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Review Article

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Environmental Pollution and Its Impact on Kidney Health: A Contemporary Review of Existing Evidence

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ABSTRACT

Kidney diseases pose a growing public health concern due to their rising prevalence, associated cardiovascular risks, and substantial economic burden on the health care system worldwide. This article discusses the intricate connection between environmental pollution and kidney diseases, stressing the global impact of accelerated industrialization and urbanization. Despite traditional risk factors like diabetes and hypertension accounting for some cases, the rising prevalence of various kidney diseases, specifically chronic kidney disease (CKD), suggests additional, previously overlooked contributors. The kidney, with its filtration functions, is particularly susceptible to the toxic effects of environmental pollutants, making long-term exposure a significant risk factor for kidney diseases. Particulate matter (PM) with a mean aerodynamic diameter of less than 2.5 µm (PM2.5) has been linked to an increased risk of acute and CKDs, glomerular diseases, urological cancers, a more accelerated decline in kidney function, and high mortality. Additionally, exposure to industrial and agricultural pollutants, biogenic toxins, and second-hand smoke is associated with an increased risk of kidney disease. These various risk factors contribute to the global burden of kidney diseases, emphasizing the need for comprehensive understanding and effective strategies to mitigate adverse environmental influences on kidney health. The article underscores the need for longitudinal studies to establish causation, detailed investigations into pollutant-specific mechanisms, and exploration of gene–environment interactions. By fostering awareness and implementing successful pollution control strategies, the public health threat posed by environmental pollutants, specifically in the context of kidney diseases, might be mitigated.
Keywords: Chronic kidney disease, industrialization, pollution, particulate matter, agricultural chemicals

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INTRODUCTION

Kidney diseases are significant contributors to the global health burden. While the traditional risk factors for the development of various kidney diseases such as diabetes mellitus and hypertension are well-known, the role of environmental pollution in kidney diseases is being increasingly recognized. According to the World Health Organization (WHO), air pollution refers to the contamination of the indoor or outdoor environment by any chemical, physical, or biological agent that alters the natural characteristics of the atmosphere.[1] Table 1 shows the common sources and recommended guideline levels for key pollutants

A seminal proof-of-concept animal study provided much-needed insight that inhaled aerosols of sufficiently small diameter may cross the alveolar tissue and reach the bloodstream to affect many organ systems.[2] Several animal studies have demonstrated kidney damage induced by exposure to diesel exhaust particles and particulate matter (PM).[3,4] Similar studies in cultured human kidney cells revealed decreased cell

Table 1. The Common Sources and Recommended Guideline Levels for Key Pollutants

Pollutants	Sources	Concentration
$PM_{2.5}$	• Natural Sources: • Forest fires, volcanic eruptions, and natural disasters such as earthquakes • Industries and Treatment Plants: industry, oil refineries, brick kilns, and power plants • Emissions: Vehicle emissions, coal combustion, burning leaves and wood, agricultural activities, biomass burning • Household Activities and Personal Habits: Tobacco smoking, cooking activities (e.g., sautéing, frying), kerosene heaters, gas stoves, fireplace operation, and Construction activities	Less than 5 $\mu g/m^3$ annual average concentration
PM_{10}	• Combustion processes (e.g., vehicle exhaust, industrial emissions, and residential heating). • Construction activities and road dust. • Natural sources such as wildfires, volcanic eruptions, and dust storms. • Agricultural activities, including plowing and field burning.	15 $\mu g/m^3$ annual mean
Ozone (O_3)	Emitted from cars, power plants, industrial boilers, refineries, and chemical plants	100 $\mu g/m^3$ 8-hour mean
Nitrogen dioxide (NO_2)	Emitted from automobile engines	100 $\mu g/m^3$ 8-hour mean
Sulphur dioxide (SO_2)	Fossil fuel consumption or industrial activities	40 $\mu g/m^3$ 24-hour mean
Carbon monoxide (CO)	Fossil fuel consumption	7 $\mu g/m^3$ 24-hour mean

