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Review Article

Air-Water Pollution Pathways and Respiratory Health: Environmental Engineering Strategies for Cleaner Lungs

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Abstract

Environmental pollution represents the largest global cause of disease and death, yet traditional research approaches have examined air and water contamination as separate domains. This literature review synthesizes evidence from peer-reviewed studies to establish a comprehensive framework for understanding air-water-lung pollution pathways and their implications for respiratory health. The review identifies two primary cross-media pathways: water-to-air transmission, including bioaerosol emissions from wastewater treatment plants dispersing pathogens over distances exceeding 3 kilometers and harmful algal bloom toxins becoming aerosolized concentrations 125-fold above safety guidelines; and air-to-water pathways, where atmospheric nutrient deposition promotes eutrophication and subsequent algal bloom formation. Evidence from Mendelian randomization studies demonstrates causal relationships between PM2.5, PM10, and NO_2 exposure and lung function decline, while developmental research establishes that prenatal pollution exposure creates lasting respiratory vulnerability. The airway microbiome emerges as both a mediator and modifier of environmental health effects. Engineering solutions including biofiltration systems and risk-based monitoring frameworks offer promising approaches for cross-media pollution control. This review concludes that effective protection of respiratory health requires integrated environmental engineering strategies that recognize the fundamental interconnectedness of air quality, water quality, and human health outcomes.

Keywords: *Air Pollution; Water Pollution; Respiratory Health; Bioaerosols; Environmental Engineering; Cross-Media Pollution; Wastewater Treatment*

Introduction

Global respiratory disease burden

Respiratory diseases represent one of the most significant global health challenges of the 21st century. The Lancet Commission on Pollution and Health established that pollution is the largest environmental cause of disease and death globally, responsible for an estimated 9 million premature deaths in 2015, with 92% occurring in low- and middle-income countries [1]. This landmark report estimated welfare losses due to pollution at more than US$4.6 trillion per year, equivalent to 6.2% of global economic output [1]. The Commission dispelled the myth that pollution is an inevitable consequence of economic development and demonstrated that pollution is a winnable battle with proper political will and evidence-based interventions [1].

The burden of air pollution on respiratory health is particularly striking. West., *et al*. documented that ambient fine particulate matter (PM2.5) ranks as the seventh most important global mortality risk factor, responsible for 2.9 million premature deaths in 2013 [2]. Their research emphasized that 87% of the global population lives in areas exceeding WHO PM2.5 guidelines, with the most polluted areas concentrated in South and East Asian megacities [2]. Wallbanks., *et al*. confirmed that over 90% of the global population currently resides in areas where environmental air pollution exceeds World Health Organization guidelines, with adverse effects ranging from acute airway irritation to complex immunomodulatory alterations [3].

The mechanisms underlying these health effects have been elucidated through decades of research. Brauer., *et al*.'s seminal study investigated the types, sizes, and locations of atmospheric particles retained in human lungs, providing direct measurements of deposited particles in human tissue [4]. Their research revealed how ultrafine particles from combustion processes translocate across alveolar epithelium to the interstitium, where they may be cleared slowly or remain permanently [4]. This foundational work established the biological basis for epidemiological observations linking particulate air pollution to increased cardiopulmonary mortality and morbidity.

Priyatham's emerging research findings confirm that particulate matter penetrates deep into lungs, triggering airway inflammation, oxidative stress, and tissue damage while potentially entering the bloodstream [5]. The research highlights air pollution as a known carcinogen linked to lung cancer through prolonged exposure to fine particles and polycyclic aromatic hydrocarbons, contributing to 4.2 million premature deaths annually [5].

