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Review Article

Indoor $PM_{2.5}$ and Microbial Contaminants in Developing Countries: Sources, Chemical Interactions, Exposure Pathways, and Health Outcomes

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ABSTRACT

Indoor air pollution has emerged as a critical but under-recognized health burden, particularly in developing countries where households are frequently exposed to unsafe concentrations of fine particulate matter ($PM_{2.5}$) and microbial contaminants. Vulnerable populations, especially women, children, and the elderly, spend most of their time indoors, yet remain unprotected by enforceable standards. This review, conducted under the Preferred Reporting Items for Systematic Reviews and Meta Analyses 2020 framework, systematically examined 102 studies published between 2000 and 2025, synthesizing evidence on pollutant chemistry, microbial ecology, exposure pathways, and health outcomes across residential, industrial-adjacent, and commercial households. Findings reveal that indoor $PM_{2.5}$ in developing countries is enriched with organic carbon, polycyclic aromatic hydrocarbons, transition metals, and secondary aerosols, often exceeding the World Health Organization guidelines by 5–10 folds. Microbial contaminants, including fungi, Gram-negative bacteria, endotoxins, and β-glucans, commonly surpass reference levels set in developed countries, with synergistic $PM_{2.5}$ microbe complexes amplifying oxidative stress, inflammation, and systemic toxicity. Comparative analysis demonstrates stark inequalities, while developed nations enforce indoor air quality standards and adopt advanced monitoring tools (e.g., condensation particle counters, scanning mobility particle sizers, and quantitative polymerase chain reaction), developing countries rely on gravimetric and culture-based approaches, resulting in chronic underestimation of risks. This review emphasizes the urgent need for indoor-specific air quality guidelines, integrated chemical and biological monitoring frameworks, and affordable mitigation technologies tailored for resource-limited settings. Addressing these gaps would not only safeguard health but also reduce economic losses associated with productivity decline and healthcare burden. By reframing indoor pollution as a dual chemical-microbial hazard, this review calls for decisive global and national action to protect vulnerable dwellers and to bridge the inequities in environmental health between developed and developing regions.

Key words: Indoor air pollution, Microbial contaminants, Particulate matter, Women's health

1. INTRODUCTION

Indoor air pollution is a severe and under-recognized public health crisis, particularly in developing countries where populations face chronic exposures yet remain poorly protected by regulations. The World Health Organization (WHO) estimates that approximately 3.2 million pre-mature deaths each year are attributable to household air pollution, with the vast majority occurring in developing regions of Asia and Africa [1]. While outdoor air quality often dominates environmental policy discussions, the indoor environment is in many ways more hazardous. People commonly assume that being inside protects them from environmental pollutants, but mounting evidence shows that indoor concentrations of harmful substances often exceed outdoor levels [2,3]. This false sense of security particularly endangers women, children, and the elderly, who spend the greatest proportion of time indoors and whose health is disproportionately impacted. A key driver of this risk is indoor fine particulate matter ($PM_{2.5}$), a complex pollutant composed of diverse chemical species. In residential households of developing countries, especially those reliant on biomass or kerosene fuels, indoor $PM_{2.5}$ is enriched with organic carbon (OC), elemental carbon (EC), polycyclic aromatic hydrocarbons (PAHs), and volatile organic compounds (VOCs), all of which are mutagenic and carcinogenic [4,5]. Industrial-adjacent households are additionally exposed to transition metals, such as Fe, Cu, Ni, and Pb, which catalyze oxidative reactions that produce reactive oxygen species (ROS), damaging DNA and proteins [6]. Commercial households located above busy markets or roadsides are often contaminated with secondary inorganic aerosols, ultrafine particles, and traffic-related soot, which penetrate deeply into alveolar regions and even translocate into the bloodstream [7]. Recent toxicological and epidemiological studies demonstrate that indoor $PM_{2.5}$ is linked to respiratory infections, asthma, cardiovascular disease, neurodegeneration, and accelerated

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aging, mediated through oxidative stress, systemic inflammation, and epigenetic modifications [8,9]. Alongside chemical pollutants, indoor spaces harbor microbial contaminants, including bacteria, fungi, and microbial fragments, such as endotoxins and β-glucans. Kitchens and bathrooms, with their high humidity, are hotspots for fungal growth (Aspergillus, Cladosporium, Penicillium), while dust-laden living rooms accumulate bacterial spores and Gram-negative bacteria [10,11]. These microbes and their by-products compromise respiratory health by inducing airway inflammation, allergic sensitization, asthma exacerbations, and chronic infections. Studies in Delhi and Lahore households reveal bacterial loads exceeding 1,000 colony-forming unit (CFU)/m^3, well above international reference levels, such as the Hong Kong Indoor Air Quality Objectives (<500 CFU/m^3) [12-15]. The situation is particularly alarming in developing countries, where poor ventilation, damp construction, and sanitation challenges exacerbate microbial proliferation. Emerging research highlights that $PM_{2.5}$ and microbial contaminants act synergistically rather than independently. Carbonaceous particles adsorb microbial fragments, while metal-rich fractions stabilize spores and endotoxins, forming bio-organic complexes that persist in the air and penetrate deeper into the lungs [16,17]. These complexes exhibit enhanced toxicity, triggering more severe oxidative stress, immune activation, and systemic inflammation than either $PM_{2.5}$ or microbes alone [18]. Experimental studies confirm that co-exposures to $PM_{2.5}$ and endotoxins amplify pulmonary injury and immune dysregulation, providing mechanistic insight into why combined indoor pollutants are especially harmful [19]. Despite this evidence, policy frameworks remain inadequate. In developed countries, where indoor pollution levels are relatively lower, governments have enforced indoor air quality standards, building codes, and microbial exposure benchmarks. The United States Environmental Protection Agency (EPA), European Union directives, and Hong Kong's IAQ objectives provide measurable thresholds and monitoring schemes that reduce risks [20,21]. In contrast, developing countries, where indoor exposures are consistently higher, lack enforceable standards and effective governance. India's national $PM_{2.5}$ standard, for example, allows 60 μg/m^3 (24-h average), far more lenient than the WHO's 2021 guideline of 15 μg/m^3, and it does not include microbial pollutants at all [1,22]. This imbalance reflects a troubling paradox: Those who face the greatest health risks receive the least regulatory protection. This review is therefore both urgent and necessary. It integrates the chemistry of indoor $PM_{2.5}$ and the biology of microbial contaminants, framing them not as isolated challenges but as synergistic hazards. By synthesizing evidence from diverse household microenvironments residential, industrial-adjacent, and commercial, it underscores the disproportionate health risks borne by vulnerable dwellers in developing countries and contrasts them with the comparatively safer, better-regulated indoor environments of developed nations. The review also emphasizes the economic dimension of indoor pollution, linking poor air quality with productivity losses, healthcare costs, and poverty cycles. By addressing these dimensions together, this review provides a comprehensive and comparative analysis that is directly relevant to scientists, healthcare providers, and policy-makers, while advocating for indoor-specific standards, affordable monitoring tools, and equitable interventions.

