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Air Pollution Worsens Respiratory Disease, Increases Infection Risk

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Introduction

Ambient air pollution poses a significant threat to respiratory health, exacerbating pre-existing conditions and contributing to the development of new ones. Fine particulate matter (PM2.5) and nitrogen dioxide (NO2) are particularly implicated in driving inflammation, oxidative stress, and airway remodeling, leading to increased symptom severity and a faster decline in lung function in individuals with chronic obstructive pulmonary disease (COPD) and asthma. Understanding these complex mechanisms is paramount for developing effective public health interventions and targeted treatments [1]. The ubiquitous presence of traffic-related air pollution (TRAP) is recognized as a major environmental risk factor for the onset and worsening of asthma, especially in vulnerable pediatric populations. Pollutants such as PM2.5, NO2, and ozone trigger inflammatory responses in the airways, leading to heightened hyperresponsiveness and increased mucus production, with early-life exposures potentially causing long-term respiratory deficits [2]. Indoor air pollution, predominantly from biomass fuel burning and tobacco smoke, significantly impacts respiratory well-being, particularly in resource-limited settings. These pollutants are major contributors to the incidence and progression of serious conditions including COPD, pneumonia, and tuberculosis, underscoring the critical need for improved indoor air quality through cleaner energy sources and smoke-free environments [3]. Exposure to particulate matter (PM) has been consistently linked to a heightened susceptibility to respiratory infections, including viral pneumonias. PM can compromise the functionality of immune cells within the lungs, rendering individuals more vulnerable to infection and potentially resulting in more severe disease outcomes, emphasizing the crucial role of air quality in mitigating severe respiratory illness [4]. Ozone (O3) pollution, especially during warmer months, demonstrably exacerbates asthma control and precipitates symptoms in susceptible individuals. As a potent oxidant, O3 irritates the airways, inducing inflammation and bronchoconstriction, with even brief exposure having considerable clinical implications for those with pre-existing respiratory ailments [5]. The persistent exposure to ambient air pollution is a recognized contributor to the genesis and advancement of interstitial lung diseases (ILDs). Fine and ultrafine particles can instigate inflammatory processes and fibrosis within the lung interstitium, resulting in irreversible lung damage and impaired gas exchange capabilities, thus positioning air pollution as a critical risk factor for ILD development [6]. The combined impacts of air pollution and climate change present an escalating challenge to respiratory health. For instance, extended pollen seasons driven by climate change, when co-occurring with air pollution, can amplify allergic respiratory responses, necessitating a comprehensive understanding of these synergistic effects for robust public health strategies [7]. Research has begun to elucidate the molecular pathways through which PM2.5 contributes to airway inflammation and oxidative stress, factors central to COPD exacerbations. Key inflammatory mediators like IL-6 and TNF-alpha, along with signaling pathways such as NF-⊠B, have been identified, offering potential targets for therapeutic interventions aimed at mitigating pollution-induced lung damage [8]. The aging demographic exhibits a heightened vulnerability to the detrimental respiratory effects of air pollution. Age-related physiological changes in lung function and immune system responses can be amplified by pollutants, leading to an increased prevalence and severity of respiratory diseases among older adults, highlighting a critical public health concern [9]. Studies are increasingly examining the role of air pollution in triggering exacerbations of cystic fibrosis (CF). Exposure to PM2.5 and other airborne pollutants can intensify airway inflammation and increase bacterial colonization, leading to more frequent and severe CF exacerbations, which ultimately compromise lung function and patient prognosis [10].

Description

Ambient air pollution significantly worsens respiratory diseases, particularly COPD and asthma, by promoting inflammation and oxidative stress through pollutants like PM2.5 and NO2. This leads to more severe symptoms and faster lung function decline, underscoring the need for interventions [1]. Traffic-related air pollution (TRAP) is a major environmental factor in asthma development and exacerbation, especially in children, causing airway inflammation and hyperresponsiveness due to pollutants like PM2.5, NO2, and ozone; early exposure may lead to lasting deficits [2]. Indoor air pollution from biomass burning and tobacco smoke heavily impacts respiratory health, especially in low-income areas, contributing to COPD, pneumonia, and tuberculosis, and necessitating cleaner fuels and smoke-free policies [3]. Particulate matter (PM) exposure increases susceptibility to respiratory infections, including viral pneumonias, by impairing lung immune cell function and leading to more severe outcomes, emphasizing air quality's role in preventing serious illness [4]. Ozone (O3) pollution, particularly during warmer periods, exacerbates asthma by causing airway irritation, inflammation, and bronchoconstriction, even with short-term exposure, significantly affecting individuals with pre-existing conditions [5]. Long-term exposure to ambient air pollution promotes the development and progression of interstitial lung diseases (ILDs) by inducing inflammation and fibrosis in the lung interstitium via fine and ultrafine particles, leading to irreversible damage and impaired gas exchange [6]. The synergistic impact of air pollution and climate change poses a growing threat, as seen in how increased pollen seasons due to climate change, combined with air pollution, can worsen allergic respiratory responses, requiring integrated public health strategies [7]. Molecular studies reveal PM2.5 induces airway inflammation and oxidative stress in COPD through mediators like IL-6 and TNF-alpha and pathways such as NF-⊠B, offering potential therapeutic targets [8]. The aging population is particularly susceptible to air pollution's adverse respiratory effects, as age-related lung and immune

changes are amplified by pollutants, resulting in higher rates and severity of respiratory diseases in older adults [9]. Air pollution, specifically PM2.5, exacerbates cystic fibrosis (CF) exacerbations by increasing airway inflammation and bacterial load, leading to poorer lung function and prognosis [10].

Conclusion

Air pollution, both ambient and indoor, significantly worsens respiratory conditions such as COPD, asthma, and interstitial lung diseases, and increases susceptibility to infections. Pollutants like PM2.5, NO2, and ozone drive inflammation and oxidative stress, leading to exacerbated symptoms and faster disease progression. Traffic-related air pollution is a key factor in childhood asthma, while indoor sources like biomass burning and tobacco smoke pose risks in developing regions. Ozone and particulate matter can trigger asthma exacerbations and increase vulnerability to infections. Long-term exposure contributes to ILDs, and the aging population is particularly at risk. Furthermore, air pollution interacts with climate change impacts like extended pollen seasons, worsening respiratory allergies. Molecular mechanisms underlying pollution's effects are being identified, offering therapeutic targets. Air pollution also exacerbates cystic fibrosis exacerbations, negatively impacting patient outcomes.

Acknowledgement

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Conflict of Interest

None.

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