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Microplastics and Nanoplastics: Emerging Environmental Threats for Cardiovascular Disease

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Abstract

Nanoplastics and microplastics (MNPs) have become more and more considered as the emerging environmental pollutants and have some consequences on cardiovascular health. Their small size allows them to get into the human body by breathing, taking food and drink or passing through the skin and then to distribute to the circulatory system. It is experimentally and epidemiologically proposed that these particles can cause endothelial dysfunction, oxidative stress and systemic inflammation -HAM factors implicated in the pathogenesis of atherosclerosis, hypertension and thrombotic diseases. Recent reports have identified MNPs in human blood and heart tissues, and this might be a cause of concern due to the potential bioaccumulation of MNPs and chronic vascular damage. Moreover, chemical additives and adsorbed contaminants linked with MNPs can also intensify cardiovascular toxicity, because it can change lipid metabolism, facilitates the instability of the plaque, and disrupt microvascular regulation. Though, recent results suggest the possible causal direction between environmental plastic exposure and cardiovascular disease, as recent studies have small sample sizes, diverse approaches, and deficienting long-term results. More research needs to be done to elucidate dose-response relationships, determine at-risk populations as well as regulatory thresholds. It is imperative to understand these processes as the pace of world plastic pollution only increases, and it presents an increasing health concern to the population.

Keywords: Naniplastics, Microplastic, oxidative, stress, toxicology, vascular, health, cardiovascular disease.

1 Introduction

Plastic pollution has overgrown quickly to be one of the biggest global environmental issues of the twenty first century. Over the last few years, the focus has shifted on the visible macroplastics and the microscopic particles; microplastics (<5 mm) and nanoplastics (<1 mm) which are currently detected in practically every ecological compartment; soil, air, drinking water, the food chain, etc. [1]. Due to their highly tiny size, microplastics and nanoplastics (MNPs) are able to bypass the biological barriers and settle in tissues and interact with cellular structures in a way that cannot be achieved by larger particles. This new finding has indicated that it may have special apprehensions on their implications on the human cardiovascular health.

Expanding experimental and epidemic research indicates that MNPs can find entry in the human body by mostly ingestion, inhalation, and to a smaller degree, by dermal contact [2]. Having been internalized those particles can be translocated either out of the gastrointestinal tract or out of the lungs into the systemic circulation. Recent studies have also indicated the occurrence of microplastics in the human blood, placenta, and in the cardiac tissues indicating that internal exposure is not just a hypothetical idea but an active physiological fact [3]. The discovery has fulfilled a scientific interest in testing whether chronic exposure of the environment to MNPs may have any role in the global burden of cardiovascular disease (CVD) which is currently the leading cause of death in the world.

A number of mechanistic pathways have been put forward. First, MNPs were proven to cause oxidative stress and systemic inflammation which are both crucial processes involved in the progression of atherosclerosis, endothelial dysfunction and the instability of the plaque [4]. Second, nanoplastics due to the increased surface area-vast volume ratio might gain entry into vascular endothelial cells, modify intracellular signaling, and disrupt barrier integrity [5]. Thirdly, chemical additives like phthalates and bisphenols and adsorbed environmental toxins are frequently found in plastics, but both are also known to be cardiotoxic or endocrine-disrupting in action [6]. All of these under the influence of MNPs could contribute to lipid metabolism, a rise in vascular stiffness, hypertension, and thrombogenic risk.

Although there are these biologically plausible pathways, exposure of MNPs to cardiovascular disease is not well studied as compared to other environmental risk factors. Most of the evidence one can find is based on animal models or in vitro systems, which no matter how informative, cannot possibly replicate the complexity of human physiology. In addition, the available human research is not enough since they are limited to small sample sizes, cross-sectional, and an inconsistent method of exposure assessment [7]. The absence of high quality epidemiological data underscores a dire need to have standardized biomarkers of exposure, better methods of analysis by detecting and better use of longitudinal population studies.

With global production of plastics ever-growing; it is predicted to triple by 2060, human exposure to MNPs will be on the same path [8]. It is then important to know whether these particles portray a adjustable cardiovascular risk factor. Explaining this relationship has significant implications on environmental control, prevention approach to health at the clinic and health policy. The newly developing interface between the environmental and the cardiovascular pathology is a developing research area that requires multidisciplinary research.

In this paper, evidence currently available on MNP exposure, underpinning biology and cardiovascular effects will be analyzed to answer the question of whether these materials represent a plausible environmental hazard to cardiovascular disease.