viability mediated by increased oxidative stress.[5] Since then, many epidemiological studies have demonstrated a possible association between air pollution and various kidney diseases. Exposure to high concentrations of PM has been linked to an increased hospitalization for acute kidney injury (AKI).[5] Studies have also noted an increased prevalence of chronic kidney disease (CKD) in areas with higher concentrations of suspended PM.[6] Trajectories of the prevalence of diabetes and hypertension closely mirror the trends in ambient air pollution, which themselves are independent risk factors for the development of CKD. Patients with traditional risk factors, such as diabetes, hypertension, pre-existing kidney diseases, other chronic illnesses, and advanced age, may be particularly vulnerable to kidney damage induced by pollution. These individuals often have compromised kidney function or additional health challenges that can exacerbate the harmful effects of pollutants on kidney health.[7] The review emphasizes the concerning increase in kidney diseases caused by air pollution, such as CKD, AKI, glomerular diseases, and urological cancers. It also explores the underlying mechanisms of pollution-induced kidney injury and suggests some important mitigating measures.Figure 1

MAIN POINTS

- Pollution has detrimental effects on kidney health, with mechanisms including oxidative stress, increased apoptosis, immune system dysfunction, and inflammation.
- Kidney diseases associated with environmental pollution include chronic kidney disease (CKD), acute kidney injury, glomerular diseases, and urological cancers, indicating a wide-ranging impact on kidney health.
- Rising pollution levels correlate with increased incidence of membranous nephropathy, adverse outcomes of systemic lupus erythematosus, an accelerated decline of glomerular filtration rate in IgA nephropathy, and contribute to higher mortality rates in CKD patients.
- Air pollution is strongly correlated with kidney disease, highlighting the urgent need for global attention and collaborative efforts to address this public health concern.
- Effective strategies to mitigate pollution's adverse effects on kidney health require collaborative initiatives, increased research investment, and the prioritization of policies aimed at fostering a healthier environment for present and future generations.

AIR POLLUTION AND KIDNEY DISEASES

Long before the advent of modern science, ancient Greeks attributed all illnesses to miasmas.[8] Miasmas were poisonous emanations from decaying carcasses, rotting vegetation, molds, and invisible dust particles. The impact of impure and unhealthy "airs" and "waters" was famously described by Hippocrates over 2400 years ago in his treatise "On Airs, Waters, and Places." Over the next centuries, many scholars recognized the ill effects brought about by coal burning. The Industrial Revolution in the eighteenth century transformed rural agrarian societies into technologically advanced urban societies, which led to an astronomical surge in air pollution.

As per the World Health Organization (WHO), 6.7 million lives are lost to air pollution every year. Pollutants with the strongest evidence for public health concern include particulate matter (PM), carbon monoxide (CO), ozone (O_3), nitrogen dioxide (NO_2), and sulfur dioxide (SO_2). Particulate matter refers to inhalable particles composed of chemical particulates, biological contaminants, dust, and gases.[9] It is generally defined by aerodynamic diameter (in micrometers), with PM2.5 and PM10 being the most common in the health-regulatory framework. Due

