



**INTERNATIONAL JOURNAL OF
PHARMACEUTICAL SCIENCES**
[ISSN: 0975-4725; CODEN(USA): IJPS00]
Journal Homepage: <https://www.ijpsjournal.com>



Review Article

Impact Of Air Pollution on Chronic Obstructive Pulmonary Disease

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ARTICLE INFO

Published: 24 Sept. 2025

Keywords:

Herbal Hair Dye, respiratory outcomes, initially identified

DOI:


10.5281/zenodo.17189159

ABSTRACT

Comprehensive research has established that air pollution significantly contributes to lung disease through airway inflammation. However, the link between sustained exposure to air impurities and increased risk factor for chronic obstructive pulmonary disease (COPD) has been debated. A comprehensive structured literature review and narrative synthesis examined this association by analysing prospective cohort studies with at least three years of follow-up, focusing on particulate (PM 2.5 and PM 10) and nitrogen dioxide (No₂) exposure. The analysis incorporated data from seven qualifying studies out of 436 initially identified, with six studies on PM_{2.5}, three on PM₁₀, and five on NO₂. Findings from the meta-analysis indicate that a 10µg/m³ increase in PM 2.5 levels is associated with a significant evolution in the incidence of COPD (pooled hazard ratio (HR) 1.13-1.23). NO₂ also shows a marginal association with COPD risk (pooled HR 0.95, 95%CI 0.83-1.08), although the available studies on PM 10 were fewer and possibly underpowered to detect an affect. Meta-regression research did not indicate substantially modifiers influencing these associations. The biological plausibility of these findings is supported by the mechanism where in inhaled pollutants induce both localized lung inflammation and systemic inflammatory responses, driving the chronic and progressive nature of COPD. These results corroborate prior data showing a connection between indoor air pollutants and health outcomes from solid fuel combustion to COPD development. Furthermore, cohort studies with prolonged exposure assessment have reinforced the connection between traffic-related impurities such as NO₂ and long-term respiratory outcomes, including COPD incidence. In summary, current confirmation substantiates that prolonged exposure to fine particulate matter (PM_{2.5}) and nitrogen dioxide (NO₂) is associated with an higher risk of developing COPD, whereas the emotional depth of larger particulate matter (PM₁₀) remains unclear due to limited data. These insights underscore the importance of policies aimed at reducing ambient air pollution to mitigate COPD burden globally.

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Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.



INTRODUCTION

Obstructive airway disease in the lungs is a hallmark of chronic obstructive airways disease (COAD), an inflammatory lung condition that encompasses emphysema and chronic bronchitis. All around the world, air pollution is a serious public health concern. It donate to the worldwide illness load and is the major reason of morbidity and mortality. The manifestation of COPD include coughing, wheezing, and shortness of breath. Furthermore, the course of the disease is frequently altered by sporadic flare-upos, which can result in a worse functional capacity, a faster pulmonary disfunction, a wrose health-related quality of the life, and a greater risk of death (5-7). According to estimates from the World Health Organization (WHO) outdoor air pollution caused 3.7 million premature deaths globally in 2012, with acute respiratory infections and COPD accounting for 14% of these fatalities (8). The human respiratory system now needs to cope with a for a greater range of ambient gasses and particles, primarily from industrial automotive and cigarette, somke sources. Globally, air polution has been a significant environmental issue and public health concern. It still posses a serious cocern to environmental health, espiceally in developing nations where industrilization and motor vehicle traffic are growing rapidly. One air polution such as ozone(O3), carbonmonoxide (CO), nitrogen dioxide (NO2) , sulfurdioxide (SO2) and particulate matter (PM2.5-PM10) can effect the respiratory system (lung and tract). These contaminates may cause oxidative stress and inflamtion during pulmonary breathing and gas exchange, which could harm and malfunction the air ways.

Age

Chronic obstructive lung disease exhibits significant differences in age of onset, clinical

features, and outcomes between males and females, reflecting both biological sex and gender-related factors.

