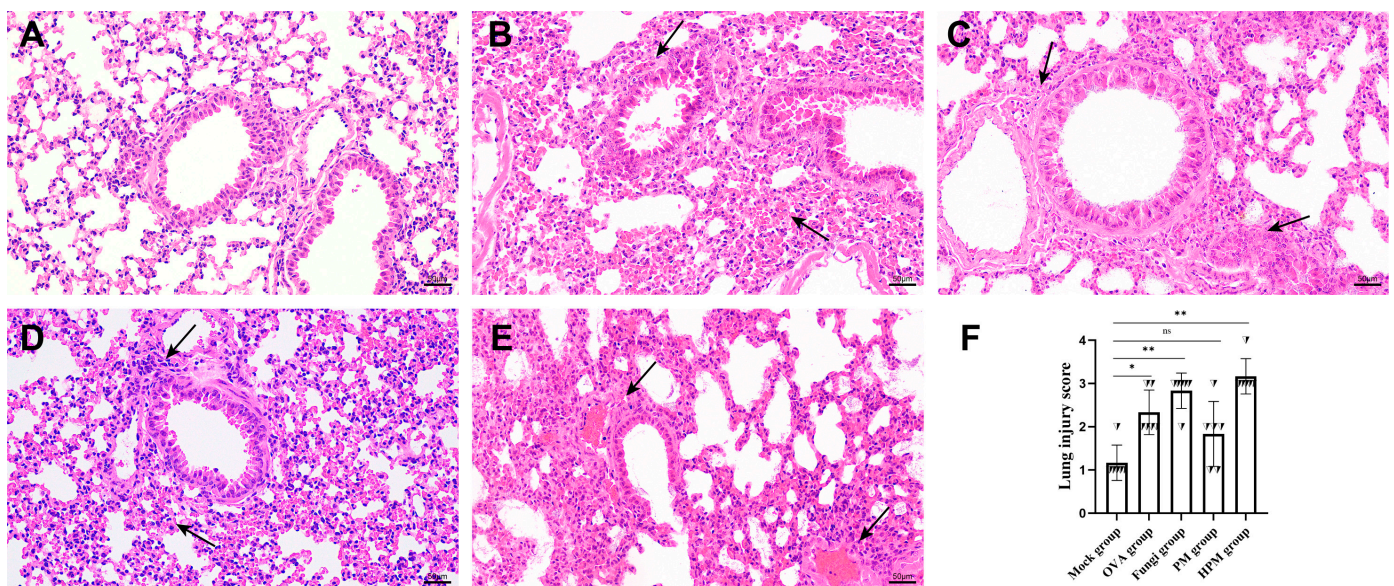


Figure 2. Microscopic lesions of lungs induced by PM from duck houses. (A) Mock group. (B) OVA group. (C) Fungi group. (D) PM group. (E) HPM group. The magnification is 200×. (F) Scores of lungs histopathological changes ($n = 6$). The arrows indicate pathological injury. The triangles represent the experimental mice. * $p < 0.05$, ** $p < 0.01$.

3.4. Analysis of Cytokines Associated with Allergic Reactions in Mice Lungs

To assess the effects of PM and fungal exposure on the expression of inflammatory cytokines, the expression of *IL-4*, *IL-5*, *IL-10*, *IL-13*, *IL-33*, and *IFN- γ* was detected via qPCR. The results are shown in Figure 3. Compared with those in the mock group, all the measured cytokines in the OVA group were significantly elevated ($p < 0.05$ or $p < 0.01$). In the PM group, the levels of *IL-5*, *IL-10*, and *IL-13* ($p < 0.05$) were significantly increased. In the HPM group, all cytokines were significantly upregulated ($p < 0.05$ or $p < 0.01$), but *IFN- γ* was significantly downregulated ($p < 0.05$). In the fungal group, the expression levels of *IL-4*, *IL-5*, *IL-10*, *IL-33*, and *IFN- γ* were highly significantly upregulated ($p < 0.01$).

