บทความปริทัศน์หรือการทบทวนวรรณกรรม (Review Article)

ฝุ่นละอองแขวนลอยในอากาศและสารละอองชีวภาพเชื้อรา: ปัจจัยร่วมการเกิดโรคระบบทางเดินหายใจ

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บทคัดย่อ

บทนำ ฝุ่นละอองแขวนลอยในอากาศเป็นตัวชี้วัดสำคัญของคุณภาพอากาศและเป็นปัจจัยหลักที่ก่อให้เกิดความเจ็บป่วยและการเสียชีวิตจากโรคระบบทางเดินหายใจ สารละอองชีวภาพเชื้อราเป็นองค์ประกอบทางชีวภาพของฝุ่นละอองแขวนลอย ซึ่งมีบทบาทสำคัญต่อมลพิษทางอากาศและภาวะผิดปกติของระบบหายใจ อย่างไรก็ตาม กลไกการปฏิสัมพันธ์ระหว่างฝุ่นละอองแขวนลอยและสารละอองชีวภาพเชื้อรายังไม่เป็นที่เข้าใจอย่างชัดเจน แม้จะมีงานวิจัยที่เพิ่มขึ้นในช่วงหลัง

วัตถุประสงค์ เพื่อสังเคราะห์และนำเสนอองค์ความรู้ปัจจุบันเกี่ยวกับผลกระทบต่อสุขภาพระบบทางเดินหายใจของฝุ่นละอองแขวนลอยในอากาศและสารละอองชีวภาพเชื้อรา

วิธีการศึกษา การทบทวนวรรณกรรมนี้อาศัยการสืบค้นจากฐานข้อมูลวิชาการที่มีความน่าเชื่อถือ เช่น PubMed และ Scopus เพื่อนำผลงานวิจัยที่เกี่ยวข้องมาวิเคราะห์และสังเคราะห์อย่างเป็นระบบ

ผลการศึกษา ฝุ่นละอองแขวนลอยในอากาศและสารละอองชีวภาพเชื้อราเป็นมลพิษมีอุบัติการร่วมกันสูง เนื่องจากคุณสมบัติทางอากาศพลศาสตร์ของอนุภาค ที่ก่อให้เกิดการสะสมในระบบทางเดินหายใจและการแทรกซึมเข้าสู่ถุงลมปอดที่อาจเสริมฤทธิ์กันเมื่อสัมผัสร่างกาย มลพิษทั้งสองสามารถกระตุ้นการเกิดภาวะเครียดออกซิเดชัน การอักเสบของเยื่อบุทางเดินหายใจ และความผิดปกติของระบบภูมิคุ้มกัน ที่มีความสัมพันธ์กับการลดลงของสมรรถภาพของปอดและก่อให้อาการทางระบบทางเดินหายใจร่วมการติดเชื้อที่สามารถก่อให้เกิดโรคมะเร็งได้ ความก้าวหน้าด้านเทคโนโลยีการตรวจวัด การประเมินความเสี่ยง และการจัดการคุณภาพอากาศช่วยสนับสนุนการพัฒนาแนวทางลดการสัมผัสมลพิษอย่างมีประสิทธิภาพ

สรุป ความสัมพันธ์ระหว่างฝุ่นละอองแขวนลอยในอากาศและสารละอองชีวภาพเชื้อรามีความซับซ้อนและมีบทบาทร่วมในการเกิดโรคระบบทางเดินหายใจในหลายระดับ จึงควรส่งเสริมการวิจัยเชิงบูรณาการและการพัฒนาเทคโนโลยีนวัตกรรมเพื่อควบคุม ลดการสัมผัส และป้องกันความเสี่ยงต่อสุขภาพ

คำสำคัญ ฝุ่นละอองแขวนลอยในอากาศ ละอองชีวภาพเชื้อรา โรคระบบทางเดินหายใจ

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Particulate matter and fungal bioaerosols: co-drivers of respiratory tract diseases

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Abstract

Introduction: Particulate matter (PM) is a primary indicator of air quality and a key determinant of respiratory morbidity and mortality. Fungal bioaerosols, the biological fraction of airborne PM, are emerging as significant yet insufficiently characterized contributors to air pollution and respiratory disorders. Although research on their prevalence and health effects has expanded in recent years, the mechanistic interactions between airborne fungal bioaerosols and PM remain poorly understood.

