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IDOSR JOURNAL OF SCIENTIFIC RESEARCH 11(1):117-125, 2026. ISSN: 2550-794X

https://doi.org/10.59298/IDOSRJSR/2026/11.1.117125 IDOSRJSR11106

Review of Urban Air Pollution and Health

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ABSTRACT

Urban air pollution remains a critical global public health challenge, driven largely by accelerated urbanization, industrial expansion, and increasing energy demands. This narrative review synthesizes existing evidence on the characteristics, sources, and health impacts of urban air pollutants, emphasizing particulate matter, nitrogen oxides, sulfur dioxide, ozone, ultrafine particles, and emerging contaminants such as microplastics. Drawing on historical perspectives, the review highlights the evolution of scientific understanding from early smog events to contemporary concerns involving complex pollutant mixtures and systemic health impacts. The review also explores measurement and monitoring strategies ranging from fixed-site stations to mobile sensors and integrated modeling, which shape exposure assessment and epidemiological research. Evidence consistently links urban air pollution to cardiovascular, respiratory, neurological, immunological, and perinatal health outcomes, with children, older adults, socioeconomically disadvantaged groups, and those with underlying disease identified as particularly vulnerable. Mechanistic pathways, including inflammation, oxidative stress, endothelial dysfunction, and neuroinflammation, provide biologically plausible explanations for observed risks. The review further assesses public health interventions, including urban planning, transportation policies, emission reduction strategies, and risk communication frameworks. Despite advancements in regulatory standards, clean technologies, and surveillance systems, substantial inequities persist across urban populations, particularly in low- and middle-income countries. Strengthening interdisciplinary approaches, improving exposure assessment, and addressing emerging contaminants are essential to reducing the global urban air pollution burden.

Keywords: Urban Air Pollution, Particulate Matter (PM), Health Outcomes, Oxidative Stress and Inflammation, and Environmental Public Health Policy.

INTRODUCTION

Urban air pollutants from anthropogenic sources pose a serious and widespread threat to human health. A narrative review synthesizes the evolution of knowledge on urban air pollution and health and describes major pollutants, their sources, and underlying mechanisms [1]. Health outcomes associated with urban air pollution are identified, along with vulnerable populations. Public health interventions and mechanisms by which pollution affects health are assessed, highlighting research gaps and methodological needs [2]. Urban air pollution refers to pollutants emitted within cities and includes particulate matter and trace gases. Health outcomes encompass morbidity and premature mortality across all population groups [6]. Exposure to urban air pollution is associated with higher cardiovascular morbidity and mortality and exacerbation of pre-existing respiratory conditions [1]. The urban health burden from air pollution remains substantial despite policy interventions, emerging technologies, and regulatory measures, particularly in low- and middle-income countries where economic growth is increasing emissions [1]. Air pollution is a complex, multicomponent mixture, yet rising emissions continue to include many well-studied toxic substances [3]. The global mental health crisis highlights the relevance of emerging pollution and phase-change contaminants, including phthalates, flame retardants, microplastics, and ultrafine particles [2]. Awareness of urban air pollution as a key public health hazard dates back to the London smog of 1952. Comprehension of the mechanisms through which pollutants affect health and identification of

additional pollutants continue to evolve [2]. Despite significant regulatory and policy interventions, urban air pollution remains a critical global public health challenge that warrants continued attention [1].

Historical Perspectives on Urban Air Pollution

Human societies often pay a heavy toll for their development in the form of health inequities caused by environmental hazards [1]. Improvements in health have sometimes lagged behind those in prosperity and life expectancy, especially when narrowly focused on biomedicine [2]. A growing body of evidence indicates that the health of urban populations demonstrates persistent disparities that are place-specific and influenced by environmental toxicants, including air pollution [1]. The growing recognition of public health as a prerequisite for a prosperous and stable society has led to calls for "urban air pollution" to be regarded as a global public health problem in its own right, more than two millennia after individuals first pointed out its detrimental effects [3].