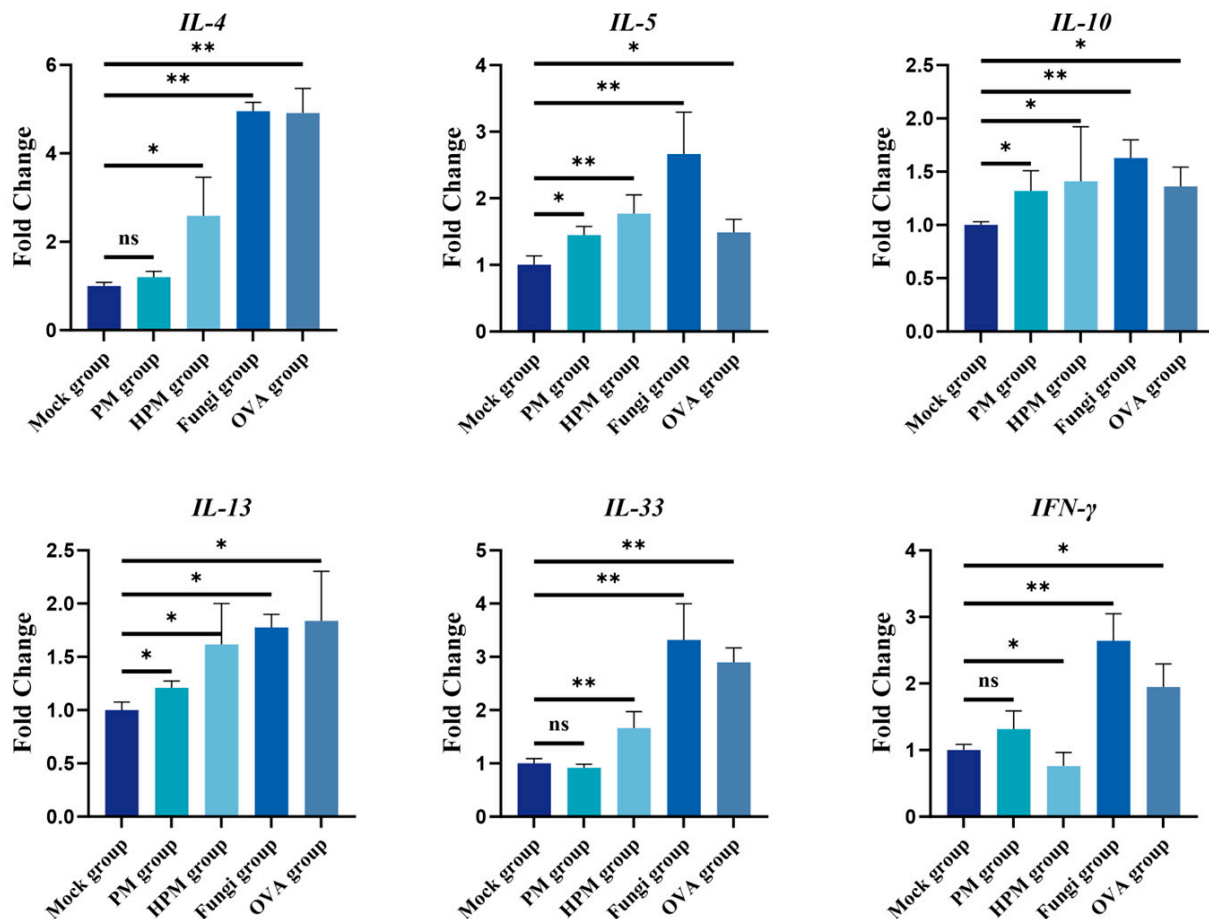


Figure 3. Changes in the expression of lung cytokines in mice. The data are expressed as the means $\pm$ SDs ($n = 3$). * $p < 0.05$, ** $p < 0.01$.

3.5. Metabolomic Analysis of BALF in the Lungs of Mice

To investigate the effects of PM exposure on the metabolic profile of mice, we used LC–MS/MS to analyze metabolite alterations in the OVA and HPM groups. PCA revealed that the LC–MS/MS system was stable, and OPLS–DA revealed significant separation of clusters (Supplementary Figures S1 and S2), indicating a significant difference between the mock group and the OVA group, as well as between the mock group and the HPM group. Comparative metabolomic profiling revealed distinct metabolic perturbations induced by OVA and HPM exposure. As shown in Figure 4A,B, in the mock vs. the OVA groups, 1010 metabolites exhibited significant alterations ($VIP > 1$, $p < 0.05$), with 373 upregulated (e.g., (R)-lipoic acid, beta-D-galactose, pyrophosphate, and carbamoyl phosphate) and 673 downregulated, including prostaglandin D2, arachidic acid, and inosine. In contrast, 526 differential metabolites ($VIP > 1$, $p < 0.05$) were identified in the mock group vs. the HPM group (228 upregulated, 298 downregulated), with notable upregula-

tion of uracil and hypoxanthine and downregulation of arachidic acid and indoxyl sulfate. Hierarchical clustering highlighted both shared and distinct metabolic signatures. Differential metabolites were systematically annotated using the KEGG database to delineate the metabolic pathway perturbations induced by OVA and HPM exposure. Enrichment analysis revealed distinct metabolic signatures between the treatment group and the mock group. As shown in Figure 5A,C, in the OVA group, purine metabolism, ABC transporters, nucleotide metabolism, oxidative phosphorylation, and nitrogen metabolism were significantly enriched. As shown in Figure 5B,D, in contrast, the HPM group exhibited prominent enrichment in nucleotide metabolism, beta-alanine metabolism, and pantothenate/CoA biosynthesis. Through comprehensive analysis of the pathways where the differential metabolites are located (including enrichment analysis and topology analysis), further screening was performed to identify the key pathways with the highest relevance to the metabolite differences. As shown in Figure 6A, the OVA group demonstrated maximal topological significance in the taurine/hypotaurine metabolism pathway ($p < 0.01$). Conversely, as shown in Figure 6B, the HPM group presented the strongest association with the pyrimidine metabolism pathway ($p < 0.01$).

