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IMPACT OF POLLUTED AMBIENT AIR ON GASTRIC MORPHOLOGICAL PARAMETERS: CURRENT EVIDENCE AND MODERN PERSPECTIVES

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Resume

Ambient air pollution is widely recognized as a major environmental determinant of chronic non-communicable diseases. Current research has predominantly focused on the adverse effects of fine particulate matter (PM2.5), toxic gases, and tobacco smoke constituents on the respiratory and cardiovascular systems. Nevertheless, evidence regarding pollution-related injury of the gastrointestinal tract, particularly gastric tissues, remains fragmented and insufficiently systematized. This review summarizes contemporary concepts of the pathogenetic mechanisms underlying gastric morphological alterations associated with polluted air exposure. Special attention is given to inflammatory and degenerative changes, microcirculatory disturbances, and impairment of the mucosal barrier. Additionally, key approaches to morphological assessment and quantitative morphometric analysis are outlined. The roles of oxidative stress, chronic low-grade inflammation, endothelial dysfunction, neurohumoral dysregulation, and immune responses are discussed, along with perspectives for preventive and corrective strategies.

Keywords

ambient air pollution, PM2.5, stomach, morphology, morphometry, oxidative stress, inflammation, microcirculation.

In recent decades, environmental factors have become leading determinants of chronic non-infectious diseases. Against the background of rapid urbanization, increased transportation load, industrial growth, and changes in energy consumption patterns, a steady rise in atmospheric pollutant concentrations has been observed. Fine particulate matter (PM2.5) is considered among the most hazardous components due to its ability to penetrate deeply into the respiratory tract and exert systemic effects through inflammatory and metabolic pathways.

Although the stomach is not the primary site of contact with aerosol pollutants, accumulated evidence suggests that it may represent an important target organ of toxic exposure. This vulnerability is related to complex neurohumoral and vascular responses, as well as the entry of particles into the gastrointestinal tract through mucociliary clearance and subsequent swallowing.

Modern morphology considers the stomach to be a highly dynamic system characterized by continuous epithelial regeneration, active barrier defense mechanisms, and complex microcirculatory regulation. Disruption of these components during chronic exposure to environmental pollutants becomes a critical factor in the development of functional disorders and structural gastric alterations. Therefore, systematizing current data on the morphological consequences of air pollution on gastric tissues remains a relevant task with significant fundamental and clinical value.

Ambient air pollution includes a broad spectrum of chemical and physical agents such as particulate matter, nitrogen and sulfur oxides, ozone, volatile organic compounds, heavy metals,

and products of incomplete fuel combustion. PM2.5 may contain polycyclic aromatic hydrocarbons, diesel exhaust constituents, sulfates, and nitrates, which possess pronounced pro-oxidant and pro-inflammatory potential.

Inhalation exposure to pollutants triggers a cascade of systemic reactions, including activation of macrophages and neutrophils, release of pro-inflammatory cytokines, and increased production of reactive oxygen species (ROS). These processes may impair endothelial function, disrupt microcirculation, and create conditions for chronic inflammation in various organs, including the stomach.

Despite the fact that the primary contact with pollutants occurs in the respiratory tract, the stomach may be affected through several mechanisms:

1. **Indirect systemic pathway.** Inflammatory mediators and endothelial dysfunction promote vascular injury and tissue hypoxia, which is particularly critical for the gastric mucosa due to its high sensitivity to ischemic factors.

2. **Particle ingestion.** Particles and toxic compounds deposit on the respiratory mucosa and subsequently enter the gastrointestinal tract through swallowing.

3. **Neurohumoral regulation.** Stress responses, autonomic imbalance, and hormonal shifts may influence gastric acid secretion, motility, and mucosal trophic regulation.

4. **Immune mechanisms.** Activation of innate immunity and impairment of mucosal immune defense (MALT) contribute to the development of chronic inflammatory processes.

Morphological manifestations depend on exposure duration and intensity, age, baseline mucosal status, and associated risk factors (smoking, nutrition, stress). The literature highlights several major groups of gastric alterations:

Chronic exposure to pollutants is associated with gastritis-like alterations, including infiltration of the lamina propria by lymphocytes and macrophages, increased numbers of plasma cells, and enhanced local production of inflammatory mediators. These changes may be accompanied by disruption of barrier function and increased epithelial permeability.

Pro-oxidant components of pollutants may initiate membrane injury, mitochondrial dysfunction, and apoptosis of epithelial cells. Morphologically, this may manifest as cytoplasmic vacuolization, loss of cellular polarity, focal epithelial destruction, and altered secretory activity of the glandular apparatus.

Microcirculatory impairment is considered a key component of the pathological process, including vascular spasm, stasis, endothelial dysfunction, and increased capillary permeability. This results in interstitial edema, tissue hypoxia, and secondary dystrophic changes. Within gastric morphology, microcirculatory disorders are regarded as factors that potentiate inflammation and delay regeneration.

The gastric mucosa is characterized by a high rate of cellular renewal. Under chronic injury, an imbalance between proliferation and apoptosis may occur, leading to changes in mucosal thickness, restructuring of the glandular system, and a reduction in functional reserve.

