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Particulate Matter (PM2.5) Exposure: Implications for Public Health and Respiratory Diseases in Urban and Rural Areas

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ABSTRACT

Fine particles smaller than 2.5 micrometers, known as particulate matter (PM2.5), are a serious threat to public health worldwide, especially when it comes to respiratory conditions. This review highlights the distinctions between urban and rural settings while synthesizing the most recent data on PM2.5 sources, exposure patterns, and health effects. PM2.5 is mostly produced by automobile emissions, industry, and construction in cities, which results in continuously high concentrations. On the other hand, transboundary pollution frequently makes PM2.5 worse in rural areas, where it is caused by burning biomass, agricultural practices, and seasonal dust. Through processes like oxidative stress, inflammation, and epigenetic modifications, PM2.5 exposure causes both acute respiratory problems like bronchitis and asthma flare-ups as well as chronic illnesses like lung cancer and chronic obstructive pulmonary disease (COPD). While rural communities experience seasonal spikes and substantial indoor pollution from biomass combustion, urban populations are exposed on a chronic basis. Children, the elderly, and low-income groups are among the vulnerable groups that suffer disproportionately. Important discoveries highlight the pressing need for improved air quality monitoring, more stringent laws, and customized interventions to lessen the negative health effects of PM2.5. To improve our understanding of toxicity mechanisms and guide policy, future research should focus on long-term exposure studies, the impact of climate change on PM2.5 dynamics, and integrative techniques like multi-omics. In order to address urban-rural disparities and lessen the effects on global health, sustainable, context-specific solutions are essential.

INTRODUCTION

A complex mixture of microscopic particles suspended in the atmosphere, particulate matter (PM) is divided into three categories based on aerodynamic diameter: PM10 (<10 µm), PM2.5 (<2.5 µm), and PM1 (<1 µm). Because PM2.5 can enter the bloodstream and travel deep into the lungs, it poses a serious risk to human health (1). Since air pollution has become a major environmental health concern and causes millions of premature deaths every year, research on PM2.5 has gained attention in recent decades. A thorough grasp of PM2.5 exposure's effects in various contexts, especially urban and rural ones, is imperative due to its widespread occurrence worldwide and its varied sources and effects (2).

The Industrial Revolution marked the start of serious research on air pollution, with the initial studies concentrating on coarse particles and visible smog. Epidemiological evidence connecting PM2.5 to cardiovascular and respiratory disorders led to its recognition as a critical pollutant in the late 20th century. Significant incidents, like the London Smog in 1952, brought attention to the fatal effects of air pollution and sparked improvements in regulation and monitoring (3). Global health organizations such as the World Health Organization (WHO) have made PM2.5 a priority and have established strict guidelines to reduce its impact (4).

With an emphasis on urban rural disparities, this review attempts to compile the most recent information on PM2.5 sources, composition, exposure patterns, and health effects. It offers researchers and policymakers insights by examining toxicity mechanisms, public health implications, and mitigation strategies. The review compares urban and rural settings to show the need for customized interventions to address the health burdens

associated with PM2.5 and suggests future research directions to address this urgent global issue.

Sources and Composition of PM2.5

Anthropogenic and Natural Sources

There are numerous anthropogenic and natural sources of PM2.5, each of which contributes unique chemical profiles and exposure risks. In urban settings, where vehicle emissions from gasoline and diesel engines release fine particles containing carbon compounds and heavy metals, anthropogenic sources predominate (5). Through material handling and combustion processes, industrial operations like manufacturing, power generation, and chemical processing release PM2.5, which frequently releases trace metals, nitrates, and sulfates (6). Particularly in densely populated areas, domestic activities such as heating and cooking with solid fuels also make a substantial contribution (7). Burning biomass, which includes wood, crop waste, and manure used for heating or cooking, is a major source of organic carbon and volatile organic compounds in rural areas (8). Dust and chemical aerosols produced by agricultural practices, such as tillage and pesticide application, raise PM2.5 levels in rural areas (9). Natural sources include sea spray, which introduces aerosols based on sodium and chloride in coastal areas, and dust storms, which move mineral particles in arid regions (10). Due to the increased frequency of wildfires brought on by climate change, both urban and rural areas are greatly impacted by the substantial PM2.5 that these fires release, which contains organic and elemental carbon (11). Even though they are less common, volcanic eruptions have the ability to release PM2.5 that is high in sulfates into the atmosphere, which can have a significant impact on air quality over long distances. Because PM2.5 from urban industrial centers can spread to rural areas, increasing exposure, transboundary pollution makes source attribution even more difficult (12).