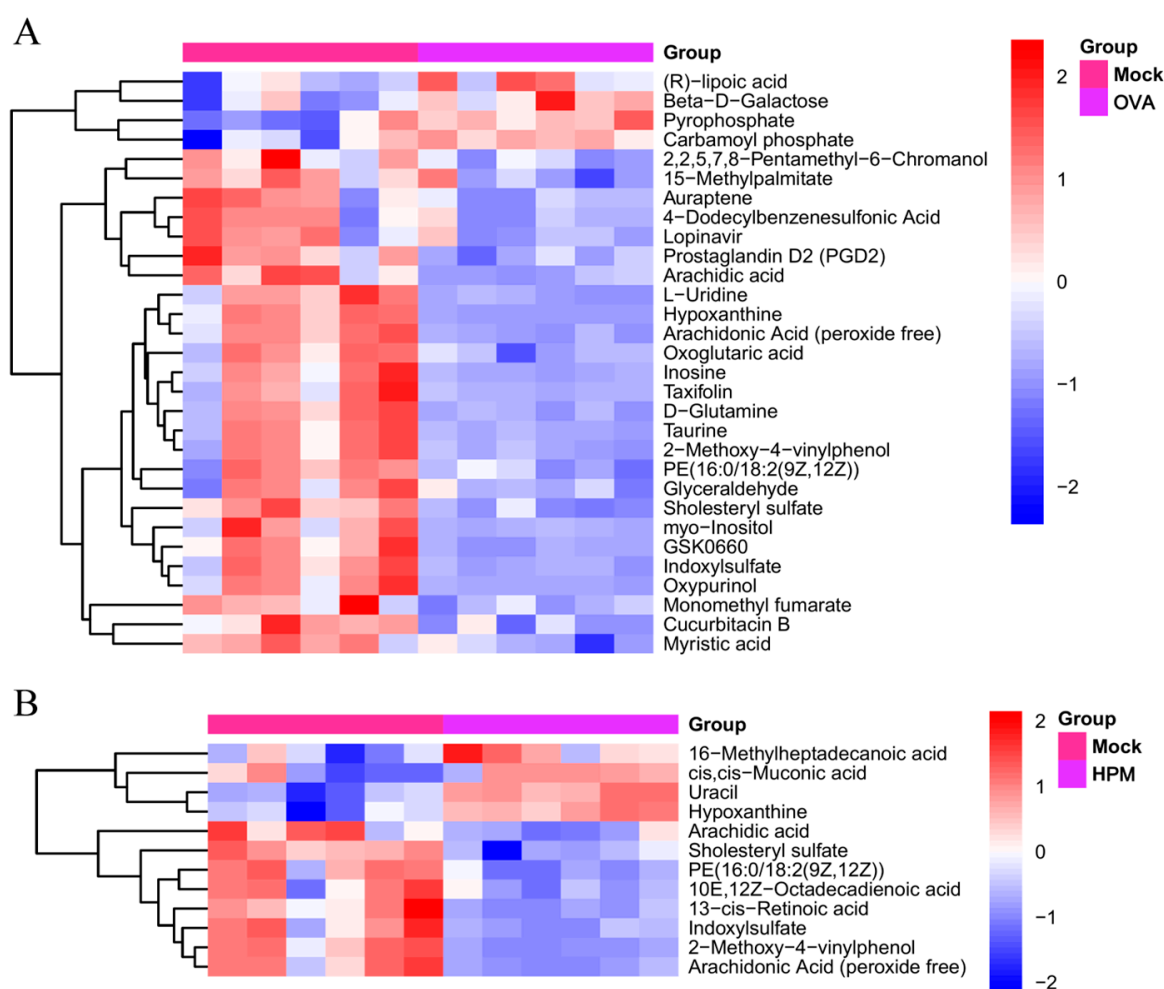


Figure 4. Heatmap of hierarchical clustering analysis results. (A) Mock group vs. OVA group. (B) Mock group vs. HPM group. Red: upregulation; blue: downregulation. (n = 6).

