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PHYSICAL ENVIRONMENTAL INFLUENCES ON SILICOSIS: A NARRATIVE REVIEW INTEGRATING COMMUNITY EXPOSURE AND WISTAR RAT EXPERIMENTAL FINDINGS IN COAL-HANDLING REGIONS

PENGARUH LINGKUNGAN FISIK TERHADAP SILIKOSIS: TINJAUAN NARATIF YANG MENGINTEGRASIKAN PAPARAN PADA MASYARAKAT DAN TEMUAN EKSPERIMENTAL TIKUS WISTAR DI KAWASAN PENANGANAN BATUBARA

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Abstract

Coal mining, handling, and transportation activities are major sources of airborne particulate matter containing respirable crystalline silica, which poses significant risks to respiratory health. Silicosis remains a serious occupational and environmental disease affecting not only workers but also communities living near coal-handling areas. Physical environmental factors, including air quality, temperature, humidity, and wind speed, play an important role in influencing dust generation, dispersion, and inhalation exposure. This narrative review aims to synthesize current evidence on the influence of physical environmental conditions on silica exposure and silicosis development, integrating findings from environmental monitoring studies, epidemiological research, and experimental Wistar rat models. A literature search was conducted using major scientific databases to identify relevant peer-reviewed articles published between 2010 and 2024. The reviewed evidence indicates that prolonged or high-intensity exposure to silica dust is strongly associated with chronic pulmonary inflammation and progressive fibrosis. Environmental conditions can exacerbate exposure risk by increasing airborne particulate concentrations and respiratory vulnerability. Experimental studies using Wistar rats provide mechanistic insights into silica-induced lung injury, supporting epidemiological observations in human populations. This review highlights the importance of integrating environmental, occupational, and biological perspectives to improve risk prediction, early detection, and preventive strategies for silicosis in coal-handling regions.

Keywords: silicosis; crystalline silica; coal handling; physical environment; Wistar rat

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Abstrak

Kegiatan pertambangan, penanganan, dan transportasi batubara merupakan sumber utama partikulat udara yang mengandung silika kristalin respirabel dan berisiko tinggi terhadap kesehatan pernapasan. Silikosis masih menjadi masalah kesehatan kerja dan lingkungan yang serius, tidak hanya pada pekerja tetapi juga pada masyarakat yang tinggal di sekitar kawasan penanganan batubara. Faktor lingkungan fisik, seperti kualitas udara, suhu, kelembaban, dan kecepatan angin, berperan penting dalam mempengaruhi pembentukan, penyebaran, dan tingkat paparan debu silika. Tinjauan naratif ini bertujuan untuk mensintesis bukti ilmiah terkait pengaruh lingkungan fisik terhadap paparan silika dan perkembangan silikosis dengan mengintegrasikan hasil penelitian pemantauan lingkungan, studi epidemiologi, dan model hewan tikus Wistar. Penelusuran literatur dilakukan pada basis data ilmiah utama untuk artikel yang dipublikasikan antara tahun 2010–2024. Hasil telaah menunjukkan bahwa paparan debu silika jangka panjang atau intensitas tinggi berhubungan erat dengan inflamasi paru kronis dan fibrosis progresif. Model tikus Wistar memberikan pemahaman mekanistik yang mendukung temuan pada manusia. Pendekatan terpadu ini penting untuk meningkatkan prediksi risiko, deteksi dini, dan upaya pencegahan silikosis di kawasan penanganan batubara.

Kata kunci: silikosis; silika kristalin; penanganan batubara; lingkungan fisik; tikus Wistar

1. INTRODUCTION

Coal mining, handling, and distribution activities are essential components of the global energy supply chain and contribute substantially to economic development, particularly in resource-rich countries such as Indonesia. Despite their economic importance, these activities are widely recognized as major sources of environmental pollution and occupational health hazards. One of the most significant concerns is the generation of airborne particulate matter containing crystalline silica, released through mechanical processes such as blasting, drilling, crushing, loading, and transportation of coal and associated rock materials (Ivanova et al., 2022; Vanka K. S. et al., 2022). When inhaled over prolonged periods, respirable crystalline silica poses a serious threat to respiratory health, leading to the development of silicosis and other chronic lung diseases.