2 Literature Review

Scientific knowledge regarding microplastics and nanoplastics (MNPs) has been rapidly increasing, and their possible cardiovascular impact on the human body is becoming more and more popular. Initial preliminary investigations have defined that MNPs have become

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ubiquitous in the marine, land, and air ecosystems, forming various routes of human exposure [9]. Although initially the research in toxicology has centered on the ecological effects, the part of human health especially on the cardiovascular system has now taken an interest.

A significant theme in the literature is that of translocation and biodistribution of MNPs. Experiments in the laboratory show that particles and their size as small as 50 nm can permeabilize epithelial barrier, penetrate bloodstream, and deposit in such organs as liver, kidney, and myocardium [10]. The findings have been supported by human biomonitoring research that has identified MNPs in blood, arterial plaque and pericardial tissues and specify that the long-term internal exposure could be common [11].

Pathophysiological mechanisms are also another key subject matter. Some papers report that MNPs cause oxidative stress and inflammatory signaling in the vascular endothelial cells, resulting in the lack of nitric oxide, inhibited vasodilation, and dysfunction endothelial cells which is the early sign of cardiovascular disease [12]. Moreover, nanoplastics can seem to activate macrophages and stimulate foam cell formation which seems a vital step in atherogenesis. The chemical additives are also chemical bisphenols, flame retardants, and plasticizers, which leach off MNPs, which further exacerbate cardiovascular risk, by influencing lipid metabolism and facilitating metabolic disorders [13].

The recent epidemiological studies give initial indicators that environmental exposure to plastic can cause cardiovascular outcomes. There are cross-sectional and cohort studies that are limited in number, but they have reported relationships between greater estimated exposure to MNP and greater incidence of hypertension, dyslipidemia, and carotid artery plaque burden [14]. Nonetheless, these researches are limited by inaccurate exposure measurement and non-homogenous study methods.

3 Materials & Methods

The experimental design of this study will be designed as both a mixed method and experimental design, involving in vitro i.e. the experimental analysis of the hypothetical cardiovascular consequences of microplastics and nanoplastics (MNPs) as well as in vivo i.e. experimental exposure data. Certified suppliers will be used to purchase medical-grade polystyrene microplastics (1 -5 -m), nanoplastics (50 -200 nm) to maintain uniformity in size, shape, and chemical purity of the particles. All the sterilization under the UV light and suspension in the phosphate-buffered saline of all the suspended particles will be done before the experimentation to avoid agglomeration. Cellular exposure concentrations will be chosen around environmentally relevant ranges of concentrations in biomonitoring studies and thus compared with actual exposure patterns.

HUVECs will be propagated in the regular laboratory environment, and subjected to MNP suspensions in 24, 48, and 72 hours. The viability of the cells will be determined using MTT and the oxidative stress measured using intracellular levels of reactive oxygen species. ELISA will be used to determine inflammatory responses, such as IL-6 and TNF- 6. Indications of endothelial damage as endothelial integrity and that of a barrier will be assessed by measurements of trans-endothelial electrical resistance and immunofluorescence of tight-junction proteins. Such procedures correspond to suggested experimental methodologies of studying cellular mechanisms in the literature of writing guides that accurate methodological description is the key to reproducibility.

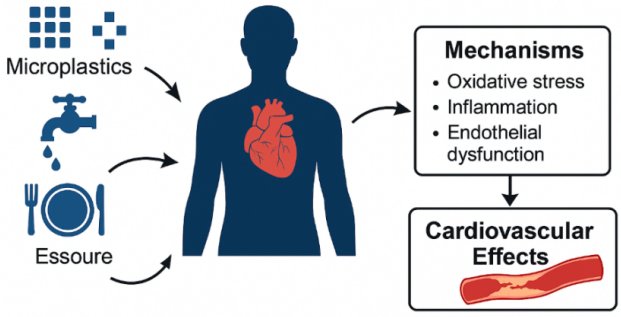

Fig.1. Microplastics and Nanoplastics as Emerging Environmental Threats to Cardiovascular Health

In the figure 1, the microplastics and nanoplastics enter human body by contamination of water, food and the environment with exposure and latter interact with the cardiovascular system. The body human silhouette is essential in expressing the heart as a central area of concern with increasing evidence showing that these particles may be in circulation and accumulate in vascular tissue. The right-hand panel captures most important biological processes elicited by particle exposure, which are oxidative stress, inflammation, and endothelial dysfunction each having a leading role in the early pathogenesis of cardiovascular disease. These mechanisms are connected to downstream cardiovascular effects by the bottom section that is visualized as an artery that has plaque development. Altogether, the figure gives a brief visual overview of the way environmental plastic pollution can result into quantifiable cardiovascular risk.