2. MATERIALS AND METHODOLOGY: SYSTEMATIC REVIEW APPROACH

This systematic review was conducted in accordance with the preferred reporting items for systematic reviews and meta-analyses (PRISMA 2020) guidelines [23] to ensure methodological transparency and reproducibility. Studies were considered eligible if they (i) reported indoor concentrations of $PM_{2.5}$ and microbial contaminants (including bacteria, fungi, endotoxins, and β-glucans) in household environments, (ii) focused on residential, industrial adjacent, or commercial household settings, (iii) provided quantitative measurements, chemical or microbial characterization, and associations with health outcomes, and lastly (iv) were published in peer reviewed journals between 2000 and 2025 in the English language. Studies were excluded if they were limited to outdoor air pollution, focused solely on occupational or non-household environments, or lacked direct indoor pollutant measurements. A comprehensive literature search was performed across PubMed, Scopus, Web of Science, and Google Scholar. The core search strategy combined controlled vocabulary and free-text terms as follows: ("indoor air quality" or "household air pollution") and ("$PM_{2.5}$" or "fine particulate matter") and ("microbial" or "bacteria" or "fungi" OR "endotoxin" OR "β-glucan") and ("residential" or "commercial" or "industrial adjacent") and ("developing countries"). Searches were conducted for studies published from January 2000 to March 2025, with the final search completed in March 2025. The initial database search yielded 2,136 records. After removal of duplicates, 1,852 articles remained for title and abstract screening. Of these, 364 full-text articles were assessed for eligibility based on the pre-defined criteria, and 102 studies were ultimately included in the qualitative synthesis. The study selection process is summarized in the PRISMA 2020 flow diagram [Figure 1]. Data extraction was performed systematically, capturing information on geographic location, household microenvironment

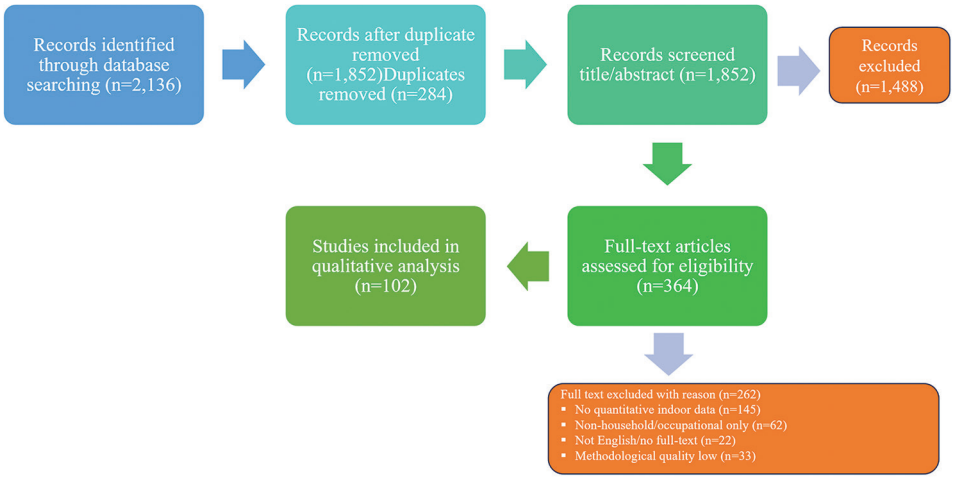

Figure 1: Preferred reporting items for systematic reviews and meta-analyses 2020 flowchart

type, sampling and analytical techniques, pollutant concentrations, chemical and microbial profiles, seasonal variability, and reported health outcomes. Comparative synthesis was undertaken to evaluate patterns across different household settings and between developing and developed urban contexts. Findings were presented through narrative synthesis, comparative tables, and graphical representations where appropriate. To assess methodological quality, each included study was evaluated using an adapted Newcastle Ottawa scale for observational research, focusing on sampling representativeness, exposure measurement reliability, and clarity of outcome reporting. Studies were categorized as high, moderate, or low quality, and quality considerations were incorporated into the interpretation of findings.

3. CHEMISTRY OF HOUSEHOLD INDOOR $PM_{2.5}$

Indoor $PM_{2.5}$ in households of developing countries is chemically complex and results from a combination of direct emissions and subsequent transformations that occur in poorly ventilated microenvironments. The primary constituents are carbonaceous aerosols, where EC and OC dominate due to biomass fuel use, such as wood, dung, and agricultural residues. Within OC, a variety of PAHs are present, many of which are carcinogenic and mutagenic. In addition, households located near industrial facilities or commercial zones accumulate substantial levels of trace metals, including lead, nickel, chromium, and manganese, as well as secondary inorganic ions, such as sulfate, nitrate, and ammonium that contribute to aerosol acidity [24,25]. Beyond these primary inputs, indoor $PM_{2.5}$ undergoes significant secondary chemical transformations. Sulfur dioxide and nitrogen oxides infiltrating from industrial and traffic emissions can react with hydroxyl radicals indoors, leading to the formation of sulfuric and nitric acids that readily condense onto particles, forming sulfate and nitrate aerosols. VOCs, such as terpenes released from cooking oils, formaldehyde from furniture or cleaning agents, and aromatic hydrocarbons from traffic infiltration, undergo oxidation with ozone, NOx, or hydroxyl radicals, producing secondary organic aerosols (SOA). These processes not only increase particle mass but also enrich their toxicity by generating aldehydes and peroxides [26,27]. Indoor $PM_{2.5}$ also acts as a reactive surface. Soot particles can reduce nitrogen dioxide to nitrous acid (HONO), which becomes an important radical precursor indoors, while ozone interactions with organic-coated particles yield oxidized products that can exacerbate respiratory toxicity. Moreover, the porous surfaces of $PM_{2.5}$ adsorb toxic co-pollutants, including PAHs, heavy metals, and microbial fragments, such as endotoxins, thereby functioning as carriers that enhance deep lung deposition [28,29]. The precise composition of household $PM_{2.5}$ varies by microenvironment: Residential households dominated by biomass smoke show higher carbonaceous content, industrial-adjacent households accumulate heavy metals and sulfates, while commercial households-often located above shops or close to markets and traffic-exhibit higher nitrates, VOCs, and PAHs [30]. The interplay of emissions, chemical transformations, and pollutant interactions underscores that indoor $PM_{2.5}$ is not merely a mass concentration but a chemically reactive entity. This evolving chemistry substantially magnifies its toxicity and explains the disproportionately high burden of disease observed in vulnerable populations living in such households.

