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Air pollution and kidney disease; a comprehensive updated overview of recent evidence on functional and structural alterations

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ABSTRACT

The association between air pollution and renal health has gained increasing attention in recent years, with evidence suggesting that prolonged exposure to air pollution poses significant threats to both renal function and structural integrity of kidneys, with both immediate and long-term consequences on kidney health. Studies have indicated that various air pollutants are linked to the onset and progression of chronic kidney disease (CKD) and other renal disorders. The evidence links short-term increases in pollutant concentrations to declines in glomerular filtration rates, while long-term exposure is associated with a heightened risk of CKD and structural damage to renal tissues. The mechanisms through which air pollution impacts renal health, systemic inflammation, oxidative stress, endothelial dysfunction, and direct nephrotoxicity highlight the urgent need for public health policies aimed at improving air quality and protecting kidney health. The evidence emphasizes air pollution as a critical environmental risk factor contributing to kidney diseases globally.

Review

Implication for health policy/practice/research/medical education:
Air pollution introduces a mixture of harmful pollutants into the environment, many of which have been identified as significant contributors to kidney damage. Pollutants like particulate matter, nitrogen dioxide, sulfur dioxide, and heavy metals enter the bloodstream upon inhalation and are subsequently transported to various organs, which damage the renal tissues. Given the far-reaching implications of air quality on overall health, addressing air pollution should be viewed as a public health priority aimed at safeguarding kidney health and preventing chronic disease. Regulatory measures to reduce exposure to harmful pollutants will be crucial in mitigating the current burden of kidney disease while promoting environmental health equity across diverse populations.

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Introduction

Air pollution has emerged as a critical public health concern globally, with significant evidence linking it to various health problems, including renal function and structure deterioration. The kidneys play an essential role in filtering blood, removing waste, and maintaining fluid and electrolyte balance. However, exposure to environmental pollutants can compromise kidney function through multiple complex mechanisms (1). Research has consistently demonstrated that exposure to elevated levels of air pollution is associated with a decline in renal function, notably manifested as a decrease in estimated glomerular filtration rate. For instance, a significant body of evidence indicates that medium- and short-term exposure to particulate matter is linked with adverse outcomes in kidney function, contributing to the development of chronic kidney disease (CKD) (2). Those with CKDs are particularly vulnerable, as reduced renal function complicates the clearance of environmental toxins from the bloodstream, exacerbating the toxic burden imposed by air pollution (3).

The epidemiological evidence further solidifies the connection between air pollution and the incidence of CKD. Multiple studies have shown that regions with higher ambient air pollution levels exhibit a marked increase in the prevalence of CKD (4). A systematic review found that chronic exposure to fine particulate matter ($PM_{2.5}$) correlates with an increased risk of CKD, as approximately 428,000 people in Europe are estimated to die prematurely due to air pollution-related complications affecting various organ systems, including the kidneys (5). Short-term exposure to air pollutants has been shown to result in immediate renal consequences. For example, a study by Miao Cai in 2023 demonstrated that each increment of 10 μg/m^3 in atmospheric PM_1 and $PM_{2.5}$ concentrations was associated with a prevalence ratio of 1.31 for developing CKD (6). Another population-based cohort study found correlations between long-term exposure to multiple air pollutants and increased rates of both renal function decline and greater incidences of CKD diagnoses (7). This correlation extends beyond merely observational; experimental data have provided insights into the underlying biological processes at play. Recent studies involving animal models exposed to $PM_{2.5}$ have shown significant declines in glomerular filtration rates alongside pathological changes such as glomerular injury and tubulointerstitial fibrosis, akin to the clinical manifestations observed in CKD (8). In this review we aimed to explicates the pathophysiological mechanisms connecting air pollution exposure to the development of renal failure, weaving in current research findings that underscore the public health implications of these environmental threats.

Search strategy

For this study, we searched PubMed, Web of Science, EBSCO, Scopus, Google Scholar, Directory of Open Access Journals (DOAJ) and Embase, using different keywords including; air pollution, chronic kidney disease, renal toxicity, oxidative stress, inflammation, endothelial dysfunction, nitrogen dioxide, sulfur dioxide, particulate matter and heavy metals.