Chemical and Physical Composition

The physical and chemical characteristics of PM2.5, a heterogeneous mixture, differ depending on its source and location. Chemically, it is made up of heavy metals (like lead, cadmium, and arsenic from industrial processes), inorganic ions (like sulfates and nitrates from industrial emissions), organic carbon (like polycyclic aromatic hydrocarbons from combustion), and elemental carbon (like soot from diesel engines or burning biomass) (13). Additionally, there are biological elements like bacteria, fungus spores, and pollen, especially in rural regions with a lot of vegetation or agricultural activity. Long-range transport is made possible by PM2.5 particles' physical smallness (<2.5 μm), which allows them to float in the atmosphere for days or weeks (14). Due to industrial and vehicular sources, urban PM2.5 typically contains a higher percentage of secondary aerosols (created by atmospheric reactions) and heavy metals (15). On the other hand, organic carbon and potassium from burning biomass frequently account for the majority of rural PM2.5, with seasonal increases occurring during harvest or wildfire seasons. The composition affects toxicity; biological components may cause allergic or inflammatory reactions, while organic compounds and heavy metals increase oxidative potential. Deposition in the respiratory tract is also influenced by particle size and surface area; smaller PM2.5 fractions present higher health risks because of their deeper lung penetration (16).

Urban vs. Rural PM2.5: Distribution and Characteristics

Urban Settings

Due to heavy traffic, industrial processes, and construction, PM2.5 concentrations in urban areas are often elevated, often ranging from 20 to 100 μg/m^3 in megacities (17). Significant amounts of black carbon and ultrafine particles are contributed by vehicle emissions, such as exhaust from cars, trucks, and buses, especially during peak traffic hours (16). PM2.5 containing sulfates, nitrates, and heavy metals is released by industrial sources like factories and power plants; emissions vary depending on the industrial activity and the application of regulations. Dust from demolition and material handling on construction sites contributes both fine and coarse particles to the urban air (18). Pollutant hotspots with complex chemical mixtures can be created by urban infrastructure, such as tall buildings, and high population density. Secondary aerosol formation, in which gases such as sulfur dioxide and nitrogen oxides react in the atmosphere to form fine particles, also affects urban PM2.5 levels and raises them during periods of stagnant weather. For urban dwellers, these factors lead to chronic exposure, which has serious consequences for cardiovascular and respiratory health (19).

Rural Settings

Although PM2.5 levels are typically lower in rural areas, they can rise to 50–150 μg/m^3 during certain activities, like burning biomass or during agricultural seasons (20). A major source of organic carbon and volatile compounds is the burning of wood, crop waste, or manure for cooking and heating (21). Dust and chemical aerosols are produced by agricultural operations like plowing, harvesting, and burning crop stubble, especially during dry seasons. Mineral-based PM2.5 is derived from natural sources such as wind-blown dust from fields or unpaved roads, whereas seasonal wildfires release substantial amounts of organic and elemental carbon (22). Depending on wind patterns, transboundary pollution from urban or industrial areas can raise PM2.5 levels in rural areas by hundreds of kilometers. Rural PM2.5 is more affected by primary emissions from burning biomass than by secondary aerosols, which can cause acute exposure events during burning seasons. In rural households, inadequate ventilation and infrastructure make indoor PM2.5 levels worse, frequently exceeding outdoor concentrations (23).