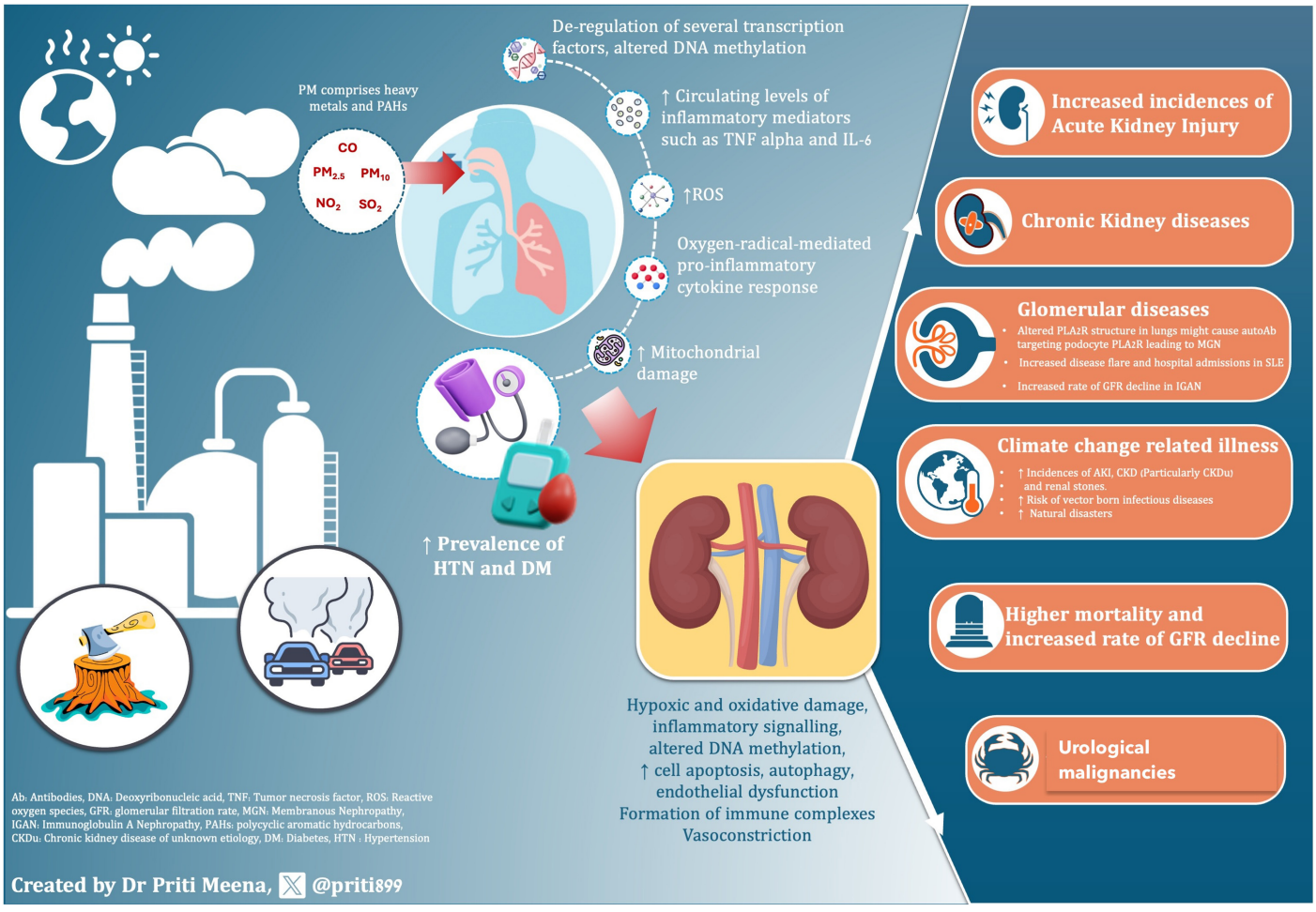

Figure 1. Outline of the various potential mechanisms of pollution-induced kidney diseases, illustrating the spectrum of impacts on kidney health.

to their tiny dimensions, PM2.5 and PM10 remain suspended in the atmosphere for prolonged periods and migrate across borders to affect populations far distant from their site of production.[10] Nanoparticle studies in rodents and human volunteers demonstrated that inhaled particles, when sufficiently small in size (4 nm or smaller in diameter), can enter systemic circulation, accumulate at sites of disease, and get excreted in urine. Another study demonstrated increased fibrosis, mesangial expansion, and decreased glomerular and tubular lumen volumes in rodents exposed to PM2.5, with effects more pronounced in hypertensive animals.

