

Indian Journal of Modern Research and Reviews

This Journal is a member of the '*Committee on Publication Ethics*'

Online ISSN:2584-184X



Research Article

Impact of Urban Air Pollution and Heavy Metal Exposure on Human Microbiota and Multisystem Physiology: A Narrative Review of the Gut–Lung Axis

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DOI: <https://doi.org/10.5281/zenodo.20445422>

Abstract

With rapidly growing cities and industrial development, urban air pollution and heavy metal exposure have become prominent environmental and human health issues. Numerous airborne contaminants such as particles, nitric and sulfur oxides, ozone, lead, cadmium, mercury and arsenic, have been associated with lung diseases, gastrointestinal system problems, cardiovascular diseases, kidney diseases, metabolic diseases and immune diseases. Furthermore, the toxins in our environment can modify the human microbiota and then disrupt physiological homeostasis by inducing dysbiosis. In this narrative review, we summarised the impacts of urban air pollution and heavy metal exposure on human microbiota and multisystem physiology, emphasizing the role of the gut-lung axis. The mechanisms underlying the association between environmental toxins and altered respiratory and gastrointestinal microbiota were discussed. Oxidative stress, chronic inflammation, epithelial barrier defects and immune dysfunction were all involved in the effect of airborne pollutants on microbiota. Dysbiosis of the gut-lung axis was reported to result in respiratory and gastrointestinal diseases, cardiovascular system dysfunction and kidney damage. Clarification of the interplay between environmental toxins, microbiota dysbiosis and multisystem physiology provides promising opportunities for devising novel prevention and treatments for air pollutant-induced diseases. Reducing the environmental exposures and recovering microbial balance should be regarded as alternative approaches for improving health.

Manuscript Information

- ISSN No: 2584-184X
- Received: 05-04-2026
- Accepted: 27-05-2026
- Published: 29-05-2026
- MRR:4(5); 2026: 350-356
- ©2026, All Rights Reserved
- Plagiarism Checked: Yes
- Peer Review Process: Yes

How to Cite this Article

Layek S, Sen A, Mondal S, Khatun T. Impact of urban air pollution and heavy metal exposure on human microbiota and multisystem physiology: A narrative review of the gut–lung axis. Indian J Mod Res Rev. 2026;4(5):350-356.

Access this Article Online



www.mrrjournal.in

KEYWORDS: Air pollution, heavy metals, gut-lung axis, microbiota, dysbiosis, oxidative stress, environmental toxicology.

INTRODUCTION

A rise in exposure of humans to toxic environmental contaminants, including air pollutants and heavy metals, in cities, has recently been attributed to industrial and urban development [1,4]. Constant growth of infrastructure used for transportation, emissions released into the atmosphere, combustion of fuels, and the collection of waste, has heavily influenced the world's environment [16, 17]. Some of the main pollutants of the air and heavy metals that cause numerous problems for human health are sulfur dioxide, lead, arsenic, Particulate Matter (PM), Nitrogen Dioxide (NO₂), Cadmium (Cd), mercury (Hg) and ozone (O₃) [2, 12, 21]. Human diseases that affect the kidneys, metabolic system, cardiovascular system, gastrointestinal system, respiratory system and the immune system are associated with long-term exposure to these pollutants [2, 3, 20].

A connection between human microbiota, which has been recently shown to be crucial for the homeostatic regulation, the gastrointestinal tract microbes and the fact that it is closely associated with the metabolism of nutrients, the immune system and protection against pathogens [5, 6, 15], has been confirmed. If the human microbiota is exposed to harmful substances and elements in the environment, their composition can be altered. The result is a disorder of the microbiota (dysbiosis) that causes various adverse physical outcomes [7, 8]. Exposure to air pollutants and heavy metals has been documented to have both direct impact and lead to dysbiosis of the lung and gut microbial communities [7, 11].