Spatial and Temporal Variations

Significant temporal and spatial variations in PM2.5 levels are caused by environmental factors and source activity. Traffic-related morning and evening rush hours in urban areas cause diurnal patterns to peak, whereas evening cooking or seasonal burning periods cause spikes in rural areas (24). There are noticeable seasonal variations, with harvest seasons raising PM2.5 in rural areas due to crop burning and winter inversions trapping PM2.5 in urban valleys. The dispersion and deposition of PM2.5 are significantly influenced by meteorological factors, such as

temperature, humidity, wind speed, and precipitation (25). For instance, rain can lower airborne concentrations of PM2.5, while low wind speeds and high humidity can increase PM2.5 accumulation. Within regions, there are clear spatial differences: urban centers have higher PM2.5 levels near major roads or industrial zones, whereas rural areas may have isolated hotspots near dusty fields or burning sites (26). Because urban sources of PM2.5 can affect rural air quality and vice versa during wildfire events, long-distance transport also contributes to spatial variability (27).

Indoor vs. Outdoor Exposure Differences

Due to different sources and ventilation, indoor PM2.5 exposure is a major concern in both urban and rural settings, and it frequently varies from outdoor levels. Cooking (particularly gas stoves), heating systems, tobacco smoke, and the infiltration of outdoor pollutants through windows or ventilation systems are all sources of PM2.5 in urban homes (28). PM2.5 can build up in poorly ventilated apartments in crowded cities, especially in low-income housing with inadequate air filtration (29). Burning biomass, such as wood, manure, or crop residues, for cooking or heating is the main source of indoor PM2.5 in rural areas. This process releases large amounts of organic carbon and particulate matter. Rural homes, which are frequently built with inexpensive materials, have inadequate ventilation, which causes indoor PM2.5 concentrations to be two to five times higher than outdoor levels, particularly when cooking (30). There are differences in outdoor-to-indoor transport as well: dust or smoke from wildfires enters homes in rural areas, whereas PM2.5 from traffic is more prevalent in urban areas. These variations underline the necessity of focused interventions, like better ventilation systems in cities and better cookstoves in rural areas (31).

Mechanisms of PM2.5-Induced Toxicity

Inhalation and Deposition Pathways

Because PM2.5 particles are small (less than 2.5 μm), they can enter the respiratory system deeply and make their way to the alveoli, which are where gas exchange takes place (32). Because of its deep deposition, PM2.5 can interact with lung epithelial cells and occasionally move into the bloodstream, which can have systemic effects (33). PM2.5's aerodynamic characteristics increase its potential for harm by allowing it to evade the filtration mechanisms of the upper respiratory tract, including cilia and mucus (34). Because they can penetrate the alveolar-capillary barrier and enter the systemic circulation, ultrafine particles (<0.1 μm) in the PM2.5 fraction are especially dangerous because they may have an impact on organs other than the lungs, such as the heart and brain (35). In addition to individual factors like lung anatomy and breathing patterns, PM2.5 deposition efficiency is also influenced by particle size, shape, and density. For instance, faster breathing or impaired lung function may result in higher deposition rates in children and people with pre-existing respiratory conditions. Since PM2.5 can cause both acute and long-term health effects due to its deep lung penetration and persistence, toxicological research must concentrate on this issue (36).