Figure 2 depicts the major sources and transformation pathways of indoor $PM_{2.5}$ in developing country environments, illustrating how emissions from biomass combustion, industrial activities, and traffic-related sources contribute primary particles enriched with carbonaceous components, PAHs, and metals. It further highlights the secondary formation of inorganic and organic aerosols through oxidation of gaseous precursors (SO_2, NO_x, and VOCs) and subsequent particle aging via surface reactions. Together, these processes generate a chemically complex and highly reactive $PM_{2.5}$ mixture, emphasizing the elevated toxicity of indoor air pollution in poorly ventilated settings.

4. MICROBIAL POLLUTANTS IN HOUSEHOLDS

Indoor air in households across developing countries is a reservoir of diverse microbial pollutants, comprising bacteria, fungi, spores, endotoxins, mycotoxins, and fragments of microbial cell walls. These contaminants arise from everyday household activities and the broader environment. In residential households, damp conditions in kitchens and bathrooms promote the growth of mold species, such as

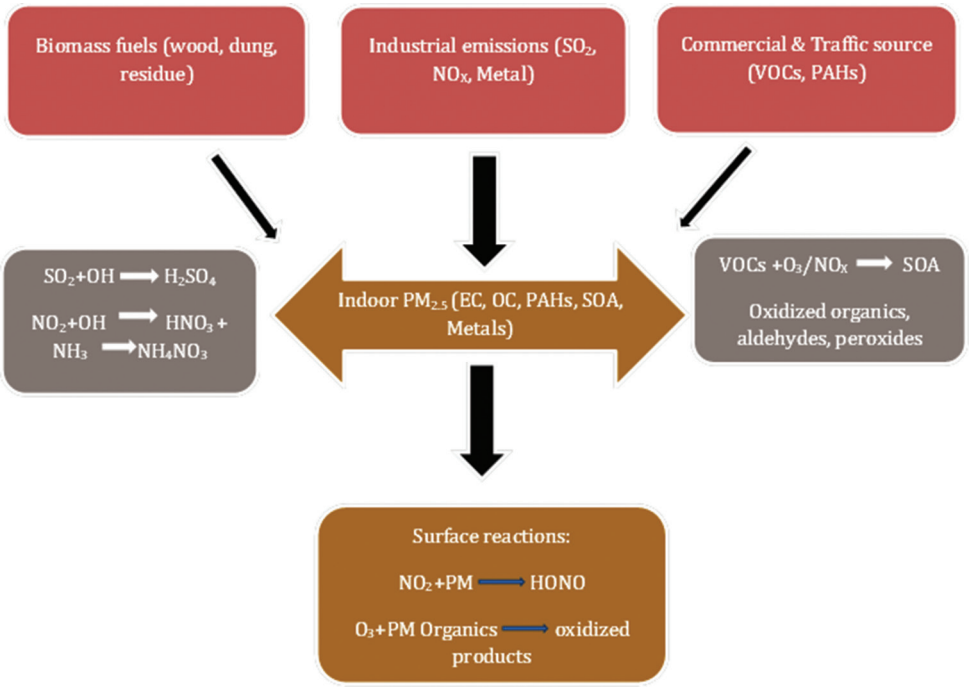

Figure 2: Chemistry of indoor $PM_{2.5}$ in households

Aspergillus, Cladosporium, and Penicillium, while human occupancy contributes bacteria like Staphylococcus and Streptococcus. In households located near industrial zones, spore-forming bacteria, such as Bacillus and actinomycetes are commonly detected, with high levels of endotoxins carried indoors via dust. Commercial households often situated above shops or next to markets and traffic, show increased loads of Gram-negative bacteria, such as *Escherichia coli* and *Pseudomonas*, reflecting their close association with food waste, wastewater leakage, and high human density. Once indoors, these microorganisms do not remain inert; they undergo transformations influenced by temperature, humidity, and ventilation. Fungal spores germinate in damp environments and release secondary metabolites, such as aflatoxins and ochratoxins, which are chemically stable and persist on particulate surfaces. Gram-negative bacteria continuously shed lipopolysaccharides (endotoxins) that remain active in dust and aerosols even after the bacteria are no longer viable. Similarly, fungal cell wall components, such as β-glucans are released during sporulation or decomposition, acting as potent immunomodulators [31-33]. These microbial products are further altered by chemical oxidants present indoors. For example, ozone attacks fungal spores, increasing the release of allergenic proteins, while nitrogen oxides lead to the formation of nitrated proteins with greater inflammatory potential [34]. Thus, microbial pollutants are subject to both biological and chemical transformations that increase their toxicity. The health relevance of these processes is magnified when microbial pollutants interact with fine particulate matter. $PM_{2.5}$, with its porous carbonaceous surface, readily adsorbs spores, endotoxins, and microbial fragments. This not only increases their stability and residence time in the air but also enables deep penetration into the alveoli when inhaled. Once combined with $PM_{2.5}$, microbial residues undergo photooxidation and metal-catalyzed reactions that generate ROS, resulting in amplified oxidative stress and inflammation compared to either pollutant alone. In residential households, this synergy is evident in biomass-fueled kitchens where fungal spores interact with smoke particles. In industrial households, endotoxin-rich spores combine with metal-laden $PM_{2.5}$ infiltrating from factories, while in commercial households, bacteria from food waste or sanitation gaps become attached to traffic-related particles [35]. Overall, microbial pollutants in households across different microenvironments cannot be viewed in isolation from indoor chemistry. They continuously evolve through biological reproduction, chemical reactions, and physical interactions with particulate matter. The resulting bio-chemical complexes are potent triggers of respiratory inflammation, allergies, and chronic disease, with the greatest risks borne by vulnerable groups, such as women, children, and the elderly who spend the most time indoors.

The above Figure 3 shows how microbial contamination varies across household microenvironments, with kitchens and bathrooms acting as major growth hotspots due to high moisture and organic residues, while damp living areas serve as persistent reservoirs where fungi and bacteria proliferate and are repeatedly re-suspended into indoor air. Sleeping rooms accumulate microbes mainly through human shedding and dust retention, leading to prolonged inhalation exposure. Overall, the figure highlights that indoor microbial pollution is unevenly distributed and largely driven by localized moisture conditions and daily household activities.