Objective: This systematic review was conducted using reputable databases, such as PubMed and Scopus, followed by comprehensive analysis and synthesis of relevant research findings.

Method: A literature review was conducted using reputable academic databases, including PubMed and Scopus. Relevant studies were systematically analyzed and synthesized.

Results: Airborne PM and fungal bioaerosols are pollutants that frequently co-occur due to their aerodynamic properties, which facilitate deposition in the respiratory tract and penetration into the alveoli, potentially resulting in synergistic effects upon exposure. Both pollutants can induce oxidative stress, inflammation of the respiratory epithelium, and immune system dysfunction, which are associated with reduced lung function, respiratory symptoms accompanied by infections, and may contribute to carcinogenesis. Advances in monitoring technologies, risk assessment, and air quality management further support the development of effective strategies to reduce exposure to these pollutants.

Conclusion: The complex interplay between airborne PM and fungal bioaerosols plays a pivotal role in the pathogenesis of respiratory diseases. A deeper mechanistic understanding and the development of innovative exposure reduction and prevention strategies are essential to mitigate their health impacts.

Keywords: Particulate matter, Fungal bioaerosol, Respiratory tract diseases

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Introduction

Air pollution remains one of the most pressing environmental and public health challenges of the 21st century. According to the World Health Organization (WHO), approximately 99% of the global population lives in areas where ambient pollutant concentrations exceed recommended air quality guidelines, contributing to an estimated 4.2 million premature deaths each year[1]. This burden is disproportionately concentrated in rapidly urbanizing and industrializing regions[2]. Among atmospheric pollutants, particulate matter (PM) serves as both a primary indicator of air quality and a principal mediator of adverse health effects. PM comprises a heterogeneous mixture of solid and liquid particles suspended in the air, capable of deep respiratory penetration and association with pulmonary malignancies[1,3]. The biological fraction of PM includes fungal bioaerosols, which can constitute up to 50% of atmospheric aerosol particles in certain environments, particularly in the indoor air as a result of human activities or ventilation processes[4]. Epidemiological studies have linked inhalation exposure to fungal spores with a range of respiratory disorders, particularly among vulnerable populations[5,6]. Despite increasing evidence of their prevalence and pathogenic potential, the mechanistic interactions between fungal bioaerosols and particulate pollutants remain poorly characterized. While both PM and fungal bioaerosols independently contribute to respiratory morbidity, current literature has yet to adequately address their complex interplay. Therefore, an integrated analytical framework encompassing their physicochemical properties, interactive mechanisms, and synergistic health impacts is warranted to better elucidate their roles in respiratory disease pathogenesis.

Objective

This review aims to synthesize current knowledge regarding the physicochemical characteristics of PM, the compositional diversity and spatial distribution of fungal bioaerosols, and their respective as well as synergistic effects on human respiratory health.

Methodology

A structured literature search was conducted using the keywords “particulate matter,” “fungal bioaerosol,” “treatment,” “prevention,” and “innovation” across the Google Scholar, PubMed, and Scopus databases, covering publications from 2010 to 2025. Inclusion criteria were limited to peer-reviewed scientific studies published in English, while non-English and non-scientific sources were excluded to maintain methodological rigor. Relevant studies were synthesized thematically, and the quality of evidence was appraised using standardized evaluation frameworks to ensure the validity, reliability, and overall relevance of the review findings.

Particulate Matters (PMs): Characteristics and Health Impacts

Air pollution constitutes one of the most critical global environmental and public health challenges, with approximately 99% of the world's population residing in regions where pollutant concentrations exceed health-based air quality standards. The dispersion and accumulation of atmospheric pollutants are strongly influenced by meteorological factors such as wind speed, wind direction, and atmospheric stability, particularly under conditions of thermal inversion. Moreover, large-scale circulation patterns can promote pollutant accumulation in topographically constrained areas, most notably valleys and urban basins where natural dispersion mechanisms are inherently limited[2].

Major pollutants, including fine (<$PM_{2.5}$) and coarse (>PM_{10}) PM, tropospheric ozone (O_3), and nitrogen dioxide (NO_2), represent primary contributors to ambient air quality degradation. Additional contaminants such as polycyclic aromatic hydrocarbons (PAHs) and volatile organic compounds (VOCs) further exacerbate the burden of air pollution[7]. Collectively, these pollutants are implicated in approximately 4.2 million premature deaths per year, primarily due to cerebrovascular, cardiovascular, and pulmonary diseases, encompassing acute and chronic respiratory diseases[1].