Cellular and Molecular Effects

Because of its chemical makeup and physical characteristics, PM2.5 causes toxicity through a variety of cellular and molecular pathways. One important mechanism is oxidative stress, which is brought on by substances like polycyclic aromatic hydrocarbons (PAHs) and heavy metals that produce reactive oxygen species (ROS) (37). Inflammation and cell death are brought on by ROS overpowering the body's antioxidant defenses, which results in lipid peroxidation, protein damage, and DNA strand breaks (38). Airway inflammation and systemic effects are caused by pro-inflammatory cytokines (e.g., IL-6, TNF-α) released by lung macrophages and epithelial cells, which mediate inflammatory responses. Additionally, PM2.5 causes epigenetic alterations like DNA methylation and histone modification, which can change the expression of genes linked to inflammation and cell repair and make people more vulnerable to chronic illnesses (39). Furthermore, PM2.5 alters the microbial communities in the lungs and promotes persistent inflammation or infections by upsetting the respiratory microbiome. Toxic metabolic byproducts can result from the activation of the aryl hydrocarbon receptor (AhR) by organic substances such as PAHs. Cellular signaling pathways, including those controlling apoptosis and immune responses, are hampered by heavy metals like lead and cadmium (40). Due to its metal-rich composition, urban PM2.5 frequently exhibits higher potency than rural PM2.5, which is dominated by organic compounds. These combined effects increase the toxicity of PM2.5 (41).

PM2.5 and Respiratory Health: Evidence from Urban and Rural Populations

Acute Health Effects

Acute respiratory effects, such as bronchitis, airway irritation, and asthma flare-ups, are strongly linked to short-term exposure to PM2.5. Increased hospital admissions and ER visits for respiratory symptoms are associated with spikes in PM2.5 levels, which frequently occur during pollution events like smog or wildfires, according to epidemiological studies (42). For instance, hospitalizations related to asthma can increase by 2–5% with a 10 $\mu g/m^3$ increase in PM2.5 concentration. Frequent exposure to traffic-related PM2.5, which contains high levels of black carbon and ultrafine particles that cause airway inflammation, has a particularly negative impact on urban populations (43). Seasonal biomass burning in rural areas is frequently associated with acute effects, such as coughing, wheezing, and decreased lung function due to smoke exposure, particularly in households that cook with wood or dung. During PM2.5 spikes, children and people with pre-existing conditions, like asthma, have worsened symptoms; urban children are more likely to visit the emergency room because of chronic exposure (44). These acute effects are caused by the inflammatory response to PM2.5, which is fueled by oxidative stress and cytokine release. The severity of symptoms varies depending on the particle composition and length of exposure (45).

Chronic Respiratory Outcomes

Seasonal biomass burning in rural areas is frequently associated with acute effects, such as coughing, wheezing,

and decreased lung function due to smoke exposure, particularly in households that cook with wood or dung. During PM2.5 spikes, children and people with pre-existing conditions, like asthma, have worsened symptoms; urban children are more likely to visit the emergency room because of chronic exposure (44). These acute effects are caused by the inflammatory response to PM2.5, which is fueled by oxidative stress and cytokine release. The severity of symptoms varies depending on the particle composition and length of exposure (45). Chronic exposure to biomass smoke, which is high in organic carbon, causes interstitial lung disease and COPD in rural areas, especially in women and older people who cook (46). Both environments increase the risk of lung cancer, but the carcinogenic elements of urban PM2.5 such as PAHs and heavy metals present a bigger risk (47). Additionally, long-term exposure damages children's lung development, which lowers lung function as an adult. These chronic outcomes are caused by the cumulative effects of PM2.5, which are mediated by epigenetic modifications and disruption of the microbiome. Urban-rural differences reflect different exposure patterns (48).

