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Mikro ve Nanoplastiklerin Hücresel Yaşlanma Süreci Üzerindeki Potansiyel Etkileri

Potential Effects of Micro and Nanoplastics on Cellular Aging Processes

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Öz

As micro and nanoplastics have become widespread environmental pollutants, their effects on human health have gained significant attention. The majority of research has shown their potential to induce oxidative stress, inflammation, DNA damage, and hormonal disruption mechanisms that are closely related to senescence and aging. Therefore, exposure to micro and nanoplastic may be an important factor in accelerating these processes. Due to their smaller size, nanoplastics can rapidly cross biological barriers, may target more organs and tissues and may lead to more profound effects compared to microplastics. Understanding aging-related effects of micro and nanoplastics, as well as developing protective strategies is essential for minimizing associated health risks.

Abstract

Mikro ve nanoplastiklerin çevrede yaygın kirleticiler haline gelmesiyle birlikte, insan sağlığı üzerindeki etkileri bilimsel çevrelerde giderek daha fazla ilgi görmeye başlamıştır. Yapılan araştırmaların büyük bir kısmı, bu plastiklerin oksidatif stres, inflamasyon, DNA hasarı ve hormonal bozulma gibi yaşlanma ve senesens ile yakından ilişkili mekanizmaları tetikleme potansiyeline sahip olduğunu göstermektedir. Bu nedenle, mikro ve nanoplastiklere maruz kalma, bu süreçlerin hızlanmasında önemli bir etken olabilir. Nanoplastikler, daha küçük boyutları sayesinde biyolojik bariyerleri hızla geçebilmekte, bu da onların daha fazla doku ve organı hedef almasına ve mikroplastiklere kıyasla daha ciddi sorunlara neden olabilir. Mikro ve nanoplastiklerin yaşlanma ile ilişkili etkilerinin anlaşılması ve bu etkilerden korunmaya yönelik stratejilerin geliştirilmesi, ilgili sağlık risklerinin en aza indirilmesi açısından büyük önem taşımaktadır.

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Dear Editor;

During the last few decades, plastic pollution has become a major threat to human health which directly affects humans via different mechanisms. Accumulation of plastic particles upon increased exposure via inhalation, ingestion and/or dermal route have the potential to impact many biological pathways and thereby raising considerable health concerns (1). Based on these concerns, the potential effects of micro and nanoplastics on longevity and cellular senescence have become a significant research interest recently.

Plastic particles ranging from 5 mm to 1 μm are considered as microplastics (MPs), whereas particles smaller than 1 μm are classified as nanoplastics (NPs) (2). Even if both are composed of the same resin, in other words, they have the same chemical properties, the physicochemical properties such as size and shape affect the toxicity profile of these particles. Having higher surface/volume ratio than MPs enables NPs to be more reactive, as well as the opportunity to pass through the biological barriers, thereby targeting more organs and tissues throughout the organism (3). For instance, it has been demonstrated that particle accumulation in the tissues was size dependent, upon to exposure to 0.5 μm-NPs 5μm-MPs (4). Even both MPs and NPs exert their toxicity mostly via similar pathways, NPs are more potentially affecting senescence and longevity than MPs, due to their smaller size.

Oxidative stress and inflammation are major contributors in MPs or NPs induced cellular senescence, because they mainly exert their cytotoxicity through these mechanisms. NPs can pass through the cell membrane and accumulate in the cells, and increases reactive oxygen species (ROS) production and induces oxidative stress (5). ROS are one of the most important factors that triggers senescence, due to the oxidation of biomolecules in the cells such as lipids, protein and DNA. As a result of the increase in intracellular ROS along with the insufficient antioxidant defense mechanisms, oxidatively damaged biomolecules may not be repaired and cells enter senescence (6). These cells may both lose their functions and accelerate aging by damaging nearby tissues. In a similar manner to NPs, MPs that cannot be localized to the cells (20μm) have been shown to accumulate in the liver and alter biomarkers of oxidative stress in mice (7). This data indicates that plastic particles can induce oxidative stress independently from localization in cells and may trigger senescence.

It is almost certain that NPs affect the aging process, because the most critical impact of exposure to NPs is DNA damage (8). This damage frequently results in cell cycle arrest, which contributes to senescence. In addition, exposure to NPs negatively affects DNA repair mechanisms, thereby reduces the capacity of tissue renewal. Similarly, increased intracellular ROS levels upon MP exposure may also trigger DNA damage and may contribute to aging process (9,10). Telomere shortening is also an indicator or aging, while in every cell division telomeres become shortened and in the late stages cells stop dividing. Oxidative stress triggered by MPs and NPs may also has the potential of suppressing the telomerase activity and accelerate the aging process.

Inflammation is known to have a major role in aging and age-related diseases. Inflammatory responses triggered by MPs and NPs are also causes stress in immune system, thereby having the potential to accelerate aging process (11). Previous studies demonstrated that exposure to both MPs and NPs cause cytokine release and triggers inflammatory processes (12,13). Furthermore, histopathological examination showed that exposure to MPs caused inflammatory cell infiltration in zebrafish liver (10). As the inflammation become chronic, proinflammatory cytokines released by aging cells may affect nearby cells and trigger a systemic aging process.

Body critically regulates the levels of hormones via endocrine system and dysregulation of the hormones affect whole organism. Endocrine disrupting chemicals such as bisphenol A and phthalates disrupt hormonal signalization and are known to be included in several resin types of plastics. These chemicals included in MPs and NPs are have the potential of leading hormonal imbalances, thereby affect both

cellular pathways and aging process (15). Particularly, estrogens, testosterone and thyroid hormone mimicking chemicals can increase the potential of aging and age-related diseases by triggering cellular stress and oxidative damage. Alterations in hormonal balance may also contribute the reduction of cells' self-renewal capacity, as well as tissue regeneration, thereby accelerates aging. For this reason, due to the endocrine disrupting nature of plastics is a crucial factor to be considered in MP/NPs induced senescence and aging.

In conclusion, the effects of MPs and NPs on human health are increasingly gaining attention in the literature. As a result of MP and NP exposure, inflammatory responses, oxidative stress, DNA damage, cellular function are triggered and poses significant health risks. In this concept, reduction in tissue renewal capacity and increase in age-related diseases may be anticipated outcomes. Particularly NPs, which has the potential of bypassing biological barriers and reaching multiple organs, suggests that their negative impact on aging process may be more profound and hazardous. For these reasons, studies mechanistically investigating the potential health risk of MPs and NPs in humans as well as protective mechanisms to reduce the risks of MP and NP exposure play a crucial role in MP/NP research. Specifically, experimental and computational modelling approaches that assess the distribution, bioavailability, and long-term effects of MPs and NPs in humans could fill the crucial gaps in MP/NP toxicity. In addition, studies conducted with the participants of high-risk human groups such as plastic industry workers and the inhabitants in urban/metropolitan areas may contribute the risk assessment of MPs and NPs. Furthermore, studies specifically designed to evaluate the effects of MPs and NPs on aging by using human derived cell lines or model organisms at a mechanistically level, are of great importance for preventing plastic related accelerated aging and taking early precautions. Future studies would significantly contribute to scientific literature and would be valuable references for protecting both human and environmental health.

Yours sincerely,

Kaynaklar

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