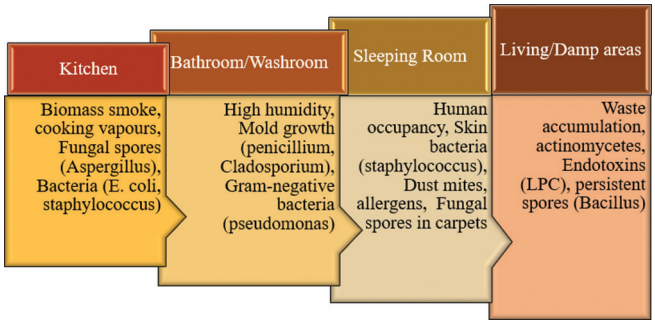

Figure 3: Zones of a household and associated microbial growth (kitchen, bathroom, sleeping room, and damp living areas)

5. HOUSEHOLD MICROENVIRONMENTS: RESIDENTIAL, INDUSTRIAL, AND COMMERCIAL HOUSES

Households represent diverse microenvironments that differ in their sources and composition of pollutants, depending on their spatial context-residential, industrial-adjacent, or commercial. In developing countries, residential households are often characterized by the widespread use of biomass and coal for cooking and heating, resulting in high levels of $PM_{2.5}$ enriched with EC, OC, and PAHs. Poor ventilation and overcrowding exacerbate pollutant concentrations, while high humidity in kitchens and bathrooms fosters microbial growth of Aspergillus, Cladosporium, and Penicillium [36,37]. In contrast, residential households in developed countries rely primarily on cleaner fuels, such as LPG and electricity, with better building insulation and ventilation systems. Here, indoor $PM_{2.5}$ levels are substantially lower, often dominated by secondary inorganic aerosols like sulfates and nitrates, and microbial growth is largely minimized by controlled humidity and HVAC systems. Industrial-adjacent households in developing countries present unique challenges. Families often reside in close proximity to small-scale factories, textile units, or metal workshops. Infiltration of sulfur dioxide, nitrogen oxides, and heavy metals (Pb, Cr, and Ni) into living spaces significantly increases indoor $PM_{2.5}$ toxicity. In addition, these environments show a high prevalence of endotoxins and spore-forming bacteria, such as Bacillus, especially where waste management is inadequate. In developed countries, strict zoning laws and industrial regulations prevent residential settlements near heavy industries. As a result, infiltration of industrial pollutants indoors is minimal, and microbial contamination is controlled through better infrastructure and sanitation. Commercial households, common in urban centers of developing countries, are typically located above shops, near markets, or along heavily trafficked roads. These dwellings are exposed simultaneously to vehicular emissions (EC, OC, PAHs), VOCs from commercial kitchens, and microbial contaminants, such as *E. coli* and *Pseudomonas* originating from poor sanitation and food waste handling. In developed countries, such mixed-use housing is less common due to urban planning and waste management practices. Indoor air in these settings is more strongly influenced by VOCs from consumer products and cleaning agents, with microbial contamination minimized by food safety regulations and hygiene standards [36-39]. The contrast between households in developing and developed countries underscores the critical role of socio-economic status, fuel choices, building infrastructure, and regulatory enforcement in shaping indoor exposures. While chemical and biological pollutants coexist in all household types, the risks are substantially greater in developing countries, where inadequate ventilation, poor sanitation, and high reliance on solid fuels amplify both the chemical toxicity of $PM_{2.5}$ and the biological hazards of microbial pollutants.

Table 1 shows that household microenvironments in developing countries consistently experience much higher and more complex indoor pollution than those in developed regions. Residential homes are dominated by $PM_{2.5}$ from biomass cooking along with moisture-driven bacterial and fungal growth, while industrial adjacent households contain metal and PAHs-rich particles along with dust-associated microbes. Commercial households are influenced mainly by traffic emissions and crowd-related microbial contamination. In comparison,

Table 1: Comparison of $PM_{2.5}$ and microbial contaminants in household microenvironments of developing and developed countries

Microenvironment	Developing countries	Developed countries
Residential houses	$PM_{2.5}$ dominated by biomass smoke (EC, OC, PAHs); microbial load includes molds (Aspergillus, Cladosporium) and bacteria (staphylococcus, streptococcus) [35,36].	Lower $PM_{2.5}$, mainly sulfates and nitrates; microbial contaminants minimized, occasional molds controlled by HVAC and sanitation [37].
Industrial Adjacent Houses	$PM_{2.5}$ enriched with heavy metals (Pb, Cr, Ni) and sulfates; microbial contaminants include endotoxins and spore-forming bacteria (Bacillus, actinomycetes) [38].	Limited infiltration of $PM_{2.5}$ from industry; Low heavy metals indoors; microbial contamination is rare due to strict industrial regulation [40].
Commercial Houses	Elevated $PM_{2.5}$ from vehicular and cooking emissions (EC, OC, VOC-derived PAHs); microbial load includes Gram-negative bacteria (*Escherichia coli, Pseudomonas*) [40]	$PM_{2.5}$ primarily from VOCs and secondary aerosols; microbial contaminants negligible, controlled by food safety and hygiene standards [40].

developed country homes generally maintain lower particulate and microbial levels due to cleaner fuels, better ventilation, and stronger building standards. Overall, the comparison highlights a compounded chemical biological exposure burden in developing countries that is largely mitigated in developed regions through infrastructure and regulation.

6. HEALTH IMPACTS ON VULNERABLE DWELLERS

The health burden of indoor $PM_{2.5}$ and microbial contaminants is most severe among vulnerable groups, particularly women, children, and the elderly. In developing countries, women are disproportionately exposed because of prolonged cooking in biomass-fueled kitchens, where $PM_{2.5}$ concentrations are several times higher than outdoor levels. These fine particles are composed of carbonaceous cores coated with organic compounds, heavy metals, and microbial residues, which make them chemically reactive. When inhaled, they penetrate deep into the lungs and deposit in the alveolar regions, initiating processes that disturb the balance of oxidative and immune defenses. $PM_{2.5}$ surfaces carry a mixture of toxic substances, such as organic compounds, trace metals, and PAHs. Once inhaled, these particles trigger the formation of reactive oxygen molecules that overwhelm antioxidant systems, a condition known as oxidative stress. This process damages cell membranes, proteins, and genetic material. At the same time, microbial contaminants, including bacterial endotoxins and fungal fragments, interact with the immune system by binding to receptors on airway cells, stimulating the release of inflammatory molecules. The combined action of these pollutants leads to persistent airway inflammation, reduced lung function, and heightened susceptibility to infections [41,42]. Children face particularly high risks because of their faster breathing rates, developing respiratory systems, and immature immune defenses. Continuous exposure to $PM_{2.5}$ and microbial fragments during early years has been linked with asthma, recurrent respiratory infections, impaired cognitive development, and slowed growth. For the elderly, who often already suffer from chronic conditions, such as cardiovascular disease or diabetes, additional exposure creates a dangerous feedback loop: Inhaled pollutants increase blood vessel stiffness, disturb heart rhythm, and aggravate existing respiratory illnesses [43]. In households near industrial areas, particulate matter enriched with heavy metals combines with endotoxin-rich microbial dust, creating a double hazard. This mixture not only provokes intense immune responses but also accelerates processes associated with cellular aging, such as chronic inflammation and tissue remodeling. Over time, these changes contribute to diseases, including chronic obstructive pulmonary disease, cardiovascular dysfunction, neurodegenerative disorders, and cancer [44]. The burden is not only medical but also socioeconomic. Women often lose working days due to respiratory illness, children miss school, and the elderly face repeated hospital visits. Together, these impacts perpetuate cycles of poor health and poverty in low-income communities.