Characteristics and Sources of Urban Pollutants

Pollution in urban areas emerges from numerous activities generating compounds with diverse physical-chemical properties and origins, which change with time and topography [6]. Particulate matter (PM) and trace gases rank as the most relevant urban pollutants for human health [4]. Urban air pollution consists mainly of PM and trace gases resulting from anthropogenic activities. Compounds released into atmospheric urban environments have small particle sizes and a wide range of vapor pressures, which expedite their settling to deposition surfaces [5]. Additionally, they are subject to oxidation and different atmospheric transformations, leading to the generation of secondary urban pollutants [4]. Particulate matter refers to materials (solid or liquid) suspended in the atmosphere. PM10, PM2.5 (particular concern for health), and PM0.1 circulation in urban environments derive from primary sources (e.g., combustion engines, construction, and industrial activities), while gases, such as nitrogen oxides (NOX) and sulfur dioxide (SO2), oxidize following emission from combustion sources and contribute to PM2.5 secondary formation[3]. Nitrogen oxides (NOX), comprising nitric oxide (NO) and nitrogen dioxide (NO2), arise from combustion processes (vehicles, industries, power plants), with a strong correlation to PM emissions. Sulfur dioxide (SO2), mainly produced from coal burning, petroleum refining, and the oil industry, follows the same behavior in urban settings. Ozone (O3) forms through the photochemical reaction of precursor gases such as NOX and volatile organic compounds (VOCs), typically in the morning and afternoon, with an added contribution from CO [5]. Environmental conditions influence the occurrence, precursors, and associated compounds of all three species significantly [1]. Registration of these species remains intricate since their concentrations must be deduced from other simpler substances subject to different but decipherable transformations [3]. Significant efforts, funds, and tools become necessary for the detection of emerging contaminants (emerging pollutants) in the air; therefore, despite further development, ultrafine particles still require much research to establish direct relationships with various health risks circulating in urban air [5].

Particulate Matter and Trace Gases

Particulate matter (PM) is a term for a mixture of solid and liquid particles of organic and inorganic substances suspended in air. PM is classified by size, with PM10 passing through a 10-micron sieve, PM2.5 passing through a 2.5-micron sieve, and PM1 that can penetrate the lungs and enter the circulatory system [5]. Ultrafine particles (generally less than 100 nm) can pass through the alveolar wall to reach the bloodstream [6]. Other proxy indicators for PM include mass, number, surface area, and composition, but mainstream macroscopic urban air pollutant studies focus on PM, NOx, SO2, and O3. PM8 encompasses the common sources and public health effects of PM10 and PM2.5. PM10 includes indoor and outdoor sources such as vegetation, sea spray, soil, and industrial, traffic, residential, and power-generation emissions [2]. PM2.5 particles from long-range transmission (secondary particles), such as dust outbreaks, vegetation fires, or biomass burning from agricultural activities, may evade convenient PM monitoring. Within urban air pollution studies, emphasis has shifted towards understanding particles smaller than 2.5, nanosized particles, and particle number concentration [3]. Traffic-related pollutants comprise PM10, PM2.5, NOx (sourced mainly from fuel combustion), and PAHs (partly from incomplete fuel combustion), while larger particles arise from nonspecific vehicular spray of road/topsoil and industrial combustion/waste process. Overall, fuel type appears more closely correlated to aerosol loading than PM size [4]. Trace gases form as combustion products and secondary pollutants [1]. Examples include nitrogen oxides (NOx), sulfur dioxide (SO2), carbon monoxide (CO), carbon dioxide (CO2), reactive organic gases (ROG), and ozone (O3). NOx derives mainly from transportation, industry, and heating; SO2 arises from coal/oil combustion and metal smelting; CO corresponds to fuel combustion; ROG has both anthropogenic and biogenic origins; and O3 is a secondary pollutant formed in sunlight-driven photochemical reactions involving VOCs and NOx [2]. Hence, NOx, SO2, and O3 are regulated in many cities worldwide, particularly megacities, still in the early stages of air-pollution control [1].