Globally, silica exposure remains a persistent occupational hazard across mining regions in Asia, Africa, Europe, and the Americas. Epidemiological evidence consistently demonstrates high rates of silicosis among exposed workers, with studies reporting substantial radiographic abnormalities and progressive massive fibrosis in heavily exposed populations (Hoy et al., 2022). In the United States alone, approximately 1.7 million workers are estimated to be exposed to respirable crystalline silica annually, particularly those engaged in high-risk tasks such as drilling and roof bolting (Madureira et al., 2023). Similar patterns are observed in low- and middle-income countries, where inadequate ventilation, dry drilling techniques, and prolonged exposure durations further amplify disease risk (Austin et al., 2021).

In Indonesia, coal-handling zones represent an important yet underexplored source of community-level exposure. Environmental assessments conducted in the Kertapati Port area of Palembang have revealed silica contents ranging from 24% to 46% in collected dust samples, indicating a substantial potential for inhalation exposure among both workers and nearby

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residents (Wahyudi et al., 2022). This finding is particularly concerning given evidence that silica-related mortality and respiratory disease risk persist even among non-smokers and increase proportionally with exposure intensity (Steenland & Ward, 2014; Requena-Mullor et al., 2021). Recent studies have further quantified respirable dust and respirable crystalline silica concentrations in coal mining operations at levels capable of inducing long-term pulmonary injury (Gbondo et al., 2024).

Animal models, particularly Wistar rats, have been extensively employed to investigate the progression and mechanisms of silicosis. Their pulmonary physiology closely resembles that of humans, making them a valuable translational model for studying chronic lung disease. Experimental approaches using repeated intranasal instillation of crystalline silica effectively reproduce occupational exposure patterns, inducing inflammatory and fibrotic changes consistent with human silicosis (Cao et al., 2023; Li et al., 2021). These models bridge the gap between in vitro studies and human epidemiological observations, providing critical insights into disease pathogenesis and potential intervention strategies.

Environmental conditions also modulate biological responses in experimental animal models. Temperature, humidity, and airflow affect thermoregulation, metabolism, immune function, and disease susceptibility in laboratory rodents. Deviations from thermoneutral conditions may induce physiological stress, while improper humidity and ventilation can alter respiratory outcomes and experimental reproducibility (Kasza et al., 2023; Lee et al., 2019). Consequently, understanding environmental influences is essential not only for exposure assessment but also for interpreting experimental findings in silicosis research.

Given the complex interplay between environmental parameters, silica exposure, and biological responses, an integrated perspective is needed to better understand silicosis risk in coal-handling regions. This narrative review synthesizes current evidence from environmental monitoring studies, epidemiological investigations, and Wistar rat experimental models to elucidate how physical environmental factors influence silica exposure and silicosis development. By integrating community-level exposure data with experimental findings, this review aims to inform risk prediction, early detection strategies, and preventive interventions, ultimately contributing to improved occupational and environmental health protection in coal-handling areas.

2. RESEARCH METHOD

This study employed a narrative review design to synthesize existing evidence on the influence of physical environmental factors on silicosis in coal-handling regions, integrating findings from environmental studies, human populations, and Wistar rat experimental models. This approach was chosen to allow flexible and integrative interpretation of heterogeneous study designs.

A literature search was conducted using PubMed, Scopus, Web of Science, ScienceDirect, and Google Scholar, covering publications from 2010 to 2024. Search terms included combinations of *silicosis, crystalline silica, coal mining, coal handling, environmental exposure, temperature, humidity, wind speed, community exposure,* and *Wistar rat*. Reference lists of relevant articles were also screened to identify additional sources.