PM serves as a principal indicator of air pollution and remains among the most extensively studied atmospheric pollutants[2]. Its chemical composition typically includes sulfates, nitrates, ammonium, sodium chloride, black carbon, mineral dust, and water[1]. In addition to direct health effects, PM influences key atmospheric processes, including cloud condensation, acid deposition, and the formation of secondary pollutants through photochemical reactions. These interactions facilitate the bioaccumulation of pollutants within terrestrial vegetation, soil matrices, and aquatic ecosystems. Furthermore, the prolonged atmospheric persistence of particulate pollutants contributes significantly to reduced visibility and atmospheric haze formation[2].

The health impacts of PM are strongly influenced by particle size. Coarse particles (PM_{10}) are primarily deposited in the upper respiratory tract via sedimentation, particularly in the pharynx, larynx, and trachea, and may also contribute to obstruction in the lower airways. Approximately 20% of larger particles can penetrate beyond the extrathoracic airways, where they are typically cleared through mucociliary mechanisms. Particles that evade clearance may interact with the bronchial epithelium and the muscularis mucosae throughout the bronchial tree, leading to adverse respiratory effects[8].

In contrast, fine particles ($PM_{2.5-10}$) are capable of deeper penetration into the lower respiratory tract, reaching the alveolar regions. Up to 50% of $PM_{2.5}$ can translocate into the systemic circulation and accumulate in lung tissue, potentially penetrating biological barriers through translocation and trans-

synaptic mechanisms[8,9]. Exposure to $PM_{2.5}$ is associated with worsening respiratory symptoms, including reduced lung function, cough, wheezing, and dyspnea[8].

Epidemiological evidence indicates that $PM_{2.5}$ significantly increases both the incidence and prevalence of asthma[10]. The underlying mechanisms are thought to involve the release of molecular mediators that affect cellular, tissue, and systemic responses following exposure, as well as the interaction of pathogenic antibodies with pro-inflammatory cell surface receptors. These processes contribute to the exacerbation of chronic inflammation through activation of signaling pathways such as extracellular signal-regulated kinase (ERK)/protein kinase B (Akt), mitogen-activated protein kinase (MAPK), and signal transducer and activator of transcription-1 (STAT-1)[9].

Bioaerosol-Airborne Particles

Aerosols have attracted increasing scientific attention due to their critical roles in atmospheric processes and expanding technological applications. In this context, growing emphasis has been placed on the characterization of bioaerosols. Depending on their aerodynamic properties, bioaerosols can penetrate the respiratory tract and deposit within pulmonary tissues, thereby inducing infections, allergic responses, toxic effects, asthma, inflammatory disorders, and potentially carcinogenic outcomes[4,11].

Bioaerosols constitute a biologically active fraction of PM, defined as airborne particles containing viable or non-viable microorganisms and their derivatives, including bacteria, viruses, fungi, pollen grains, fungal spores, and microbial fragments such as endotoxins and peptidoglycans[12]. The concentration, diversity, and taxonomic composition of bioaerosols are governed by complex interactions among biological and environmental determinants, including geographic location, atmospheric chemistry, meteorological conditions, anthropogenic activities, and the physicochemical characteristics of local soils and vegetation[13]. The biological fraction of atmospheric aerosols has been estimated to account for up to 50% of total PM, with particle diameters reaching approximately 100 μm[4]. Depending on their aerodynamic properties, these particles can be inhaled and deposited along distinct regions of the respiratory tract from the trachea to the bronchioles, thereby influencing their pathogenic potential.

Fungal spores represent a predominant subset of atmospheric bioaerosols and have been epidemiologically linked to the onset and exacerbation of various respiratory and allergic disorders[12]. A study in Thailand identified dominant fungal genera such as *Penicillium* (14.36%), *Aspergillus*, *Cladosporium*, *Rhizopus*, and *Fusarium*, with pathogenic species accounting for approximately 28% of airborne fungal isolates. These fungi contribute not only to structural material degradation but also to

adverse respiratory health outcomes. Marked spatial and temporal variability in fungal community composition has been reported between indoor and outdoor environments and across different landscape types, often corresponding to fluctuations in PM_{10} and $PM_{2.5}$ concentrations. Intensive incense burning has also been identified as a major source of indoor particulate pollution[14]. Although several studies have documented associations between PM and fungal bioaerosol abundance[15,16], the mechanistic pathways underlying their interactions and potential synergistic health effects remain insufficiently elucidated.