The gut-lung axis is a novel field of research that involves a bidirectional interaction between the gastrointestinal tract and the respiratory tract. The immune system, the metabolites produced by gut microbes, cytokines and inflammatory signals mediate the interaction [9, 10, 23]. Gut dysbiosis has been identified to affect immune system function and contribute to lung diseases, such as respiratory infection, COPD and asthma [18, 23]. Conversely, inflammatory effects in the lung cause loss of intestinal permeability, gut microbiota composition change and lead to systemic inflammation [10, 14].

Heavy metal poisoning aggravates these disease conditions in the body through mechanisms involving accumulation, free radical production (oxidative stress), damage of mitochondria, and activation of inflammation [12, 13, 25]. Inhaled airborne contaminants or toxins cause increased permeability of the intestinal epithelium and development of the intestinal barrier dysfunction [14, 19]. This process results in an increase of the microbial metabolic products that can contribute to systemic inflammation and damage in multiple organs and organ systems, including the development of cardiovascular, kidney diseases and altered immune system function [20, 24, 28].

MATERIALS AND METHODS

A narrative literature review was performed to systematically investigate the impact of environmental urban air pollution and heavy metal exposure on human microbiota and multisystem physiology, especially relating to the gut-lung axis. Electronic databases (PubMed, Scopus, Google Scholar, Web of Science) were searched for relevant literature. The search terms used to

query the literature database include 'urban air pollution', 'heavy metals', 'microbiota', 'gut-lung axis', 'dysbiosis', 'oxidative stress', 'respiratory diseases', 'gastrointestinal diseases' and 'environmental toxicology'. Review articles, observational, experimental and clinical studies published in the English language were included.

The identified literature was evaluated and relevant studies examining the link between environmental toxicants, the alteration in microbiota and inflammatory pathways and multisystem physiological outcome were selected. Irrelevant studies, duplicate records, non-English publications and case studies were excluded. The identified papers were then analyzed through a narrative review to outline the existing data regarding environmental toxins, microbial dysbiosis, oxidative stress, inflammation and resulting physiological outcomes.

Urban Air Pollution and Human Exposure

Urban air pollution is a major global public health concern that has resulted from the industrial and urbanization processes in many developing countries. [16, 17] Increasing volumes of industrial and vehicular exhaust emissions, fossil fuel and biomass combustion, and construction work have been associated with the deterioration of air quality in urban areas. [1, 4] Pollution of urban air and the resulting morbidities and mortalities of population across the globe particularly in cities, have also been studied. [17, 22]

Particulate matter (PM. And PM) is viewed as one of the potent pollutants of urban air, due to the smaller sizes of the particles that allow penetration deep into the lungs and the circulatory system [2, 3, 21]. Fine particulate matter can adsorb and bind heavy metals and organic pollutants, microbes, and may exacerbate the reactive oxygen species and inflammatory response in the body. [13, 25] Among the other significant air pollutants are the NO_x (NO and NO₂), SO, CO, ozone (O) and the VOCs that are the causes of multiple abnormalities in respiration and the body. [4, 16] Man is exposed to the pollutants through inhalation while it may also be through indirect route from food, water and the soil. [17] Multiple respiratory chronic diseases like asthma, chronic obstructive pulmonary diseases (COPD) and lung fibrosis has been observed with the prolonged exposure of man to the pollutants. [2, 18, 21] New evidences showed that the inhaled air pollutants can even affect the cardiovascular, GI, renal, nervous and metabolic conditions through their systemic inflammatory effects. [20, 22] It has also been demonstrated that air pollutants alter the microbial communities and their composition and diversity in both the respiratory and GI tracts. [7, 8, 27]

The effects of fine particulate matter on lung microenvironment result in the disruption of the mucosal barrier integrity and the depletion of immune responses and thereby leads to the respiratory microbial dysbiosis. [8, 18] Pollutant ingestion may be associated with dysbiosis of the gut microflora, damaging the gut barrier and promoting inflammatory and physiological dysfunction of multi-organ system. [14, 26] Vulnerable populations to air pollution including the children, the elderly and chronic ill people. [3, 16] Higher risk of exposure among populations with low socioeconomic status, working occupation and population

density and low levels of enforcement of the environmental regulation in the developing and industrial world has also been observed.^[17,22] The environmental pollutant is considered as major global public health problem.