Main sources of air pollution affecting renal health

Air pollution has become a significant environmental concern with profound implications for human health, particularly regarding renal health. Various pollutants, originating from diverse sources, have been shown to adversely impact kidney function and structure, leading to conditions such as chronic renal failure (9). Prior studies showed particulate matter, which primarily categorized into $PM_{2.5}$ and PM_{10}, is considered one of the most critical pollutants for kidney health. $PM_{2.5}$ refers to airborne particles with a diameter of less than 2.5 micrometers, which can penetrate deep into the lungs and enter the bloodstream, directly impacting renal tissues. Major sources of particulate matter include vehicle emissions, industrial discharge, and combustion of biomass and fossil fuels (10). Numerous studies have linked exposure to elevated levels of particulate matter with increased instances of renal impairment. The comprehensive study indicated that chronic exposure to $PM_{2.5}$ elevated the risk of developing kidney disease, with every 10 μg/m^3 increase in $PM_{2.5}$ concentration correlating to a 7% increase in CKD incidence (11,12). Meanwhile, nitrogen dioxide (NO_2) and sulfur dioxide (SO_2) are gaseous pollutants primarily produced from combustion processes, including vehicle exhaust and industrial activities. NO_2 exposure is particularly of concern, as studies have shown that it can lead to increased inflammation and oxidative stress, significantly contributing to kidney dysfunction (13). The relationship between NO2 exposure and renal health has been well-documented. A longitudinal study identified that long-term exposure to NO_2 is strongly associated with an increased risk of CKD hospitalization, manifesting through acute inflammation and vascular changes detrimental to kidney function (14). Similarly, SO_2 has been found to trigger oxidative stress responses, inducing renal cell damage that exacerbates pre-existing kidney conditions (15). Additionally, heavy metals, like lead, cadmium, and arsenic, are environmental toxins that can severely impact renal health. These metals typically enter the environment through industrial emissions, mining operations, and waste disposal practices (16). Likewise, cadmium exposure has been connected to renal tubular damage and reduced glomerular filtration rates, highlighting its role in renal failure development (17). Recent studies also showed that volatile organic compounds (VOCs) are organic chemicals that evaporate easily at room temperature and are found in a variety of everyday products, including paints, solvents, and gasoline. Sources of VOCs include industrial processes,

vehicle emissions, and even household products (18). A recent study by Ni et al has shown a correlation between increased VOC exposure and compromised kidney function, underscoring the significant health risks associated with these pollutants (19). Another study by Ni et al identified that high VOC exposure increased the prevalence of kidney stones, suggesting a direct link between airborne VOCs and renal pathologies. This relationship, coupled with the ubiquitous nature of VOCs in urban environments, poses a considerable risk to public health (20).

Molecular mechanisms of kidney injury due to air pollution

The kidneys, highly vascularized organs, are particularly susceptible to the adverse effects of these pollutants, as they filter a considerable volume of blood and expose themselves to concentrated toxins. The initial mechanism of injury involves the inflammatory response triggered by airborne pollutants (21). For instance, $PM_{2.5}$ can stimulate inflammatory cytokine production, leading to oxidative stress within renal tissues. This oxidative stress results in direct cellular damage and promotes apoptosis of renal cells, which may escalate into conditions such as glomerulosclerosis and interstitial fibrosis (22). The study by Tsai et al found that prolonged exposure to particulate matter correlates with an upregulation of inflammatory markers such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), while concurrently diminishing renal filtration function (23). Furthermore, studies illustrate that pollutants can induced systemic phenomena such as hypertension and diabetes mellitus—both of which aggravate renal injury and precipitate CKD progression (24). High levels of NO_2, commonly associated with vehicular emissions and urban pollution, have been shown to exacerbate blood pressure regulation, potentially leading to hypertensive nephropathy and associated kidney damage (25). Besides, chemokines and cell adhesion molecules also play crucial roles in mediating inflammation and renal cell signaling. Monocyte chemoattractant protein-1, a chemokine, is vital for the recruitment of monocytes to the nephron during inflammatory states. Elevated urinary levels of monocyte chemoattractant protein-1 have been linked to renal interstitial inflammation and fibrosis, marking it as a potential biomarker for renal injury (26). Cell adhesion molecules such as vascular cell adhesion molecule-1 facilitate the adherence of immune cells to vascular endothelium, further exacerbating inflammation during kidney injury. Their upregulation reflects the endothelial dysfunction associated with CKD, leading to increased leukocyte infiltration and tissue damage (27).