To objectify morphological changes in experimental models and clinico-morphological studies, the following morphometric parameters are commonly used:

- thickness of the mucosa and submucosal layer;
- depth and density of gastric glands;
- extent (area) of inflammatory infiltration;
- ratio of chief to parietal cells;
- quantitative assessment of apoptosis and proliferation (e.g., Ki-67 and caspase-3 markers in immunohistochemistry);
- microcirculatory parameters (capillary density, vessel diameter, severity of stasis).

Morphometric analysis enables the transition from descriptive morphology to quantitative assessment, substantially increasing the reliability and comparability of results.

Several key pathogenetic mechanisms of pollution-related gastric injury have been described:

Systemic inflammation with elevated levels of IL-6, TNF-α, and other mediators may alter local immune regulation and contribute to chronic gastritis development.

Vascular mechanisms play an essential role: endothelial impairment leads to vasospasm and compromised trophic supply of the mucosa. Consequently, mucosal injury may progress even without direct contact with toxic particles.

Chronic environmental stress and toxic exposure may disturb sympathetic-parasympathetic balance, influencing acid secretion and gastric motility.

ANALYTICAL DISCUSSION AND FUTURE PERSPECTIVES

Integrating the available evidence, the stomach may be considered a significant “secondary” target organ of ambient air pollution. A key issue is the potential synergy between environmental and behavioral factors: polluted air exposure frequently coexists with smoking, unbalanced nutrition, and psychoemotional stress. Therefore, gastric injury should not be interpreted as the effect of a single agent but rather as a multifactorial process.

Future research priorities include:

- development of standardized experimental models of PM2.5 exposure;
- implementation of immunohistochemical and molecular approaches to clarify damage mechanisms;
- investigation of the role of gastric microbiota in the formation of inflammatory changes;
- identification of effective corrective strategies (antioxidant protection, anti-inflammatory approaches, endothelial protection).

CONCLUSION

Current evidence indicates that polluted ambient air may induce structural and functional changes in the gastric mucosa. Morphological manifestations include inflammatory infiltration, epithelial dystrophic alterations, and microcirculatory disturbances. Morphometric analysis

provides an objective assessment of injury severity and allows the detection of early signs of mucosal remodeling. Further development of clinico-morphological research requires integration of morphology, toxicology, and molecular medicine, which will support scientifically grounded preventive and corrective approaches under unfavorable environmental conditions.

REFERENCES

1. Халимова, Ю. С. (2025). ВОЗДЕЙСТВИЕ ЗАГРЯЗНЁННЫХ СРЕД НА ИММУНОЛОГИЧЕСКИЕ ОРГАНЫ ЧЕЛОВЕКА (ОБЗОР ЛИТЕРАТУРЫ). В Вестник фундаментальной и клинической медицины (Т. 6, Выпуск 20, сс. 1535–1540). Zenodo. https://doi.org/10.5281/zenodo.18063029
2. Khalimova, Y. S. (2025). IMPACT OF SMOG EXPOSURE ON THE MORPHOLOGICAL STATE OF THE HUMAN BODY. *SHOKH LIBRARY*, *1*(12).
3. Gao, W., et al. (2025). Ambient particulate matter exposure impairs gut barrier function and promotes inflammatory injury. *International Journal of Molecular Sciences*.
4. Kou, Y., et al. (2024). Exposure to air pollution and non-neoplastic digestive diseases: Population evidence and mechanisms. *Frontiers in Public Health*.
5. Kou, Y., et al. (2025). Long-term exposure to air pollution and gastrointestinal disease. *BMC Public Health*.
6. Li, J., et al. (2024). Air pollutants, genetic susceptibility, and the risk of incident peptic ulcer and chronic gastritis. *Environment International*.
7. Li, J., et al. (2024). Air pollutants, genetic susceptibility, and the risk of incident peptic ulcer and chronic gastritis. *Environment International* (ScienceDirect abstract).
8. Liao, R., et al. (2025). Fine particulate matter (PM2.5) disrupts intestinal barrier function by inducing oxidative stress and inflammatory signaling. *International Journal of Molecular Sciences*, 26(17), 8271.
9. Loffredo, L., et al. (2025). Tobacco smoke exposure and oxidative stress: The role of gut barrier disruption and systemic inflammation. *Antioxidants*.
10. Kim, D. H., et al. (2025). Gastroesophageal disease risk and inhalational exposure: Evidence linking air pollutants and upper GI pathology. *Scientific Reports*.
11. Shi, H., et al. (2025). Underlying lung–gut metabolic communication and the impact of PM2.5 exposure on microbiota and metabolites. *The Innovation (Medicine)*.
12. Thomas, T. (2025). Air pollution and disrupted microbiomes: Tracing the impact on human health. *Cureus*.
13. Vivarelli, F., et al. (2024). Effects of unburned tobacco smoke on oxidative stress, inflammatory responses, and DNA damage. *Frontiers in Pharmacology*.