Heavy Metals and Environmental Toxicity

There has been a great deal of research focused on human physiological and toxic mechanisms related to heavy metals, as well as their occurrence in the environment, entrance into the food chain, and modes of exposure at work sites.^[11,12] Arsenic, cadmium, lead and mercury have been identified as the most relevant of the clinically important environmental toxic metals.^[12] Toxic metals are widespread environmental pollutants that cause physiological abnormalities in multiple organ systems of the body including, but not limited to, gastrointestinal tract, respiratory system, circulatory system, renal system, neurological system, and the immune system as they are easily acquired, are slow to eliminate from the body, and have high bioaccumulation capabilities.^[11,13,25] These toxic metals gain entry into the human body by ingestion of polluted water and food, and by inhalation of contaminated urban dust, and exposure at workplaces (i.e., in industrial sites).^[17]

High-level accumulation of toxic heavy metals has also been detected in the human body via contaminated drinking water and food supply as a consequence of widespread environmental pollution of water and soil resources caused by mining and industrial activities, farming and inappropriate garbage management.^[12,17] Consequently, these pollutants have entered into the food chains and resulted in higher chances for chronic exposure in the humans. The primary toxic mechanism that causes such damage is usually linked to radical generation, causing oxidative damage in cells. Heavy metals also may disrupt cell signaling pathways, interference of the energy metabolism in mitochondria and trigger inflammatory responses. Damage to proteins, lipids and DNA leads to multi-functional disruption of cells.^[13,25]

Exposure to these toxic heavy metals can result in, gastrointestinal disorders, inflammatory diseases, lung disease, heart problems, kidney dysfunction, impaired neurological functions, and immunodeficiency.^[11,20,28] In addition, it has been established that environmental toxic agents can substantially alter human microbiota's composition and function.^[7,11] Heavy metal exposure could lead to an increase in the opportunistic pathogens and a decrease in beneficial commensal species in the human gut thus causing a dysbiosis of the human gut microbiota.^[7,15] In turn, gut dysbiosis could induce increased intestinal permeability, systemic inflammatory responses, impaired metabolic homeostasis, malfunction in host microbe interactions, and also affect integrity of mucosal barrier.^[14,19]

The interaction between the gut-lung axis as well as immune system dysregulation triggered by the metal-induced dysbiosis in turn could enhance susceptible for respiratory system diseases.^[9,10,23] Due to their wide distribution and easy entrance to the human body along with their potential for accumulation and adverse impacts to health, the toxicity of heavy metals poses a globally public health threat.^[17,22]

Human microbiota and Dysbiosis

A community of many microorganisms, including bacteria, viruses, fungi and archaea living on different parts of our bodies such as in the gut, respiratory system, skin and oral cavity is known as human microbiota.^[5,6] Among the microbial community, gut microbiota is a significant group in regulating host physiology and in maintaining host homeostasis through metabolism of nutrients, immune system regulation, active molecule synthesis and epithelial barrier integrity maintenance.^[6,15] The proper host function and protection from pathogenic microorganisms depend on a balanced microbiota.^[5]

Microbial communities can be regulated by factors such as food habits, way of living, medicines, infectious diseases and pollutants in the environment like air pollution and heavy metals.^[7,8] Air pollutants and toxic metals have been identified as factors which are responsible for disrupting a balanced microbiota (known as microbial dysbiosis).^[7,11] Microbial dysbiosis is a term that signifies the imbalance in a healthy microbial community which are related to the depletion of beneficial bacteria and proliferation of pathogenic species.^[15]

