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Prenatal Microplastic Exposure Impairs Offspring Neurodevelopment through the Placenta-Brain Axis

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Abstract

Microplastics have emerged as environmental pollutants capable of crossing the maternal-fetal barrier and exerting adverse effects on fetal neurodevelopment. This review aims to elucidate how prenatal microplastic exposure impairs fetal brain development via the placenta-brain axis and to delineate its underlying neurotoxic mechanisms. Following maternal exposure via inhalation, ingestion, or dermal contact, microplastics enter the placenta via the blood and lymphatic systems. They are more likely to cross the placental barrier via energy-dependent active transport mechanisms. Microplastics can infiltrate fetal brain tissue through placental blood exchange or amniotic fluid ingestion, causing direct neural damage. They disrupt placental homeostasis, indirectly inhibiting fetal nervous system development. Prenatal microplastic exposure can interfere with core developmental events, including neural tube formation, cell proliferation and differentiation, neuronal migration, synaptogenesis, and myelination, leading to offspring phenotypes such as reduced spatial memory, decreased learning ability, impaired motor coordination, and anxiety-like behaviors. Furthermore, we conducted a bioinformatic analysis of transcriptomic data from microplastic-exposed human placenta (GSE220756) and mouse prefrontal cortex (GSE160012) retrieved from the GEO database, identifying shared regulatory pathways centered on aberrant vascular development, immune-inflammatory imbalance, and apoptotic dysregulation, particularly angiogenic impairment, which may serve as a critical node linking placental dysfunction to fetal neurotoxicity. However, limitations persist: small bioinformatic sample sizes, confounded lactational sampling in animal studies, exposure doses exceeding environmental relevance, and immature nanoplastic detection. Future work should expand the clinical specimens, establish standardized prenatal exposure models, integrate multi-omics with machine learning to identify predictive biomarkers, and validate the roles of angiogenic signaling.

Keywords

microplastics, prenatal exposure, placenta-brain axis, fetal neurodevelopment, neurotoxicity

1. Introduction

As a ubiquitous class of anthropogenic pollutants, microplastics (encompassing both micro- and nanoscale plastic particles, <5 mm) now permeate virtually every environmental compartment. The exponential growth of the plastics industry—production having expanded twentyfold over five decades to a cumulative 9.2 billion tonnes—has generated a disposal crisis: with recycling and incineration addressing only 9% and 12% of waste respectively, the residual 79% accumulates across air, water, and soil matrices, establishing continuous human

exposure pathways [1], endangering public health. In particular, fetuses in the gestational developmental stage are particularly susceptible to harmful environmental exposures. Recent studies have confirmed that microplastics can pass through the maternal-fetal barrier and transfer to various fetal organs [2, 3], with detection in human placenta, amniotic fluid, maternal blood, and meconium, providing powerful evidence [4-6]. Nowadays, the significant rise in neurodevelopmental disorders among children has drawn growing attention to prenatal microplastic-induced alterations in offspring neural development [7]. During pregnancy, the fetal brain is in a critical developmental stage and highly sensitive to environmental influences. As an environmental pollutant, microplastics are believed to exert irreversible effects on the developing brains of infants and young children. It was also proven that prenatal microplastic exposure exerts more severe effects on offspring nervous system development compared to lactational exposure [7-9], consequently establishing a vulnerable neurodevelopmental substrate that manifests as autism spectrum disorder, anxiety-like behavioral profiles, impaired social cognition, and decrements in learning and memory—trajectories that may extend into neurodegenerative vulnerability, including Alzheimer's disease. However, the specific transmembrane mechanisms of microplastic placental transport and their exact neurotoxic effects on the fetal brain are still not fully understood [10]. This review aims to investigate how prenatal microplastic exposure affects fetal brain development in a gestational window-specific manner through the placenta-brain axis and its underlying neurotoxic mechanisms, and identify shared pathways of placental and brain injury by mining placental exposure data and prefrontal cortex data from microplastic-exposed samples

2. Maternal-Fetal Interface: Microplastic Trans-barrier Transport into the Brain

During gestation, the developing embryo is highly susceptible to exogenous pollutants. This vulnerability arises from the incomplete maturation of biological barriers and the dynamic processes of organ development. Following maternal exposure, microplastics penetrate the placental barrier, passing through the blood-brain barrier, and ultimately impair the progression of nervous system development.