Nitrogen Oxides, Sulfur Dioxide, and Ozone

Nitrogen oxides, sulfur dioxide, and ozone are common urban air pollutants linked to short-term health effects such as increased mortality and morbidity [7]. Concentrations of nitrogen dioxide and ozone typically peak

during the daytime, while sulfur dioxide concentrations are higher at night. Ozone is formed through the photochemical reaction of precursor pollutants, nitrogen oxides, volatile organic compounds, and carbon monoxide under sunlight, resulting in elevated levels on hot summer days [8]. Other nitrogen oxides (NOx) are also involved in ozone formation [8]. Primary nitrogen oxides are emitted directly into the atmosphere as a consequence of incomplete combustion processes in automobiles, generators, and various industrial activities. Sulfur dioxide originates from the burning of fossil fuels, primarily coal and oil, in power generation and residential heating [7]. Emissions consist mainly of sulfur-dioxide-containing fuels, and burning heavy fuel oils generates excessive levels of nitrogen oxides and sulfur dioxide [3]. Ozone is a naturally occurring compound. Sources of event-triggering nitrogen oxides and sulfur dioxide, and ozone precursors from combustion systems, kitchens, and smaller generators can also be found in residential vehicular emissions and emissions triggered from secondary combustion processes in indoor activities are important event sources, especially in domestic premises [1].

Emerging Contaminants and Ultrafine Particles

Urban air quality and health remain an active field of research. Emerging contaminants of urban concern include airborne microplastics and biological material, as well as ultrafine particles (UFP) [8]. Microplastics are small plastic particles less than 5 mm, generated from either the fragmentation of larger items or the direct release of small material such as cosmetic products [6]. Globally, urban areas act as a source of microplastics, where data suggests the numbers of particles released are approximately in the range of 12% to 37%. These airborne microplastics are very challenging to detect, for which there are no specific detection methods available. Nonetheless, airborne microplastics released from urban areas and UFP have been associated with lung health effects [1]. UFPs are defined as particles smaller than 100 nm, with many urban sources [5]. UFP can be emitted directly or formed from gas-to-particle conversion in the atmosphere [5]. Direct sources of emissions include road traffic (combustion engines, brake and tyre wear), industrial activities, domestic combustion, evaporation of liquid fuels, and cooking. Secondary formation routes can occur from gas-phase reactions of volatile organic compounds, nitrogen oxides, sulphur dioxide, and ammonia [9]. Due to their high surface-to-volume ratio, the presence of harmful compounds on their surfaces, and the ability to penetrate deep into the respiratory tract, UFPs have raised widespread concerns regarding their potential adverse health effects. UFPs are not currently regulated in any jurisdiction worldwide [5].

Measurement and Monitoring in Urban Environments

Urban air quality is monitored and modeled to characterize human exposure, the primary determinant of health impacts [8]. Fixed monitoring networks, consisting of large and expensive instruments located at a few sites, provide detailed, long-term data of good statistical reliability [3]. Major urban networks typically monitor between 5 and 20 criteria pollutants, but spatial coverage remains limited and may not represent nearby conditions. Regulatory sites prioritize compliance over health (J. Kelly & C. Fussell, 2020) [1]. Personal and mobile monitoring approaches often combined with complementary modeling enable denser and more varied sampling of relevant microenvironments [9]. Wearable instruments offer personalized exposure estimates reflecting individual activity patterns. Data from low-cost sensors, primarily targeted at particulate matter, are increasingly integrated with models to describe urban and indoor microenvironments, extend coverage, and support exposure assessment [8].

Fixed Monitoring Networks

Fixed air quality monitoring stations are operated by governmental authorities in major cities to collect long-term data on ambient concentrations of target air pollutants, including criteria pollutants [2]. They facilitate analysis of long-term trends and serve as a basis for epidemiological studies [3]. However, these networks have been criticized for limited spatial coverage, poor representation of exposure at a small-scale level, and insufficient ability to characterize evolving patterns in complex urban areas [5]. Fixed stations monitor at a relatively low temporal resolution. A community sensor network in Imperial County, California, performed well in terms of community science and pollutant episode reporting, but lacked good precision [9]. National legislation and international treaties set standards for the operation of fixed monitoring networks, representing major milestones in urban air quality legislation and engagement with scientific advice [7]. A broader understanding of urban air pollution as a large systemic health risk arose in response to new findings about health effects, risk typologies, and population susceptibility. Human involvement in the monitoring process remains a prominent factor affecting sensor network performance, as approaches on statistical models exhibit lower resilience compared to data-driven methods [8].