Air pollutants (like PM, O₃ and NO₂) can lead to changes in respiratory and gut microbiota by having impacts on the immune system via oxidative stress and inflammation.^[8,26,27] Air pollutants can also trigger the development of respiratory tract inflammation and affect mucosal immunity and epithelial barrier integrity in the respiratory tract, which lead to an imbalance of microbes.^[18] Similarly, ingestion of pollutants into the gastrointestinal tract and systemic inflammatory mediators will also influence the bacterial composition of the intestinal microbiota and cause increase of intestinal permeability.^[14,19]

Heavy metals (like Lead, Cadmium, Mercury, and Arsenic) might also further disturb microbiota by promoting antibiotic-resistant or opportunistic bacteria growth while depleting beneficial probiotic bacteria involved in anti-inflammation and metabolism.^[7,11,12] And consequently altering microbial metabolism, it leads to the subsequent increase of oxidative stress, inflammation, immune dysfunction, metabolic disorders.^[13,24]

With increasing evidence in recent studies, microbial dysbiosis is now shown to be closely related to chronic diseases such as asthma, COPD, IBD, obesity, cardiovascular disease and renal disorders.^[18,24,28] The gut-lung axis which links gut and lung microbiota may affect by the inflammatory cytokines and their mediators, microbial products and immune signaling pathways^[9,10,23]. The exploration on relationship between pollution exposure and alteration of microbiota may offer new direction to treat systemic illness.

The Gut-Lung Axis

The term gut-lung axis represents the bi-directional inflammatory, microbial and immune signaling pathway between the gut and the lung.^[9,10,23] Discoveries over recent years have shown that the gut microbiota can profoundly impact the lung immune response, subsequently modulating pulmonary inflammation, immunity and the risk of developing lung disease.^[10,18] Likewise, the pulmonary immune response

and lung diseases and inflammatory states in the respiratory tract can affect the gut microbiome and integrity of the intestinal barrier^[9,23] This suggests a complex interaction between both organ systems.

Mediators of the gut-lung axis include the microbial metabolites, cytokines, immune cells and neuronal signaling.^[10] Beneficial microbes produce short-chain fatty acids (SCFAs) and other anti-inflammatory microbial metabolites in the gut to support immune homeostasis.^[23] This from a normal gut microbiota can induce a systemic immune response which ultimately modulates response in the respiratory tract^[9,10] Consequently, a loss in gut microbial balance can lead to immune system dysfunction and promote the inflammatory state in the respiratory tract^[18]

Environmental pollutants such as particulate matter and heavy metals may influence the gut-lung axis through multiple mechanisms. [8,11] Evidence from inhaling pollutants has demonstrated the creation of inflammatory stimuli, oxidative stress and epithelial injury in the lungs.^[13,25]

Subsequently, the inflammation occurring in the lungs was reported to cause the release of inflammatory signals into the circulation^[13,25] these circulating inflammatory signals further induce alterations in intestinal permeability and gut microbiota composition and, as a result, promote intestinal dysbiosis.^[14,19] Gut dysbiosis generated by the inhaled pollutant could then promote negative effects in the respiratory immune system, particularly via altered immune cell responsiveness and modulation of microbial metabolites.^[7,27]

It has been confirmed that there is a link between gut-lung axis dysfunction and a number of lung disorders such as asthma, COPD, allergic airways disease, respiratory infection etc.^[18,23] Alterations in intestinal microbiota can potentially weaken the defense of the organism against respiratory infections and inflammation of the lung.^[9,10] Consequently, systemic inflammation initiated in the gut, may eventually produce chronic organ system disease and system-wide functional deficit.^[20,24]

Restoration of gut microbiota balance through changes in diet, prebiotics, probiotics and life-style modification may help improve function of gut-lung axis and alleviate inflammation^[10,30] These results offer prospects in the development of preventive and treatment strategies for pollutant-associated and chronic diseases.