Fungal Bioaerosol: Characteristics and Health Impacts

Airborne fungi are ubiquitously distributed in both indoor and outdoor environments and originate from a wide range of natural and anthropogenic sources and are commonly identified as dominant pathogenic taxa[17]. Fungal concentrations are modulated by local vegetation, human activities, and air pollution levels, while exhibiting limited diurnal or seasonal variation[18]. In Thailand, multiple investigations have characterized fungal bioaerosol distribution across diverse microenvironments, including hospitals[19], recycling facilities[20], ancient temples[14], and subway stations[21]. In the Bangkok metropolitan area, encompassing Bangkok Noi, Chatuchak, Pathum Wan, Phra Khanong, and Lat Krabang, airborne fungal spore concentrations were systematically monitored between May 2012 and April 2013. During this period, mean fungal spore levels frequently exceeded 10^3 spores/m^3, surpassing thresholds recommended by the World Health Organization (WHO) and other international guidelines. Elevated airborne fungal concentrations were positively associated with increased prevalence of allergic rhinitis and other respiratory hypersensitivity disorders[22]. Moreover, Ramírez et al. (2025) reported that the abundance of allergenic and pathogenic fungal bioaerosols is closely associated with elevated ambient particulate matter (PM) levels in Colombia[12]. Their findings identified Aspergillus fumigatus as a predominant species associated with respiratory irritation and pulmonary infections. These observations underscore the potential for synergistic interactions between fungal bioaerosols and particulate matter in modulating adverse respiratory health outcomes, including pulmonary aspergillosis[23].

Interaction Between Fungal Bioaerosol and PMs

Recent investigations have demonstrated the coexistence of fungal-associated PM in ambient air samples[24-26]. The emission of fungal particles into the atmosphere occurs through two principal mechanisms. Passive release is mediated by external physical forces and mechanical disturbance of fungal colonies, e.g., handling, vibration, or air movement, which facilitate the detachment of particles from surfaces. In contrast, active release is driven by intrinsic

biological forces within the fungus, spores or hyphal fragmentation, typically involving the rapid discharge of energy resulting from osmotic pressure differentials and surface tension effects[4]. Airborne fungal particles exhibit a wide aerodynamic size spectrum, encompassing large hyphal fragments and multicellular spores (>10 μm), unicellular spores (1-10 μm), and submicron fragments (<1 μm). This size heterogeneity governs their deposition patterns within the respiratory tract and their atmospheric residence time. The unicellular spores are efficiently removed by the ventilation system filters in the indoor environment, deposit slowly onto upward surfaces, and can be resuspended with moderate efficiency from flooring during human occupancy, in contrast to multicellular spores, which sediment rapidly onto upward surfaces and can be strongly resuspended during human occupancy. This finding indicated aerodynamic behavior and long-range transport potential in the atmospheric system of fungal spores, which are small particles, facilitating into the human lung, e.g., *Aspergillus*, *Cladosporium* and *Penicillium* spores[27].

Although the molecular and physicochemical mechanisms underlying PM-fungal bioaerosol interactions remain poorly elucidated, insights from bacterial aerosol studies suggest that these associations are primarily mediated by electrostatic, hydrophobic, and van der Waals forces between PM surfaces and microbial cell walls. Adsorption energy analyses indicate negative adsorption energy values for PM-bacteria complexes, implying thermodynamically favorable attachment processes[28]. By analogy, similar physicochemical interactions likely facilitate the formation of PM-fungal bioaerosol aggregates, which may enhance atmospheric stability and potentially amplify adverse health effects upon inhalation exposure.

The Effects of PMs and Fungal Bioaerosol on Human Health

The combined health effects of PM and fungal bioaerosols remain incompletely understood; however, the adverse impacts of each pollutant have been well documented. Ambient PM represents a principal component of air pollution, comprising a heterogeneous mixture of solid and liquid particles with diverse physicochemical properties. The mechanisms underlying the adverse physiological effects of PM have not been fully elucidated or systematically integrated[29]. Although inhalation is the primary exposure route, the exact processes through which PM exacerbates respiratory symptoms in patients with chronic airway diseases remain uncertain.