Personal and Mobile Monitoring

Contaminated air from industry and traffic threatens urban dwellers' health. Yet, many lack access to detailed exposure data, limiting understanding and intervention [7]. Whereas centralized networks estimate population exposure, location, and time, they drive personal risk. Mobile measurements promise tailored insights, revealing pollution dynamics and measuring unseen pollutants like nanosized particles [5]. Wearable monitors with small

sensors estimate individuals' exposure in real time, enhancing risk perception and participant engagement 10. Personal and mobile monitoring, either indoor or outdoor, stationary or portable, detects air pollution where and when individuals breathe [3]. These methods, though still evolving, support outdoor, indoor, and indoor-outdoor measurements and various solutions for pedestrians, workers, students, and other microenvironments [11].

Modeling and Data Assimilation

Estimating exposure to urban air pollution is essential for epidemiology studies and environmental-health research [12]. Data assimilation addresses the challenges of providing a comprehensive description of urban air quality. The integration of transport models, emission estimates, and observations improves predictability and reduces uncertainty for processes such as air-conditioning pollutant removal and wildfire emissions [13]. Integrating satellite images with transport and chemical models characterizes space-time variability in atmospheric and indoor conditions and provides complementary spatial information across various measurements [13].

Health Outcomes Associated with Urban Air Pollution

Air pollution is associated with a wide range of adverse health effects. This section summarizes the health impacts linked to urban air pollutants, the strength of evidence, and the consistency of findings across studies and populations particularly vulnerable to pollution exposure [7]. Pollutants that have been associated with cardiovascular impacts include particulate matter, nitrogen dioxide, and ozone; respiratory and immunological effects have similarly been well documented for particulate matter, nitrogen dioxide, and sulfur dioxide. Pollutants linked to neurological effects include particulate matter, nitrogen dioxide, and manganese [2]. The health impacts associated with urban air pollutants, their strength of evidence, and the populations particularly vulnerable to them are summarized below [16]. Cardiovascular effects. Urban air pollution has been associated with cardiovascular morbidity and mortality. Epidemiologic studies have linked air pollution exposure to various cardiovascular events, such as myocardial infarction, stroke, and arrhythmia [14]. Significant associations have also been observed with cardiovascular disease incidence and hospital admissions for cardiac-related conditions. For all cardiovascular endpoints, robust positive associations have been reported for particulate matter [2]. For nitrogen dioxide, positive associations have been observed for myocardial infarction, stroke, and cardiac-related hospital admissions [16]. For ozone, cardiovascular incidence and hospital admissions are both positively associated with exposure. Models have documented dose–response relationships for cardiopulmonary mortality [1]. Respiratory and immunological effects. Urban air pollution exposure has been linked to adverse respiratory and immunological effects. For ozone, positive associations of exposure with asthma incidence, asthma attacks, and respiratory tract infections have been reported [6]. Exposure to nitrogen dioxide has also been associated with asthma incidence. Particulate matter has been positively linked to hospital admissions for obstructive lung disease and chronic obstructive pulmonary disease. Air pollution drives systemic immune responses and modifies the immune system, which may increase susceptibility to infections [9]. Neurological and other systemic effects. Urban air pollution is associated with neurological effects and other systemic health impacts. Associations are particularly well established for particulate matter, with links to neuroinflammation [15], neurodegeneration, dementia, and cognitive decline documented in different epidemiologic studies. Effects have also been reported for prenatal exposure, including low birth weight, preterm delivery, and gestational diabetes. Other pollutants of concern include nitrogen dioxide and manganese [1].