While Figure 4 illustrates the health impacts associated with indoor $PM_{2.5}$ exposure, Table 2 further demonstrates that the combined effects of $PM_{2.5}$ and microbial contaminants are most pronounced among vulnerable household populations due to variations in exposure intensity and biological susceptibility. Women, particularly those engaged in cooking activities, experience the highest cumulative $PM_{2.5}$ exposure, which contributes to chronic respiratory impairment and increased cardiovascular stress. Children exhibit heightened vulnerability as their developing lungs and higher inhalation rates facilitate deeper particle penetration, while concurrent exposure to microbial allergens promotes asthma and recurrent respiratory infections. Elderly individuals and those with pre-existing health conditions are especially susceptible to inflammation-driven disease exacerbations. Thus, the table highlights that co-exposure to fine particulate matter and microbial pollutants disproportionately affects vulnerable groups, establishing indoor air pollution as a significant yet largely preventable public health burden in developing countries.

7. CHEMISTRY AND RELEVANCE OF INDOOR $PM_{2.5}$ AND MICROBIAL POLLUTANTS IN HOUSEHOLDS

The toxicity of indoor $PM_{2.5}$ is strongly linked to its chemical composition, which differs substantially across regions and household types. In developing countries, indoor $PM_{2.5}$ often originates from biomass combustion, characterized by a high fraction of OC, EC, and PAHs [52,53]. These organic-rich particles serve as reactive surfaces for the adsorption of microbial fragments, such as endotoxins and fungal spores, thereby increasing their persistence and delivery to the respiratory tract. By contrast, in developed countries, indoor $PM_{2.5}$ tends to have lower mass concentrations, with a composition dominated by secondary inorganic aerosols, such as sulfates, nitrates, and ammonium, which have lower affinity for microbial binding [54]. The interaction of microbial contaminants with $PM_{2.5}$ is chemically mediated. Fungal spores (Aspergillus, Penicillium) bind readily to soot and EC surfaces, while Gram-negative bacterial endotoxins (lipopolysaccharides, LPS) attach to metal-rich $PM_{2.5}$ fractions. Transition metals, such as Fe, Cu, and Ni, can catalyze the oxidation of microbial cell wall components, generating ROS that amplify inflammatory potential [55]. These bio-organic-metal complexes are more toxic than either $PM_{2.5}$ or microbes alone. In households with poor ventilation, high humidity accelerates these reactions, allowing microbial proteins to undergo nitration and oxidation, producing compounds with enhanced allergenic properties [56]. Figure 5 shows that indoor $PM_{2.5}$ and microbial pollutants interact closely to form combined bioorganic complexes rather than acting as separate exposures. Carbon particles rich in PAHs readily trap fungal spores,

Table 2: Comparative effects of indoor $PM_{2.5}$ and microbial contaminants on vulnerable dwellers

Vulnerable group	$PM_{2.5}$ effects	Microbial contaminants effects
Women (biomass cooking, household chores)	Chronic exposure in poorly ventilated kitchens→asthma, COPD, cardiovascular stress, adverse pregnancy outcomes [45,46].	Exposure to mold spores and endotoxins in damp kitchens/bathrooms→allergic rhinitis, skin irritation, respiratory infections [47].
Children (developing lungs, higher breathing rates)	$PM_{2.5}$, penetrates alveoli→reduced lung growth, asthma, neurodevelopmental impairment, higher lifetime disease risk [48].	Early-life exposure to endotoxins, β-glucans, fungal-fragments→recurrent infections, wheezing, immune dysregulation [49,50].
Elderly (pre-existing condition, reduced immunity)	Accumulated lifetime exposure→worsens cardiovascular disease, hypertension, arrhythmia, accelerates aging processes [51].	Exposure to microbial pollutants→higher risk of pneumonia, chronic inflammation, exacerbation of COPD or diabetes complications [51].

COPD: Chronic obstructive pulmonary disease

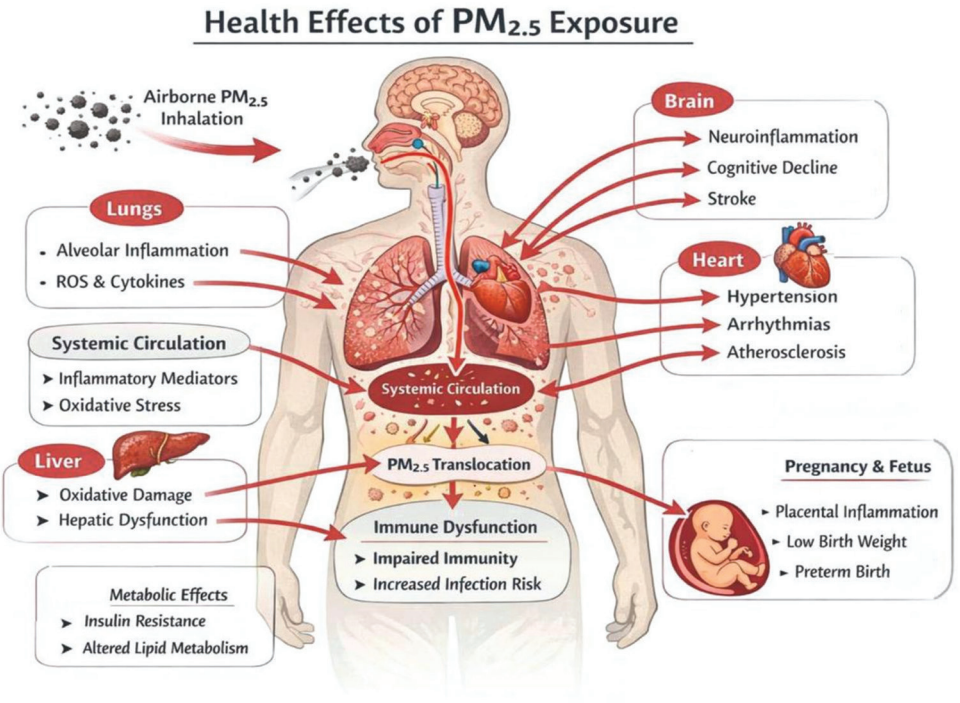

Figure 4: Differential health impacts of indoor $PM_{2.5}$ across different organs and systems

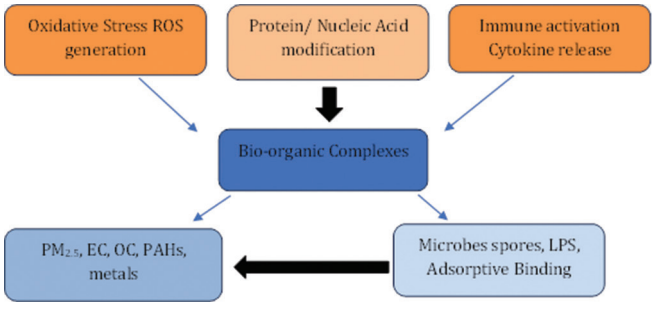

Figure 5: Mechanistic pathways of $PM_{2.5}$, microbial co-exposure toxicity

endotoxins, and β-glucans on their surfaces, while metal components within $PM_{2.5}$ enhance chemical reactivity and promote the generation of ROS. When inhaled, these composite particles penetrate deep into the lungs and simultaneously deliver toxic chemicals and microbial fragments, leading to strong oxidative stress and immune responses. This interaction accelerates inflammation and molecular damage in airway tissues, helping to explain why households with both high particulate pollution and damp microbial growth experience more severe respiratory and systemic health effects than would be expected from either pollutant alone.

