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*Billionaires often say they'd trade all their wealth to be young again. But most of them don't invest in aging science. Nathan Cheng, engineer ([source](#)).*

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### **This month's theme: Microplastics and Aging**

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Microplastics are tiny plastic particles less than 5 millimeters in size that originate from the breakdown of larger plastic waste or are manufactured for use in products like cosmetics and cleaning agents. These particles have become widespread in the environment and have been detected in food, water, air, and even inside the human body, including the lungs, blood, and placenta. Their small size allows them to enter the body through ingestion or inhalation, where they may accumulate and cause harm. Microplastics do not biodegrade and can persist in the environment for hundreds to thousands of years, continuously fragmenting into smaller particles without ever fully disappearing.



Microplastics can trigger damage, disrupt the gut microbiome, and carry toxic chemicals such as [bisphenol A \(BPA\)](#) and phthalates, which are known to interfere with the endocrine system. Additionally, they may serve as carriers for pathogens and heavy metals, further increasing their potential health risks. While research is ongoing, early studies suggest that [microplastics could contribute to immune dysfunction, respiratory issues, hormonal imbalance, and possibly even cancer, making them an emerging threat to human health.](#)

Emerging research suggests that microplastics may contribute to the acceleration of human aging by disrupting several key biological processes. Once inside the body, microplastics can trigger chronic low-grade inflammation, known as "inflammaging," which is a recognized contributor to age-related diseases such as cardiovascular disorders, neurodegeneration, and cancer. They also promote oxidative stress by increasing the production of reactive oxygen species, leading to damage to DNA, proteins, and lipids, factors closely linked to cellular aging. Furthermore, microplastics have been shown to impair mitochondrial function, reducing cellular energy production and contributing to the decline in tissue function observed with age. In addition, they may induce cellular senescence, a state in which cells stop dividing and begin releasing harmful inflammatory molecules, further accelerating tissue damage. The endocrine-disrupting chemicals carried by microplastics, such as bisphenol A (BPA) and phthalates, can also interfere with hormone regulation, potentially affecting metabolism, reproduction, and other systems tied to the aging process. While further studies are needed to fully understand the long-term impact, current evidence already establishes that microplastic exposure may be a significant environmental factor contributing to premature aging and age-related decline.

## Accumulation of Microplastics in Aging Tissues

The accumulation of microplastics (MPs) in aging tissues has become a pressing environmental and biomedical concern. As microplastics become increasingly prevalent in the environment, emerging evidence suggests their systemic uptake and potential to exacerbate aging-related physiological processes, particularly through oxidative stress, cellular senescence, and chronic inflammation. Aging tissues may be particularly vulnerable due to declining barrier functions, impaired clearance mechanisms, and altered immune responses.

Microplastics enter the body principally through ingestion or inhalation. Once internalized, they may: Bypass biological barriers, especially if under 5 µm. Accumulate in organs such as the liver, gut, and even the brain. Generate reactive oxygen species (ROS), which induce oxidative damage. Trigger senescence pathways in fibroblasts and immune cells. Alter extracellular matrix composition (ECM), leading to impaired tissue repair and elasticity.

### 1. Skin Aging and Fibroblast Senescence

[A 2024 study](#) demonstrated that polystyrene microplastics disrupted skin barrier function and induced fibroblast senescence. This led to downregulation of key ECM genes such as *COL1A1*, contributing to premature skin aging

### 2. [Systemic Aging and Cognitive Decline in Animal Models](#)

Chronic oral exposure to polyethylene terephthalate (PET) microplastics (MPs) in [senescence-prone OXYS rats](#) accelerated features of age-related diseases, such as cataracts, macular degeneration, and memory impairment, suggesting systemic aging effects beyond the site of entry.

### 3. Environmentally Persistent Free Radicals (EPFRs) from Aged MPs

[A critical review highlighted that aged MPs can carry and generate EPFRs, which may further contribute to oxidative stress and toxicity when they accumulate in biological systems](#)

## Effect in the brain

The most worrying effect known today is that microplastics can cross the blood-brain barrier, and they remain in the brain until death. Even worse, a [study showed that people with Alzheimer's disease have higher levels of microplastics in the brain](#). This doesn't prove that microplastics aggravate neurodegenerative diseases because neurodegenerative diseases could facilitate the penetration of microplastics. But it is at least worrying.

## Synergistic Effects with Other Environmental Pollutants

Microplastics (MPs) are not only toxic in isolation but also serve as vectors for co-pollutants like heavy metals (HMs), persistent organic pollutants (POPs), and

pharmaceuticals. [In aged populations](#)—characterized by reduced detoxification capacity and compromised gut and immune barriers—the combined toxic burden of MPs and these contaminants may exacerbate health risks such as inflammation, oxidative damage, and organ degeneration.

[Microplastics act as sorption \(sort of absorption\) substrates due to their high surface-area-to-volume ratio and hydrophobicity.](#) Upon aging, especially under UV or thermal exposure, MPs:

- Become rougher and more porous.
- Develop oxygen-containing functional groups that increase affinity for metals and organics.
- Undergo surface oxidation, enhancing adsorption of cadmium ( $\text{Cd}^{2+}$ ), chromium ( $\text{Cr}$ ), lead ( $\text{Pb}^{2+}$ ), and various endocrine-disrupting chemicals.

[Once internalized in the body, these composite particles \(MPs + contaminants\):](#)

- Induce oxidative stress through reactive oxygen species (ROS).
- Trigger autophagy and pyroptosis (inflammatory cell death).
- Compromise the intestinal and blood-brain barriers, especially in aging tissues.

## Conclusion

It is too late to stop microplastics with our current technical and scientific capacities. Plastics are everywhere, and they will continue to degrade in the coming years. We must urgently collect more knowledge about the effects in animal models (mice), and thanks to epidemiological studies. We must urgently study how to mitigate absorption in the body, especially in the brain.

The only good news is that it seems to have no important negative effect yet. Indeed, life expectancy continues to rise even in places where microplastics are in large quantities. It could be that most microplastics are not very harmful. It could even be that in very specific cases, some microplastics have a few positive consequences (let's dream, artificial is not always bad). However, as long as we do not study this enough, we take an enormous risk of slowly damaging our bodies from the inside because of the environmental changes we created.

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### The good news of the month. A single gene to rejuvenate human cells.

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Shift Bioscience has discovered [SB000](#), a single gene capable of rejuvenating cells without activating pluripotency, avoiding the risks associated with OSKM (Yamanaka factors). SB000 matches OSKM in reversing cellular age while preserving cell identity and function. It works across multiple cell types and enhances functions like collagen

production. The discovery was made using an AI-driven platform based on transcriptomic aging clocks.

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For more information

- [Heales](#), [Longevity Escape Velocity Foundation](#), [International Longevity Alliance](#), [Longevity](#), and [Lifespan.io](#)
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