POLLUTION AND RISK OF DIABETES AND HYPERTENSION

The increasing prevalence of diabetes in recent years has become a worldwide concern, impacting populations in developed as well as developing nations. Recent studies indicate a concerning correlation between air pollution and the likelihood of developing diabetes mellitus.[11] A cross-sectional study in Iran found that prolonged exposure to ambient PM10 over 5 years was associated with an increased odds of developing diabetes mellitus.[12] Similarly, a prospective cohort study in Hong Kong demonstrated that high levels of PM2.5 were linked to a greater risk of both prevalent and incident cases of diabetes mellitus over nearly a decade.[13] Moreover, the escalation of air pollution concentrations can trigger various physiological disturbances, including disruptions in the autonomic nervous system, oxidative stress, inflammation, endoplasmic reticulum stress, apoptosis, and metabolic imbalances in glucose and insulin homeostasis.[14] Long-term exposure to PM2.5 has been found to promote insulin resistance (IR), impaired glucose tolerance (IGT), inflammation, and mitochondrial alterations, all of which are risk factors for diabetes mellitus.[11] Animal studies have shown that PM2.5 exposure leads to impaired hepatic glycogen storage, glucose intolerance, and IR.[15] The underlying mechanisms include the activation of inflammatory pathways mediated by Jun N-terminal kinase (JNK), NF-κB, and TLR4, which inhibit insulin receptor substrate 1 (IRS1)-mediated signal transduction. Additionally, PM2.5 exposure suppresses the expression of peroxisome proliferator-activated receptors PPARγ and PPARα in the liver, further contributing to insulin resistance and glucose metabolism disturbances.[16] Reactive oxygen species can also interfere with the insulin signaling pathway by triggering β-cell inflammatory responses via the NF-κB signaling pathway.[17]

Not only is pollution causing increased incidences of diabetes mellitus, but studies have shown an association between ambient air pollution and cardiometabolic diseases. In a study of 40 112 participants with a median follow-up of 12.6 years, PM2.5 was shown to be strongly associated with cardiometabolic diseases and with transitions from a healthy to disease state and then to death.[18] Exposure to ambient air pollution is also found to be associated with elevated blood pressure (BP), potentially contributing to increased cardiovascular mortality and morbidity risks. A study explored the hourly relationship between pollutants and BP, showing significant fluctuations.[19] Systolic BP decreases after exposure to SO_2 and CO, while O_3 and NO_2 lead to increases. The PURE study across 21 countries showed a 4% rise in hypertension with 3-year pollution exposure, particularly in cases where PM2.5 levels exceeded 62 µg/m^3.[20] Long-term exposure to PM2.5, PM10, and NO_2 is linked to high BP and heightened hypertension prevalence among rural Chinese adults.[17]

GLOMERULAR DISEASES AND POLLUTION

Xu et al[17] presented data on 71 151 native kidney biopsies across 282 cities in China over more than a decade, revealing that adjusted odds for membranous nephropathy (MN) exhibited a concerning annual increase of 13% over the study duration. Variations in 3-year average PM2.5 exposure, ranging from 6 to 114 µg/m^3, were observed among the various cities. The data showed that each incremental 10 µg/m^3 rise in PM2.5 concentration caused a 14% higher likelihood of MN, particularly accentuated in regions with PM2.5 concentrations surpassing 70 µg/m^3. A study in a Taiwanese cohort corroborated these findings, showing an increased risk of nephrotic syndrome in tandem with increasing levels of NO, NO_2, and PM2.5 concentrations.[21] Expanding on this, a 12-year population-based cohort study showed a stepwise increase in adjusted hazard ratios (HRs) for nephrotic syndrome in children across quartiles of SO_2, total hydrocarbon, and methane concentrations.[22] Another multi-center cohort study by Luo et al indicated PM2.5 as an independent risk factor for kidney function decline in patients with IgA nephropathy.[23] Furthermore, the impact of air pollutants on the progression and worsening of clinical manifestations of systemic lupus erythematosus (SLE) and the subsequent hospitalization of individuals with SLE has been explored in studies involving adults.[24-26] In a study from Taiwan by Jung et al,[24] an increase in NO2, CO, and PM2.5 showed positive associations with the development of SLE, whereas O_3 and SO_2 exhibited negative associations. Another study by Cakmak et al[26] showed that SO_2, CO, and PM2.5 were positively linked to higher hospital admissions with a primary diagnosis of SLE. Additionally, a Chinese study suggested that elevated PM2.5 concentrations might be connected to an increased risk of SLE relapse, while elevated levels of NO_2 and SO_2 exposure could be associated with an increased likelihood of first-time hospital admissions for SLE.[25] Notably, urinary casts and anti-double-stranded deoxyribonucleic acid antibodies, 2 common biomarkers of disease flare, were linked to short-term changes in PM2.5 exposure.[25] In tandem, these findings highlight an alarming link between rising pollution levels and the increasing occurrence of glomerular diseases. Hydrocarbon exposure has also been implicated as a potential risk factor for developing Goodpasture's Syndrome.[27] In a study analyzing 43 cases of Goodpasture's Syndrome associated with hydrocarbon exposure, the mean age of affected individuals was 28 years, with a slight predominance of males.[28] The types of hydrocarbons identified varied widely, and exposure durations ranged from brief encounters to prolonged periods spanning years. Most patients in this cohort experienced pulmonary hemorrhage and tested positive for antiglomerular basement membrane antibodies. These findings indicate that hydrocarbons could act as environmental triggers, possibly initiating or worsening the autoimmune response seen in Goodpasture's Syndrome.