From a relevance perspective, these interactions are particularly significant in developing countries, where households face combined exposures: Biomass smoke ($PM_{2.5}$ rich in carbonaceous and metal fractions), poor ventilation (leading to microbial growth), and infiltration of outdoor pollutants (traffic and industrial emissions). In contrast, developed countries experience lower levels of $PM_{2.5}$ indoors, dominated by secondary inorganic aerosols, while microbial exposure is mitigated by air conditioning, HVAC systems, and better sanitation. This disparity highlights the importance of comparative studies that not only assess concentrations but also analyze chemical composition and toxic potential. In continuation of the discussion, Table 3 presents a comparative overview of studies from different cities, providing a broader perspective on the spatial variability of indoor $PM_{2.5}$ and microbial contamination. The comparison clearly indicates that households in developing regions consistently experience substantially higher $PM_{2.5}$ concentrations, largely driven by biomass cooking, traffic emissions, and industrial activities, along with elevated bacterial and fungal loads associated with dampness and inadequate ventilation. In evaluation, households in developed cities generally report much

Table 3: Comparative studies on indoor $PM_{2.5}$ and microbial contaminants in households across different cities

City/country	Household type	$PM_{2.5}$ chemistry	Microbial contaminants	References
Delhi, India	Biomass-fueled kitchens	High $PM_{2.5}$ (150–500 μg/m^3); rich in OC, EC, PAHs; trace metals (Fe, Cu)	High fungal spores (Aspergillus, Cladosporium); endotoxin levels elevated.	[57,58]
Lucknow-India	Industrial-adjacent households	$PM_{2.5}$ enriched with heavy metals (Pb, Cr, Ni); carbonaceous particles from traffic.	Gram-negative bacteria (*Escherichia* coli), actinomycetes, spore-forming Bacillus.	[30,59-61]
Beijing-China	Urban residential homes	$PM_{2.5}$ moderate indoors (~80 μg/m^3) dominated by nitrates and sulfates in winter.	Fungal spores frequent; β-glucans in settled dust.	[62]
Bangkok, Thailand	Mixed residential-commercial	$PM_{2.5}$ (70–200 μg/m^3) from traffic and cooking oils; high VOC derivatives.	Bacterial endotoxins (LPS) linked to food/waste handling.	[63-65]
London, UK	Modern residential households	Low indoor $PM_{2.5}$ (<20 μg/m^3); dominated by sulfates and nitrates.	Minimal microbial load; Cladosporium in damp basements.	[66-69]
Los Angeles, USA	Suburban households	Indoor $PM_{2.5}$ moderate (~25 μg/m^3); EC and OC infiltration from traffic.	Low microbial load; occasional endotoxins in poorly ventilated homes.	[70-72]

lower particulate and microbial levels, with contamination primarily restricted to specific moisture-affected or poorly ventilated spaces. By bringing together evidence from diverse urban settings, the table emphasizes that indoor air pollution represents a far more severe and widespread public health challenge in developing countries, characterized by a compounded chemical-biological exposure burden.

8. EXPOSURE ASSESSMENT, MONITORING APPROACHES, AND POLICY RELEVANCE

Table 4 links real-world indoor exposures with practical monitoring and policy implications for developing country microenvironments. It shows that typical household $PM_{2.5}$ and microbial levels frequently exceed health-based reference benchmarks, particularly in homes using solid fuels or affected by dampness and poor ventilation. Alongside these exposure ranges, the table outlines appropriate monitoring approaches, from calibrated low-cost sensors and basic bioaerosol sampling to reference-grade methods, ensuring reliable assessment even in resource-limited settings. By directly connecting measured concentrations with regulatory gaps and potential interventions, emphasizing that indoor air pollution in developing countries remains largely underregulated despite its high health burden.

Assessment of indoor exposure to $PM_{2.5}$ and microbial contaminants is central to understanding their health relevance. Indoor $PM_{2.5}$ levels are highly variable across different cities and strongly reflect fuel use, ventilation, and ambient infiltration. In developed cities, such as London, average indoor $PM_{2.5}$ concentrations have been reported around 10 μg/m^3, which is close to but still above the WHO annual guideline of 5 μg/m^3 [66]. In contrast, homes in Delhi show much higher concentrations, with kitchen measurements averaging nearly 278 μg/m^3 during cooking, far exceeding the WHO 24-h limit of 15 μg/m^3 and India's own national 24-h standard of 60 μg/m^3 [83]. Dhaka households that rely on biomass fuels often experience short-term spikes exceeding 1000 μg/m^3, indicating extreme exposure conditions [12]. Similarly, Nigerian households in Ogbomoso reported mean values of 41.6 μg/m^3, which, while lower than Indian or Bangladeshi kitchens, still exceed WHO limits [81]. In Los Angeles, indoor $PM_{2.5}$ values are generally lower, but wildfire smoke episodes can elevate indoor concentrations above the US EPA 24-h standard of 35 μg/m^3 [78]. These comparisons highlight that developing-country households face persistent, chronically high exposures, while developed-country households experience episodic exceedances linked to specific events. Microbial contaminants also vary widely across cities. UK airtight dwellings reported relatively low concentrations, with mean bacterial levels around 213 CFU/m^3 and fungal levels around 18 CFU/m^3, both well within Hong Kong's "Excellent" objectives (<500 CFU/m^3 for bacteria and fungi) [57]. Hong Kong residences themselves showed mean bacterial counts near 677 CFU/m^3 and fungal counts near 218 CFU/m^3, approaching the "Good" objective of <1,000 CFU/m^3 [76]. By contrast, Delhi homes exhibited much higher seasonal averages, with bacterial loads between 740 and 1,650 CFU/m^3 and fungal levels between 780 and 1,275 CFU/m^3, consistently exceeding international reference objectives [83]. In Lahore, kitchens and living rooms demonstrated extreme variability, with bacterial counts ranging from 472 to over 14,000 CFU/m^3 and fungal counts between 236 and 1887 CFU/m^3 [12]. These data confirm that microbial exposure indoors is a widespread problem in developing regions, strongly linked to dampness, sanitation gaps, and inadequate ventilation. Exposure assessment methods differ significantly between developed and developing contexts. In developed countries, high-resolution instruments, such as condensation particle counters, scanning mobility particle sizers, and aerosol mass spectrometers provide detailed information on size, number, and chemical composition of $PM_{2.5}$, while microbial contaminants are increasingly characterized by molecular approaches like quantitative polymerase chain reaction (qPCR), metagenomics, and ELISA-based endotoxin assays [14]. In developing countries, however, exposure assessment still relies heavily on gravimetric samplers for $PM_{2.5}$ mass and culture-based microbial techniques, which underestimate both the true microbial burden and the toxic potential of $PM_{2.5}$ This disparity makes it difficult to compare data across countries, but it highlights the urgent need for affordable, integrated monitoring approaches that combine PM chemistry with microbial markers in resource-limited settings. From a policy perspective, indoor $PM_{2.5}$ and microbial exposures carry critical socio-economic implications. In residential households, biomass smoke not only increases chronic respiratory and cardiovascular risks but also exacerbates microbial contamination in damp kitchens and bathrooms. In industrial-adjacent households, toxic metal-rich particles infiltrate homes and interact with microbial residues, producing highly inflammatory complexes. In commercial households above shops and markets, traffic emissions, food-related particulates, and poor sanitation combine to create a double burden of chemical and microbial exposure. Developed countries typically face much lower exposure levels, but episodic challenges, such as wildfire smoke or mold in damp basements still require continuous monitoring and mitigation. Overall, the contrast between cities in developing and developed nations demonstrates that exposure assessment must be microenvironment-specific and context-aware. While London flats illustrate that targeted feedback and monitoring can reduce indoor $PM_{2.5}$ levels by double digits, Delhi kitchens and Dhaka biomass