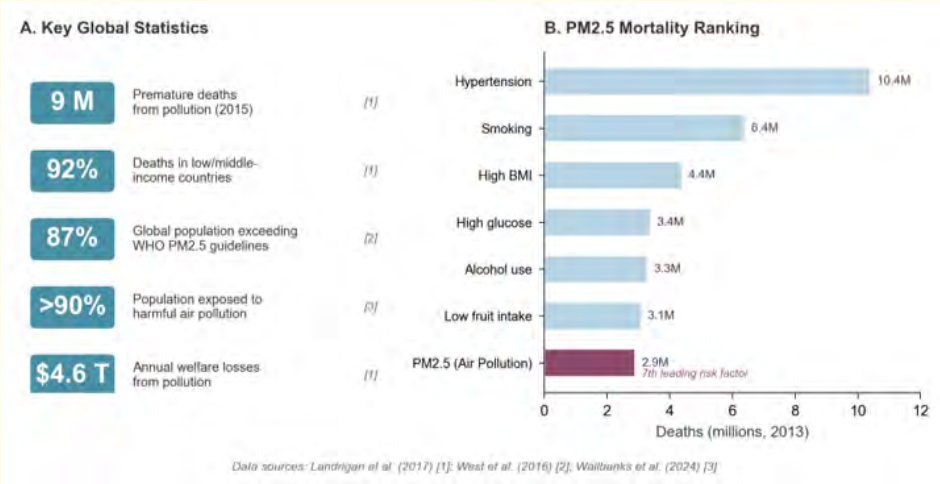

Figure 1

The interconnected nature of environmental pollution

Traditional approaches to environmental health research have predominantly operated within disciplinary silos, examining air pollution and water contamination as separate domains. However, mounting evidence demonstrates the fundamental interconnectedness of environmental media. Caudeville., *et al*. developed the PLAINE platform, a GIS-based multimedia exposure model demonstrating how pollutants move through soil, water, air, and food sources, ultimately contributing to human exposure through multiple pathways simultaneously [6]. Their spatial stochastic model identified major risk hotspots where cadmium concentrations posed significant health concerns through multiple exposure pathways [6].

This integrated perspective aligns with emerging exposome science. Huang and Jiang introduced the concept of the human exposome as a comprehensive framework encompassing all external and internal exposures from conception to death, including chemical, physical, biological, and social factors [7]. Their approach represents a paradigm shift from single-exposure studies to comprehensive exposure assessment methodologies that consider the totality of human environmental experiences [7].

Briggs proposed a comprehensive framework for integrated environmental health impact assessment (IEHIA) to address complex, systemic risks that traditional risk assessment methods cannot adequately handle [8]. The framework incorporates four key stages-issue-framing, design, execution, and appraisal (IDEA process)-emphasizing stakeholder participation throughout [8]. Unlike conventional approaches focusing on single agents or pathways, this methodology considers multiple environmental factors, their interactions, and adaptive responses within social-ecological systems [8].

The limitations of single-media approaches become particularly evident when examining respiratory health impacts from water-to-air transmission pathways. Vermeulen., *et al*. demonstrated that *Legionella* transmission through atmospheric dispersion from wastewater treatment facilities occurs over distances exceeding 3 kilometers, with significant population exposure and increased disease risk [9]. Similarly, Lim., *et al*. documented how harmful algal blooms in freshwater systems generate spray aerosols containing cyanotoxins, creating novel inhalation exposure pathways previously unrecognized [10].

Lin., *et al*.'s systematic review analyzed 85 papers revealing that over 80% of global sewage is discharged without treatment, contributing to more than 50 diseases [11]. They documented that 80% of diseases and 50% of child deaths worldwide are related to poor water quality, emphasizing the global burden of waterborne diseases that can also manifest as respiratory health risks through aerosolization [11].

Scope and Objectives

This literature review synthesizes evidence from 25 peer-reviewed studies published between 2001 and 2025, focusing specifically on the air-water-lung nexus in pollution pathways. The primary objectives are: (1) to establish a comprehensive theoretical framework for understanding air-water-lung pollution pathways; (2) to critically analyze key pollution pathways affecting respiratory health; and (3) to evaluate environmental engineering solutions across multiple environmental media.

Theoretical framework: The air-water-lung nexus

Pollutant transfer mechanisms

The air-water-lung nexus represents a dynamic system where pollutants continuously exchange between atmospheric and aquatic compartments through physical, chemical, and biological processes. Caudeville., *et al*.'s spatial stochastic multimedia exposure model demonstrates how pollutants move through environmental compartments, with Monte Carlo simulations quantifying exposure doses and associated health risks [6]. Their work identified two major risk hotspots in France-the former Metaleurop industrial site and the Lille agglomeration-through integrated spatial analysis [6].

Qiu., *et al*. investigated atmospheric nitrogen and phosphorus deposition effects on water quality in China's middle route project of the South-to-North water diversion [12]. Their research revealed that meteorological factors and nutrient deposition account for 18% of water quality variation, demonstrating that atmospheric deposition contribution to water pollution is increasing and requires attention [12]. Total nitrogen sedimentation flux showed obvious seasonal variation consistent with rainfall patterns, establishing a direct air-to-water pollution pathway [12].