Short-term effects of air pollution on renal function

Numerous studies indicated that short-term exposure to elevated levels of air pollution is associated with a decline in kidney function, particularly through reduced glomerular filtration rate (28). For instance, a study demonstrated that short-term increases in particulate matter and sulfur dioxide concentrations correlated with significant drops in the glomerular filtration rate levels among exposed populations. This transient decrease in kidney function can reflect immediate physiological responses to pollutant exposure, as toxins enter the bloodstream, causing systemic inflammation and oxidative stress that adversely affect the kidneys (29). Furthermore, it has been documented that the main pollutants affecting renal function include $PM_{2.5}$ and nitrogen dioxide, with studies showing that increases in the concentrations of these pollutants are linked to a measurable deterioration in kidney function. The immediate consequences of this exposure suggest mechanisms through which air pollution translates to renal impairment, emphasizing the need for continual monitoring and regulation of air quality to protect public health (30).

Long-term impact of air pollution on kidney health

The long-term implications of air pollution on kidney health are particularly concerning, as sustained exposure to pollutants can culminate in chronic health conditions. Epidemiological studies have established a clear association between long-term pollution exposure and heightened risk for renal failure. An increase in the annual average concentration of $PM_{2.5}$ has been linked to an elevated incidence of renal failure, with findings suggesting that each increment of $PM_{2.5}$ is associated with a 7% increased risk of developing the condition (31,32). Moreover, the cumulative effects of air pollution contribute to structural changes within the kidneys, potentially leading to irreversible damage. Chronic exposure to pollutants such as $PM_{2.5}$ and NO_2 has been shown to induce glomerulosclerosis and tubular damage, promoting renal fibrosis and impacting overall kidney architecture (33). The consistent overload of pollutants fosters an environment conducive to chronic inflammation, which further accelerates renal deterioration and increases susceptibility to advanced-stage kidney disease (34).

Harmful effects of air pollution on kidney

The primary pathways through which air pollution affects the kidneys include systemic inflammation, oxidative stress, endothelial dysfunction, and direct nephrotoxic effects from heavy metals (35). One of the foremost mechanisms is the induction of systemic inflammation. Airborne pollutants, especially particulate matter, can penetrate the respiratory tract and enter the bloodstream, triggering immune responses that lead to the production of pro-inflammatory cytokines and chemokines that circulate in the blood and reach the kidneys (36). Since the renal system receiving a significant part of heart output, making them particularly susceptible to inflammation. In general, an increasing in the plasma cytokine levels may

lead to glomerulonephritis, characterized by inflammation of the glomerulus, ultimately resulting in impaired renal function over time (37). Several studies have shown that elevated levels of inflammatory markers, such as C-reactive protein and pro-inflammatory cytokines such as IL-6 and TNF-alpha, across with other inflammatory mediator are associated with particulate matter exposure and are indicative of increased risk for renal impairment (38).

Another critical mechanism is oxidative stress, which occurs when there is an imbalance between the production of reactive oxygen species (ROS) and the body's ability to neutralize them. Inhaled pollutants translocate into systemic circulation via the lungs, initiating inflammatory responses that leading to oxidative damage that impairs renal vasculature and alters intracellular signaling pathways essential for maintaining renal tissue integrity (39,40). As mentioned above, air pollutants, particularly PM, NO_2, and VOCs, can enhance ROS production within the renal system, leading to cellular damage. It should remember that, the kidneys have a limited capacity to detoxify ROS, making them particularly susceptible to oxidative injury (41). This oxidative stress can lead to cellular damage by damaging lipids, proteins, and DNA, disrupting normal cellular functions, apoptosis of renal cells (42). Prolonged oxidative damage can cause significant structural changes to the kidneys, such as tubulointerstitial fibrosis, which is often seen in patients with CKD. This fibrotic change contributes to the deterioration of renal function, further precipitating the progression of kidney failure (43).