Table 4: Exposure assessment, monitoring approaches, and policy relevance for indoor $PM_{2.5}$ and microbial contaminants in developing country microenvironments

Section/item	Parameter and unit	Benchmark/ guideline (reference)	Typical indoor range in developing country settings*	Recommended monitoring approach (minimum→advanced)	Policy relevance/ interpretation	References
A. Benchmarks for interpreting indoor measurements						
WHO Global Air Quality Guidelines (2021)	$PM_{2.5}$ ($\mu g/m^3$)	Annual: 24-h	Often exceeded indoors during cooking/poor ventilation	Gravimetric (FRM/FEM) or reference grade continuous; add personal sampling for high-risk groups	Health protective indoor target where no indoor standard exists	[1]
US EPA NAAQS (final 2024)	$PM_{2.5}$ ($\mu g/m^3$)	Annual 24 h	Indoor peaks can exceed 35 during wildfire smoke or cooking events	Reference grade continuous (BAM/TEOM/FRM) for comparability; event logging	Supports advisories and filtration guidance for episodic high exposures	[73]
India NAAQS (CPCB, 2009; current)	$PM_{2.5}$ ($\mu g/m^3$)	Annual: 24 h	Solid fuel kitchens frequently >60; living areas often 50–200+	FRM/FEM where feasible; calibrated low-cost sensors for screening+co-location QA/QC	Makes the regulatory gap explicit; supports indoor-specific standards and clean cooking policies	[74]
EU Ambient Air Quality Directive (in force) + revision trajectory	PM2.5 ($\mu g/m^3$)	Current annual limit: 25; policy proposal toward 10 by ~2030	Many LMIC indoor settings far exceed 25	Use as an international benchmark; adopt interim indoor targets (e.g., 15–10) for programs	Provides a feasible tightening pathway for policy roadmaps	[75,76]
Hong Kong IAQ Certification Scheme	Bioaerosols (CFU/m^3)	Airborne bacteria: ≤500 (Excellent), ≤1,000 (Good); mould/fungi often assessed against 500	Delhi/Lahore-type homes often exceed 1,000; damp homes show high fungal counts	Impaction sampling on culture media+RH/ temperature logging; duplicates and blanks	Actionable, audit-friendly thresholds for ventilation/moisture control	[80]
Singapore SS 554:2016	Fungi (CFU/m^3)	≤500 (commonly used audit criterion)	Damp, poorly ventilated homes frequently exceed 500	Impaction/filtration culture+moisture inspection; consider qPCR when culture underestimates	Triggers building remediation and indoor moisture standards	[78]
B. Monitoring toolkit for exposure assessment (field ready)						
$PM_{2.5}$ mass-reference (comparability)	$PM_{2.5}$ ($\mu g/m^3$)	FRM/FEM comparability	-	Filter-based gravimetric (24-h) + optional speciation (OC/EC, ions, metals, PAHs)	Enables compliance style comparison; supports source attribution and risk assessment	[1]
$PM_{2.5}$ mass-time resolved peaks	$PM_{2.5}$ ($\mu g/m^3$)	Peak and pattern detection	-	BAM/TEOM or high-grade optical with RH correction; integrate time activity diaries	Identifies cooking/ traffic/smoke peaks; evaluates interventions (LPG, chimneys, filtration)	[73,79]
Personal exposure (dose proxy)	Personal $PM_{2.5}$ ($\mu g/m^3$)	Closer to inhaled dose than area monitors	-	Wearable PM monitor+activity logs (or GPS); repeated measures across seasons	Improves exposure-response inference; essential for women/ children-focused assessments	[79,80]
Bioaerosols-culture-based (policy friendly)	Bacteria/fungi (CFU/m^3)	Operational thresholds (500–1,000)	-	Andersen/impaction sampling; standardized media and incubation; field blanks	Useful for audits and compliance reporting; supports dampness/ventilation policies	[12,77]
Bioaerosols-biomarkers and molecular	Endotoxin (EU/m^3), β-glucan, qPCR/ sequence reads	No universal standards	-	Filter sampling+LAL (endotoxin), β-glucan assays; qPCR/amplicon sequencing	Captures non-culturable fraction; strengthens mechanistic interpretation and health linkage	[81]

(*Contd...*)

Table 4: (*Continued*)

C. Illustrative exposure ranges reported in developing country homes (context)**						
Section/item	**Parameter and unit**	**Benchmark/ guideline (reference)**	**Typical indoor range in developing country settings***	**Recommended monitoring approach (minimum→advanced)**	**Policy relevance/ interpretation**	**References**
Solid fuel households (India; multi-site)	24-h kitchen $PM_{2.5}$ ($\mu g/m^3$)	-	Kitchen: Mean ~609; median ~472; living area mean~163 (reported values depend on kitchen type/ventilation)	Area gravimetric or calibrated monitors; record fuel, kitchen configuration, and ventilation	Quantifies benefits of clean cooking and ventilation; informs subsidy/transition programs	[79]
Urban LMIC homes (Nigeria example)	Indoor $PM_{2.5}$ ($\mu g/m^3$)	-	~41.6 mean over multi-week monitoring (urban background influence)	Co-located indoor/outdoor monitoring+building survey	Shows outdoor to indoor coupling; supports airshed control plus indoor filtration	[82]
Delhi residential houses (bioaerosols)	Bacteria/fungi (CFU/m^3)	HK/SG (500-1000) as comparators	Bacteria: ~738–1654 (seasonal means); fungi: ~776–1275 (seasonal means)	Six stage/impactor culture; RH/temperature; seasonal sampling	Supports need for microbial IAQ benchmarks and moisture control policies	[12]

*Typical ranges are indicative and vary strongly with fuel type, ventilation, building tightness, season, occupancy, and outdoor infiltration. **Reported ranges are provided for context only; sampler type, culture media, and incubation protocols can shift CFU counts, standardize methods, and report QA/QC. qPCR: Quantitative polymerase chain reaction, EPA: Environmental protection agency

homes show that chronic exceedances of WHO standards remain the dominant exposure pathway. Addressing this disparity requires interventions that prioritize clean fuels, better ventilation, humidity control, and low-cost integrated monitors capable of capturing both $PM_{2.5}$ chemistry and microbial diversity [12,66,83].