Plaas and Paerl's critical review examined how toxic *Cyanobacteria* threaten both water and air quality through aerosolization of cyanotoxins [13]. Their research demonstrated that freshwater spray aerosol can incorporate cyanobacterial cells and toxins, creating

inhalation exposure pathways as cyanobacterial harmful algal blooms (CyanoHABs) increase temporally and spatially due to nutrient over enrichment and climate change [13].

Respiratory exposure pathways

Direct inhalation of airborne pollutants from water sources represents a critical exposure pathway. Vermeulen., *et al*. utilized atmospheric dispersion modeling to investigate *Legionella* transmission from 776 wastewater treatment plants across the Netherlands during 2013 - 2018 [9]. Using the OPS atmospheric dispersion model, they found that Legionnaires' disease cases were significantly more exposed to WWTP aerosols than controls (OR: 1.32, 95% CI: 1.06 - 1.63), with aerosols dispersing over distances of at least 3 kilometers [9].

Van den Berg., *et al*. developed a risk matrix to identify WWTPs at high risk for *Legionella* growth and emission based on biological treatment type, industry type, water temperature, and aeration processes [14]. Their inventory of 778 WWTPs revealed that 18% of moderate-to-high risk facilities tested positive for *Legionella* species, with industries processing nutrient-rich wastewater at 30 - 38°C representing the highest risk [14].

Particle size distribution critically determines respiratory tract deposition patterns. Brauer., *et al*.'s research on particle retention in human lungs revealed how particles are translocated across alveolar epithelium to the interstitium [4]. This mechanism is particularly relevant for understanding how particles generated from water treatment processes affect deep lung structures and contribute to chronic respiratory disease development.

Yi., *et al*.'s population-based study of 3,915 household members in Guangdong, China revealed how the airway microbiome mediates environmental exposure effects on respiratory health [15]. Using induced sputum samples, they demonstrated that fungal microbiota (*Penicillium* and *Cladosporium*) mediate PM2.5 concentration effects on respiratory symptoms, establishing the airway microbiome as both mediator and modifier of environmental health effects [15].

Conceptual model

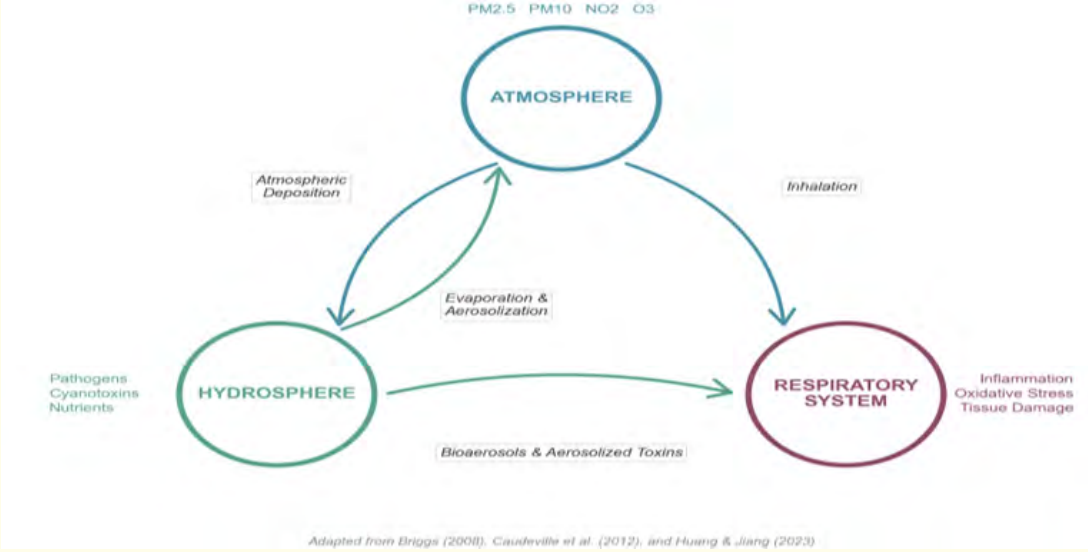

Figure 2

The integrated air-water-lung nexus model synthesizes multiple pathways into a comprehensive framework. Briggs emphasized that unlike conventional approaches, integrated assessment must consider multicausality, non-linearity, and temporal dynamics in environmental health relationships [8]. This approach supports more effective policy development for complex environmental health issues such as climate change and emerging systemic risks [8].

