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COMMENTARY

The hidden threat of microplastics in the bloodstream

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The increasing prevalence of microplastics (MPs) in the environment has raised significant concerns about their potential impact on human health.[1] A recent study published in *Science Advances* titled "Microplastics in the bloodstream can induce cerebral thrombosis by causing cell obstruction and lead to neurobehavioral abnormalities"[2] has shed new light on the mechanisms by which these tiny particles can disrupt critical physiological functions. This commentary aims to explore the implications of this study and discuss the broader context of microplastic pollution on human health.

Microplastics are defined as plastic particles smaller than 5 millimeters in diameter, they originate from various sources, including the degradation of larger plastic materials, microbeads in personal care products, and synthetic fibers from clothing.[1] Previous studies have demonstrated that MPs can enter the human body through various pathways, including ingestion, inhalation, and skin contact.[3] These particles can cross biological barriers, and accumulate in tissues, potentially leading to adverse health effects. Early studies also highlighted the ability of MPs to induce inflammation and oxidative stress, which are key factors in the development of chronic diseases. For example, MPs have been shown to activate immune cells, leading to the release of pro-inflammatory cytokines and the disruption of gut microbiota[4] These findings have underscored the importance of understanding the interactions between MPs and physiological systems. In addition, recent advances in the detection and quantification of MPs have revealed their widespread presence in various environmental compartments and human tissues and blood. Previous research has shown that microplastics can cross biological barriers, such as the blood-brain barrier, and interact with physiological systems, potentially leading to adverse health effects.

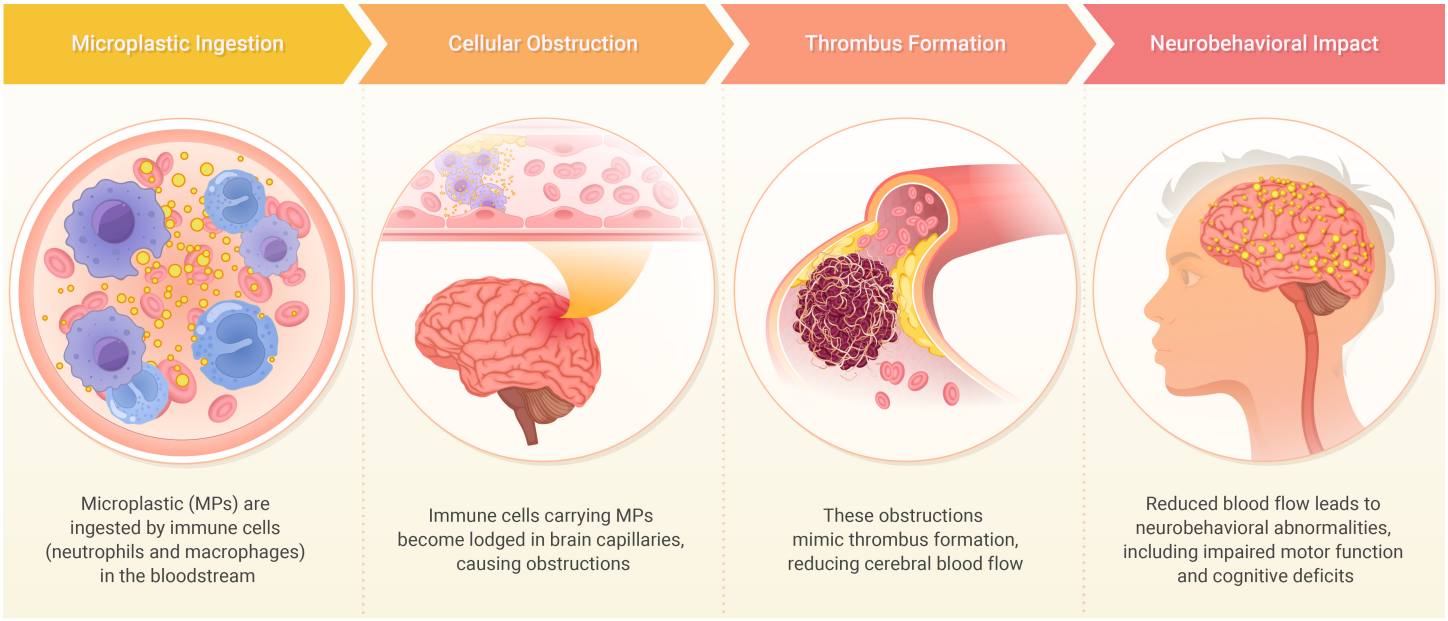

Figure 1. Mechanistic overview of microplastic-induced cerebral thrombosis and neurobehavioral abnormalities (1) Microplastic ingestion: MPs are ingested by immune cells (neutrophils and macrophages) in the bloodstream. (2) Cellular obstruction: Immune cells carrying MPs become lodged in brain capillaries, causing obstructions. (3) Thrombus formation: These obstructions mimic thrombus formation, reducing cerebral blood flow. (4) Neurobehavioral impact: educed blood flow leads to neurobehavioral abnormalities, including impaired motor function and cognitive deficits.