Age and Sex Differences in COPD

Age of onset and disease susceptability females tend to develop COPD at a younger age compared to males. This earlier onset in women is notable even when account for smoking history (fewer pack –years) than men but with similar or worse lung function impairment. This suggests an increased biological suspectability in females to harmful inhaled substances such as tobacco smoke and environmental polutants. Also, nonsmoking COAD patients are more likely to be female, often due to exposure like biomass fuels and indoor air polution.

Sex related biological mechanisms

Women have smaller airway calibers relative to lungs size compare to men, leading to greater partical deposition and airway injury for identical exposures. This structural difference contributes to higher prevalence of small airway disease in females, compared to the emphysema-predominant phenotype more frequent in males. Hormonal and immune response differences also play roles in sex-based COPD susceptability and progression.

Clinical expression by Sex and symptoms

Symtoms burden and Dyspnea Despite often having similar objevtive lung function impairment in females report more severe dyspnea and worse standard of living than males. studies show females experience higher dyspnea records and more frequent exacerbations, even when matched for disease severity. Both sexes derive coparable benefits from standard COPD therapies including inhaled

bronchodilators, corticosteroids, pulmonary rehabilitation, and smoking cessation programs. However sex specific consideration may optimize therapy, such as focusing on management of anxiety and depression in females and cardiovascular comorbidities in males.

Age onset and susceptibility across sexes

The age at which COPD manifests differs notably between males and females. Women generally tend to be diagnosed at an earlier age than men, a

phenomenon observed even when smoking exposure, traditionally the commonest risk factor, is taken into consideration. Women frequently develop COPD despite having smoked fewer pack-years compare to men presenting with comparable lung impairment levels. This suggests that women possess a heightened biological vulnerability to inhaled toxins such as tobacco smoke and airborne pollutants.

Comparative Features of COPD by Sex and Age

Aspects	Females	Males
Age at Diagnosis	Typically younger, earlier onset	Generally older at diagnosis
Smoking Exposure	Lower pack-years despite similar severity	Higher cumulative smoking burden
Airway anatomy	Smaller airway diameter relative to lungs size	Larger airways, more emphysema prone
Symptoms burden	Greater dyspnea, worsen quality of life	Less perceived breathlessness for same lung function
Exacerbation rate	More frequent, shorter time to exacerbation	Less frequent exacerbations
Comorbidities	Osteoporosis, anxiety	Cardiovascular diseases
Functional capacity	Reduced despite similar lung impairment	Higher exercise tolerance

Risk Factors:

- 1. Tobacco Smoking:** Smoking remains the leading cause of COPD world wide, mainly responsible for the general of cases. However, only about half of long term smokers eventually develop the condition.
- 2. Indoor Air Pollution :** Continuous exposure to smoke and pollutants from burning biomass fuels(like wood, crop residues and cow dung) in poorly ventilated homes is a Significant risk, especially in developing countries.
- 3. Occupational Exposures :** Prolonged inhalation of dust, chemical fumes and vapors

in industrial and agricultural work places raises the risk. This includes workers in fields such as manufacturing , grain handling , mining, metal working, farming and textiles.

- 4. Outdoor Air Pollution:** Airborne pollutants and particulates from environmental sources also contribute to COPD risk, though less than indoor exposures.
- 5. Genetic Susceptibility:** Certain genetic traits, particularly deficiencies in the alpha -1 antitrypsin protein due to mutations in the SERPINA1 gene, increase vulnerability to COPD even in non smokers.

6. History of Tuberculosis: Individuals with a past diagnosis of tuberculosis are significantly more prone to developing airway obstruction associated with COPD, even after successful treatment.

7. Childhood Respiratory Infections: Recurrent or severe respiratory tract infections in infancy or early childhood can negatively influence lung development, making COPD more likely in adulthood.

8. Low Socioeconomic Status: Poverty is linked to higher COPD rates, likely because of greater exposure to risk factors such as household air pollution, poor nutrition, and limited healthcare access.