At the cellular level, PM deposition in the airways and centriacinar regions of the lungs can trigger inflammatory airway diseases such as bronchial asthma and chronic obstructive pulmonary disease (COPD), as well as more severe conditions including pulmonary fibrosis and lung cancer. PM exposure induces oxidative damage through the generation of reactive oxygen species

(ROS) and the activation of oxidative stress pathways, resulting in dysregulation of both innate and adaptive immune responses[3] (Figure 1a). PM exposure produces both acute and chronic health effects, particularly in children, whose developing lungs are highly susceptible to environmental insults. Zwozdziak (2016) assessed lung function indices-forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), peak expiratory flow (PEF), and mid-expiratory flows (MEF_{25}, MEF_{50}, MEF_{75})-in relation to short-term indoor PMs exposure. The PM_1 fraction was identified as a more sensitive indicator of acute effects compared with $PM_{2.5}$, and was associated with pronounced impairments in small airway function among schoolchildren[8]. Indoor PM, predominantly consisting of ultraviolet light-absorbing PM (UVPM) and $PM_{2.5}$, was correlated with a 4.1% reduction in the FEV_1/FVC ratio. Specifically, an 11.3 μg/m^3 increase in UVPM corresponded to a 6.4% decrease in predicted FEV_1/FVC and a 14.7% reduction in FEF_{25-75}, while a 17.7 μg/m^3 increase in indoor $PM_{2.5}$ was associated with a 6.1% decrease in FEV_1/FVC and a 12.9% decrease in FEF_{25-75}. In contrast, outdoor PM showed no significant relationship with lung function, suggesting that indoor PM exerts a stronger association with impaired pulmonary performance in asthmatic children[30]. Similarly, elevated concentrations of ambient $PM_{2.5}$ and PM_{10} have been linked to increased respiratory symptoms such as coughing, wheezing, breathlessness, and chest illness among school-aged children in Tanzania[31].

In Thailand, studies have demonstrated an association between exposure to indoor air pollution in child day care centers (DCCs) and respiratory symptoms among children under six years of age across all three seasons. Notably, the frequency of rhinitis was positively correlated with PM_{10} concentrations. Airborne fungal levels were within WHO guideline limits during the summer; however, they exceeded recommended levels in approximately 40% of sites during the rainy season and 70% during the winter. Despite these findings, no significant association was observed between visible indicators of dampness or mold and measured airborne fungal concentrations[32].

Fungal bioaerosols, particularly within indoor environments, contribute to human exposure primarily through inhalation and dermal contact[29]. Humans inhale thousands of fungal spores daily, often in conjunction with other airborne pollutants. Such exposure can elicit immune responses ranging from allergic sensitization to infection and chronic inflammation, manifesting clinically as toxicosis, asthma, hypersensitivity pneumonitis, or invasive fungal diseases including aspergillosis[17,6] (Figure 1b).

Most fungal spores deposit within the extrathoracic airway regions; however, *Aspergillus* and *Cladosporium* species can penetrate deeply into the alveolar zone, causing conditions such as allergic bronchopulmonary aspergillosis (ABPA)

and allergic asthma[33]. These disorders are characterized by dysregulated type 2 immune responses. Genetic polymorphisms in airway epithelial receptors and immune-regulatory pathways can impair clearance of A. *fumigatus*, predisposing individuals to aberrant Th2-mediated inflammation[34].

Pathogen-associated molecular patterns (PAMPs) derived from *Aspergillus* including β-glucan, galactomannan, galactosaminogalactan, and proteases are recognized by pattern recognition receptors (PRRs) such as Dectin-1 and toll-like receptors (TLRs) expressed on lung epithelial and dendritic cells. Fungal proteases disrupt epithelial integrity, triggering the release of alarmins such as interleukin (IL)-33, IL-25, and thymic stromal lymphopoietin (TSLP), which in turn activate type 2 innate lymphoid cells (ILC2s) and $CD4^+$ Th2 lymphocytes. Activated dendritic cells further amplify the response by promoting allergen-specific Th2 differentiation.

Eosinophils play a central role in ABPA pathogenesis: their interaction with *A. fumigatus* induces galectin-10 release and Charcot-Leyden crystal (CLC) formation. Subsequent eosinophil extracellular trap (EET) formation increases mucus viscosity and contributes to bronchial obstruction. The ensuing type 2 cytokine milieu-characterized by IL-4, IL-5, and IL-13 secretion-drives IgE class switching, mast cell degranulation, eosinophil recruitment, and mucus hypersecretion. Collectively, these processes culminate in airway inflammation, mucus plugging, and bronchiectasis, with high-attenuation mucus (HAM) serving as a radiologic hallmark of advanced disease.