Respiratory Effects

It has been established that exposure to air pollution, heavy metals, and other respiratory events occur in close proximity.^[16,21,2] Respiratory exposure to pollutants with heavy metals, toxic gases and particulate matter can contribute to damage to the respiratory epithelium, a breakdown of mucociliary clearance, and inflammation of the airway.^[23] PM_{2.5} has the ability to enter into the alveolar space, causing an inflammation and oxidative stress within the lung.^[3,13] The chronic inhalation of polluted air can cause numerous chronic airway diseases such as; asthma, COPD, bronchitis, pulmonary fibrosis and respiratory infections.^[18,21] Air pollution can promote airway hyper responsiveness, hypersecretion of mucus

and remodeling within the airway and cause exacerbated pre-existing diseases of the lung.^[21,23] The patient that suffers with chronic lung diseases are extremely susceptible to the pollutants effects. Cd, As, Hg, Pb and other heavy metals can cause further respiratory toxicity. The inhalation of heavy metals will cause inflammation and damage within the lungs.^[11,12] The long term exposure of humans and other beings to heavy metals cause damage to lung tissue, inflammation within the airway, and a heightened sensitivity to respiratory infections.^[12,20] Some heavy metals will accumulate within lung tissue and cause detrimental lung toxicity through free radical mediated mechanisms.^[13,25] The local pulmonary environment, including respiratory microbiota, and the immune response can also be negatively affected by environmental pollution.^[8,18] The respiratory microbiota can be disrupted, increasing sensitivity to infections. Intestinal dysbiosis, induced by air pollution, may affect lung immunity via the gut-lung axis, increasing risk of respiratory illness^[9,10,23] The primary mechanism of lung damage by environmental pollutants is oxidative stress.^[13,25] The generation of excessive reactive oxygen species causes damage to lipids, DNA and protein within the lung tissue, triggering inflammatory cytokine release.^[20,25]

Gastrointestinal Effects

The gastrointestinal tract is directly affected by environmental pollutants and heavy metals by direct toxic actions and inflammatory mechanisms and changes in the intestinal microbiota population. ^[11,12,26] Constant contact with environmental pollutants in the air, food, water and soil may induce imbalance in the intestinal system and several gastrointestinal diseases.^[16,17]

The environmental pollutants enter the gastrointestinal tract by swallowing and mucociliary Clearance in the airway may carry the pollutants into the intestinal tract causing direct effects on the gastrointestinal mucosa and on the intestinal microecology.^[7,15]

In addition particulate matters and environmental toxins impair the integrity of intestinal epithelial and can be responsible for increased permeability in intestinal epithelium (leaky gut).^[14,19] In this condition bacterial translocation, entry of pollutants and pro-inflammatory molecules into circulation can be occur, thus provoking chronic inflammation and immune responses.^[14] Moreover these compounds are responsible for damaging intestinal epithelia, causing mucosal inflammation and exacerbating intestinal injury via oxidative stress.^[13,25]

Heavy metals including arsenic (As), cadmium (Cd), lead (Pb) and mercury (Hg) cause direct injury to the gastrointestinal tract. ^[11,12] Their chronic exposure can result in mal-digestion and mal-absorption and alter the intestinal flora.^[11] The beneficial bacteria could be decreased and the opportunistic microbes would proliferate. Dysbiosis and subsequent gastrointestinal inflammation is established.^[7,15] Subsequently altered bacterial metabolites synthesized under these circumstances may promote immune responses in distal organs.^[24]

Environmental pollutant induced dysbiosis is also the cause for IBD, IBS, colitis and metabolic diseases. [15,26] This intestinal inflammation also induces immune responses in remote organs via gut-lung axis. [9,10,23] As a result chronic gastrointestinal inflammation cause systemic physiological imbalance in a multitude of organs beyond digestive system [20,24]