Critical exposure points include wastewater treatment facilities, which serve as major sources of bioaerosol generation. El-Bestawy., *et al*. quantified bioaerosol emissions from an Egyptian WWTP, finding bacterial counts up to 42.7 × 10^3 CFU/m^3, classified as "very poor" according to the index of microbial air contamination [16]. Poopedi., *et al*. identified 36 potential airborne bacterial pathogens at South African WWTPs, with 78% classified as risk group 2 according to biological hazard regulations [17].

Freshwater systems experiencing harmful algal blooms represent another critical node. Olson., *et al*. demonstrated that microcystin toxins can be incorporated into lake spray aerosols at concentrations reaching 50 ± 20 ng/m^3-exceeding EPA's "do not drink" recommendation by over 125-fold [18]. This provides critical evidence for airborne transmission pathways of HAB toxins.

Key pollution pathways affecting respiratory health

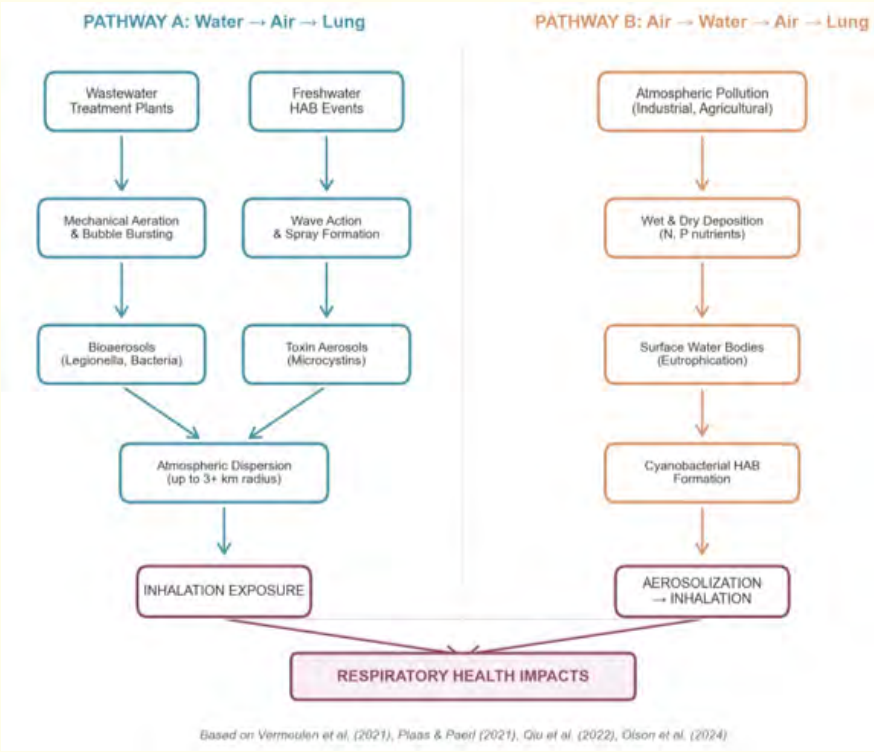

Figure 3

Water-to-air-to-lung pathways

Bioaerosols from wastewater systems

Wastewater treatment plants generate significant bioaerosol emissions that impact both worker and community health. Vermeulen., *et al*.'s 6-year case-control study across the Netherlands provided robust epidemiological evidence that WWTP aerosols containing *Legionella*

disperse over substantial distances, with Legionnaires' disease cases significantly more exposed than controls [9]. The association remained significant even after excluding known outbreak-related facilities, indicating widespread transmission from multiple WWTPs [9].

El-Bestawy., *et al*. conducted quantitative analysis at an Alexandria WWTP, collecting 170 samples across 10 monitoring sites [16]. They identified pathogenic microorganisms including *Lysinibacillus fusiformis*, *Bacillus cereus*, *Escherichia coli*, and various *Aspergillus* species posing health risks to workers [16]. Similarly, Poopedi., *et al*. used Illumina MiSeq sequencing to investigate bacterial diversity at South African WWTPs with different aeration systems, finding mechanical aeration tanks showed significantly higher pathogen diversity (72% of identified pathogens) compared to diffused aeration systems (17%) [17].