Cardiovascular Impacts

Urban air pollution is a major, growing public health threat [2]. It is a complex mix of chemicals, biological agents, and materials originating from a variety of natural and anthropogenic sources. The concentration and composition of the mixture depend on local sources of emissions, atmospheric conditions, and geographic location. Urban air pollution has been linked to numerous adverse health effects, which vary depending on the nature and intensity of exposure and the population group affected [5]. Potential health impacts include cardiovascular, respiratory, and neurological diseases, together with aggravation of pre-existing illnesses. Furthermore, compromised immune responses, allergies, and cancer development are also of concern. Children, elderly people, and individuals with pre-existing health conditions, as well as certain occupational groups, are particularly vulnerable [16, 17].

Respiratory and Immunological Effects

Exposure to urban air pollution causes respiratory and immunological effects that vary across exposure types and population subgroups [9]. Numerous studies link urban air pollution to respiratory disease in early life, aggravating asthma and other symptoms in childhood and contributing to related hospitalizations and deaths. Evidence also suggests that air pollution causes chronic obstructive pulmonary disease (COPD), pneumonia, and respiratory infections; reduces growth in lung function; and modifies the immune response [18]. A major challenge in understanding the respiratory effects of air pollution is the variety of pollutants involved and the different exposure circumstances that vary in composition and concentration [16]. Epidemiological studies draw

links to respiratory effects through a variety of urban exposure factors or pollutants, including nitrogen dioxide as a marker of traffic exposure, suspended particulate doses, and weather and season [11].

Neurological and Other Systemic Effects

The possible connection between urban air pollution and adverse neurological outcomes has received increasing attention in recent years [2]. Emerging epidemiological studies suggest associations with neuroinflammatory processes, neurodevelopmental impairment, cognitive deficits, and progressive neurodegenerative diseases. Investigating the effects of air pollution on populations living at current ambient concentrations and elucidating underlying biological mechanisms are emerging research priorities [3]. Apart from the brain, other organ systems are increasingly considered susceptible to air pollution. For example, the occurrence of type 2 diabetes mellitus has been associated with exposure to particulate matter and nitrogen oxides [4]. In addition, pregnant women exposed to air pollution show alterations in placental morphology, a trimester-dependent increase in preeclampsia incidence, and impaired fetal growth. Recent evidence suggests a direct route of translocation of particles from the lungs to the fetal environment, indicating a fetal exposure that requires further attention [7]. The inflammatory influences of air pollution exposure appear to extend beyond the respiratory system. In a meta-analysis of toxicological studies, the effects of air pollution were shown to modulate the immune response but did not directly influence susceptibility to infectious diseases [3].

Vulnerable Populations and Health Inequities

Environmentally vulnerable populations are those at greater risk of experiencing adverse health effects of pollution or environment-related diseases [8]. The prevalence of environmental risk factors that contribute to communicable diseases or injuries can vary widely across individuals of different age or gender groups, the socially or economically disadvantaged, or specific occupational, geographic, or cultural groups [5]. Children, the elderly, the poor, and those with existing health problems were identified as particularly vulnerable [19]. Environmental injustice that overlays and compounds a larger and growing set of health inequities afflicts various racial or ethnic groups, specific cultural or immigrant populations, and individuals living at the intersection of two or more cultural groups [2]. Vulnerable populations in urban areas may be disproportionately exposed to high levels of air pollutants, such as particulate matter, respiratory gases, and trace elements, such as lead and mercury [5]. Economically disadvantaged people and people who are nonwhite are disproportionately exposed to air pollution; these same population characteristics generally characterize the most polluted neighborhoods within cities, as described by various U.S. studies [9].