Restoring the microbial balance by changing diets, probiotics and decreasing exposure to environmental pollutant can help in prevention of gastrointestinal injuries induced by environmental pollutants. [10,30] Therefore the understanding of the intestinal damage resulting from environmental toxicants is essential for prevention and treatment for pollution related diseases [16,17]

Cardiovascular and Renal Effects

Cardiovascular and renal damage is driven by inflammation, oxidative stress, Immune system dysregulation and endothelial dysfunction induced by environmental pollutants and heavy metals [2,13,20] Epidemiologic and clinical data indicate increasing linkage between long term air pollution and heavy metal exposure to chronic kidney disease, ischemic heart disease, hypertension and atherosclerosis [17,20,22]

In the lung, inhaled fine particulate matter and gaseous pollutants may enter into the systemic circulation, interact with vascular tissue and thus lead to endothelial damage, poor vascular function and chronic inflammation.[2,3] The vascular dysregulation could initiate the development of vascular stiffness, thrombus formation and chronic vascular disease progression.[20] High level air pollution in urban environments is linked with elevated risk of cardiac arrhythmias, hypertension, myocardial infarction and cerebrovascular diseases [2,20,22]

The cardiovascular toxicity of heavy metals like cadmium, arsenic, mercury and lead might compound the toxicity on the cardiovascular system [11,12] Accumulation of heavy metals in target tissues directly disturbs cellular signal transduction pathways, inhibits endogenous antioxidant defense mechanisms, induces mitochondrial dysfunction and reduces nitric oxide availability which leads to the impairment of cardiac and vascular function.[13,25] Chronic heavy metal exposure has been found to be linked with enhanced production of inflammatory cytokines and metabolic disturbances.[20]

The kidney, with its function of filtration and excretion of exogenous and endogenous toxic substances is highly susceptible to the damaging effects of environmental toxicants [28] Accumulation of heavy metals in the kidney may cause oxidative damage and tubular injury, promote inflammatory response and lead to reduced renal perfusion ultimately resulting in chronic kidney injury.[11,28] Reduced glomerular filtration rate (GFR) and proteinuria were noted during chronic heavy metal exposure.[28] Chronic inflammation from air pollution, on the other hand may augment vascular dysfunction within the renal microcirculation and compromise kidney function.[20,22]

The dysbiosis in microbial communities has recently emerged as a contributing factor that alters cardiovascular and renal function via immune signaling, microbial metabolites and metabolic pathways [24,28] Gut dysbiosis was found to be

correlated with endothelial dysfunction, metabolic abnormalities and chronic inflammation which support the concept of environmental pollution associated multisystem organ failure.[7,24] As such environmental toxins could serve as significant risk factors in the ongoing global cardiovascular and renal epidemic.[17,22]

Mechanisms: Oxidative stress and inflammation

It is now thought that the pathogenesis of air pollutant-mediated toxicity and heavy metal intoxication is, to a large degree, a mechanism of oxidant stress and sustained inflammation [13,25] By generating overproduction of ROS in tissues/organs it upsets homeostatic regulation, triggering inflammatory responses in organs.[2,13] If this oxidative stress persists, it causes cellular damage such as protein modification, lipid peroxidation, DNA damage and organelle damage; leading to the failure of systems throughout the body [13,25]

Both fine particulate matter and gaseous air pollutants may directly trigger an oxidative insult to vascular and pulmonary tissue via generation of reactive oxygen species, resulting in disruption of mitochondrial function. [3,20] Fine particles penetrate deep into lung and translocate into the systemic circulation, initiating endothelial damage and inflammatory responses in vessels [2,3] These actions induce systemic inflammation and vascular dysfunction [20]

Cadmium, arsenic, mercury, lead and other heavy metals exacerbate an oxidant insult by inhibiting the endogenous antioxidant defenses of organs, thus enhancing the generation of ROS [11,13,25] Also environmental toxins have the ability to initiate inflammatory cascades involving activation of immune cells and signalling pathways, leading to the release of cytokines and resulting tissue damage [20] The persistence of these cascades may lead to a sustained, low-level inflammation which, coupled with a dysfunctional immune system, may lead to irreversible tissue damage [13]