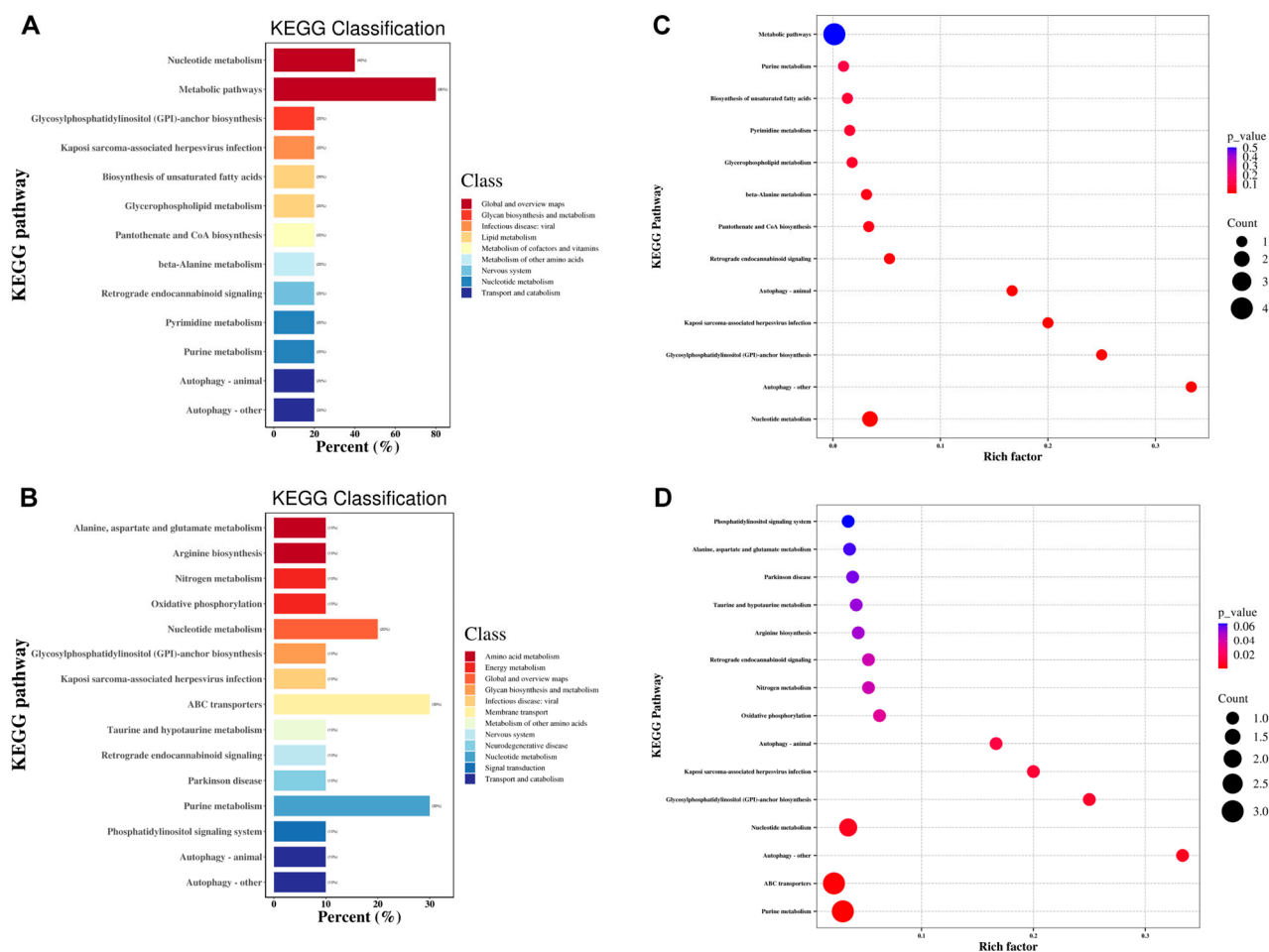


Figure 5. KEGG classification analysis for the (A) mock group vs. OVA group and (B) mock group vs. HPM group. The x-axis represents the percentage of differential metabolites annotated to a specific pathway relative to the total number of annotated differential metabolites, while the y-axis displays the names of the enriched KEGG metabolic pathways. KEGG enrichment analysis for the (C) mock group vs. OVA group and (D) mock group vs. HPM group. The size of the dots indicates the number of differential metabolites enriched in each pathway. The color reflects the p value, with smaller p values corresponding to more red hues, indicating more significant enrichment.

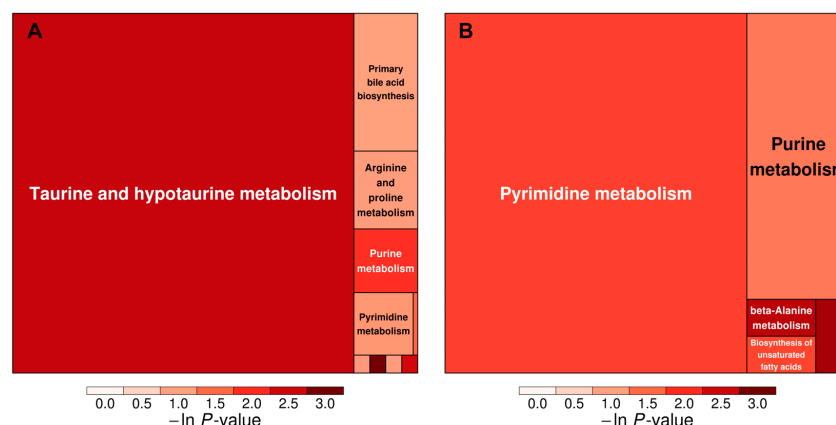


Figure 6. Metabolic pathway analysis. (A) Mock group vs. OVA group. (B) Mock group vs. HPM group. Each square represents a metabolic pathway. The size of the square indicates the pathway's influence factor in the topological analysis, and a larger size denotes a greater influence factor. The color reflects the p value of the enrichment analysis ($-\ln(p)$), with darker colors indicating smaller p values and greater enrichment significance.

4. Discussion

China is a large poultry farming country worldwide. Meat duck farming produces significant quantities of microbial aerosols, some of which contain pathogenic bacteria that pose significant threats to human and poultry health. According to the global air quality guidelines (AQGs) set by the WHO, the short-term (24 h) AQG level for PM_{2.5} is less than 15 µg/m³ [36]. The average PM_{2.5} concentration of the duck houses was $2.31 \pm 0.87 \times 10^2$ µg/m³ [27], far exceeding the short-term exposure limit and was higher than that in chicken houses from the same region [26]. Furthermore, the microbial community composition differed significantly between the two environments. The biological aerosol components in PM_{2.5} from poultry houses contain not only common animal pathogens but also a large number of microbial allergens [27]. In addition, Qu et al. [27] reported that PM in duck houses contains a substantial proportion of fungal components, such as *Alternaria*, accounting for 11.1%. These components were identified as major contributors to pulmonary inflammation and oxidative stress [20]. In this study, we further investigated the impact of duck house PM on allergic reactions. The HPM, PM, and fungal groups of mice exposed to aerosolized duck house PM presented varying degrees of allergic symptoms, including nose scratching and coughing. These allergic reactions are known to trigger pulmonary inflammation through IgE-mediated pathways [37]. Consistent with previous studies [38,39], histopathological examination of the lungs revealed that exposure to duck house PM and fungi caused obvious lung damage characterized by alveolar epithelial hyperplasia, thickening of the alveolar walls, and inflammatory cell infiltration.

To further analyze the effects of PM on allergic responses in mice, we measured the levels of IgE, His, and LTs in the serum. In type I immediate hypersensitivity reactions, IgE bound to FcεRI on mast cells and basophils is cross-linked by allergens, leading to the release of inflammatory mediators and triggering allergic responses. Therefore, IgE is a critical determinant of allergic inflammatory responses and plays a key role in activating effector cells for systemic allergic reactions and other allergic diseases [40]. Reference [41] suggested that the PM could affect mast cell activation by enhancing FcεRI-mediated signaling and releasing active mediators such as His and LTs. In the present study, the serum IgE levels were significantly increased in all the experimental groups, with the fungal group showing the highest IgE levels. The activation of mast cells and basophils during the allergic response leads to the release of a variety of bioactive mediators, including His and LTs [42]. His is capable of inducing vasodilation and increased permeability, leading to allergic reactions such as sneezing and rhinorrhea [43]. LTs can cause airway constriction and inflammatory responses, playing a critical role, especially in diseases such as allergic asthma [44]. Similarly, our findings demonstrated that the PM and its fungal components of duck houses significantly increase the concentration of both mediators. These findings suggest that exposure to duck house PM induces systemic allergic responses in mice, with more pronounced effects observed in the fungal group.