Air pollution can also cause endothelial dysfunction, primarily affecting the kidney's vasculature. Pollutants such as NO_2 and $PM_{2.5}$ disrupt nitric oxide (NO) signaling pathways, which are essential for maintaining vascular homeostasis and regulating renal blood flow (44). Endothelial cells lining the renal blood vessels become impaired, leading to increased vascular resistance and decreased glomerular filtration pressure. This dysfunction results in increased vascular resistance and decreased glomerular filtration pressure, further compromising kidney function and leading to toxin accumulation in the bloodstream (45,46). Previous investigations also have demonstrated that populations with elevated exposure to nitrogen dioxide increased markers of endothelial dysfunction, correlating with incidences of renal impairment (47). Meanwhile, certain pollutants, particularly heavy metals such as lead and cadmium, exert direct nephrotoxic effects that can severely impact kidney function. These metals can accumulate in renal tissues, causing structural and functional disruptions (48). For instance, cadmium exposure is known to induce tubular damage and interstitial fibrosis, leading to a decline in the kidneys' filtering capacity. Lead, another notorious nephrotoxin, has been linked to increased risks of hypertension and nephropathy. Its negative effects on kidney function result from both direct cellular toxicity and its ability to interfere with renal hemodynamics (49).

Symptoms caused by air pollution

One of the most common symptoms experienced by individuals with CKD is fatigue. As kidney function declines, the body becomes less efficient at filtering out waste products and toxins, which contribute to a general feeling of weakness and tiredness (50). Studies have shown that exposure to air pollutants can exacerbate fatigue, particularly in vulnerable populations. This persistent exhaustion may hinder daily activities and significantly impact overall well-being. Patients also may experience edema due to the increased permeability of blood vessels and disturbances in hydrostatic pressure within the kidneys (51,52). Along with the progression of the disease patients also complain of changes in urination patterns. Patients also may present with nausea and loss of appetite are common symptoms associated with the accumulation of waste products in the bloodstream due to reduced kidney function. Toxins, including urea and creatinine, can lead to unpleasant gastrointestinal symptoms (53). Hypertension, regulate blood pressure through the renin-angiotensin-aldosterone system. hypertension due to the air pollution may be due to some pollutants such as nitrogen dioxide and ozone (54). In addition, CKD can lead to mineral and bone disorders due to imbalances in calcium, phosphorus, and vitamin D metabolism. The patients therefore experience bone pain, weakness, and an increased risk of fractures. Regardless of bone disease due to CKD, the heavy metals also responsible for bone health disturbances in this setting (55,56). Finally, recent studies discuss on the relationship between cognitive impairment and air pollution which is also accompanied by neuroinflammation and neurodegenerative diseases (57).

Conclusion

The effects of air pollution on kidney function are multifaceted and complex, involving pathways of inflammation, oxidative stress, endothelial dysfunction, and direct toxicity. Pollutants such as particulate matter, nitrogen dioxide, sulfur dioxide, and heavy metals have all been implicated in the degradation of renal health, contributing to CKD and other renal disorders. The interplay between air pollution exposure and the development of CKD is a complex interaction involving direct kidney injury mechanisms, exacerbation of comorbid conditions, and the interplay of social determinants of health. The cumulative evidence supports the assertion that air pollution is indeed a significant risk factor for renal failure, emphasizing the need for public health interventions aime d at reducing exposure and improving air quality.

Authors' contribution

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Writing–original draft: All authors.
Writing–review and editing: All authors.

Conflicts of interest

The authors declare that they have no competing interests.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work, the authors utilized Perplexity to refine grammar points and language style in writing. Subsequently, the authors thoroughly reviewed and edited the content as necessary, assuming full responsibility for the publication's content.

Ethical issues

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors.

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