These cytokines may subsequently impact numerous organ systems including lungs, cardiovascular system, kidneys and the gut [20,24] The inflammatory signalling cascades may be further exaggerated by microbiota dysbiosis. [7,14] Changes in microbial composition may compromise barrier integrity of the gut and allow bacterial products to enter the circulation. This stimulates systemic inflammatory responses. [14,19]

In addition, microbes present and their metabolites may influence mitochondrial and redox state of peripheral organs. [24,30]

These interactive cellular mechanisms of oxidant stress, inflammatory signalling, endothelial dysfunction and microbiota dysbiosis are the probable central pathways mediating air pollutant- and heavy metal-induced multisystem diseases [13,20,25]

Future Prospects

The accumulating evidence establishing the link between pollution, microbial dysbiosis and multisystem physiological disorder shows that research in this field will continue to be a highly multidisciplinary effort. Although we have advanced considerably in knowing the link between environmental

toxicants and human health, the mechanistic paths in pollutant-induced microbial changes and multisystem diseases are still not fully clear. Future research directions should identify distinct microbial signatures to pollutant exposures to air pollutants and heavy metal.

Molecular technologies such as metagenomics, metabolomics and microbiome sequencing would help in further delineating the link between environmental toxicants, host immune system and microbial communities, as well as long-term and population-based studies in evaluating the chronic health impacts of environmental toxicants. The gut-lung axis should be a focus for future therapeutic and preventive strategies. Modulation of the gut-lung axis with probiotics, prebiotics, diet changes and lifestyle modifications can potentially treat and/or prevent multisystem inflammation and promote physiological homeostasis.

Antioxidative and anti-inflammatory treatments can be explored in treating and/or preventing pollution-induced tissue damage mediated by oxidative stress. Better pollution controls, monitoring and regulations for reducing human exposures to environmental toxicants are warranted. Increased awareness on environmental health risk factors and protective actions would benefit from prevention of pollution-induced illness. It requires the collaborations of environmental scientists, microbiologists, physiologists, clinicians and policy-makers to address the global concern on the health impacts of pollution. Future microbiota-target treatments and environmental interventions should be explored for prevention of diseases and promotion of better multisystem health status in communities chronically exposed to urban pollution and heavy metals.

CONCLUSION

Environmental pollution of urban air and exposures to heavy metals remain an outstanding challenge in environmental health, having critical consequences on human microbiota and physiology. Growing evidence indicates that toxicants at the environmental level mediate disruption in the host's microbiota (microbial dysbiosis), oxidative stress and inflammatory activation and dyshomeostasis in the immune system.

This set of mechanisms results in damage to respiratory, gastrointestinal, cardiovascular and renal systems through the cooperation of multiple physiological routes. Guts-lung axis is the most important strategy to study the link between gut and lungs, depending on how it impacts under exposures to the environment, with a potential key role played by the modulation of gut microbiome and inflammatory signals in the pathogenesis of diseases relevant to pollution.

Therefore, the clarification of these multiple links and connections between pollutants and microbiota at both the peripheral and systemic levels could provide the foundation to the design of effective preventative strategies and specific therapy to combat environmental illnesses, and future studies should focus on modulating the microbiome and reducing the environmental toxicant burden to enhance human health.

Acknowledgment

The authors wish to thank all the scientists and authors whose published work helped in creating this narrative review.

Conflict of interest

The authors reported no conflict of interest.

Funding

The authors received no funding for this research work, authorship and publication of this article.

Author contributions

Shahan Layek: conceptualization, coordination, writing-review and editing. Argha Sen: writing-microbiota portion of review and writing-review & editing. Silvia Mondal: writing-environment and pollution-based parts and writing-review & editing. Tania Khatun: literature gathering and writing-review & editing.

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