Eligible studies included peer-reviewed articles written in English that examined coal-related silica exposure, silicosis outcomes, environmental parameters, human populations, or

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experimental animal models. Editorials, conference abstracts without full text, and non-respiratory studies were excluded.

Relevant information was extracted narratively, focusing on exposure characteristics, environmental conditions, study population or animal model, and respiratory outcomes. The findings were synthesized thematically to integrate evidence on environmental determinants, exposure pathways, and biological responses related to silicosis. As this review analyzed published literature only, ethical approval was not required.

3. RESULTS AND DISCUSSION

3.1. Influence of the Physical Environment on Respiratory Health

The literature synthesis demonstrates that the physical environment is a critical determinant of public health, particularly respiratory health. Environmental components such as air quality, ambient temperature, relative humidity, wind speed, noise, and lighting collectively shape external conditions that directly influence human physiological function and the body's ability to maintain respiratory homeostasis. These factors interact to determine thermal comfort, inhaled air quality, and the effectiveness of respiratory defense mechanisms.

Air pollution, especially exposure to fine particulate matter such as PM_{10} and $PM_{2.5}$, is consistently associated with increased incidence of both acute and chronic respiratory diseases. These particles are capable of penetrating deep into the lower respiratory tract and alveolar regions, triggering inflammatory responses, oxidative stress, and progressive impairment of lung function. Numerous studies have reported strong associations between elevated particulate concentrations and higher prevalence of chronic obstructive pulmonary disease (COPD), asthma, chronic bronchitis, and pulmonary fibrosis across different age groups, with greater vulnerability observed among children, the elderly, and individuals with pre-existing respiratory conditions (Andrade et al., 2023; Zhang et al., 2022).

Meteorological factors further modulate the health effects of air pollution. Elevated ambient temperatures can accelerate atmospheric chemical reactions and increase ground-level ozone formation, exacerbating respiratory irritation. Relative humidity that deviates from optimal levels impairs mucociliary clearance, increases susceptibility to respiratory infections, and worsens symptoms in individuals with asthma and COPD. Low wind speed contributes to air stagnation and pollutant accumulation, while high wind speed may facilitate long-range dispersion of dust and airborne allergens into residential areas (Liu et al., 2024).

3.2. Silica Dust as a Primary Risk Factor for Silicosis

Respirable crystalline silica dust is identified as one of the most hazardous environmental and occupational airborne pollutants affecting the respiratory system. Its microscopic size, lack of color, and absence of odor make exposure difficult to detect without specialized monitoring, particularly in workplaces with insufficient dust control. Respirable silica particles can remain suspended in the air for extended periods and are easily inhaled into the alveolar spaces.

The reviewed literature indicates that mining, construction, and coal handling and transportation activities are major sources of silica dust exposure in both occupational settings and surrounding residential environments. Processes such as excavation, crushing, grinding, hauling, and stockpiling significantly elevate airborne silica concentrations, especially in open

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environments influenced by wind speed and direction (Gbondo et al., 2024; Requena-Mullor et al., 2021).

Importantly, silica exposure is not confined to workers. Communities residing near mining sites, coal ports, and transport corridors are chronically exposed to airborne silica transported by wind and industrial activities. Studies conducted in Indonesia have reported increased respiratory complaints, higher rates of acute respiratory infections, and early radiological indicators consistent with silicosis among populations living near coal mining and port areas. These findings highlight that silica dust constitutes a broader environmental health concern rather than an exclusively occupational issue (Putro et al., 2024; Salawati, 2017).

3.3. Pathophysiological Mechanisms of Silicosis Induced by Silica Dust Exposure

The synthesis of available evidence confirms that silicosis develops through chronic inflammatory and progressive fibrotic mechanisms within lung tissue. Respirable silica particles deposited in the alveoli are phagocytosed by alveolar macrophages as part of the lung's innate defense system. However, the cytotoxic and abrasive properties of crystalline silica lead to macrophage injury and apoptosis, impairing effective particle clearance.