Mechanistic Pathways Linking Pollution to Health

Health implications of urban air pollution involve diverse outcomes driven by different pathophysiological processes, all share common features: inflammation and oxidative stress [7]. The pollutants behind these responses include such major constituents. Potential mechanisms by which urban air pollution affects health are being increasingly explored, contributing to a better understanding of whether and how the observed effects may operate at the individual level, as specified in Koch et al. 2022[8]. For example, ambient levels of particulate matter (PM) are consistently associated with cardiovascular disease (Vanos et al. 2019), and evidence suggests that short-term exposure induces myocardial infarction and ischemic strokes in susceptible people [7]. The acute effects of PM on cardiovascular health are believed to involve PM-induced systemic inflammation and oxidative stress, which in turn favor endothelial dysfunction and atherogenesis [7]. Long-term exposure may affect other phases of the atherogenic process, such as plaque rupture. Neuroinflammation is now firmly established as a correlate of aging and a key risk factor for cognitive decline in older people [8]. Emerging evidence associates PM exposure with neuroinflammatory changes and neurodegeneration in models of aging, Alzheimer's disease, and exposure to neurotoxic agents [6]. Furthermore, a small but growing body of literature examines perinatal exposure to cardiovascular risk factors and the possible interactions with maternal obesity and advanced maternal age on the risk of adverse neurodevelopmental outcomes to describe neuroinflammation and neurodegeneration, focusing on a panel of novel neurodegeneration-related biomarkers [9].

Inflammation and Oxidative Stress

Inflammation is recognized as a common underlying mechanism mediating the adverse health effects associated with urban air pollution exposure [17]. The primary mediators of inflammation, inducing diverse biological responses, are reactive oxygen species (ROS) and other oxidants, which are crucial in initiating a myriad of deleterious effects due to the complex chemical composition of urban air pollutants [13]. Significantly elevated levels of ROS and oxidative stress have been observed after exposure to urban air contaminants such as particulate matter [12]. The deposition of urban particulate matter into the respiratory tract stimulates the generation of ROS through multiple pathways inside tissue-resident and infiltrating immune cells, epithelial cells, fibroblasts, and other cell types, subsequently propagating intricate signal transformations through different cell types and organ systems [11]. Heterogeneous pollutants may induce similar patterns of signal transduction through

overlapping mechanisms of ROS generation and detoxification, potentially contributing to the initiation of a generalized pulmonary and systemic inflammatory process after urban air pollution exposure [10].

Endothelial Dysfunction and Atherogenesis

Endothelial dysfunction is characterized by a disturbance in the normal function of the endothelium (the inner lining of blood vessel walls) [17]. It constitutes a key early event in the formation of atherosclerosis, leading to significantly increased cardiovascular disease risk [20]. In the setting of air-pollution exposure, the improvement of endothelial function has been linked with a substantial decrease in cardiovascular-disease risk. The air-pollution-induced endothelial dysfunction is associated with excessive vascular permeability, impaired vascular vasomotor function, altered vascular remodeling, and depleted endothelial progenitor-cell populations [21]. Among a variety of air pollutants investigated, fine-particulate matter has been revealed to be a predominant pollutant responsible for vascular injury and promotion of atherosclerosis because blockage of fine-particulate-matter infiltration into the body abrogates air-pollution-induced vascular injury [10]. Fine-particulate-matter-induced endothelial dysfunction also involves several chemical components such as sulfate, nitrate, and heavy metals, including manganese, copper, arsenic, and zinc, in addition to particle size [12]. Particle pollution has been shown to induce systemic inflammation, oxidative stress, and lipid peroxidation in the circulation, and these changes are sufficient to promote early atherosclerosis [17]. The systemic vascular effects of environmental particulate matter exposure depend on the activation of reactive oxygen species-generating NADPH oxidase and activation of TLR4 pathways [16].

Neuroinflammation and Neurodegeneration

Several pathways have been proposed to explain the relationship between urban air pollution and neurological disorders. Systemic inflammation and oxidative stress remain widely recognized mechanisms [4]. Histopathological examinations in human brains have revealed glial activation and tau hyperphosphorylation associated with urban air pollution exposure [22]. Long-term exposure to particulate matter has been linked to neuroinflammation, altered immune responses, blood–brain barrier disruption, and increased accumulation of amyloid beta-42 and alpha-synuclein [2]. The brain serves as a target organ for coarse particulate matter, and both coarse and fine fractions of particulate matter deposited in the lungs have been shown to induce neurotoxic effects [23]. In experimental models, diesel engine exhaust was found to accelerate plaque formation associated with Alzheimer's disease, and ultrafine particles were observed to cross cellular membranes, triggering inflammation in lung and brain tissues [25]. Neurodegeneration attributable to environmental pollutants occurs at the early stages of the disease, and the central nervous system is an underappreciated target of urban air pollutants[7]. Children exposed to high ambient levels of air pollution develop early neurological alterations, including free radical generation, DNA damage, and neurodegeneration, which are typical hallmarks of aging and neurodegenerative diseases [24]. The presence of these biomarkers in exposed children, despite clinically appearing healthy, indicates that urban-related factors are among the most important determinants of neurological health.

