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**The Impact of Microplastics and Nanoplastics on Ovarian
Reserve and Placental Function: From Epigenetic Alterations
to Clinical Pregnancy Outcomes**

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Abstract

Microplastics (MPs) and nanoplastics (NPs) are ubiquitous environmental contaminants recently detected in human follicular fluid, placental tissue, and fetal membranes. This congress paper synthesizes current mechanistic evidence demonstrating that MNPs act as potent reproductive toxicants through two interconnected pathological axes. In the ovary, MNPs induce granulosa cell pyroptosis, ovarian fibrosis, and steroidogenic dysregulation, ultimately depleting the finite follicular reserve. In the placenta, MNPs impair trophoblast syncytialization, inhibit extravillous trophoblast invasion, and trigger ferroptosis, leading to placental insufficiency. Epigenetic modifications, including aberrant DNA methylation and histone remodeling, serve as central mediators of these effects and potentially facilitate transgenerational transmission. Clinical data increasingly correlate placental MNP burden with spontaneous miscarriage, fetal growth restriction, and preterm delivery. This review establishes an integrated framework linking environmental exposure to adverse obstetrical outcomes and underscores the urgent need for regulatory intervention and prospective human biomonitoring.

Keywords: Microplastics, Nanoplastics, Ovarian Reserve, Placental Function, Epigenetics.



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1. Introduction

The exponential increase in global plastic production has resulted in the pervasive environmental dissemination of microplastics (<5 mm) and nanoplastics (<1 µm). Humans are continuously exposed to these particles via ingestion, inhalation, and dermal contact. Alarming, recent biomonitoring has confirmed the accumulation of MNPs within the human reproductive system, specifically in ovarian follicular fluid and the placenta (Li et al, 2025; Chen et al, 2025). Given the concurrent global decline in fertility rates, understanding the reproductive toxicity of MNPs is of paramount clinical and public health importance. However, a cohesive understanding bridging molecular initiating events to adverse obstetrical outcomes remains fragmented. This paper provides a concise, mechanism-focused review of how MNPs impair ovarian reserve and placental function, with a specific emphasis on epigenetic alterations, and translates these findings into clinical pregnancy outcomes.

2. Ovarian Reserve Depletion by MNPs

The ovarian reserve, defined by the number and quality of primordial follicles, is highly vulnerable to environmental toxicants.

2.1 Granulosa Cell Injury and Follicular Loss

Granulosa cells provide essential trophic support for oocytes. An (2021) first demonstrated that polystyrene microplastics (PS-MPs) induce dose-dependent apoptosis and fibrosis in rat ovaries via oxidative stress. Expanding on this, Peng et al (2024) elucidated that MNPs activate the NLRP3/Caspase-1 inflammasome axis, triggering pyroptosis, while simultaneously inhibiting the PI3K/AKT survival pathway and activating YAP1-ACSL4-mediated ferroptosis. The resulting ovarian fibrosis, driven by aberrant Wnt/β-catenin signaling, physically replaces functional follicular tissue, accelerating follicular atresia.

2.2 Steroidogenic Disruption and Oocyte Quality

MNP exposure significantly dysregulates key steroidogenic enzymes, upregulating 3βHSD and Cyp17a1 (which increases androgen synthesis) while suppressing 17βHSD, leading to hormonal imbalance (Peng et al, 2024). Clinically, Gao et al (2025) established a robust negative correlation between follicular fluid MP concentration and serum anti-Müllerian hormone (AMH) levels, as well as antral follicle count (AFC), confirming that MNP burden is an independent risk factor for diminished ovarian reserve. Furthermore, Williams et al (2025) reported that MNPs impair oocyte maturation and reduce blastocyst formation rates through gut microbiota-mediated metabolic disruption.



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3. Placental Dysfunction Induced by MNPs

The placenta is a critical transient organ, and its functional integrity is essential for fetal development. MNPs induce multi-faceted trophoblast dysfunction.

3.1 Impaired Syncytialization and Invasion

Syncytiotrophoblast formation is essential for endocrine and exchange functions. Cheng et al (2025) demonstrated that gestational MNP exposure activates the PERK/eIF2 α /ATF4 pathway of the unfolded protein response (UPR), directly suppressing syncytin-1/2 expression and inhibiting trophoblast fusion. Concomitantly, Nacka-Aleksić et al (2025) reported that 40 nm NPs internalized by trophoblasts induce a mesenchymal-to-epithelial transition (MET) shift, characterized by downregulation of MMP-2 and integrins, which impairs extravillous trophoblast (EVT) invasion and spiral artery remodeling.

3.2 Ferroptosis and Barrier Integrity

Wang et al (2025) showed that PS-MPs induce trophoblast ferroptosis via the HIF-1 α /TFRC pathway, leading to impaired angiogenesis and pre-eclampsia-like symptoms in murine models. Additionally, single-cell transcriptomics by Zhang et al (2025) revealed that MNPs significantly downregulate tight junction proteins (ZO-1, Claudin-3), disrupting the placental barrier and altering hemodynamics, which synergistically contributes to fetal hypoxia.