Differential Impacts: Urban vs. Rural

Due to variations in exposure patterns and particle composition, PM2.5 has different health effects on urban and rural populations. Due to metal-rich and secondary aerosol-laden PM2.5 from industry and traffic, chronic exposure to high PM2.5 levels (20–100 μg/m^3) in urban areas is associated with higher rates of chronic diseases like lung cancer and COPD [79]. Due to systemic inflammation caused by PM2.5 translocation into the bloodstream, urban dwellers are also at increased cardiovascular risk (49). On the other hand, burning biomass and agricultural practices cause seasonal PM2.5 spikes (50–150 μg/m2) in rural areas, which can cause acute respiratory effects and seasonal exacerbations of chronic conditions (49). Women and children are disproportionately affected by indoor PM2.5 from burning biomass in rural homes, which is frequently two to five times higher than outdoor levels and raises the risk of acute respiratory infections and COPD (50). Complexity is increased by transboundary pollution, as wildfire smoke affects both environments and urban PM2.5 can increase exposure in rural areas. With rural areas needing clean cooking technologies and urban areas needing emission controls, these disparities underscore the necessity of context-specific health interventions (51).

Vulnerable Populations

Because of physiological, socioeconomic, or occupational factors, some groups are more vulnerable to the negative health effects of PM2.5. Children are particularly at risk because of the increased PM2.5 deposition caused by their developing lungs and faster breathing rates, which raises the risk of asthma and stunts lung growth (52). Due to their weakened immune systems and diminished lung capacity, older adults are more susceptible to COPD exacerbations and death; a 10 μg/m^3 increase in PM2.5 is associated with a 1-2 percent increase in respiratory mortality. Exposure to PM2.5 during pregnancy increases the risk of poor birth outcomes, including low birth weight, which can put babies at risk for respiratory problems (53). Low-income groups are disproportionately exposed because they frequently reside close to sources of pollution, such as factories or highways in cities, or depend on biomass fuels in rural areas. Acute and chronic respiratory conditions are more common in occupational groups like farmers who are exposed to dust from agriculture or city traffic workers who breathe in exhaust (54). These weaknesses highlight the necessity of focused interventions, like policies to lessen exposure disparities and air purifiers for high-risk populations (55).

Broader Public Health Implications

Global and Regional Burden of Disease

Every year, PM2.5 causes 4.2 million premature deaths, with Asia and Africa bearing the brunt of these effects. Countries with lower and middle incomes have higher disability-adjusted life years (DALYs) from PM2.5 (56).

Socioeconomic and Health Inequalities

Health problems linked to PM2.5 are exacerbated by disparities in healthcare access between urban and rural areas. Particularly in less developed countries, healthcare expenses and decreased productivity result in large economic losses (57).

Monitoring, Regulation, and Risk Assessment

Measurement Techniques and Air Quality Monitoring Networks

Low-cost sensors, satellites, and fixed stations are used to measure PM2.5. While rural areas lack adequate infrastructure, urban areas have better monitoring systems (58).

Urban vs. Rural Monitoring Challenges

Due to a lack of monitoring tools, data is scarce in rural areas, making it challenging to assess exposure. Everywhere, it is difficult to enforce policies and conduct real-time monitoring (59).

Risk Assessment and Guidelines

The WHO has set a 5 μg/m^3 annual limit for PM2.5. According to exposure-response models, health risks rise sharply above 10 μg/m^3 (60).

Mitigation and Prevention Strategies

Policy and Regulatory Interventions

Factory emission controls and traffic restrictions are examples of urban policies. Clean cooking technologies and improved crop residue management are promoted by rural strategies (61).

Technological and Infrastructure Solutions

PM2.5 is decreased by renewable energy sources and emission controls, such as scrubbers. Air quality management is enhanced by smart city systems and increased rural monitoring (62).

Community-Level and Individual Strategies

Campaigns to raise awareness promote the use of less biomass. Exposure can be decreased with personal protective equipment such as masks and air purifiers (63).