ROLE OF POLLUTION IN CHRONIC KIDNEY DISEASE AND ACUTE KIDNEY INJURY

Numerous studies have reported a link between exposure to PM and a decline in estimated glomerular filtration rate (eGFR). A county-level cross-sectional study done in the United States (US) reported a higher prevalence of CKD in areas with high average PM2.5 concentrations.[6] In a large cohort of US veterans, a linear relationship between PM2.5 exposure and severe kidney outcomes was noted. Importantly, a 10-µg/m3 rise in PM2.5 levels was associated with an increased risk of CKD, eGFR decline ≥30%, and kidney failure.[29] A similar cohort study from Taiwan also showed an increased risk of developing CKD with an increased concentration of PM2.5.[25] In a prospective study by Mehta et al, a 2.1 interquartile range higher 1-year PM 2.5-µg/m^3 exposure was associated with a decline of eGFR by 1.87 ml/min/1.73m^2.[30] Extrapolating these findings to a more polluted area, which is not uncommon these days, would translate to a more rapid decline in eGFR, which could increase the incidence of kidney failure.

Additionally, air pollution from traffic and brake wear emissions are important sources of toxic metals like arsenic, cadmium, molybdenum, lead, antimony, uranium, and zinc. Chronic exposure to heavy metals has been linked to a higher incidence of CKD of unknown origin (CKDu).[31] "Hot-spots" of CKDu have been reported in India, Central America, the Middle East, Africa, Sri Lanka, and North America.[32] The definitive cause of kidney disease is unclear, with the possible involvement of an unidentified toxic or infectious agent, but epidemiological studies suggest that this may represent a form of "heat-induced nephropathy." Histopathology often reveals chronic tubulointerstitial nephritis with secondary glomerulosclerosis. Higher global temperatures do not just affect CKD; higher ambient temperatures have also been linked to an increased incidence of AKI.[28] In a case-crossover study, extreme heat exposure was associated with a 1.7-3.1% higher risk of kidney disease-related visits to emergency departments, with the strength of association greatest for AKI, nephrolithiasis, and urinary tract infections.[13] Lee et al noted an increased risk of first hospitalization

for AKI for a 5 μg/m^3 increase in PM2.5, a 10-ppb increase in NO_2, and a 10-ppb increase in O_3. Similar findings were replicated by a South Korean study demonstrating increased emergency visits for AKI in patients exposed to high concentrations of PM2.5.[33] The use of hospital diagnosis codes has its limitations—poor sensitivity, underestimation of AKI, including unaccountability for cases not requiring hospitalization, and gaps for timing and etiology of AKI. Nevertheless, these studies offer much-needed insight into the possible association between air pollution and AKI.

