Epigenetics Research: Open Access

OPEN ACCESS Freely available online

Opinion Article

Microplastic Accumulation and Cellular Communication Disruption in Human Pulmonary Systems

Ingrid Valdez*

Department of Respiratory Biology and Environmental Health Sciences, Universidad del Pacífico Andino, Quito, Ecuador

DESCRIPTION

The human respiratory system is continuously exposed to airborne particles originating from natural sources and human-made pollution. In recent years, attention has increased regarding the presence of microplastics in the atmosphere and their potential impact on lung biology. These microscopic plastic fragments originate from the breakdown of larger plastic materials, synthetic textiles, industrial emissions, and urban waste degradation. Once airborne, they can be inhaled and deposited within different regions of the respiratory tract, raising concerns about their influence on cellular communication and pulmonary function.

Microplastics vary in shape, composition, and size, allowing them to penetrate deep into the respiratory system. Particles smaller than 10 micrometers can bypass the upper airway defenses and reach the bronchioles and alveolar spaces. The deposition of these particles in lung tissue introduces foreign materials into a highly sensitive biological environment, where gas exchange and immune surveillance must operate efficiently.

One of the primary effects of microplastic exposure is disruption of intercellular communication within lung tissues. Pulmonary cells rely on chemical signaling pathways to coordinate functions such as mucus production, airway constriction, immune defense, and tissue repair. When microplastic particles accumulate, they can interfere with these signaling processes by inducing abnormal cellular responses or physically obstructing communication pathways.

Alveolar epithelial cells are particularly important for maintaining respiratory efficiency. These cells regulate gas exchange and act as a barrier against environmental contaminants. Exposure to microplastics can alter their signaling behavior, leading to impaired coordination with immune cells such as alveolar macrophages. This disruption can reduce the efficiency of pathogen clearance and increase vulnerability to respiratory infections.

Macrophages play a central role in identifying and removing foreign particles from lung tissue. When exposed to microplastics, these immune cells attempt to engulf and degrade the particles through phagocytosis. However, microplastics are resistant to biological degradation, leading to prolonged activation of macrophages. This persistent activation may result in altered cytokine signaling patterns and chronic inflammatory responses within lung tissue.

Inflammatory signaling pathways are strongly influenced by microplastic exposure. The presence of foreign particles stimulates the release of signaling molecules such as interleukins and tumor necrosis factors. These molecules regulate immune responses but can become dysregulated when exposure is continuous. Prolonged inflammatory signaling may damage surrounding healthy tissue and disrupt normal pulmonary function.

The structural integrity of lung tissue can also be affected by microplastic accumulation. Repeated exposure may lead to remodeling of airway structures, including thickening of airway walls and changes in epithelial cell organization. These structural changes can interfere with efficient airflow and reduce overall respiratory capacity.

Oxidative stress is another important mechanism associated with microplastic exposure. When lung cells interact with plastic particles, they may produce increased levels of reactive oxygen species. These reactive molecules can damage cellular membranes, proteins, and nucleic acids. In response, antioxidant defense systems are activated, but prolonged exposure can overwhelm these protective mechanisms, leading to cellular dysfunction.

Communication between epithelial cells and endothelial cells is essential for maintaining proper oxygen exchange and vascular regulation. Microplastics may disrupt signaling between these cell types, affecting blood vessel behavior in the lungs. This disruption can contribute to impaired oxygen delivery and reduced respiratory efficiency under stress conditions.

Recent research suggests that microplastics may also act as carriers for other environmental pollutants. Toxic chemicals such as heavy metals and organic compounds can adhere to plastic surfaces, increasing their potential biological impact.

Correspondence to: Ingrid Valdez, Department of Respiratory Biology and Environmental Health Sciences, Universidad del Pacífico Andino, Quito, Ecuador, E-mail: ingrid.valdez.upa@researchmail.ec

Received: 01-Jun-2026, Manuscript No. EROA-26-42358; **Editor assigned:** 03-Jun-2026, PreQC No. EROA-26-42358 (PQ); **Reviewed:** 17-Jun-2026, QC No. EROA-26-42358; **Revised:** 24-Jun-2026, Manuscript No. EROA-26-42358 (R); **Published:** 01-Jul-2026, DOI: 10.35248/EROA.26.8.258

Citation: Valdez I (2026). Microplastic Accumulation and Cellular Communication Disruption in Human Pulmonary Systems. J Epigenetics Res. 8:258.

When inhaled, these combined pollutants may intensify cellular stress responses and further disrupt intercellular communication.

The immune response in the lungs involves a coordinated interaction between innate and adaptive immune systems. Microplastic exposure may alter this balance by continuously activating innate immune responses while suppressing adaptive immune regulation. This imbalance can contribute to chronic inflammation and reduced ability to respond effectively to infections.

Another concern is the potential for microplastics to influence mucus production in the respiratory tract. Goblet cells responsible for mucus secretion may respond to irritation by increasing production, leading to excessive mucus accumulation. This can obstruct airflow and impair respiratory efficiency, especially in individuals with preexisting respiratory conditions.

Pulmonary fibrosis is a potential long-term outcome of chronic inflammatory responses induced by environmental particle exposure. Persistent tissue irritation may stimulate fibroblast activation and excessive collagen deposition. This leads to stiffening of lung tissue and reduced elasticity, which can significantly affect breathing capacity.

Airway smooth muscle cells also play a role in respiratory regulation. Disruption of signaling in these cells can affect airway constriction and relaxation cycles. This may lead to increased sensitivity to environmental triggers and contribute to respiratory discomfort or breathing difficulties.

CONCLUSION

Microplastic accumulation in the human respiratory system can disrupt cellular communication, induce inflammatory responses, and impair pulmonary function. Through mechanisms involving immune activation, oxidative stress, and structural changes, these particles influence lung health in multiple ways. Continued research is necessary to fully understand long-term effects and develop strategies to minimize exposure in urban and industrial environments.