4. Epigenetic Mechanisms as a Central Regulatory Hub

The chronic, low-dose nature of MNP exposure suggests that epigenetic modifications serve as a "molecular memory" driving sustained reproductive toxicity.

4.1 DNA Methylation and Histone Modifications

Smith et al (2026) reviewed evidence that MNP exposure alters the expression of DNA methyltransferases (e.g., MET-2), leading to aberrant promoter methylation of genes critical for folliculogenesis (BMP15) and placentation (PEG10). Furthermore, co-exposure of PS-NPs with plastic additives induces transgenerational toxicity via SET-2-mediated H3K4 trimethylation, while HMGB1 acetylation links epigenetic changes to inflammatory fibrosis in reproductive tissues (Smith et al, 2026).



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4.2 Non-Coding RNA Dysregulation

The expression profiles of microRNAs and tRNA-derived small RNAs (tsRNAs) are significantly perturbed in trophoblasts and granulosa cells following MNP exposure. In model organisms, germline microRNA38 has been directly implicated in mediating the epigenetic inheritance of NP-induced reproductive deficits, raising concerns for analogous pathways in humans (Novillo-Del Álamo et al, 2026).

5. Integrated Summary of Mechanisms and Clinical Outcomes

To provide a structured overview of the multi-layered pathological effects discussed, Table 1 summarizes the major target tissues, their pathological outcomes, the underlying molecular pathways, and the corresponding key references.

Table 1. Comprehensive Summary of MNP-Induced Pathological Mechanisms and Clinical Correlates in Female Reproduction

Target Tissue / Domain	Major Pathological Effect	Key Molecular Pathways	Key References
Ovary	Granulosa Cell Pyroptosis & Apoptosis	ROS/NLRP3/Caspase-1; PI3K/AKT inhibition	An, 2021; Peng et al, 2024
	Ovarian Fibrosis	Wnt/ β -catenin activation	An, 2021
	Steroidogenic Dysregulation	Cyp17a1 $\uparrow$; 3 β HSD $\uparrow$ / 17 β HSD $\downarrow$	Peng et al, 2024
	Diminished Ovarian Reserve (DOR)	AMH $\downarrow$; Antral Follicle Count (AFC) $\downarrow$	Gao et al, 2025
Placenta	Syncytialization Failure	PERK/eIF2 α /ATF4 (UPR pathway)	Cheng et al, 2025
	Impaired EVT Invasion	MMP-2 $\downarrow$; Integrins $\downarrow$; E-cadherin $\uparrow$ (MET shift)	Nacka-Aleksić et al, 2025
	Ferroptosis & Angiogenesis Deficit	HIF-1 α /TFRC pathway	Wang et al, 2025
	Placental Barrier Disruption	Tight junction proteins $\downarrow$ (ZO-1, Claudin-3)	Zhang et al, 2025
Epigenetics	Aberrant DNA Methylation	MET-2 $\downarrow$; Promoter	Smith et al,



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		hypermethylation	2026
	Histone Modifications	H3K4 trimethylation; HMGB1 acetylation	Smith et al, 2026
	Non-Coding RNA Dysregulation	miRNAs; tsRNAs dysregulation	Novillo-Del Álamo et al, 2026
Clinical	Spontaneous Miscarriage	Elevated MP burden in chorionic villi	Chen et al, 2025
	Fetal Growth Restriction (FGR)	Placental senescence & ferroptosis	Wang et al, 2025; Zhang et al, 2025
	Preterm Birth & Low Birth Weight	Placental insufficiency & hypoperfusion	Novillo-Del Álamo et al, 2026

8. Conclusion and Future Directions

This synthesis establishes that MNPs are not inert particles but active reproductive toxicants. They deplete ovarian reserve through pyroptosis, fibrosis, and hormonal disruption, while simultaneously compromising placental integrity via syncytialization failure, ferroptosis, and barrier dysfunction. Epigenetic reprogramming acts as a persistent mechanistic bridge, potentially propagating these effects across generations without direct continued exposure. The clinical correlates, miscarriage, growth restriction, and preterm birth, represent a significant, yet under-recognized, threat to global reproductive health. Addressing these challenges requires an urgent shift in research priorities. Future investigations must move beyond cross-sectional designs to establish large-scale prospective pregnancy cohorts with rigorous, standardized MNP exposure assessment. Furthermore, advanced multi-omics approaches are needed to fully decipher the complex interplay between particle toxicity, leached chemical additives, and host epigenetic responses. From a clinical standpoint, biomarkers of MNP exposure and early placental dysfunction must be developed to enable timely intervention. On a global scale, these findings provide a compelling biological rationale for policymakers to accelerate the reduction of plastic pollution and regulate MNP emissions to safeguard future generations.

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