

COMBINED IMPACT OF PM2.5 AND CIGARETTE SMOKE ON GASTRIC  
MUCOSAL MORPHOLOGY: MECHANISMS OF INJURY AND MODERN  
APPROACHES TO CORRECTION

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**Abstract**

**Background:** Air pollution and cigarette smoking are among the most significant modifiable environmental risk factors affecting human health. Fine particulate matter (PM2.5) and cigarette smoke share similar physicochemical properties and biological effects, including oxidative stress, inflammation, and epithelial barrier disruption. However, their combined impact on the gastrointestinal system, particularly gastric mucosa, remains insufficiently explored.

**Objective:** This study aims to analyze the morphological and functional alterations in gastric tissues under the combined exposure to PM2.5 and cigarette smoke, and to summarize modern approaches for correcting these pathological changes.

**Methods:** A comprehensive literature review was conducted focusing on experimental and clinical studies investigating the effects of PM2.5 and cigarette smoke on gastric mucosa. Special attention was given to mechanisms of oxidative stress, inflammatory response, apoptosis, microcirculatory disturbances, and epithelial barrier dysfunction.

**Results:** Combined exposure to PM2.5 and cigarette smoke leads to pronounced morphological damage of gastric mucosa, including epithelial desquamation, glandular dystrophy, inflammatory infiltration, and mucosal barrier disruption. These effects are mediated through synergistic activation of oxidative stress pathways, pro-inflammatory cytokine release, mitochondrial dysfunction, and impairment of tight junction proteins. Furthermore, alterations in cell proliferation and apoptosis contribute to mucosal instability and impaired regeneration.

**Conclusion:** The combined effect of PM2.5 and cigarette smoke results in significant structural and functional impairment of gastric mucosa. Modern therapeutic strategies, including antioxidant therapy, anti-inflammatory agents, and barrier-protective interventions, show promising potential in mitigating these effects. Further experimental studies are required to develop targeted therapeutic approaches.

**Keywords:** PM2.5, cigarette smoke, gastric mucosa, oxidative stress, inflammation, apoptosis, epithelial barrier, morphology

**Introduction**

In recent decades, environmental pollution and tobacco smoking have become critical global health concerns. Fine particulate matter (PM2.5) and cigarette smoke are recognized as major contributors to morbidity and mortality worldwide. According to global health data, exposure to these factors is associated with respiratory, cardiovascular, and metabolic disorders. However, their role in gastrointestinal pathology, particularly in gastric mucosal damage, has gained increasing attention.

PM2.5 consists of particles with an aerodynamic diameter of less than 2.5  $\mu\text{m}$ , allowing them to penetrate deeply into the respiratory tract and enter systemic circulation. Similarly, cigarette smoke contains thousands of toxic compounds, including nicotine, carbon monoxide, heavy metals, and carcinogenic hydrocarbons. Importantly, both PM2.5 and cigarette smoke share common biological mechanisms, including the generation of reactive oxygen species (ROS), induction of inflammatory responses, and disruption of cellular integrity.

Recent studies suggest that combined exposure to PM2.5 and cigarette smoke produces a synergistic toxic effect, leading to more severe tissue damage compared to individual exposure.



The gastric mucosa, being a highly dynamic and sensitive tissue, is particularly vulnerable to such environmental insults.

### Materials and Methods

This study is based on a systematic analysis of contemporary scientific literature, including experimental and clinical research published in international databases. The analysis focused on studies investigating the effects of PM2.5 and cigarette smoke on gastric tissues, with emphasis on morphological, biochemical, and immunological changes.

### Results and Discussion

Combined exposure to PM2.5 and cigarette smoke induces significant structural damage in gastric tissues. Histological examination reveals to epithelial desquamation with cytoplasmic vacuolization on mucosal layer. It changed nuclear pyknosis and karyolysis and it help to make focal erosions.

These changes indicate disruption of epithelial integrity and loss of protective function.

The gastric mucosal barrier plays a critical role in protecting tissues from acid and enzymatic damage. Under combined exposure, mucin production decreases, mucus layer becomes thinner with the tight junction proteins (ZO-1, occludin) are disrupted. This leads to increased epithelial permeability and vulnerability to injury.

These changes impair nutrient supply and delay tissue repair processes.

### Conclusion

The combined exposure to PM2.5 and cigarette smoke leads to severe morphological and functional alterations in gastric mucosa. These effects are mediated through oxidative stress, inflammation, microcirculatory impairment, and epithelial barrier disruption. Understanding these mechanisms provides a scientific basis for developing effective therapeutic strategies.

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