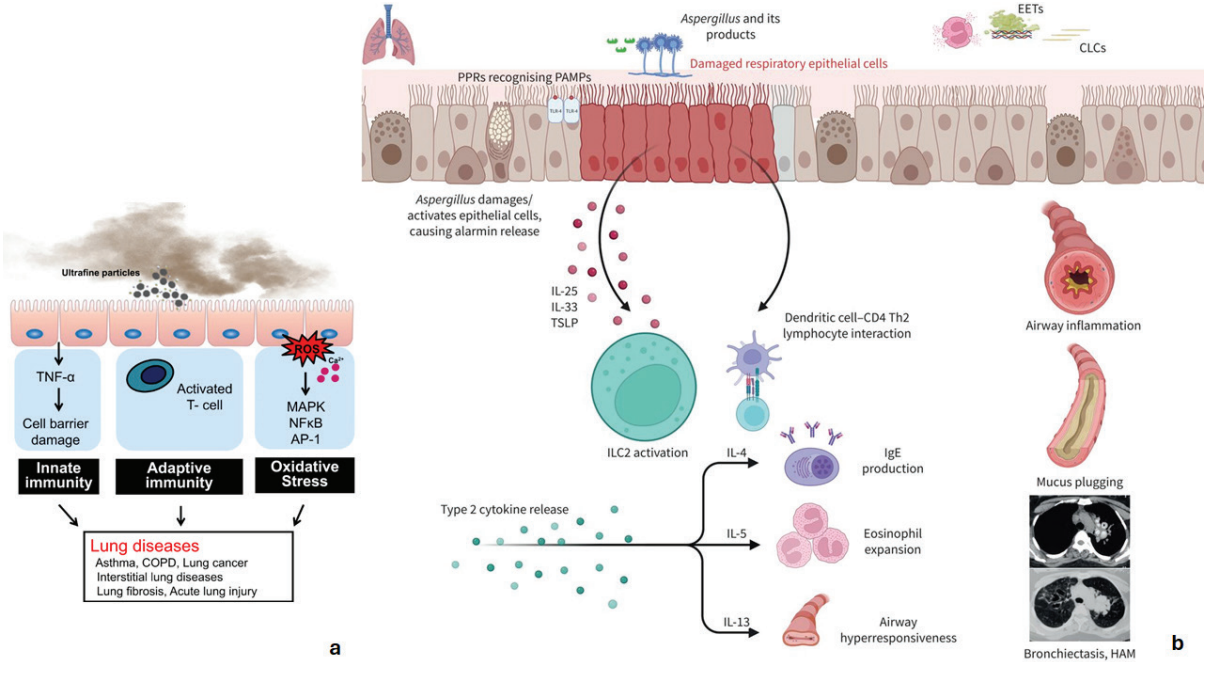


Figure 1 Proposed mechanisms underlying PM-induced lung diseases (a)[3] and the pathogenesis of allergic bronchopulmonary aspergillosis (ABPA) is initiated by *Aspergillus* colonization, which subsequently triggers dysregulated type 2 immune responses (b)[34].

Therapeutic Treatments of Respiratory Diseases.

Exposure to PM and fungal bioaerosols can lead to progressive respiratory damage, culminating in severe outcomes such as lung fibrosis and lung cancer. Common clinical manifestations include coughing, wheezing, breathlessness, and other forms of chest distress[35]. Globally, fungal infections account for approximately one-third of all infectious disease-related mortalities[36]. Managing asthma and allergic rhinitis as a unified airway disease has been shown to improve outcomes in both conditions, as restoration of nasal function enhances the air-conditioning and filtration of inhaled air[37,38]. Common therapeutic agents include antihistamines and corticosteroid nasal sprays (e.g., fluticasone), while chronic obstructive pulmonary disease (COPD) management typically involves long-acting beta-agonists (LABAs), long-acting muscarinic antagonists (LAMAs), and inhaled corticosteroids (ICSs)[38].

In Thailand, the Delphi consensus method has been applied to optimize treatment strategies for allergic rhinitis and urticaria. Second-generation antihistamines are predominantly used due to their reduced sedative properties and minimal anticholinergic side effects. For chronic urticaria, the preferred approach is up-dosing of a single antihistamine rather than combining multiple agents. Orally disintegrating antihistamines are particularly emphasized for their ease of administration and potential to enhance patient adherence[39].