Damaged macrophages release a cascade of proinflammatory mediators, including tumor necrosis factor-alpha (TNF-α), interleukin-1β (IL-1β), interleukin-6 (IL-6), and reactive oxygen species (ROS). Activation of the NLRP3 inflammasome amplifies inflammatory signaling, while upregulation of transforming growth factor-beta (TGF-β) stimulates fibroblast proliferation and excessive collagen deposition. These processes culminate in the formation of fibrotic nodules and diffuse pulmonary fibrosis, resulting in reduced lung elasticity and impaired gas exchange (Hoy et al., 2022; Martínez-López et al., 2023).

Notably, silicosis is a progressive disease that may continue to advance even after cessation of exposure. High-intensity exposure over short durations can result in acute silicosis with rapid respiratory deterioration and high mortality risk, whereas prolonged low-level exposure more commonly leads to chronic or accelerated forms of the disease.

3.4. Role of Exposure Duration and Concentration

Exposure duration and airborne silica concentration are key determinants of silicosis severity. Long-term exposure, even at relatively low concentrations, promotes cumulative silica retention in lung tissue, leading to persistent inflammation and progressive fibrosis. The cumulative exposure dose largely explains why silicosis is frequently diagnosed at advanced stages, when lung damage has become irreversible.

Conversely, short-term exposure to extremely high concentrations can precipitate acute silicosis characterized by intense alveolar inflammation and rapid respiratory failure. The interaction between exposure duration and concentration accounts for the heterogeneous clinical manifestations observed among miners, construction workers, and environmentally exposed populations. Inadequate use of personal protective equipment and ineffective dust suppression strategies further increase disease risk (Baum & Arnold, 2023; Cao et al., 2023).

3.5. Impact of Silica Dust Exposure on Workers and Surrounding Communities

Case studies involving mining and construction workers consistently report higher prevalence of silicosis and impaired lung function compared to the general population. Symptoms such as chronic cough, progressive dyspnea, and reduced pulmonary capacity

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commonly appear after 5–10 years of exposure. Radiological imaging and spirometry often reveal fibrotic changes and ventilatory impairment among affected workers (Estaurina et al., 2024; Sinaga et al., 2020).

Beyond occupational settings, communities living near industrial and coal-handling areas face elevated risks due to environmental dispersion of silica-containing dust. Coal port operations and transportation activities significantly increase ambient PM_{10}, $PM_{2.5}$, and silica concentrations. These exposures adversely affect human health and contribute to environmental degradation, including vegetation stress, soil contamination, and disruption of aquatic ecosystems due to particulate deposition (Wahyudi, 2022; Setiawan et al., 2018).

3.6. Wistar Rats as an Experimental Model for Silicosis

The reviewed evidence supports the use of Wistar rats as a relevant experimental model for silicosis research due to similarities in inflammatory and fibrotic lung responses between rodents and humans. Silica exposure in Wistar rats induces histopathological changes including inflammatory cell infiltration, elevated proinflammatory cytokine expression, and excessive collagen deposition, closely resembling human silicosis pathology. This model enables detailed investigation of molecular mechanisms and preclinical evaluation of antifibrotic therapies (Vanka et al., 2022; Jiao et al., 2021).

Nevertheless, interspecies differences in lung anatomy, lifespan, and metabolic processes represent important limitations. Consequently, findings from animal models should be interpreted cautiously and integrated with epidemiological and clinical human data to ensure translational relevance.

4. CONCLUSION

Occupational environments and residential areas located near industrial and mining activities demonstrate heightened vulnerability to adverse physical environmental conditions. Inadequate ventilation, high industrial activity density, and insufficient emission control measures lead to sustained exposure to harmful airborne pollutants. This exposure affects not only workers but also surrounding communities, contributing to long-term declines in lung capacity, increased respiratory morbidity, and a growing public health burden.

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