9. KNOWLEDGE GAPS, RESEARCH GAPS, RESEARCH NEEDS, AND COMPARATIVE PERSPECTIVES

Despite advances in indoor air quality research, important gaps remain, especially in developing countries where exposures are highest but scientific data and regulatory enforcement are weakest. Most studies continue to measure $PM_{2.5}$ only as mass concentration ($\mu g/m^3$), ignoring its chemical composition-EC, OC, PAHs, and metals-that determines toxicity and biological reactivity [84]. Similarly, microbial contaminants are still monitored predominantly through culture-based methods, which underestimate the true microbial burden by excluding non-culturable or dormant microbes. By contrast, developed countries increasingly employ advanced techniques, such as qPCR, endotoxin assays, β-glucan assays, and metagenomic sequencing, enabling more accurate characterization of microbial diversity [85,86]. Another major gap is the absence of high-resolution temporal monitoring. In developing-country settings, gravimetric 24-h $PM_{2.5}$ samples dominate, which obscure acute exposure spikes during cooking or waste burning. For example, studies in Delhi reported kitchen $PM_{2.5}$ averages around 278 $\mu g/m^3$ during cooking, while Dhaka households using biomass fuels frequently experienced short-term episodes exceeding 1,000 $\mu g/m^3$-orders of magnitude higher than WHO's 24-h guideline of 15 $\mu g/m^3$ [12,83]. Such peaks are particularly harmful for women and children, who spend long periods in kitchens and living spaces during cooking hours. Similarly, microbial exposures fluctuate seasonally and with indoor dampness, but few studies capture these variations with real-time methods [87,88]. There is also a scarcity of integrated studies combining $PM_{2.5}$ chemistry and microbial diversity. Evidence shows that carbonaceous and metal-rich particles readily adsorb fungal spores and bacterial endotoxins, producing bio-organic complexes that drive oxidative stress and immune activation [89-91]. Yet, epidemiological studies still tend to examine particles and microbes separately, underestimating the true risk of combined exposures. Finally, socio-economic assessments linking indoor exposures with productivity loss, healthcare costs, and gendered burdens remain limited in developing countries. Without such data, policy-makers struggle to prioritize IAQ interventions. In addition to scientific gaps, there is a serious lack of policy frameworks addressing indoor air quality in developing countries. Most governments rely on ambient standards (e.g., India's $PM_{2.5}$ 24-h limit of 60 $\mu g/m^3$), which are far more lenient than WHO's guidelines, and fail to account for microbial contaminants [92]. Stronger government action could dramatically change this scenario: Subsidizing and mandating a transition from biomass to clean fuels, such as LPG or electricity can drastically reduce household $PM_{2.5}$, enforcing building and ventilation codes can reduce microbial growth; adopting microbial standards, like Hong Kong's IAQ objectives (<500 CFU/m^3 bacteria, <500 CFU/m^3 fungi), can regulate microbial safety [93], and enforcing buffer zones around industries can reduce toxic dust infiltration. Governments can also mandate IAQ monitoring in public schools, hospitals, and social housing to protect the most vulnerable groups. The benefits of strict IAQ regulations would be profound and equitable. Vulnerable dwellers, women exposed to cooking smoke, children with developing lungs, and the elderly with chronic illnesses would be the first to experience health gains [94]. Stronger IAQ rules would also reduce the economic burden, cutting hospital visits, workdays lost, and school absenteeism. Over time, healthier homes contribute to stronger workforces, reduced poverty cycles, and a better quality of life. Developed countries, through policies on ventilation, microbial safety, and smoke-free indoor laws, have already demonstrated how government action transforms toxic homes into safer living environments [95,96]. Replicating these models in developing nations is not only possible but urgently necessary.

10. CONCLUSION

Indoor $PM_{2.5}$ and microbial contaminants together form a dual burden of chemical and biological pollutants that has been largely ignored in policy and research, despite their outsized health impact in developing

countries. Unlike developed nations where indoor risks are episodic (e.g., wildfire smoke in California, damp basements in the UK), exposures in developing countries are chronic, severe, and synergistic. Biomass cooking releases reactive carbonaceous $PM_{2.5}$, industrial dust infiltrates homes with heavy metals, and damp kitchens and bathrooms promote microbial growth. These pollutants interact to form bio-organic complexes, heightening toxicity through oxidative stress, immune activation, and systemic disease. This review is important because it reframes indoor pollution not as two separate challenges but as an integrated chemical-biological hazard. By synthesizing comparative evidence across developed and developing cities, it demonstrates that equity in research, monitoring, and regulation is urgently needed. Women, children, and elderly dwellers in developing countries will benefit the most from interventions that lower household exposures. Public health agencies and WHO will gain consolidated evidence to expand air quality guidelines beyond PM mass to microbial metrics. National governments can design effective and affordable interventions, such as clean cooking fuels, ventilation standards, and microbial controls. Researchers and healthcare providers will benefit from a clearer framework to study combined exposures and related health outcomes. The call to action is clear: Indoor air quality must be mainstreamed into air and health laws; resource-limited regions need affordable integrated monitoring technologies; clean fuels, ventilation, humidity control, and waste management should be prioritized; and the WHO, together with international partners, must help establish indoor-specific standards, fund interdisciplinary studies, and support developing-country institutions. Ultimately, stricter rules, better monitoring, and equitable interventions will protect the most vulnerable dwellers-transforming homes from sites of hidden exposure into safe and healthy spaces.

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Anam Taushiba: Data analysis and writing original draft.

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The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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16. HUMAN ETHICAL APPROVAL AND CONSENT TO PARTICIPATE

Not applicable.

17. CONSENT FOR PUBLICATION

All the participants were informed about publishing their results and consent was taken from all the participants.

18. COMPETING INTERESTS

The authors declare that they do not possess any identifiable conflicting financial interests or personal relationships that may have potentially influenced the research presented in this manuscript.

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