Møller., *et al*. conducted comprehensive occupational health assessment among 44 WWTP workers across four facilities, correlating bioaerosol exposures with serum biomarkers [19]. They found significant positive correlation between C-reactive protein and bacterial exposure ($p = 0.013$), with viable hazardous microorganisms including *Clostridium perfringens* and *Aspergillus fumigatus* identified in respiratory fractions [19]. This research provides direct evidence linking occupational bioaerosol exposure to inflammatory health responses.

Gleason and Cohn reviewed Legionnaires' disease and public water systems, emphasizing that *Legionella* has remained an "emerging pathogen" despite being identified 45 years ago [20]. They analyzed epidemiological data from outbreaks, particularly focusing on Flint, Michigan, examining public water infrastructure's role in disease transmission and providing recommendations for improved monitoring [20].

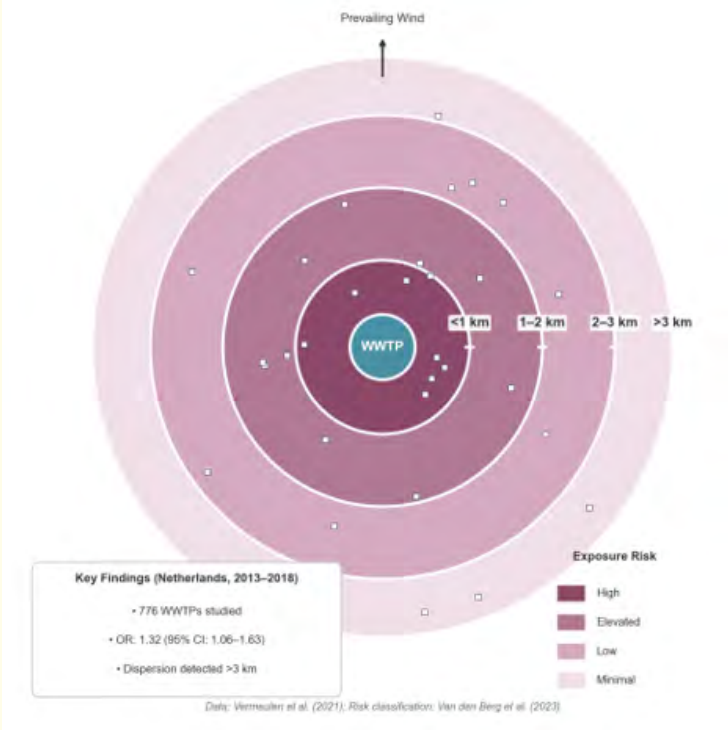

Figure 4

Harmful algal blooms and aerosolized toxins

Harmful algal bloom aerosols represent an emerging environmental health concern. Lim., *et al*. synthesized current knowledge on HAB aerosols, explaining mechanisms of aerosolization through wind-driven wave action creating spray aerosols containing toxins [10]. They examined formation of sea spray aerosols and lake spray aerosols that incorporate organic material and whole cells during bubble bursting at air-water interfaces [10]. As HAB frequency increases due to warming climate and eutrophication, this research provides crucial insights for public health protection [10].

Olson., *et al*. provided the first laboratory demonstration that microcystin toxins from HABs can be incorporated into lake spray aerosols [18]. Collecting water samples from Mona Lake during a severe HAB event (> 200 µg/L microcystin), they used LC-MS/MS to identify enrichment of hydrophobic microcystin congeners in aerosol particles [18]. The finding that microcystin concentrations exceeded EPA drinking water recommendations by over 125-fold demonstrates significant inhalation exposure potential [18].

Plaas and Paerl's comprehensive review established CyanoHABs as growing threats to both water and air quality [13]. They documented how toxigenic *Cyanobacteria* (*Cylindrospermopsis*, *Microcystis*, *Planktothrix*) produce cyanotoxins that become airborne through spray aerosol formation, with microcystin being the most widespread cyanotoxin [13]. The authors emphasized that CyanoHAB respiratory health impacts remain understudied compared to marine harmful algal blooms [13].