In addition to IgE, His, and LTs, inflammatory cytokines, particularly Th2-type cytokines such as *IL-4*, *IL-5*, *IL-10*, *IL-13*, and *IL-33*, play crucial roles in allergic reactions [45,46]. The upregulation of these Th2 cytokines serves as a molecular hallmark of allergic inflammation and facilitates its progression. PM can aggravate allergic reactions by increasing Th2 inflammatory responses through the activation of NF-κB signaling [47]. The experimental data in the present study demonstrated significant upregulation of most cytokines, including *IL-4*, *IL-5*, *IL-10*, *IL-13*, and *IL-33*, in the treatment groups, similar to the cytokine changes reported in PM-associated allergic inflammation [48–50]. As for *IFN-γ*, a cytokine secreted by Th1 cells, can reduce the production of *IL-4* and IgE, inhibiting allergic reactions [51]. In the fungal and OVA groups, the body may have inhibited Th2 overactivation by upregulating *IFN-γ*, thereby limiting the severity of allergic reactions and

maintaining immune balance. Conversely, the downregulation of *IFN- γ* in the HPM group can be attributed to the dominant activation of Th2 cells [52]. Although HPM-exposed mice exhibit severe allergic reactions, the downregulation of *IFN- γ* may be due to the complex composition of HPM, which hinders the body's ability to mount antiallergic responses effectively. These findings suggest that exposure to duck house PM leads to alterations in the expression levels of cytokines associated with allergic responses. In addition, cytokine upregulation was significantly greater in the fungal group than in the PM and HPM groups, suggesting that the fungal components in the PM from the duck house may be the primary drivers of the allergic reaction.

The metabolomic investigation of BALF from mice exposed to OVA and HPM provided critical insights into the metabolic perturbations associated with allergic responses triggered by PM in duck houses. In the OVA group, the significant enrichment of purine metabolism and oxidative phosphorylation pathways reflects the metabolic demands of immune activation, a hallmark of allergic responses [53]. Notably, taurine, a potent reactive oxygen species (ROS) scavenger [54], was significantly depleted in this study. This depletion is correlated with elevated ROS levels, exacerbating oxidative stress and subsequent ferroptosis, as well as cellular damage [55]. Previous studies have shown that PM exposure induces oxidative stress by increasing ROS levels [56], and PM exacerbates allergic responses by promoting IgE-mediated mast cell activation via the ROS/Gadd45b/JNK signaling axis [57]. In the HPM group, the upregulation of hypoxanthine indicated increased ROS levels and aggravated allergic reactions. The decrease in arachidonic acid in the HPM group suggests its rapid metabolic conversion into proinflammatory lipid mediators, such as LTs. The observed metabolic changes in the HPM group revealed a dual mechanism underlying allergic exacerbation. The upregulation of hypoxanthine directly indicates elevated ROS levels, which are known to aggravate allergic reactions through oxidative stress pathways [58]. The concurrent downregulation of arachidonic acid suggests its accelerated conversion into proinflammatory lipid mediators, particularly LTs [59]. The changes in these two metabolites both led to an increase in the active mediators of allergic reactions, indicating that exposure to PM from duck houses has the potential to induce allergic reactions. In addition, consistent with the reported role of uracil as a potent inducer of His release, the upregulation of uracil in our study suggests that pyrimidine metabolism is involved in allergic responses. Recent studies on SPF chickens exposed to chicken house PM have demonstrated that PM exposure induces lung injury and fibrosis by dysregulating pulmonary metabolic and inflammatory gene expression through the arachidonic acid metabolism pathway, ferroptosis, and the mTOR signaling pathway [60]. This provides a direction for subsequent research in this trial.

5. Conclusions

In summary, this study demonstrated that PM from duck houses can induce allergic reactions, pulmonary inflammation, and perturbations in pulmonary metabolomic profiles in mice. Higher concentrations of PM cause more severe allergic reactions, and fungi in PM may play a significant role in inducing allergic reactions. The results of this study will deepen the understanding of the allergic reactions caused by PM from duck houses and the potential health risks.

Supplementary Materials: The following supporting information can be downloaded at: <https://www.mdpi.com/article/10.3390/vetsci13020142/s1>, Figure S1: Score scatter plot 3D of PCA model with QC.; Figure S2: Score scatter plot and permutation plot test of OPLS-DA model for mock group vs. OVA group (a,c); mock group vs. HPM group (b,d).