Public Health Interventions and Policy Implications

Air pollution remains one of the world's most serious environmental hazards. People living in urban areas across the globe are exposed to a complex mixture of airborne contaminants that hinder optimal health [10]. Targeted interventions can improve urban air quality rapidly [12]. Integrating health considerations into urban planning, land use, and housing can significantly enhance public health [11]. Empirical evidence demonstrates that changes in emissions or exposure influence population health. Strong connections exist between urban air pollution and cardiovascular, respiratory, and neurological health outcomes [1]. Tracking the historical evolution of knowledge enables a more robust understanding of urban air pollution and associated health outcomes [12]. Policy frameworks that produce identifiable health improvements can motivate and bolster governmental action at all levels to address air quality pollutants. Equally important, however, are strategies that invoke fundamental changes to the way cities are constructed and urban life is lived [13]. Such strategies have the potential to yield substantial and, importantly, equitable health dividends. A systematic, comprehensive review of the adverse health effects of urban air pollution can thereby serve as a vital tool for addressing one of the great public health challenges of the current era [15].

Urban Planning and Transportation

Urban planning and transportation shape urban air pollution exposure. A broader literature search established evidence that urban and transport planning influences health [25]. Aspects such as noise, green space, active transportation, land use intensity, and proximity to major roads were shown to affect urban air pollution exposure. American Metropolitan Statistical Areas with high-capacity public transport systems saw lower mortality rates [17]. Since transportation policies may be the only sector over which Local Public Health Authorities exert significant influence, inclusion of health measures into these policies appears to be a feasible route to promote such

policies once the effect has been established [26]. Authorities on urban planning and transportation have proposed similar health-based urban and transport metrics, intending to encourage healthy planning initiatives [16].

Emission Reductions and Clean Technologies

Public health interventions targeting urban air pollution are inherently challenging. Striving for clean air in cities necessitates major changes in the supply of energy and transport [14]. The fact that all major contributions to urban air pollution regional transport, traffic, wood burning, industrial sources, and so forth, are subject to management by regulators provides an opportunity for pollution reduction: A range of clean technologies exists that could considerably lessen urban air pollution and thereby the associated health effects. But these technologies must be adopted, and emissions must be controlled. Clean technology exists [25]. Numerous studies have demonstrated that emissions reductions can lead to real air-quality improvements. The connection between exposure and health effects is well established [19]. The potential for health improvements from exposure reduction has been recognized, yet implementation remains elusive. Achieving clean air for urban populations is primarily a policy challenge, requiring changes in political will, resources, and the strength of institutions [18]. The pace and extent of change need to be sensitive to health inequalities, reducing adverse health outcomes in the most vulnerable populations first, and having a positive influence on health inequities. Local and regional particle pollution problems will not be solved by action on greenhouse-gas emissions alone [17].

Health Surveillance and Risk Communication

Air pollution has a long history of regulation in the industrialized world. However, the evidence base for the health effects of air pollution continues to evolve, with new pollutants emerging, the effects of known pollutants being better quantified, and greater recognition of the adverse health effects experienced by many urban residents. In light of above-ground and below-ground population changes, air pollution poses a particular and growing challenge for cities, where large segments of the population are highly exposed [20]. Consequently, health surveillance and risk communication are crucial to facilitate public engagement in identifying solutions. As a complement to mitigation measures, they enhance awareness of the issue and stimulate demand for further action [23]. Surveillance aims to identify and document the extent of health impacts, guiding targeted interventions by monitoring changes in circumstances, exposure, and outcomes over time [16]. Surveillance systems addressing urban air pollution already exist for multiple health endpoints, particularly in developing countries, indicating widespread recognition of urban air pollution as a contemporary health hazard. Risk communication serves to convey information that may help the public avoid adverse health effects [20]. Such communication appears particularly important for urban air quality; despite national and local legislation supporting monitoring and reduction, high concentrations persist, and awareness of air quality, its health effects, measures to mitigate exposure, and countermeasures remains low[22]. High-profile incidents, such as the 2015 Paris Climate Agreement, 1 also underscore the importance of policy uptake, as recommendations cannot exert any direct influence without policy implementation and engagement by the public and health professionals [23].

