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Microplastics and endocrine disruption: Emerging risks for human fertility

Anbuchelvan SK[1]*, Udayakumaran H[2], Xin AY[3], Loshni Y[4], Rashid F[5]

Corresponding author:

Subasri Krishnarajan Anbuchelvan

MBBS student, Quest International University, Perak, Malaysia

Email: subasrianbuchelvank11@gmail.com ORCID

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ABSTRACT

Background

Microplastics (MPs) and nanoplastics (NPs) are pervasive environmental contaminants that are increasingly recognised for their potential role in endocrine disruption and reproductive toxicity. These particles, ubiquitous in food, water, and air, harbour endocrine-disrupting chemicals (EDCs) and have demonstrated effects on hormonal regulation, gametogenesis, and fertility outcomes in both experimental and observational studies. This short communication highlights current knowledge on the association between microplastic exposure, endocrine disruption, and fertility issues, drawing on recent evidence.

Keywords

Disrupt, effect, endocrine, exposure, hormone, human, reproductive

Background

The widespread use of plastics has led to the accumulation of microplastics (MPs) and nanoplastics (NPs) in various ecosystems, resulting in significant human exposure through ingestion, inhalation, and dermal contact. MPs and NPs can enter the human body via multiple routes, including inhalation, accidental ingestion, and dermal exposure. These particles can absorb and transport endocrine-disrupting chemicals (EDCs) such as bisphenols, phthalates, and heavy metals, allowing them to interfere with hormonal systems upon entering biological tissues. The consequences of such interactions have critical implications for reproductive health, demanding focused research and action. [1-3].

Microplastics as vectors for endocrine disrupting chemicals

MPs and NPs have a large surface area-to-volume ratio, which increases their capacity to adsorb environmental EDCs. Since there are no covalent bonds to the plastic matrix, it is prone to leaching into the body following exposure. Once inside, structurally similar to endogenous hormones, EDCs can bind to hormone receptors, disrupting normal endocrine function. Moreover, in comparison to endogenous hormones, EDCs exhibit higher efficacy even at lower concentrations, which makes them more likely to disrupt general hormonal regulation. [1-3]

Altered hormone synthesis and signalling:

Interference with hypothalamic-pituitary axes, such as the HPG axis, leads to downstream dysregulation of gonadal hormone production and feedback loops. Dysregulation of gonadal hormones can lead to subfertility in females and increase the risk of malignancy of the uterus. Similarly, if there is a disturbance to the HPT axis, abnormalities related to the individual's overall metabolism and growth occur. [2, 4, 5]

Oxidative stress:

MPs induce the generation of reactive oxygen species, which contribute to structural and functional damage in reproductive organs. Furthermore, ROS species have also been shown to activate other pathways, such as mitogen-activated protein kinases, which may have detrimental effects on male reproductive organs, leading to lower sperm volume and quality. Oxidative stress can also result from some aspects, like chromium contained within MPs, whose effects are injurious to the anterior pituitary. [5, 6]

Epigenetic changes and gene dysregulation also occurs by the modulation of gene expression related to reproductive hormone pathways, demonstrated in various animal and cell studies. [2, 5]

Evidence linking microplastics to fertility issues

Male fertility

Spermatogenesis Impairment: MPs disrupt the blood–testis barrier by damaging the cells in the testis, reduce sperm count and motility, lead to the production of deformed sperm (two tailed sperm, microcephalic sperm, macrocephalic sperm, sperm with the absence of tail etc.) and decrease testosterone level by increasing activity of Reactive Oxidative Species (ROS), observed in both animal models and emerging human studies. [4, 7]

Inflammation and DNA damage:

MPs and associated NPs trigger inflammatory responses such as the release of IL-1β, IL-6 and tumour necrosis factor (TNFα) in the testes, with evidence of increased DNA fragmentation due to reduction of essential transcriptional factors, failure of gene expression and regulation such as DNA repair and reduced sperm viability, caused by ubiquitination of the protective system present in the seminal fluid which are essential for the maintenance of the sperm quality. [7]

Female fertility

Ovarian dysfunction: Exposure to MPs affects follicle development, steroidogenesis, and ovulatory cycles, leading to decreased fertility rates and altered hormonal profiles. Furthermore, MPs and NPs accumulation could lead to granulosa-cell apoptosis, elevated oxidative stress, suppress gene expression for follicular and oocyte development, further impairing fertility and increasing ovarian reserve decline. [4, 5]

Placental and offspring effects:

MPs detected in the human placenta raise concerns about potential impacts on embryo development and offspring health, as maternal exposure during gestation has been shown to have multi-generational effects in rodent models. These effects include placental inflammation, lipid and amino acid metabolic abnormalities in offspring, which may also create a hypoxic microenvironment in the embryo, impaired nutrient transport, and may cause reproductive abnormalities. [8]

Disrupted Endometrial Function:

Endometrial hyperplasia, fibrosis, and impaired embryo implantation have been reported following chronic MP exposure [4]

Collectively, findings underscore that MPs and NPs, via direct and indirect mechanisms, can compromise the reproductive axis in both sexes, leading to subfertility, endocrine disruption, and potential transgenerational effects. The precise risk to humans remains under active investigation, with animal models providing essential insights into potential pathways. According to animal studies, MPs and NPs have a significant impact on the reproductive and endocrine systems, particularly in

smaller organisms. Therefore, potential health effects in humans should not be disregarded, as increasing evidence suggests that ingested MPs and NPs can bioaccumulate in mammalian tissues, particularly in reproductive organs and endocrine glands, leading to irreversible oxidative damage and inflammation. This concern is heightened by the widespread problem of plastic pollution, often described as a 'plastic epidemic'. So, there is a clear need for comprehensive research into their long-term impacts.

Conclusion

The convergence of experimental, epidemiological, and mechanistic evidence emphasises the harmful effects of microplastics and their associated chemicals on endocrine and reproductive health. Although gaps remain concerning dose-response relationships and safe exposure limits for humans, the precautionary principle supports implementing mitigation strategies to reduce microplastic pollution and human contact.

Abbreviation

Endocrine-disrupting chemicals (EDCs), Microplastics (MPs), Nanoplastics (NPs)

Authors' contribution

a. Study planning: SKA, HU, FR
b. Manuscript writing: SKA, HU, AYX, YL, FR
c. Manuscript revision: SKA, HU
d. Final approval: SKA, HU, AYX, YL, FR
e. Agreement to be accountable for all aspects of the work: SKA, HU, AYX, YL, FR

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Author information

[1]Subasri Krishnarajan Anbuchelvan, MBBS student, Quest International University, Perak, Malaysia. ORCID
[2]Hashka Udayakumaran, MBBS student, Quest International University, Perak, Malaysia. ORCID
[3]Ang Yu Xin, MBBS student, Quest International University, Perak, Malaysia. ORCID
[4]Yoshita Loshni, MBBS student, Quest International University, Perak, Malaysia. ORCID
[5]Faria Rashid, MBBS student, St George's University of London, England. ORCID

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