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References

1. Bhatnagar, A. Cardiovascular Effects of Particulate Air Pollution. *Annu. Rev. Med.* **2022**, *73*, 393–406. [CrossRef] [PubMed]
2. Zareba, Ł.; Piszczatowska, K.; Dżaman, K.; Soroczynska, K.; Motamedi, P.; Szczepański, M.J.; Ludwig, N. The Relationship between Fine Particle Matter (PM_{2.5}) Exposure and Upper Respiratory Tract Diseases. *J. Pers. Med.* **2024**, *14*, 98. [CrossRef] [PubMed]
3. Ham, D.; Bae, H.J.; Kim, S.; Lim, H.; Choi, J.; Kwon, H.J.; Bae, S. Spatial associations of daily PM (2.5) concentration with cardiovascular and pulmonary morbidity in Korea. *Chemosphere* **2024**, *367*, 143669. [CrossRef]
4. Fang, Z.F.; Wang, Z.N.; Chen, Z.; Peng, Y.; Fu, Y.; Yang, Y.; Han, H.L.; Teng, Y.B.; Zhou, W.; Xu, D.; et al. Fine particulate matter contributes to COPD-like pathophysiology: Experimental evidence from rats exposed to diesel exhaust particles. *Respir. Res.* **2024**, *25*, 14. [CrossRef]
5. Glencross, D.A.; Ho, T.R.; Camiña, N.; Hawrylowicz, C.M.; Pfeffer, P.E. Air pollution and its effects on the immune system. *Free Radic. Biol. Med.* **2020**, *151*, 56–68. [CrossRef]
6. Li, J.; Kong, Y.; Guo, Z.; Qu, L.; Zhang, Z.; Qu, Z.; Wang, H.; Chai, T.; Li, N. Maternal exposure to particulate matter from duck houses restricts fetal growth due to inflammatory damage and oxidative stress. *Ecotoxicol. Environ. Saf.* **2024**, *273*, 116114. [CrossRef]
7. Xu, R.; Huang, S.; Shi, C.; Wang, R.; Liu, T.; Li, Y.; Zheng, Y.; Lv, Z.; Wei, J.; Sun, H.; et al. Extreme Temperature Events, Fine Particulate Matter, and Myocardial Infarction Mortality. *Circulation* **2023**, *148*, 312–323. [CrossRef]
8. World Health Organization Air Pollution. Available online: https://www.who.int/health-topics/air-pollution#tab=tab_2 (accessed on 25 February 2025).
9. Li, Z.; Wang, C.; Li, B.; Shi, Z.; Zheng, W.; Teng, G. Concentration and size distribution of particulate matter in a new aviary system for laying hens in China. *J. Air Waste Manag. Assoc.* **2020**, *70*, 379–392. [CrossRef]
10. Basinas, I.; Sigsgaard, T.; Kromhout, H.; Heederik, D.; Wouters, I.M.; Schlünssen, V. A comprehensive review of levels and determinants of personal exposure to dust and endotoxin in livestock farming. *J. Expo. Sci. Environ. Epidemiol.* **2015**, *25*, 123–137. [CrossRef] [PubMed]
11. Radon, K.; Weber, C.; Iversen, M.; Danuser, B.; Pedersen, S.; Nowak, D. Exposure assessment and lung function in pig and poultry farmers. *Occup. Environ. Med.* **2001**, *58*, 405–410. [CrossRef]
12. Muraro, A.; Fernandez-Rivas, M.; Beyer, K.; Cardona, V.; Clark, A.; Eller, E.; Hourihane, J.O.B.; Jutel, M.; Sheikh, A.; Agache, I.; et al. The urgent need for a harmonized severity scoring system for acute allergic reactions. *Allergy* **2018**, *73*, 1792–1800. [CrossRef] [PubMed]
13. Dispenza, M.C. Classification of hypersensitivity reactions. *Allergy Asthma Proc.* **2019**, *40*, 470–473. [CrossRef] [PubMed]
14. Kumar, S.; Verma, A.K.; Das, M.; Dwivedi, P.D. Molecular mechanisms of IgE mediated food allergy. *Int. Immunopharmacol.* **2012**, *13*, 432–439. [CrossRef]
15. Galli, S.J.; Tsai, M.; Piliponsky, A.M. The development of allergic inflammation. *Nature* **2008**, *454*, 445–454. [CrossRef]
16. Hu, Y.; Xu, Z.; Jiang, F.; Li, S.; Liu, S.; Wu, M.; Yan, C.; Tan, J.; Yu, G.; Hu, Y.; et al. Relative impact of meteorological factors and air pollutants on childhood allergic diseases in Shanghai, China. *Sci. Total Environ.* **2020**, *706*, 135975. [CrossRef]

