The Growing Burden of Climate Change and Air Pollution on COPD Morbidity and Mortality

Adityo Wibowo[1]

[1]Department of Pulmonology and Respiratory Medicine, Faculty of Medicine, Universitas Lampung

Abstract

Chronic obstructive pulmonary disease (COPD) arises from interactions between genetic susceptibility and long-term exposure to harmful particles or gases. While smoking is known as the primary risk factor, environmental and occupational pollutants (particulate matter, biomass smoke, and diesel emissions) significantly contribute to disease development and progression. Climate change further worsens COPD by increasing ambient particulate matter, ground-level ozone, and extreme temperature events. These exposures induce oxidative stress, airway inflammation, mucus hypersecretion, and structural lung injury, leading to accelerated lung function decline and more frequent exacerbations. Extreme heat and cold further increase hospitalization and mortality in COPD patients, while indoor environmental factors driven by air-conditioning use, humidity, and poor ventilation exacerbate the symptoms. These findings highlight the growing impact of environmental pollution and climate change on COPD burden worldwide.

Keywords: COPD, climate change, environmental pollution

Korespondensi: Adityo Wibowo l Fakultas Kedokteran Universitas Lampung l email : aditpulmo@gmail.com

Introduction

Chronic obstructive pulmonary disease (COPD) is a heterogeneous and progressive respiratory disorder characterized by persistent airflow limitation and chronic respiratory symptoms such as dyspnea, cough, sputum production, and frequent exacerbations. The disease results from structural and functional abnormalities of the airways (chronic bronchitis, bronchiolitis) and alveoli (emphysema), leading to irreversible obstruction and impaired gas exchange. Moreover, early-life factors such as recurrent respiratory infections, impaired lung development, and premature exposure to environmental toxins may reduce maximal lung growth and predispose individuals to COPD later in life.[1]

COPD pathogenesis is primarily driven by prolonged exposure to noxious particles or gases that evoke abnormal and sustained inflammatory responses in the lung. Cigarette smoking remains the predominant risk factor, accounting for nearly 80% of cases in industrialized and developing countries. However, other contributing factors, including genetic susceptibility such as α1-antitrypsin deficiency and polymorphisms in genes regulating inflammation or protease–antiprotease balance, play important roles in disease onset and progression.[1]

Environmental and occupational exposures further exacerbate COPD risk, with chronic inhalation of air pollution, biomass fuel smoke, vehicle emissions, industrial fumes, and dust being well-established contributors. Occupational environments such as mining, construction, and manufacturing are particularly high-risk due to continuous exposure to particulate and chemical irritants.[2,3]

Evidence suggests that climate change and the increasing burden of ambient particulate matter (PM10, PM2.5, and PM0.1) are emerging

contributors to COPD. Rising temperatures, droughts, and wildfires have elevated levels of airborne pollutants, which can induce oxidative stress, airway inflammation, and accelerated decline in lung function. Taken together, the interplay of genetic, environmental, and developmental factors highlights the complex, multifactorial nature of COPD pathogenesis and emphasizes its increasing global health impact in the context of ongoing climate and environmental changes.[4,5]

Environmental Pollution and Lung Injury

Environmental pollution significantly contributes to lung damage beyond the effects of smoking. Climate change adversely impacts lung function through various direct and indirect mechanisms, increasing risks for both healthy individuals and those with pre-existing respiratory conditions. Rising global temperatures lead to more frequent and severe extreme weather events, such as heat waves, wildfires, and droughts, which raise the levels of respiratory irritants and airborne particulate matter.[6,7]

Air pollution from vehicle emissions, industrial sources, and climate-related events has been consistently associated with faster lung function decline, higher rates of acute respiratory failure, and greater overall mortality from cardiopulmonary diseases. This burden is expected to increase in the coming decades as climate change continues to affect air quality, environmental exposures, and the spread of allergens and infectious disease vectors.[7,8]

Air conditioning significantly contributes to increased air pollution, especially in urban areas. Air conditioners are available in one-third of homes worldwide, with higher usage rates in high-income countries. The global average usage of air conditioning is currently about 28%, projected to rise to 41-55% by 2050. Increased air conditioning use leads to higher emissions of nitrogen oxides (NOx) and particulate matter (PM). NOx is a precursor to secondary particulate formation and ozone, which worsens ambient air pollution. This creates a negative feedback loop where urban cooling efforts paradoxically worsen local air quality and atmospheric pollutant levels, thereby increasing respiratory and cardiovascular health risks.[9-11]

In Indonesia, particulate matter (PM2.5) pollution is a major health concern, especially in large cities. Data from 2023-2024 shows that average PM2.5 levels in Indonesia significantly exceed the World Health Organization (WHO) guideline of 5 $\mu g/m^3$. The population-weighted average PM2.5 concentration in Indonesia in 2024 was about 35.5 $\mu g/m^3$, roughly seven times higher than the WHO-recommended limit. This high level of particulate matter poses not only serious health risks but also exacerbates climate change by affecting atmospheric conditions. PM2.5 particles absorb and scatter sunlight, changing the Earth's radiative balance, and serve as nuclei for cloud formation, which influences precipitation patterns and regional weather. Moreover, these pollutants contribute to atmospheric warming by increasing greenhouse gases and

intensifying droughts, which in turn worsen air quality through increased wildfires and dust emissions.[12,13]