In this study,[2] the authors employed advanced imaging techniques to investigate the impact of microplastics on cerebral blood flow and neurobehavioral functions in mice. The researchers used fluorescently labeled polystyrene microplastics of three sizes (5 μm, 2 μm, and 80 nm) and injected them intravenously into mice at concentrations mimicking human exposure levels. Using laser speckle contrast imaging and miniature two-photon microscopy, they observed the movement and interaction of microplastics within cerebral blood vessels in real-time.

The study revealed several critical findings. First, circulating microplastics were phagocytosed by immune cells, which then became trapped in the capillaries of the brain cortex, obstructing blood flow. These obstructions, resembling thrombus formation, led to reduced cerebral blood perfusion within 30 minutes of injection. Second, the study demonstrated that larger microplastics (5 μm) caused more prolonged obstructions compared to smaller particles (2 μm and 80 nm). This size-dependent effect highlights the importance of particle size in determining the severity of vascular blockages. Behavioral experiments showed that mice exposed to microplastics exhibited significant impairments in locomotion, memory, and motor coordination. These neurobehavioral abnormalities were largely reversible within four weeks, although some vascular obstructions persisted at lower densities. Third, the study also identified that the altered morphology and adhesive properties of immune cells after phagocytosing microplastics contributed to the vascular blockages.

The findings of this study raise significant concerns about the potential health risks of microplastic exposure. While the study was conducted in mice, the similarities between mouse and human immune systems and vascular structures suggest that similar mechanisms could occur in humans. The potential for microplastics to induce cerebral thrombosis and neurobehavioral abnormalities underscores the need for further research to understand the long-term effects of microplastic exposure on human health. The study

highlights the importance of considering the size and concentration of microplastics when assessing their health risks. Smaller particles, while potentially more easily cleared from the body, may still pose significant risks due to their ability to penetrate deeper into tissues. Additionally, the study emphasizes the need for more research on the interactions between microplastics and immune cells, as these interactions may play a crucial role in the development of vascular and neurological disorders. We suggest that future studies could focus on several key areas. First, studies could investigate the long-term effects of microplastic exposure on human health, particularly in individuals with pre-existing cardiovascular conditions or narrowed blood vessels. Second, research could explore the mechanisms by which microplastics interact with immune cells and other physiological systems, potentially leading to chronic inflammation and disease. Finally, studies could examine the potential for microplastics to accumulate in human tissues over time and the cumulative effects of such accumulation.

We acknowledge that the detection of MPs in human blood remains a subject of debate within the scientific community. While several studies have reported the presence of MPs in human blood, there are significant challenges related to the specificity and reliability of detection methods. For instance, the use of different analytical techniques, such as pyrolysis-gas chromatography–mass spectrometry (Py-GC-MS)[3] and Fourier-transform infrared (μ-FTIR) spectroscopy,[5] can lead to variable results. Additionally, the potential for contamination during sample collection and processing further complicates the interpretation of these findings. Both MPs and PM2.5 can induce oxidative stress, inflammation, and immune responses, which are central to their toxic effects. However, MPs are distinguished by their ability to persist in the environment and accumulate in tissues over time, potentially leading to chronic exposure. In contrast, PM2.5 toxicity is often associated with acute exposure events and primarily affects the respiratory and cardiovascular systems. We suggest that future research could explore the synergistic effects of MPs and other particulate pollutants, as well as their cumulative impacts on human health.

In conclusion, the study by Huang et al.[2] provides critical insights into the mechanisms by which microplastics can disrupt cerebral blood flow and induce neurobehavioral abnormalities (Figure 1). The findings highlight the potential health risks associated with microplastic exposure and underscore the need for further research to understand the long-term effects of these particles on human health. As microplastic pollution continues to increase, it is imperative that we develop strategies to mitigate their release into the environment and reduce human exposure. This study serves as a stark reminder of the hidden threats posed by microplastics and the urgent need for action to protect human health.

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AUTHOR CONTRIBUTIONS

All authors conceived the original idea, wrote and reviewed the manuscript.

DECLARATION OF INTERESTS

The authors declare no competing interests.