17. Liu, L.; Liu, C.; Chen, R.; Feng, R.; Zhou, Y.; Wang, L.; Hong, J.; Cao, L.; Lu, Y.; Dong, X.; et al. Associations of ambient air pollution and daily outpatient visits for pediatric atopic dermatitis in Shanghai, China. *Ecotoxicol. Environ. Saf.* **2024**, *286*, 117231. [CrossRef]
18. Villeneuve, P.J.; Chen, L.; Rowe, B.H.; Coates, F. Outdoor air pollution and emergency department visits for asthma among children and adults: A case-crossover study in northern Alberta, Canada. *Environ. Health* **2007**, *6*, 40. [CrossRef]
19. Rick, E.M.; Woolnough, K.; Pashley, C.H.; Wardlaw, A.J. Allergic Fungal Airway Disease. *J. Investig. Allergol. Clin. Immunol.* **2016**, *26*, 344–354. [CrossRef]
20. Wu, B.; Dong, Y.; Wang, M.; Yang, W.; Hu, L.; Zhou, D.; Lv, J.; Chai, T. Pathological damage, immune-related protein expression, and oxidative stress in lungs of BALB/c mice induced by haze PM2.5 biological components exposure. *Atmos. Environ.* **2020**, *223*, 117230. [CrossRef]
21. Cramer, R.; Garbani, M.; Rhyner, C.; Huitema, C. Fungi: The neglected allergenic sources. *Allergy* **2014**, *69*, 176–185. [CrossRef]
22. Fakhimahmadi, A.; Roth-Walter, F.; Hofstetter, G.; Wiederstein, M.; Jensen, S.A.; Berger, M.; Szepannek, N.; Bianchini, R.; Pali-Schöll, I.; Jensen-Jarolim, E.; et al. Mould allergen Alt a 1 spiked with the micronutrient retinoic acid reduces Th2 response and ameliorates *Alternaria* allergy in BALB/c mice. *Allergy* **2024**, *79*, 2144–2156. [CrossRef]
23. Okada, N.; Yamamoto, Y.; Oguma, T.; Tanaka, J.; Tomomatsu, K.; Shiraishi, Y.; Matsuse, H.; Shimoda, T.; Kimura, H.; Watai, K.; et al. Allergic bronchopulmonary aspergillosis with atopic, nonatopic, and sans asthma-Factor analysis. *Allergy* **2023**, *78*, 2933–2943. [CrossRef] [PubMed]
24. Wang, K.; Shen, D.; Dai, P.; Li, C. Particulate matter in poultry house on poultry respiratory disease: A systematic review. *Poult. Sci.* **2023**, *102*, 102556. [CrossRef] [PubMed]
25. Santibáñez-Andrade, M.; Quezada-Maldonado, E.M.; Quintana-Belmares, R.; Morales-Bárceñas, R.; Rosas-Pérez, I.; Amador-Muñoz, O.; Miranda, J.; Sánchez-Pérez, Y.; García-Cuellar, C.M. Sampling, composition, and biological effects of Mexico City airborne particulate matter from multiple periods. *Sci. Total Environ.* **2024**, *926*, 171933. [CrossRef] [PubMed]
26. Wang, X.; Chen, L.; Yang, G.; Cai, Y.; Yu, G. Bacterial and fungal aerosols in poultry houses: PM (2.5) metagenomics via single-molecule real-time sequencing. *Poult. Sci.* **2024**, *103*, 104348. [CrossRef]
27. Qu, Z.; Wang, H.; Li, N.; Guo, Z.; Li, J.; Lv, X.; Cui, Y.; Chai, T. Detection and High-Throughput Microbial Analysis of Particulate Matter in Houses and Downwind Areas of Duck Farms. *Indoor Air* **2024**, *2024*, 7774679. [CrossRef]
28. Andersen, A.A. New sampler for the collection, sizing, and enumeration of viable airborne particles. *J. Bacteriol.* **1958**, *76*, 471–484. [CrossRef]
29. Santona, A.; Mhmoud, N.A.; Siddig, E.E.; Deligios, M.; Fiamma, M.; Paglietti, B.; Bakhiet, S.M.; Rubino, S.; Fahal, A.H. Metagenomic detection of eumycetoma causative agents from households of patients residing in two Sudanese endemic villages in White Nile State. *PLOS Neglected Trop. Dis.* **2022**, *16*, e0010385. [CrossRef]
30. Procop, G.W.; Church, D.L.; Hall, G.S.; Janda, W.M. *Koneman's Color Atlas and Textbook of Diagnostic Microbiology*, 7th ed.; Jones and Bartlett Learning: Burlington, MA, USA, 2017; 1825p.
31. Woudenberg, J.H.; Groenewald, J.Z.; Binder, M.; Crous, P.W. *Alternaria* redefined. *Stud. Mycol.* **2013**, *75*, 171–212. [CrossRef]
32. Cárdenas-Torres, F.I.; Cabrera-Chávez, F.; Arvizu-Flores, A.A.; Flores-Mendoza, L.K.; Lopez-Teros, V.; Astiazaran-Garcia, H.; Gracia-Valenzuela, M.H.; Figueroa-Salcido, O.G.; Arámburo-Gálvez, J.G.; Ontiveros, N. Assessment of the Route of Exposure to Ovalbumin and Cow's Milk Proteins on the Induction of IgE Responses in BALB/c Mice. *Biology* **2022**, *11*, 542. [CrossRef]
33. Casaro, M.; Souza, V.R.; Oliveira, F.A.; Ferreira, C.M. OVA-Induced Allergic Airway Inflammation Mouse Model. *Methods Mol. Biol.* **2019**, *1916*, 297–301.
34. Chen, C.; Sun, N.; Li, Y.; Jia, X. A BALB/c mouse model for assessing the potential allergenicity of proteins: Comparison of allergen dose, sensitization frequency, timepoint and sex. *Food Chem. Toxicol.* **2013**, *62*, 41–47. [CrossRef]
35. Mincham, K.T.; Scott, N.M.; Lauzon-Joset, J.F.; Leffler, J.; Stumbles, P.A.; Holt, P.G.; Strickland, D.H. Early life ovalbumin sensitization and aerosol challenge for the induction of allergic airway inflammation in a BALB/c murine model. *Bio-Protoc* **2019**, *9*, e3181. [CrossRef]
36. World Health Organization. WHO Global Air Quality Guidelines: Particulate Matter (PM2.5 and PM10), Ozone, Nitrogen Dioxide, Sulfur Dioxide and Carbon Monoxide. Available online: <https://www.who.int/publications/i/item/9789240034228> (accessed on 25 February 2025).
37. Reber, L.L.; Hernandez, J.D.; Galli, S.J. The pathophysiology of anaphylaxis. *J. Allergy Clin. Immunol.* **2017**, *140*, 335–348. [CrossRef]
38. Li, Y.; Lin, B.; Hao, D.; Du, Z.; Wang, Q.; Song, Z.; Li, X.; Li, K.; Wang, J.; Zhang, Q.; et al. Short-term PM (2.5) exposure induces transient lung injury and repair. *J. Hazard. Mater.* **2023**, *459*, 132227. [CrossRef] [PubMed]
39. Wu, S.S.; Yin, L.; Han, K.; Jiang, B.; Meng, Q.; Aschner, M.; Li, X.; Chen, R. NAT10 accelerates pulmonary fibrosis through N4-acetylated TGFβ1-initiated epithelial-to-mesenchymal transition upon ambient fine particulate matter exposure. *Environ. Pollut.* **2023**, *322*, 121149.