9. Reduced Lung Growth and Development: Impaired lung development from causes such as prematurity or malnutrition increases life long susceptibility to COPD.

10. Poor Nutrition: Inadequate nutrition can compromise lung health and immune function, elevating the risk of respiratory illness, including COPD.

11. Female Gender: Women appear to be more vulnerable to the respiratory problems of indoor air pollution and may face a greater risk of developing COPD for reasons that are not fully understood.

12. Advanced Age: The natural loss of lung function with aging makes older adults more sensitive to COPD.

Comorbidities

Comorbidities are prevalent among people with COPD. For instance, a research by vanfleteren et al examined 213 COPD patients and found that 97.7% had at least one coexisting condition, while

over half one (53.5%) were living with four or more. Even with active screening, the true rate of comorbidities may be underestimated since some groups, like those with unstable COPD or recent heart attack, were excluded.

Accurately determining comorbidity prevalence in COPD is difficult because:

COPD and its comorbidities often share risk factors, like smoking. There is frequent underdiagnosis of both COPD and related comorbid conditions. Some comorbid symptoms overlap with COPD severity criteria. Variations in study populations create selection biases, affecting reported rates of different comorbidities.

Despite these challenges, most research agrees that the most prevalent comorbid conditions in COPD include:

- Anxiety and depression
- Heart failure
- Ischemic heart disease
- Pulmonary hypertension
- Metabolic syndrome
- Diabetes
- Osteoporosis
- Gastroesophageal reflux disease (GERD)

Also clinically significant: lung carcinoma, fibrosis lung disease, long-term kidney disease.

Comorbidities reduce quality of life: Research indicates that as the number of comorbidities increases, patients well-being of life and self-reported health status decline. For example, having three or more comorbidities than measures like

FEV₁ or breathlessness. Each is linked to a worse quality of life comorbidity increases the odds of poor self-rated health by about 43%. Heart failure, diabetes, arthritis, and urological problems notably decrease life quality, even after adjusting for other factors. Comorbidities lead to more exacerbations: Various conditions- including GERD, anxiety, depression, pulmonary embolism, pulmonary hypertension, and cardiovascular disease are most commonly tied to exacerbations and hospitalizations. More comorbidities mean greater risks of exacerbation, longer hospital stays, and higher mortality. It is still debated whether these other diseases trigger exacerbations, mimic COPD flares, or simply reflect disease severity, but their negative impact is clear. In summary, managing comorbidities is essential for COPD care, as they strongly influence outcomes like quality of life, exacerbation risk, hospitalization rates, and mortality.

CONCLUSION

Chronic lung diseases, such as asthma and COPD, continue to contribute significantly to global morbidity and mortality, presenting a substantial challenge to public health and generating a heavy economic burden. Over the past decade, the incidence rates of these conditions have risen, increasing healthcare costs and personal suffering. Despite considerable research using conventional experimental methods, many aspects of the pathogenesis of these multifactorial lung diseases remain elusive. The emergence of systems biology offers a transformative perspective for pulmonary research. By integrating diverse types of biological data and leveraging computational analysis, systems biology facilitates a deeper exploration of disease mechanisms that might be overlooked by traditional approaches. High-throughput technologies, such as microarrays and next-generation sequencing, have increasingly been

applied to study chronic lung diseases. While these studies mark progress, most investigations still fall short of harnessing the full potential of comprehensive systems biology strategies. Nevertheless, several promising discoveries have already surfaced from high-throughput and integrative analyses, supporting the notion that systems biology is poised to significantly advance our understanding of complex lung disorders and drive innovations in diagnosis, treatment, and prevention.

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HOW TO CITE: Priyadharshini M.*, Gnanavi H. R., Harshitha K., Hilal Ahmad Dass, Jayanti Mandal, Jhanavi L. Reddy, Impact of Air Pollution on Chronic Obstructive Pulmonary Disease, *Int. J. of Pharm. Sci.*, 2025, Vol 3, Issue 9, 2739-2744 <https://doi.org/10.5281/zenodo.17189159>