UROLOGICAL MALIGNANCIES

Outdoor air pollution has been linked to lung cancer, and there are concerns regarding its potential connection to urological cancers. However, the current evidence stems from occupational studies, leaving the impact of air pollution on the risk of urological cancers in the general population uncertain. A study using data from 14 European cohorts within the ESCAPE study aimed to explore potential associations between outdoor air pollution at residential locations and the occurrence of kidney parenchymal cancer in the general population.[34] This meta-analysis revealed elevated risk linked to higher concentrations of PM, though statistical significance was not attained. Moreover, the study suggested that exposure to outdoor PM at the residence might be linked to an increased risk of kidney parenchymal cancer; however, the observed association could, to some extent, be attributed to chance. Conversely, a study pooling 6 European cohorts (N = 302 493) found no association between the elemental components of PM2.5 and kidney parenchyma cancer.[35] In a systematic review of 20 studies, the primary method of exposure assessment was through modeling air pollutants. While the majority of studies have indicated a positive association between air pollution and the likelihood of developing urological malignancies, only a small proportion of these studies attained statistical significance. Of note, an adjusted odds ratio of 1.13 (1.03-1.23) has been reported for a 4.4 μg.m-3 exposure for bladder cancer mortality.[36]

RISING POLLUTION AND MORTALITY FROM KIDNEY DISEASES

The United Nations estimated that in 2012, around 12.6 million deaths worldwide occurred due to environmental factors—almost 1 in 4 of all global deaths—either due to working or living in an unhealthy environment.[37] The presence of air, water, and soil pollution, as well as climate change and exposure to industrial chemicals and ultraviolet radiation, contribute to an increased susceptibility to over 100 diseases and injuries. This heightened susceptibility is particularly high in pediatric and elderly populations. In China, each 10 μg/m^3 increase in log 0–1 mean concentrations of air pollutants has been shown to increase mortality from kidney disease by 1.33% for PM1, 0.49% for PM2.5, 0.32% for PM10, 1.26% for NO2, and 2.9% for SO_2.[38] Similar study by Min et al done in 136 locations (Portugal, Canada, South Korea, Taiwan, Japan, and the UK) between 1987 and 2018 shown increased relative risk of AKI-related deaths.[39] The KiPP study (a Sri Lanka–US collaboration) conducted in endemic areas for CKDu suggests that a significant contributor to the disease probably the agrochemical compounds present in drinking water sources, primarily wells. These include Diazinon, organophosphates including dichloro-diphenyl-trichloroeth ane, Propanil, and banned substances like Endosulfan II etc.[40] In 2016, CKDu was reported to be the 8th leading cause of in-hospital mortality in the country.[41]

PATHOPHYSIOLOGY OF POLLUTION AND KIDNEY DAMAGE

Various studies have demonstrated that exposure to PM2.5 can result in oxidative stress, inflammation, cell autophagy, and apoptosis, potentially causing harm to kidney tissue.[42] Additionally, inert gold nanoparticles, serving as a model for PM2.5, have been observed in urine shortly after exposure, suggesting direct kidney filtration and subsequent tissue damage.[43] Sulfur dioxide exposure has been linked to structural damage in kidney cells, while hydrocarbons may contribute to glomerulonephritis via immune complex deposition and direct toxicity.[39] PM2.5-induced thrombus formation and vascular dysfunction may exacerbate cardiovascular events, further impacting kidney health.[2] Other pollutants like SO2 and diesel exhaust particles (DEPs) have also been implicated in kidney cell damage, oxidative stress, inflammation, and worsening kidney injury.[44] Gene expression studies on mice have shown that ozone inhalation alters genes involved in the antioxidant response, inflammatory signaling, and endothelial dysfunction, particularly in the kidneys, shedding light on potential mechanisms underlying ozone-related kidney vulnerability.[41] Regarding glomerular diseases, the association between PM2.5 exposure and the incidence of MN has been noted, although the precise mechanism remains unclear.[45] MN, an autoimmune disorder characterized by autoantibodies against secretory phospholipase A2 receptor (PLA2R), involves immune complex deposition in the glomerular basement membrane. Particulate matter exposure may modify PLA2R structure in the lungs, triggering autoantibody production targeting PLA2R, potentially leading to immune complex formation, cell damage, and MN development.[46] Animal studies further support PM toxicity mechanisms, showing autoantibody and immune complex production upon fine PM exposure.[43] PM2.5 exposure also elevates circulating inflammatory mediators, promoting systemic inflammation and oxidative stress, which could contribute to distant organ damage, including the kidneys.[47] Research involving traffic policemen has indicated increased levels of inflammatory mediators such as tumor necrosis factor (TNF) and IL-6 in their blood in response to PM2.5 exposure.[24]