40. Wang, J.; Zhou, Y.; Zhang, H.; Hu, L.; Liu, J.; Wang, L.; Wang, T.; Zhang, H.; Cong, L.; Wang, Q. Pathogenesis of allergic diseases and implications for therapeutic interventions. *Signal Transduct. Target. Ther.* **2023**, *8*, 138. [[CrossRef](#)] [[PubMed](#)]
41. Jin, Y.; Zhu, M.; Guo, Y.; Foreman, D.; Feng, F.; Duan, G.; Wu, W.; Zhang, W. Fine particulate matter (PM (2.5)) enhances FcεRI-mediated signaling and mast cell function. *Cell. Signal.* **2019**, *57*, 102–109. [[CrossRef](#)] [[PubMed](#)]
42. Akdis, C.A. Allergy and hypersensitivity: Mechanisms of allergic disease. *Curr. Opin. Immunol.* **2006**, *18*, 718–726. [[CrossRef](#)]
43. Lieberman, P. The basics of histamine biology. *Ann. Allergy Asthma Immunol.* **2011**, *106*, S2–S5. [[CrossRef](#)]
44. Jo-Watanabe, A.; Okuno, T.; Yokomizo, T. The Role of Leukotrienes as Potential Therapeutic Targets in Allergic Disorders. *Int. J. Mol. Sci.* **2019**, *20*, 3580. [[CrossRef](#)]
45. Walker, J.A.; McKenzie, A.N.J. T(H)2 cell development and function. *Nat. Rev. Immunol.* **2018**, *18*, 121–133. [[CrossRef](#)]
46. Han, M.; Ma, J.; Ouyang, S.; Wang, Y.; Zheng, T.; Lu, P.; Zheng, Z.; Zhao, W.; Li, H.; Wu, Y.; et al. The kinase p38α functions in dendritic cells to regulate Th2-cell differentiation and allergic inflammation. *Cell. Mol. Immunol.* **2022**, *19*, 805–819. [[CrossRef](#)] [[PubMed](#)]
47. Piao, C.H.; Fan, Y.; Nguyen, T.V.; Song, C.H.; Kim, H.T.; Chai, O.H. PM2.5 exposure regulates Th1/Th2/Th17 cytokine production through NF-κB signaling in combined allergic rhinitis and asthma syndrome. *Int. Immunopharmacol.* **2023**, *119*, 110254. [[CrossRef](#)] [[PubMed](#)]
48. Zhao, J.; Xie, Y.; Qian, C.; Li, L.; Jiang, R.; Kan, H.; Chen, R.; Song, W. Imbalance of Th1 and Th2 cells in cardiac injury induced by ambient fine particles. *Toxicol. Lett.* **2012**, *208*, 225–231. [[CrossRef](#)]
49. Ma, Q.Y.; Huang, D.Y.; Zhang, H.J.; Wang, S.; Chen, X.F. Exposure to particulate matter 2.5 (PM2.5) induced macrophage-dependent inflammation, characterized by increased Th1/Th17 cytokine secretion and cytotoxicity. *Int. Immunopharmacol.* **2017**, *50*, 139–145. [[CrossRef](#)]
50. Zhao, Y.X.; Zhang, H.R.; Yang, X.N.; Zhang, Y.H.; Feng, S.; Yu, F.X.; Yan, X.X. Fine Particulate Matter-Induced Exacerbation of Allergic Asthma via Activation of T-cell Immunoglobulin and Mucin Domain 1. *Chin. Med. J.* **2018**, *131*, 2461–2473. [[CrossRef](#)] [[PubMed](#)]
51. Oboki, K.; Ohno, T.; Saito, H.; Nakae, S. Th17 and allergy. *Allergol. Int.* **2008**, *57*, 121–134. [[CrossRef](#)]
52. Zhang, B.; Zeng, M.; Zhang, Q.; Wang, R.; Jia, J.; Cao, B.; Liu, M.; Guo, P.; Zhang, Y.; Zheng, X.; et al. Ephedrae Herba polysaccharides inhibit the inflammation of ovalbumin induced asthma by regulating Th1/Th2 and Th17/Treg cell immune imbalance. *Mol. Immunol.* **2022**, *152*, 14–26. [[CrossRef](#)]
53. Toskala, E. Immunology. *Int. Forum Allergy Rhinol.* **2014**, *4*, 21380. [[CrossRef](#)]
54. Chesney, R.W.; Han, X.; Patters, A.B. Taurine and the renal system. *J. Biomed. Sci.* **2010**, *17*, S4. [[CrossRef](#)] [[PubMed](#)]
55. Endale, H.T.; Tesfaye, W.; Mengstie, T.A. ROS induced lipid peroxidation and their role in ferroptosis. *Front. Cell Dev. Biol.* **2023**, *11*, 1226044. [[CrossRef](#)] [[PubMed](#)]
56. Liu, K.; Hua, S.; Song, L. PM2.5 Exposure and Asthma Development: The Key Role of Oxidative Stress. *Oxidative Med. Cell Longev.* **2022**, *2022*, 3618806. [[CrossRef](#)]
57. Wang, Y.; Tang, N.; Mao, M.; Zhou, Y.; Wu, Y.; Li, J.; Zhang, W.; Peng, C.; Chen, X.; Li, J. Fine particulate matter (PM2.5) promotes IgE-mediated mast cell activation through ROS/Gadd45b/JNK axis. *J. Dermatol. Sci.* **2021**, *102*, 47–57. [[CrossRef](#)]
58. Bortolotti, M.; Polito, L.; Battelli, M.G.; Bolognesi, A. Xanthine oxidoreductase: One enzyme for multiple physiological tasks. *Redox Biol.* **2021**, *41*, 101882. [[CrossRef](#)]
59. Dahlke, P.; Peltner, L.K.; Jordan, P.M.; Werz, O. Differential impact of 5-lipoxygenase-activating protein antagonists on the biosynthesis of leukotrienes and of specialized pro-resolving mediators. *Front. Pharmacol.* **2023**, *14*, 1219160. [[CrossRef](#)]
60. Wu, B.; Zheng, W.; Bai, Y.; Huang, H.; Chai, T. Integrated analysis of transcriptome and metabolome reveal the complex molecular regulatory network of chicken house PM2.5 induced lung injury. *Res. Vet. Sci.* **2026**, *200*, 106039. [[CrossRef](#)] [[PubMed](#)]

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