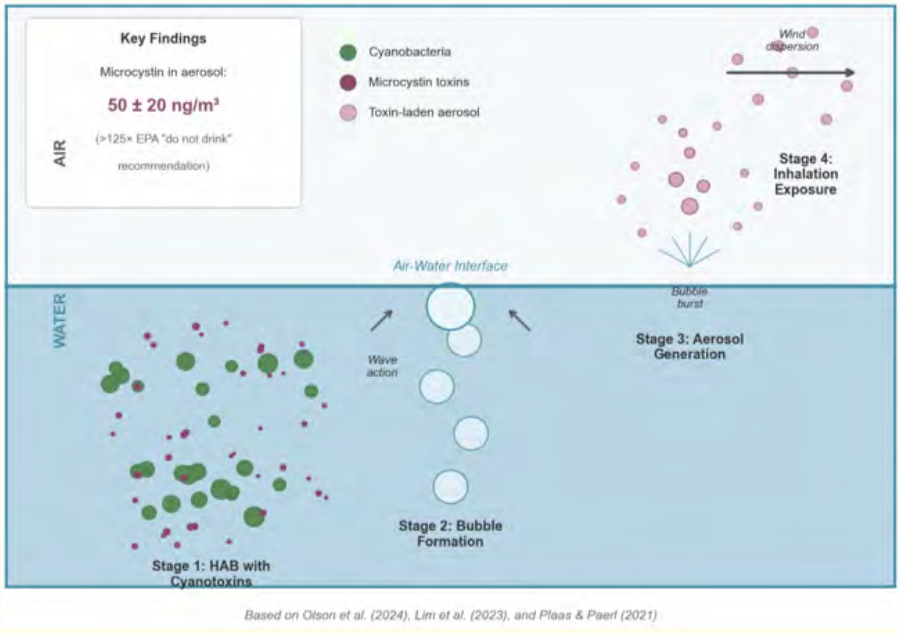

Figure 5

Air-to-water-to-lung pathways

Nutrient pollution and secondary effects

Atmospheric deposition of nutrients significantly impacts water quality and subsequent air quality through secondary processes. Qiu., *et al*.'s two-year study at China's South-to-North water diversion project analyzed dry and wet deposition samples, revealing that nitrogen and phosphorus deposition significantly correlated with water quality factors [12]. They demonstrated that atmospheric deposition's ecological impact on water pollution is increasing, particularly in engineered aquatic systems with minimal human disturbance [12].

This nutrient loading promotes eutrophication and harmful algal bloom formation. Plaas and Paerl emphasized that CyanoHABs are increasing temporally and spatially due to nutrient over enrichment, creating conditions for aerosolized cyanotoxins [13]. The cascade from atmospheric nitrogen deposition to water eutrophication to algal bloom formation to respiratory toxin exposure represents a complete air-to-water-to-air-to-lung pathway.

Climate change and pathogen dynamics

Climate change amplifies pollution transfer between environmental media. Wu., *et al*. reviewed scientific evidence on climate change impacts on infectious diseases based on publications from 1990 - 2015 [21]. They documented how long-term weather shifts and extreme events multiply existing health problems and create new disease transmission pathways [21]. The review identified how temperature, precipitation, and extreme weather variations alter infectious disease patterns [21].

Quinete and Hauser-Davis examined how drinking water contaminants may compromise immune system function, particularly relevant during the COVID-19 pandemic [22]. They discussed major immunotoxic contaminants including PFAS, plastics, and metals that enter drinking water, exploring mechanisms through which these pollutants affect immune function and potentially compromise responses to viral infections [22].

Landrigan., *et al*.'s comprehensive assessment of ocean pollution examined threats to human health from a complex mixture of toxic metals, plastics, chemicals, and sewage, with over 80% arising from land-based sources [23]. They demonstrated that pollution is heaviest near coasts and most concentrated along shores of low- and middle-income countries, establishing ocean pollution as an insufficiently recognized component of global environmental health threats [23].

Respiratory health mechanisms and vulnerable populations

Mechanisms of pollution-induced respiratory disease

Li., *et al*. provided robust causal evidence for air pollution effects on respiratory health using Mendelian randomization with genetic instrumental variables [24]. Analyzing 400,102 participants, they demonstrated significant causal relationships between PM2.5, PM10, and NO_2 with lung function decline [24]. PM10 negatively affected FEV1 (OR: 0.934), FVC (OR: 0.941), and FEV1/FVC ratio (OR: 0.965), while PM2.5 and NO_2 increased COPD incidence (OR: 1.273 and 1.357, respectively) [24]. Importantly, they identified Interleukin-17A as a mediator in the PM10-lung function relationship [24].