IMPACT OF CHANGING CLIMATE ON KIDNEYS

By last century's end, the temperature rise of 1-4.5°C, mainly attributed to human-induced greenhouse gas emissions (GHG) from fossil fuels and extensive deforestation, raised considerable alarm and underscored the urgent need for action. Throughout the tweniteth century, these emissions drove global climate change, manifesting in rising temperatures,

extreme weather events, and various ecological disruptions.[48] The resulting climate change, exacerbated by air pollution exacerbates, poses a serious threat to kidney health.[49] The prevalence of heat-related kidney diseases, including AKI and CKD (as discussed above), is on the ascent worldwide, particularly in regions exposed to extreme heat.[23] Heat stroke is one of the most recognized heat-associated illnesses and is commonly complicated by AKI.[50] Simultaneously, association between nephrolithiasis and increased ambient temperatures has also been shown in various studies.[51] Dehydration, a risk factor for kidney stones, leads to increased urine concentration and acidification, heightening the risk of uric acid nephrolithiasis.[52] Climate change is anticipated to broaden the stone belt, markedly increasing the risk of kidney stones.[51] Vector-borne diseases thriving in warmer climates, such as malaria and dengue, are significant contributors to AKI, especially in Low-Income Countries (LIC) and Lower-Middle-Income Countries (LMIC).[53] Modelling studies indicate that rising temperatures favor the transmission of these diseases, increasing the likelihood of AKI incidence in affected regions.[54] Climate change exacerbates the threat posed by the mosquito-borne Zika virus, compounding risks to human health.[55] The impact of extreme weather events, intensified by climate change, increases the risk of AKI through mosquito-borne diseases as well as diarrheal and rodent-borne illnesses causing epidemic outbreaks and consequent AKI in low-income and tropical nations.[56] The forecasted increase in the frequency and intensity of extreme weather events poses an imminent threat to health systems globally. The potential disruption of crucial infrastructure like power, water supplies, transportation, and telecommunication services during such events increases the vulnerability of people with chronic medical conditions, particularly dialysis patients, amplifying the risks associated with missed dialysis sessions.[57]

WATER POLLUTION

Water contamination by heavy metals, perfluorinated compounds, pesticides, industrial hydrocarbons, and pathogens poses significant risks to human health, particularly kidney function. Exposure to these pollutants occurs through drinking water, consuming contaminated aquatic life, and skin or mucosal contact.

Heavy metals such as arsenic, cadmium, lead, mercury, and uranium are well-documented nephrotoxins.[58] Arsenic, a common groundwater contaminant, has been linked to CKD and ESKD.[59] Cadmium, introduced into the environment through mining, fossil fuel combustion, and waste incineration, historically caused severe health crises, such as Japan's "itai-itai" disease.[60] Lead contamination in water often results from leaching plumbing materials and industrial runoff, and it is associated with CKD, increased serum creatinine, and decreased eGFR.[61] Mercury exposure, mainly through the consumption of contaminated fish, leads to kidney dysfunction, though specific links to CKD are less clear. In a study, mercury overload in domestic water was found to be associated with higher proteinuria and low hemoglobin and hematocrit levels compared to the control group.[62] Uranium, predominantly from natural erosion and mining, is associated with kidney dysfunction, supported by animal studies. Per- and polyfluoroalkyl substances (PFAS), persistent environmental pollutants found in various consumer products, are significant water contaminants. Some studies link PFAS exposure to CKD and reduced GFR, although the relationship remains ambiguous.[63] Industrial chemicals such as trichloroethylene and tetrachloroethylene, used in degreasing and dry cleaning, also demonstrate nephrotoxic effects.[64] Biological contaminants, including bacteria like Leptospira and parasitic worms from the genus Schistosoma, are implicated in CKD development.[65,66] Exposure to pesticides through contaminated water affects farmers and agricultural workers, with several herbicides and insecticides linked to CKD and ESKD.[67] These include alachlor, atrazine, glyphosate, paraquat, pendimethalin, and methyl parathion. Organochlorine insecticides like hexachlorocyclohexane and endosulfan, the herbicide dicamba, and the insecticide DDT also impact kidney health.[68]