Several provincial capitals across Indonesia experience significantly elevated levels of air pollution. The high levels of PM2.5 air pollution become a concern that people living there could gain more than two extra years of life if the air met the WHO clean-air standards. The main causes include factory emissions, vehicle exhaust (especially from diesel engines), burning of waste, dust from construction, and coal-powered plants. Pollution often gets worse during the dry season, heavy rain periods, and droughts, causing thick haze and many days of unhealthy air.[14,15]

Climate Change and The Effect on COPD Patients

Climate change contributes to the worsening of air pollution. Rising temperatures influence both the formation and dispersion of pollutants by altering wind patterns, solar radiation, and precipitation, particularly in urban environments. As a result, climate change has increased ground-level ozone concentrations in already polluted regions, raised emissions of volatile organic compounds (VOCs), and reduced nitrogen oxide (NOx) sequestration. These factors directly contribute to higher mortality and morbidity rates and indirectly exacerbate or increase the risk of pulmonary diseases linked to extreme weather conditions.[10,11]

Rapid climate change induces high levels of particulate matter (PM10, PM2.5, and PM0.1), which has been associated with increased inflammation, oxidative stress, and even progressive fibrotic changes in the respiratory tract. Extreme temperatures and humidity fluctuations further exacerbate respiratory symptoms and acute exacerbations, especially in populations with COPD.[16,17]

Increased exposure to fine particulate matter (PM) and diesel exhaust particles (DEP), significantly worsens COPD through multiple harmful mechanisms. Micro particles of PM2.5 and 0.1 deposits deeply in the small airways and alveoli, inducing chronic inflammation, oxidative stress, and immune cell recruitment (neutrophil, eosinophils, and macrophages), which contribute to airway remodeling and fibrosis. Studies show that PM exposure leads to goblet cell hyperplasia and mucus hypersecretion, increasing airway obstruction and lung resistance. It also promotes fibrotic changes around small airways and parenchymal tissue damage, and worsening airflow limitation.[18,19]

Clinically, exposure to particulate matter (PM) induces COPD symptoms, leads to a decline in lung function, and elevates the incidence of acute exacerbations and hospitalizations. The oxidative stress induced by PM results in mitochondrial impairment and disruption of the epithelial barrier, further worsening pulmonary injury. Prolonged exposure increases the risk of COPD onset and accelerates disease progression. Patients with COPD who are chronically exposed to high levels of particulate matter experience worsening respiratory symptoms, impaired lung mechanics, and decreased quality of life.[19,20]

Studies found that individuals with COPD have an increased risk of

mortality due to exposure to extreme temperatures. During summer, high temperatures can raise the risk of death from COPD by up to 25%. In contrast, cold weather increases the risk of mortality during winter by about 20%, with one study revealing a 4.8% rise in mortality for every 1°C drop in temperature below 11°C over the following month.[21]

The specific causes of death among individuals with COPD during extreme temperature events are not yet well understood. Mortality may be related to increased cardiac stress, but other factors, such as infection-related exacerbations, increased airway inflammation, damage to the airway epithelium, and cold-induced bronchoconstriction, are also likely to play important roles. Beyond mortality, exposure to both heat and cold has been linked to a higher risk of COPD-related hospital admissions. For instance, a study in New York City reported a 7.6% increase in COPD hospitalizations for every 1 °C rise above a threshold of 29 °C. Similarly, research from Taiwan showed a 0.8% increase in COPD exacerbations for every 1 °C decrease in the average daily temperature.[21-23]

Climate change influences both the emission and the spread of primary air pollutants, especially particulate matter, and also increases the formation of secondary pollutants such as ground-level ozone. Growing evidence shows that air pollution is associated with worse symptoms, higher exacerbation rates, and an increased risk of death in individuals with COPD. Several studies have also reported synergistic effects between heat and air pollution, leading to higher rates of all-cause mortality, respiratory-related mortality, and hospital admissions in the general population. In Taipei, Taiwan, elevated levels of ozone and PM2.5 were found to increase the risk of hospital admissions on hotter days.[10,23]

Studies show that many people with more severe disease of COPD spend most of their time indoors due to physical limitations or dependence on oxygen devices. Indoor temperature and humidity can vary widely even when outdoor conditions are similar, and are influenced by factors such as building design (e.g., insulation, heating, sunshades, and ventilation), socioeconomic conditions, and daily activities like cooking or bathing. Indoor temperatures are often higher than outdoor temperatures, even during heatwaves, and high indoor humidity can worsen heat-related stress by reducing the body's ability to cool itself. Elevated humidity may also promote the growth of microorganisms inside the home. Together, these climate-related indoor environmental changes can worsen the clinical condition of patients with COPD.[16,24]

Conclusion

In summary, climate change has become a growing concern that worsens air pollution and increases exposure to extreme temperatures. These climate-related extremes, such as severe heat or cold, can directly affect the airways of people with COPD, triggering symptoms and exacerbations. Together, these environmental pressures now play a significant role in the overall burden and progression of the disease.

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