Wallbanks., *et al*. described mechanisms by which particulate matter, ozone, and nitrogen dioxide affect lung function parameters including airway caliber, resistance, conductance, lung volumes, and gas exchange [3]. Their review emphasized that pollution effects vary across populations, with children, older adults, and those with pre-existing conditions being most vulnerable [3]. Both acute airway irritation and complex immunomodulatory alterations result from pollution exposure [3].

Early life vulnerability

Kim., *et al*. examined the critical link between prenatal/perinatal air pollution exposure and later-life respiratory disease development [25]. Their perspective review provided epidemiological evidence that pregnancy and fetal development stages are highly susceptible to environmental exposure, leading to long-term health impacts [25]. Research demonstrated associations between early-life air pollutant exposure and adverse birth outcomes including preterm birth, low birth weight, and lung developmental defects [25]. This work established the developmental origins of health and disease (DOHaD) paradigm for respiratory conditions [25].

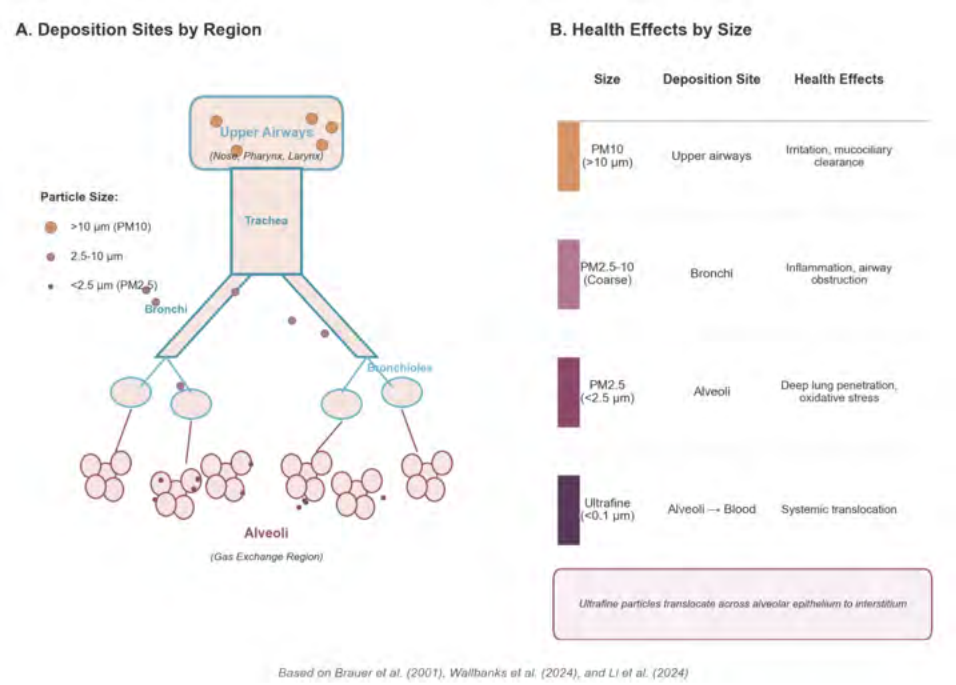

Figure 6

Environmental engineering solutions

Engineering approaches for cross-media pollution control

Addressing the air-water-lung nexus requires integrated engineering solutions. Sheoran., *et al*. reviewed biofiltration as a sustainable pollution management strategy for removing odor, VOCs, and other air pollutants [26]. Their comprehensive analysis emphasized biofiltration's advantages: low cost, straightforward technique, high reduction efficacy, low energy requirements, and minimal residual consequences [26]. Contaminants are adsorbed on medium surfaces and metabolized to benign outcomes through immobilized bacteria and fungi [26].

Applications span multiple sectors including wastewater treatment, food processing, pharmaceutical manufacturing, and agricultural operations [26]. The authors demonstrated that biofiltration provides an economical alternative to costly chemical and microfilter techniques, particularly effective for carcinogenic pollutants [26].

Risk assessment and monitoring frameworks

Van den Berg., *et al*.'s risk matrix for *Legionella* surveillance provides a systematic approach for WWTP monitoring based on biological treatment type, industry type, water temperature, and aeration processes [14]. Their framework enables prioritization of high-risk facilities for targeted intervention, with sequence typing revealing diverse *Legionella* strains [14].