Future Perspectives and Research Directions

Innovative research and interdisciplinary approaches are needed to address the global PM2.5 challenge in order to close knowledge gaps and create workable solutions. To

gain a better understanding of the long-term health effects of PM2.5 exposure, longitudinal cohort studies are crucial, especially for underrepresented rural populations where data is limited (64). In order to capture the emergence of chronic diseases and the effects on successive generations, these studies ought to concentrate on cumulative exposure effects and monitor health outcomes over decades (65). Given that rising temperatures, shifting precipitation patterns, and an increase in the frequency of wildfires are likely to worsen PM2.5 levels and change exposure patterns, more research is necessary to understand how climate change is affecting PM2.5 dynamics (66). For instance, studies should look into how transboundary PM2.5 transport between urban and rural areas is impacted by climate-driven changes in wind patterns (67). Promising approaches to clarify PM2.5 toxicity mechanisms at the molecular level include multi-omics (e.g., proteomics, metabolomics, genomics) (68). By identifying biomarkers of exposure and susceptibility, these methods can enhance risk assessments and provide individualized health interventions (69). The study of cumulative environmental exposures, or exposomics, can also offer a comprehensive understanding of the ways in which PM2.5 interacts with other pollutants, lifestyle choices, and socioeconomic circumstances (70). To enhance exposure assessment and guide policy, research should also give top priority to the creation of affordable, scalable air quality monitoring technologies, especially for rural areas (71).

In order to address urban-rural disparities, community-driven interventions and participatory research are essential. Adoption and efficacy can be increased by involving local communities in the planning and execution of air quality initiatives, such as clean cookstove campaigns or urban green spaces (72). In order to support air quality improvements in low-resource settings, policy-oriented research should assess the effects of current regulations, such as WHO guidelines and emission standards, and investigate creative financing models (73). Furthermore, incorporating machine learning and artificial intelligence into air quality modeling can enhance forecasts of PM2.5 levels and health hazards, allowing for proactive mitigation measures (74).

The study of how PM2.5 interacts with other environmental stressors, like heat waves or co-pollutants like ozone, to potentially intensify health effects is one emerging field (75). Given the substantial contribution of indoor PM2.5 in rural environments, research should also examine the role of indoor air quality interventions. Last but not least, international cooperation is required to combat transboundary pollution, necessitating uniform air quality standards and frameworks for exchanging data among countries. These lines of inquiry, along with advancements in technology and policy, are essential to lowering the worldwide health burden of PM2.5 and attaining long-lasting improvements in air quality (76).

CONCLUSION

With significant effects on respiratory health and wider societal repercussions, PM2.5 continues to be a major worldwide public health concern. Asthma, COPD, and lung cancer are among the acute and chronic health effects that are caused by its varied sources, which include biomass burning and agricultural activities in rural areas, and vehicle and industrial emissions in urban areas. While rural communities struggle with seasonal spikes and substantial indoor pollution from biomass combustion, which disproportionately affects vulnerable groups like women and children, urban populations experience chronic, high-level exposure to metal-rich PM2.5, which exacerbates the prevalence of chronic diseases. PM2.5's systemic impact is highlighted by its toxicity, which is mediated by oxidative stress, inflammation, and epigenetic modifications. Ultrafine particles present particular risks because of their capacity to enter the bloodstream.

Interventions for PM2.5 must be customized and situation-specific. While rural areas need sustainable agricultural practices and easily accessible clean cooking technologies, urban areas need stricter emission controls, such as low-emission zones and industrial regulations. Improved monitoring is essential for precise exposure assessment and efficient policy enforcement, especially in rural areas with inadequate infrastructure. Targeted protections are necessary for vulnerable groups, such as children, the elderly, and low-income communities, because socioeconomic disparities increase the health burden of PM2.5. Addressing transboundary pollution and bringing air quality standards into line require international cooperation, particularly in areas like Asia and Africa, where PM2.5 levels are high.

Future development depends on combining cutting-edge technologies like AI-based air quality modeling, community-driven solutions, and sophisticated research like multi-omics and exposomics. Policymakers and researchers can lessen the negative health effects of PM2.5 and lessen global inequality by giving priority to long-term studies, the effects of climate change, and equitable interventions. Enhancing air quality sustainably requires a coordinated effort to close the gap between urban and rural areas, safeguard public health, and guarantee a healthier future for everybody.

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