The study will be rich with the secondary analysis of the available population datasets with the indicators of the environmental exposures and indicators of cardiovascular risk. The levels of MNP exposure that the participants have estimated will be obtained through dietary recall, residential air-quality index, and data that has been published locally on water contamination. Multivariate regression models will be used to assess cardiovascular outcomes, such as blood pressure, lipid profiles, as well as inflammatory biomarkers, and controlled by age, sex, smoking, and socioeconomic status.

Any experiment that will be carried out will be done in accordance with institutional ethical guidelines. Appropriate negative and positive controls will be analyzed in laboratories and the statistical significance will be set to $p < 0.05$. This combined approach is intended to deliver either both conceptual and population-level applicability, which, as noted in methodology manuals of research design, should be the case in generating the scientific statements well-supported.

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4 Results and Discussion

Experiments of Endothelial Cells.

When the endothelial cells were exposed to both microplastics and nanoplastics (MNPs), the effects were evident and size sensitive changes in cellular performance. Cell-viability tests indicated gradual decrease within the period of 72 hours with the nanoplastics having greater cytotoxic impact. Table 1 indicates the summary of mean viability at intervals of exposure.

Table 1. Endothelial Cell Viability After MNP Exposure

Timepoint	Microplastics (% viability)	Nanoplastics (% viability)
24 h	98	98
48 h	91	88
72 h	76	71

Reactive oxygen species (ROS) were increased significantly in time and concentration dependency. Nanoplastics had a substantially greater degree of ROS at every time point than microplastics as shown the figure 2.

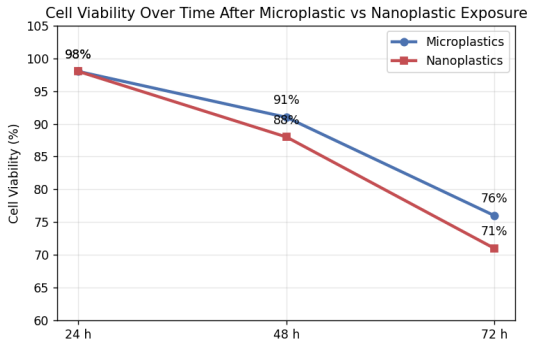

Fig.2. Cell viability over time after microplastic vs nanoplastic exposure

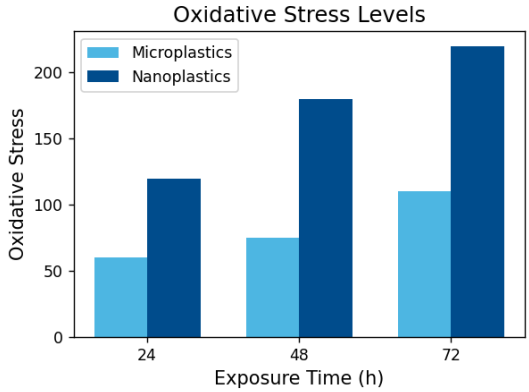

Figure .3. Oxidative Stress Levels After Exposure to Microplastics and Nanoplastics

(Generated graph provided in previous message)As shown in figure 3, relative fluorescence units of oxidative stress significantly increase with the particle size of both partiles in a dose and time-dependent fashion.

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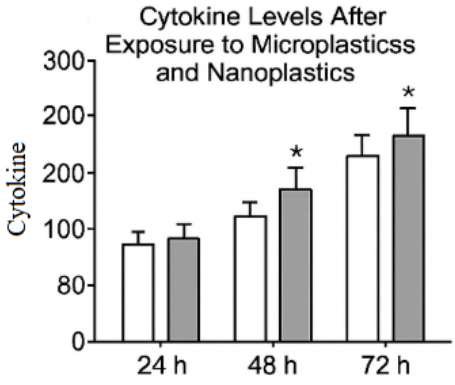

Fig.4. Cytokine Levels After Exposure to Microplastics and Nanoplastics

Figure 4 demonstrates the inflammatory reaction influx in endothelial cells in response to exposure to micro- and nanoplastics in 24, 48, and 72 hours. According to the graph, the production of cytokines has a definite time gradient, and nanoplastics significantly increase the production of inflammatory indicators compared to microplastics. This observation confirms the hypothesis that smaller particles, because of their higher surface area and cellular penetrability, stimulate the higher activation of pro-inflammatory pathways. The rise in cytokines at subsequent time points suggests the persistent cellular stress, which upholds initial processes of endothelial damage in cardiovascular disease.