Research Gaps and Future Directions

Urban air pollution is an important public health topic, yet significant research gaps remain [2]. Further studies can improve methods for monitoring urban air pollution, assess its effects on additional health endpoints, strengthen mechanistic understanding, and enhance modeling of urban microenvironments to facilitate exposure health assessments [6]. Identification of priority research areas would support the growing urban air pollution-health community in guiding future investigations toward the most pressing and useful questions [25-27]. Methods for monitoring outdoor urban air pollution remain imperfect [21]. Understanding the precise relationship between urban air quality and diverse health effects remains in its infancy. Continuing investigations into key biological pathways and specific health outcomes are greatly needed. Procedures for estimating exposure to urban pollution at high spatial and temporal resolution remain limited [16]. Enhancing the characterization of urban microenvironments through further research would foster the development of novel conceptual models and facilitate urban exposure health assessments [27-33]

CONCLUSION

Urban air pollution continues to pose a significant threat to global public health, despite decades of scientific progress, regulatory action, and technological innovation. This narrative review demonstrates that contemporary urban environments expose residents to a complex mix of pollutants ranging from well-characterized particulate matter and trace gases to emerging contaminants such as ultrafine particles and airborne microplastics, each contributing to a wide spectrum of adverse health outcomes. Robust evidence links these pollutants to cardiovascular diseases, respiratory dysfunction, neurological impairment, metabolic disorders, and adverse perinatal outcomes, with biological mechanisms rooted in inflammation, oxidative stress, endothelial injury, and neuroinflammatory processes. The review also underscores that vulnerability to pollution is not evenly distributed. Children, the elderly, individuals with pre-existing illnesses, low-income populations, and minority communities bear a disproportionate burden, reflecting entrenched environmental injustices and systemic

inequities in urban settings. Addressing these disparities requires targeted policies that prioritize at-risk groups and foster equitable access to clean air. Advances in air-quality monitoring, including mobile sensors, personal exposure devices, satellite-derived data, and integrated modeling, have improved the precision and spatial resolution of exposure assessment. However, gaps remain in capturing real-time pollution dynamics, identifying emerging contaminants, and evaluating cumulative exposures across microenvironments. Continued innovation in measurement techniques is essential for accurate risk assessment and effective intervention. Effective public health interventions must go beyond traditional emission control to incorporate broader urban planning approaches that reshape transportation systems, reduce reliance on fossil fuels, expand green spaces, and enhance active mobility. Clean technologies and renewable energy solutions provide viable pathways to emission reduction, but their success hinges on strong political will, institutional capacity, and community engagement. Health surveillance, transparent risk communication, and long-term epidemiological tracking remain critical for guiding policy action and building public trust. In conclusion, urban air pollution is both a scientific and ethical challenge, one that demands coordinated action across environmental science, public health, urban planning, and policy. Reducing the global burden of disease associated with urban air pollution will require innovations in monitoring, a deeper understanding of mechanistic pathways, targeted protections for vulnerable populations, and transformative urban policies aimed at creating healthier, more resilient cities. Continued interdisciplinary research and evidence-informed policymaking are essential to achieving sustainable improvements in air quality and health equity worldwide.

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CITE AS: Rukundo Sande Kibuuka. (2026). Review of Urban Air Pollution and Health. IDOSR JOURNAL OF SCIENTIFIC RESEARCH 11(1):117-125. https://doi.org/10.59298/IDOSRJSR/2026/11.1.117125