Xiao., *et al*. demonstrated comprehensive pollution tracking using data from China's TAP project, documenting a 48% decrease in national PM2.5 exposure during 2013 - 2020 [27]. Improved air quality prevented 308,000 long-term and 16,000 short-term exposure-related deaths in 2020 compared to 2013 [27]. However, they found O_3 pollution worsened with average April-September exposure

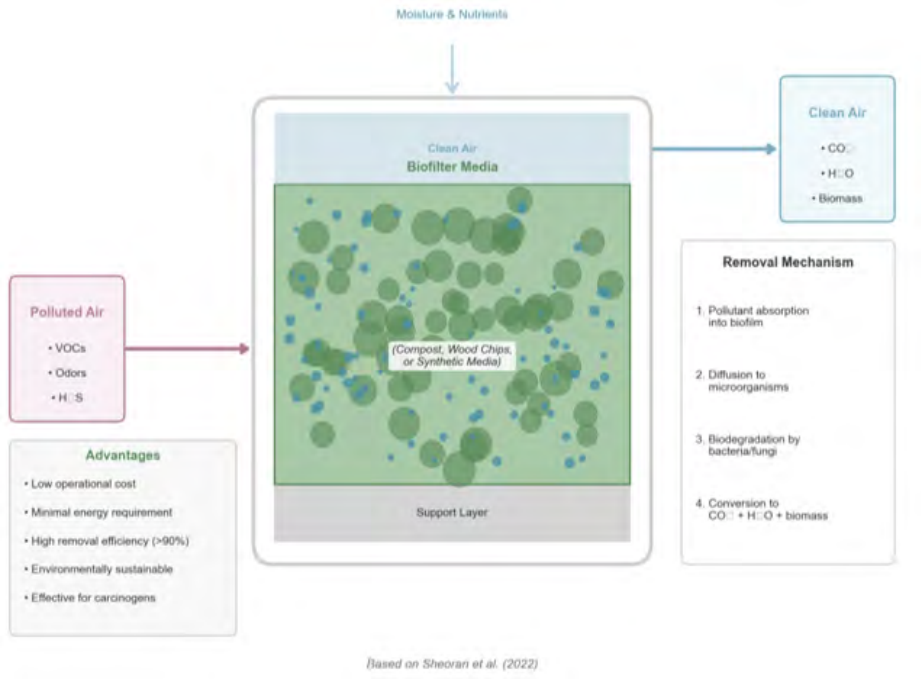

Figure 7

increasing by 17%, and deaths attributable to short-term O_3 exposure exceeded PM2.5 deaths since 2018 [27]. This research demonstrates complex trade-offs in air pollution control requiring integrated approaches [27].

Conclusion and Future Directions

Key findings: This literature review establishes the fundamental interconnectedness of air and water pollution in determining respiratory health outcomes. The evidence synthesized from 25 peer-reviewed studies demonstrates:

1. **Water-to-air pathways pose significant respiratory risks**: Wastewater treatment plants emit bioaerosols containing pathogenic bacteria that disperse over distances exceeding 3 kilometers [9], while harmful algal blooms generate aerosolized cyanotoxins at concentrations far exceeding safety guidelines [18].
2. **Air-to-water pathways create cascading effects**: Atmospheric nutrient deposition promotes eutrophication and algal bloom formation [12,13], ultimately manifesting as respiratory health risks through toxin aerosolization.
3. **The airway microbiome mediates environmental health effects**: Environmental exposures shape respiratory tract microbial communities, which influence individual susceptibility to pollution effects [15].
4. **Causal evidence links air pollution to respiratory disease**: Mendelian randomization studies provide robust evidence for causal relationships between PM2.5, PM10, NO_2 and lung function decline [24].
5. **Early life exposure creates lasting vulnerability**: Prenatal and perinatal air pollution exposure leads to long-term respiratory health impacts through developmental mechanisms [25].

Research gaps and future priorities

Critical knowledge gaps remain in understanding cross-media pollutant transfer and quantifying synergistic health effects. Future research should prioritize:

- Longitudinal studies tracking health outcomes across environmental media.
- Development of integrated monitoring systems for air-water quality.
- Evaluation of engineering intervention effectiveness across multiple pathways.
- Climate change adaptation strategies for environmental health protection.

Implications for practice

The evidence supports adoption of integrated environmental engineering approaches that address multiple pollution sources and exposure routes simultaneously. Biofiltration and other sustainable technologies offer promising solutions for cross-media pollution control [26], while risk assessment frameworks enable targeted intervention at high-risk facilities [14]. Policy development must recognize the fundamental connections between air quality, water quality, and respiratory health to effectively protect vulnerable populations.

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