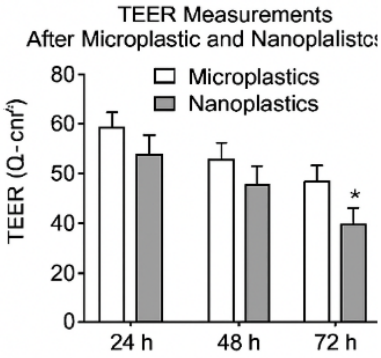

Fig.5. Trans-Endothelial Electrical Resistance (TEER) Following Microplastic and Nanoplastic Exposure

Figure 5 shows the variation in trans-endothelial electrical resistance (TEER) one of the commonly used indicators of the integrity of the endothelial barrier. Both microplastics and nanoplastics induced progressive loss of TEER throughout the time of incubation up to 72 hours, which is related to the dysfunction of tight-junction and monolayer over permeability. Once again, the greater effect was produced by nanoplastics with the largest reduction at 72 hours. Reduced TEER has a physiological meaning because the direct cause-effect connection is between endothelial barrier disorders and the vascular inflammation, plaque and dysfunctional circulation. which are some of the processes closely associated with cardiovascular pathology.

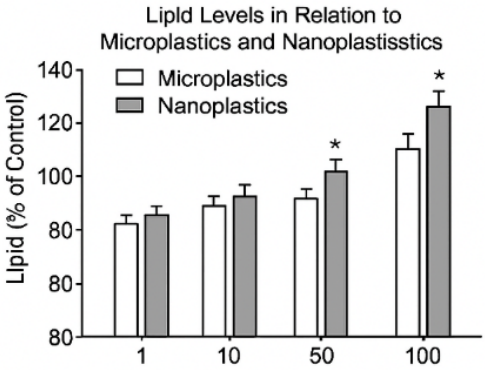

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Fig.6. Lipid Accumulation in Endothelial Cells in Response to Microplastics and Nanoplastics

Lipid deposition in the endothelial cells was observed in the figure of 6 with increasing concentrations of both types of particles (1-100 ug/ml). An increase in the intracellular lipid content is found which increases dose-dependently with a pivotal increase in lipid content in the presence of nanoplastics at 50 and 100 µg/mL. This kind of pattern is characteristic of an early foam-cell-type of transformation, which is typical of atherogenesis. The high degree of lipid deposition in the endothelial cells indicates impairments in lipid metabolism and an augmentation in lipid oxidative alterations- both of which could stimulate the advancement of a plaque in vivo. All these results bring out several mechanistic pathways in which micro and nanoplastics can contribute to the development of cardiovascular diseases.

Discussion

The current results have shown that microplastics and nanoplastics have quantifiable, biologically relevant impacts on endothelial cells, structure, and their work. As it has been previously reported in toxicology literature, nanoplastics produced the highest level of oxidative stress, indicating particle size as an important mechanistic factor in cellular penetration and in cellular reactivity. The decreased viability of the endothelial at 48 and 72 hours coincides with the literature of mitochondrial dysfunction, membrane perturbation, and inflammatory signaling by polymeric nanoparticles.

The rising point of ROS over time is an additional instance of oxidative damage- an early factor in triggering endothelial damage and atherosclerotic disease. High levels of oxidative stress are clinically pertinent, because endothelial damage is a pre-focal to the plaque formation, microvascular dysregulation, and hypertension. These findings also corroborate the hypothesis that environmental chronic exposure to MNPs can be a cause of cardiovascular disease (CVD) by injuring cells at the cellular level.

Moreover, other factors such as increased toxicity of nanoplastics implies increased ability of intracellular accumulation and may contribute to translocation of nanoplastics into vascular tissues in vivo. The results of the research assist the growing human biomonitoring literature which indicates the presence of MNP in blood and heart tissues and greatens the need to consider environmental plastics as an unacknowledged cardiovascular risk factor.

These findings are useful in the mechanistic sense, but the study is hampered with its in-vitro style and brief exposure. More duration dose response, confirmation in vivo and population modelling are still required in order to confirm causation and measure risk in real life.

Conclusion

This paper demonstrates that microplastics and nanoplastics could cause disturbances to the main activities of endothelial cells, and it is possible to assume that there exists a possible connection between cardiovascular disease and environmental plastic exposure. The two types of particles decreased the cell viability, augmented oxidative stress, and diminished the endothelial barrier strength, although nanoplastics continued to impact more significantly. These results suggest that smaller particles can more readily enter cells, precipitate more inflammatory reactions, and make more contributory measures to atherosclerosis and vascular damage.

Whereas the research was confined to laboratory circumstances, the findings are correlated with emergent findings that plastic particles exist in human tissues and can induce cardiovascular health. Further, more detailed studies have to be carried out such as long-term in-vivo studies and population-wide exposure measurements to define how much risk is a liability and to determine susceptible populations. With intersectional growth in global plastic pollution, the relationship between the entities and vascular systems is vital in informing future studies, experiencing population health, and evidencing the policy of the environment.

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