Aspergillus sp. can cause chronic pulmonary aspergillosis, a severe angioinvasive and tissue-invasive condition often requiring lifelong antifungal therapy. The antifungal agents approved by the U.S. Food and Drug Administration (FDA) for aspergillosis and related fungal respiratory diseases include liposomal amphotericin B (AmB), amphotericin B lipid complex (ABLC), voriconazole, itraconazole, posaconazole, isavuconazole, caspofungin, and micafungin. However, azole therapies have notable limitations due to poor tissue penetration, suboptimal drug concentrations at infection sites, low solubility, rapid clearance, and potential toxicities-particularly hepatotoxicity and cardiotoxicity with azoles, and nephrotoxicity with AmB. Combination therapy using terbinafine alongside triazoles has shown synergistic effects, improved treatment efficacy while potentially reducing resistance[40,41].

Innovative Implementation and Prevention of Fungal Bioaerosol and PMs

Indoor air quality (IAQ) represents a critical component of environmental health, as pollutant concentrations indoors can reach up to 100 times higher than outdoor levels. Conventional methods for detecting airborne fungal spores are often costly, labor-intensive, and insufficiently sensitive, thereby limiting their effectiveness for large-scale and continuous environmental monitoring. Recent advancements in micro-nano biosensor systems have provided promising alternatives

for real-time detection and monitoring. These systems integrate miniaturized sensor architectures, advanced nanomaterials, and microfluidic technologies, such as microfluidic biosensors. They are designed to directly sample airborne aerosols into devices capable of capturing exhaled breath condensate, incorporating microfluidic detectors and lab-on-chip cartridges with microfluidic interfaces. This integrated approach enables diagnostic analysis of exhaled breath aerosols with enhanced sensitivity, specificity, and portability[42]. Consequently, such technological innovations hold substantial potential for applications in both indoor and outdoor air quality surveillance.

To mitigate PM and fungal bioaerosol exposure, nanomaterial-based air filtration systems have shown considerable efficacy. For instance, nano-silver/chitosan-titanium dioxide (nano-Ag/CS-TiO_2) composite filters have demonstrated broad-spectrum pollutant reduction capabilities, effectively removing $PM_{2.5}$, PM_{10}, carbon monoxide (CO), carbon dioxide (CO_2), VOCs, and microbial aerosols. Beyond filtration, these systems enable real-time monitoring of air pollutants, facilitating the formulation of evidence-based interventions to sustain healthier indoor environments[43]. Furthermore, a hybrid air purification system incorporating a bioplastic-based photocatalytic film-composed of polylactic acid (PLA) embedded with TiO_2 nanoparticles and silver (Ag/TiO_2/PLA biofilm) has been developed to enhance the degradation efficiency of airborne contaminants. When combined with a photocatalytic oxidation system, this integrated approach significantly improves the removal of PMs and microbial aerosols, underscoring its potential as an advanced strategy for IAQ management[44].

Conclusion

Air pollution remains a pressing global health challenge, with PM serving as a key indicator due to its diverse chemical and biological composition. Fine ($PM_{2.5}$) and coarse (PM_{10}) particles are strongly correlated with adverse cardiovascular, respiratory, and oncological outcomes. Among the biological constituents of PM, fungal bioaerosols represent a major contributor to allergic rhinitis, asthma, hypersensitivity pneumonitis, and invasive fungal infections. Their distribution is influenced by both environmental (meteorological, geographic) and anthropogenic (industrial, urban, and indoor) factors, with particularly high concentrations documented across various settings in urban Thailand.

Emerging evidence highlights interactive dynamics between PM and fungal bioaerosols, where particle size, morphology, and surface properties modulate their deposition in the respiratory tract and their potential to form complex aggregates. These interactions may amplify toxicity through shared pathogenic mechanisms, including oxidative stress, immune dysregulation, and airway inflammation. Vulnerable populations such as children, the elderly, and individuals with chronic respiratory

or immune disorders, are disproportionately affected by these combined exposures.

Therapeutic strategies for allergic respiratory diseases primarily rely on antihistamines and corticosteroid nasal sprays, while fungal infections require targeted antifungal therapies.

In parallel, the development of innovative monitoring and preventive technologies, including nano-engineered filtration and biosensor systems, is increasingly vital for mitigating the health impacts of PM and bioaerosols amid accelerating industrialization and urbanization.

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