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ENVIRONMENTAL FINE PARTICULATE MATTER (PM2.5) AS A DETERMINANT OF GASTRIC MUCOSAL REMODELING: MORPHOLOGICAL MECHANISMS, MICROVASCULAR DYSFUNCTION, AND TRANSLATIONAL IMPLICATIONS

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Abstract: Air pollution has become one of the most significant environmental determinants of global morbidity and mortality. Among atmospheric pollutants, fine particulate matter with an aerodynamic diameter of ≤2.5 μm (PM2.5) is considered particularly hazardous due to its high surface reactivity, ability to carry toxic compounds, and capacity to induce systemic biological responses. While the respiratory and cardiovascular consequences of PM2.5 exposure have been extensively investigated, the gastrointestinal tract—particularly the stomach—has received comparatively limited scientific attention despite growing evidence of its vulnerability to environmental pollutants.

This study provides a comprehensive analysis of the morphological and pathophysiological effects of PM2.5 exposure on gastric tissues. The objective of this review is to synthesize current experimental, epidemiological, and mechanistic evidence describing how airborne particulate pollutants influence gastric mucosal integrity, inflammatory responses, microcirculatory function, and epithelial regeneration. Particular emphasis is placed on the role of oxidative stress, endothelial dysfunction, systemic inflammatory signaling, and gut barrier disruption as key mechanisms linking air pollution to gastric pathology.

Recent experimental models demonstrate that chronic exposure to particulate pollutants may induce structural alterations in gastric tissues including epithelial degeneration, inflammatory infiltration, vascular remodeling, and impaired mucosal regeneration. In addition, morphometric analyses indicate significant changes in mucosal thickness, glandular architecture, and microvascular density. These findings suggest that the stomach represents an important secondary target of environmental toxicants, mediated through both direct and systemic biological pathways.

Understanding the morphological consequences of air pollution for gastrointestinal tissues is essential for developing preventive strategies and improving environmental health policies. Future interdisciplinary research integrating environmental toxicology, molecular pathology, and clinical gastroenterology will be crucial for clarifying mechanisms of gastric injury and identifying potential therapeutic targets.

Keywords: PM2.5, air pollution, gastric mucosa, environmental toxicology, morphometry, oxidative stress, inflammation, microcirculation.

Introduction

Environmental pollution represents a major global health challenge, with increasing industrialization, urbanization, and transportation emissions contributing to rising concentrations of atmospheric pollutants. According to the World Health Organization, air pollution is responsible for millions of premature deaths annually and is strongly associated with a broad spectrum of chronic diseases.

Fine particulate matter (PM2.5) has attracted particular scientific attention due to its ability to penetrate deeply into biological systems and initiate systemic inflammatory responses. These particles are composed of complex mixtures of organic compounds, metals, sulfates, nitrates, and other reactive chemical species capable of inducing oxidative stress and cellular injury.

Although the respiratory tract represents the primary site of pollutant entry, accumulating evidence indicates that PM2.5 exposure affects multiple organ systems. Epidemiological studies have demonstrated associations between particulate pollution and cardiovascular diseases, metabolic disorders, neurodegeneration, and immune dysregulation.

In recent years, the gastrointestinal tract has emerged as a potential target of environmental pollutants. The stomach, as a central organ of digestive physiology, plays a critical role in nutrient processing, barrier defense, and immune regulation. Gastric mucosa is characterized by rapid epithelial turnover, complex glandular structures, and highly regulated microcirculation.

However, chronic exposure to environmental pollutants may disrupt these physiological processes. Several experimental studies suggest that particulate matter may affect gastric tissues through systemic inflammation, neurohumoral pathways, and translocation of swallowed particles cleared from the respiratory tract.

Despite growing interest in this topic, the morphological consequences of air pollution for gastric tissues remain insufficiently characterized. Therefore, this review aims to analyze current evidence regarding the structural and morphometric alterations of the gastric mucosa associated with PM2.5 exposure and to discuss the underlying biological mechanisms.

Materials and Methods

This study was conducted as a systematic analytical review of contemporary scientific literature addressing the relationship between atmospheric pollution and gastrointestinal pathology.

Scientific publications were identified through major international databases including **PubMed, Scopus, Web of Science, and Google Scholar**. The search strategy included combinations of keywords such as:

- air pollution
- PM2.5 exposure
- gastric mucosa
- gastrointestinal inflammation
- oxidative stress
- microcirculation
- environmental toxicology

The review included peer-reviewed studies published between **2020 and 2025**, encompassing epidemiological investigations, experimental animal models, and mechanistic molecular studies. Articles focusing on morphological, histopathological, or morphometric changes in gastrointestinal tissues were prioritized.

Data extraction focused on several key parameters:

- structural alterations of gastric mucosa
- inflammatory responses and immune signaling
- oxidative stress markers
- microvascular dysfunction
- epithelial barrier integrity.

The collected data were synthesized and analyzed to identify common mechanisms and emerging research trends.

Results

Systemic Effects of Fine Particulate Matter

Fine particulate matter is characterized by a high surface area capable of adsorbing toxic substances including heavy metals, polycyclic aromatic hydrocarbons, and organic pollutants. Once inhaled, PM2.5 particles interact with respiratory epithelial cells and immune cells, initiating inflammatory responses and oxidative stress.

These systemic responses may extend beyond the lungs through circulating cytokines, inflammatory mediators, and oxidative molecules that affect distant organs.

Pathways Linking Air Pollution to Gastric Injury

Several biological mechanisms may explain how air pollution influences gastric tissues.

First, inhaled particles deposited in the respiratory tract may be transported through mucociliary clearance and subsequently swallowed, allowing direct interaction with gastric mucosa.

Second, systemic inflammatory mediators released during pulmonary responses may alter gastrointestinal immune regulation.

Third, endothelial dysfunction induced by particulate exposure may impair microvascular perfusion within gastric tissues.

Finally, environmental stressors may affect autonomic nervous system regulation of gastric secretion and motility.

Morphological Alterations of Gastric Tissues

Experimental studies have identified multiple structural changes in gastric mucosa associated with pollutant exposure.

Common morphological findings include:

- inflammatory infiltration of lamina propria
- epithelial cell degeneration

- glandular structural alterations
- mucosal barrier disruption
- vascular congestion.

These alterations suggest that chronic pollutant exposure may contribute to progressive remodeling of gastric tissues.

Discussion

The findings of this review indicate that air pollution may represent an important environmental risk factor for gastrointestinal pathology. The stomach appears particularly vulnerable due to its exposure to both systemic inflammatory signals and swallowed particulate contaminants.

Oxidative stress plays a central role in pollutant-induced gastric injury. Reactive oxygen species generated by particulate exposure can damage cellular membranes, proteins, and DNA. These processes activate inflammatory pathways and disrupt epithelial barrier function.

Another important mechanism involves endothelial dysfunction and microvascular impairment. Reduced tissue perfusion may exacerbate hypoxia and impair regenerative processes in gastric mucosa.

These findings highlight the need for further interdisciplinary research integrating environmental science, gastroenterology, and molecular pathology.

Conclusion

Fine particulate air pollution represents a significant environmental determinant of gastric mucosal injury. Chronic exposure to PM2.5 may induce inflammatory, degenerative, and vascular changes within gastric tissues, ultimately leading to structural remodeling and impaired physiological function.

Understanding these mechanisms is essential for developing preventive strategies and improving environmental health policies aimed at reducing pollution-related gastrointestinal diseases.

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