Although there is no definitive evidence that air and water pollution cause different kidney damage patterns in different geographical regions, geographical location can significantly affect the levels and types of pollution due to factors such as industrial activities, traffic density, population size, and local regulations. Different regions may have varying sources and types of pollutants, leading to distinct patterns of exposure and health outcomes.

Air pollution is most common in large cities where emissions from various sources are concentrated. Sometimes, mountains or tall buildings prevent air pollution from spreading out. Ozone, for example, can travel far from its initial source, moving with air masses.[69] Another example is Balkan endemic nephropathy (BEN), a type of endemic nephropathy associated with upper urothelial cancer, which has a specific geographical distribution along the tributaries of the Danube River.[70] Several hypotheses on the cause of BEN have been suggested, such as mycotoxins, heavy metals, viruses, and trace element insufficiencies.

MITIGATING MEASURES

The association between air pollution and kidney diseases calls for urgent global attention. Integrating PM2.5–kidney diseases as a recognized risk-outcome pair in assessing health hazards due to air pollution is essential. To prevent kidney diseases and other non-communicable diseases (NCDs) caused by pollution, substantial investments and technical support are essential, especially in LMICs.[71] Strengthening data systems, challenging vested interests, undertaking comprehensive research with kidney disease associations, and aligning global strategies with local action plans are imperative steps.[72] Incorporating pollution mitigation initiatives within the Global Action Plan for the Prevention and Control of NCDs is crucial.[73] Multiple tools

have already been developed to address pollution control and mitigate its detrimental impact on human health. The model for disadvantaged countries can be derived from the successful techniques implemented by high- and middle-income countries with legal frameworks, policies, regulations, and improvements in technology.[57,58] Mandated emission reductions, stringent air-quality standards, incentives for transitioning to non-polluting energy sources, and the promotion of public transportation promotion are key measures.[74] These strategies, if globally adopted, can empower the world to overcome the health and ecological challenges tied to industrial development. Raising awareness among policymakers, industry stakeholders, and the public in emerging economies is crucial in addressing environmental pollutants as a significant public health concern. It is essential to prioritize the implementation of initiatives like promoting renewable energy and public transportation, as well as emphasizing research on the health effects of pollutants. Addressing information disparities and promoting policy dialogues at regional and international levels is of utmost importance. To improve the health status of individuals and prevent kidney damage due to pollution, stricter air and water quality regulations should be enforced, and the use of personal protective equipment (PPE) in high-risk areas should be promoted. Regular monitoring of kidney function is essential, as early detection can help manage and mitigate the effects of exposure. Although there are currently no specific biomarkers or therapeutic agents that can accurately identify or treat early signs of kidney damage due to pollution, ongoing research in this area is crucial. Public awareness campaigns should inform individuals in high-risk areas about the hazards of pollution on kidney health. Further studies are needed to identify specific compounds within PM responsible for kidney damage and to develop strategies to prevent or treat these adverse effects. Recognizing the role of environmental health in kidney disease at the policy level will drive the implementation of preventive measures, such as stricter regulation of pollutants and protections for outdoor workers exposed to high temperatures due to climate change. Addressing kidney diseases linked to pollution necessitates a collective effort to develop and implement effective strategies, highlighting the importance of global environmental preservation.

Figure 1 provides the outline of the various potential mechanisms of pollution-induced kidney diseases, illustrating the spectrum of impacts on kidney health.

Conclusion

In conclusion, the nexus between air pollution and kidney health needs immediate global action. The evidence of pollution's harmful effects on kidney health is robust and consistently growing. Mechanisms include oxidative stress and inflammation, exacerbated by climate change. Combating these pathological changes requires collaborative efforts, research investment, and policy prioritization for a healthier future.

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