2.1 Maternal Exposure and Transplacental Transport of Microplastics

Consistent detection of microplastics in human placentas has been documented [4, 5, 11]. When examining pathways by which microplastics enter the human placenta, blood circulation plays a crucial role in this process [10], originating from maternal exposure routes—including inhalation, ingestion, and dermal contact [4, 11]. Microplastics enter the bloodstream and are transported to the placenta via systemic circulation. The identification of plastic polymers in human blood lends further weight to this process [12]. The lymphatic system may also play a role in this process: microplastics have been detected in the tonsils, and cross-species studies have shown that intestinal M cells can mediate transcytosis, with Peyer's patches possessing particulate-sampling functions [13, 14]. One hypothetical mechanism showed that microplastics, via M cell-mediated endocytosis or paracellular transport, enter the maternal respiratory system and the gastrointestinal tract (GIT), then access the bloodstream via dendritic cell-mediated transport, followed by transplacental infiltration into the placental tissue [4]. However, the lymphatic pathway remains purely theoretical at present, and no studies have directly traced the dynamic process by which microplastics transfer from the maternal lymphatic system to the placenta.

Upon reaching the placental interface, microplastics encounter four distinct transport modalities across the placental barrier: passive diffusion, energy-dependent active transport, phagocytosis/pinocytosis, and biotransformation [15]. Although the invasion mechanisms of microplastics into the placenta in depth remain elusive and complex [16], that microplastics traverse the placental barrier and exert fetal effects is no longer conjectural [2, 17]. The investigations by Braun and Ragusa demonstrated particle recovery from every placental region examined, as well as from maternal blood, meconium, fetal appendages, and umbilical vein blood—findings that collectively provide macroscopic proof of transplacental passage [4-6]. While Ragusa's 2022 research confirmed the presence of microplastics on the surface of placental villi, within cells of different placental layers, especially endothelial cells surrounding fetal microvessels, and in the extracellular environment, providing ultrastructural evidence of the complete transmembrane transport of microplastics [18]. Although passive diffusion cannot be entirely excluded, the preponderance of evidence suggests that energy-dependent active transport represents the principal driver of this transplacental translocation [10], and the syncytiotrophoblast is a key player in the process. In vitro models have demonstrated that microplastic particles accumulate in the syncytiotrophoblast and are translocated across the placenta via phagocytosis or endocytosis

[19, 20]. As exogenous foreign bodies, MPs' toxicity depends on particle size, surface charge, chemical composition, and the formation of protein corona and microbial films, which determine their ability to cross the cell monolayer and interact with cells [10, 12, 21, 22]. Among these factors, particle size plays a critical role: Sheng's study demonstrated that smaller polystyrene nanoparticles (PS-NPs) induced greater toxicity in human placental cells, likely due to their enhanced cellular accumulation. Surface charge is another key determinant—Shen reported that NH_2-functionalized PS-NPs exhibited higher cytotoxicity, stronger PKA inhibition, and greater oxidative stress than COOH-modified or pristine counterparts in vitro [23]. Beyond intrinsic physicochemical properties, the biological identity of microplastics is further modified upon entering physiological environments. When nanoparticles adsorb human proteins, they form a "protein corona" that can alter their original surface properties and promote cellular transport [24].

2.2 Direct Microplastic Exposure in Fetal Brain Development Following Transplacental Transfer

Pregnancy represents a critical window of vulnerability for fetal brain development, during which microplastics may reach the fetal central nervous system through multiple exposure routes. The primary conduit involves transplacental exchange followed by transport across the blood-brain barrier (BBB). During early gestation, the BBB remains immature and highly permeable, lacking fully established tight junctions; consequently, microplastics that have infiltrated the placental tissue can readily access the fetal circulation and subsequently the developing neural tissue through blood exchange [4, 6]. As gestation progresses into the second trimester, the BBB begins to form through the assembly of tight junctions between brain endothelial cells [25]. Nevertheless, polystyrene nanoparticles (PS-NPs) have been demonstrated to circumvent this nascent barrier in mouse models, either by directly disrupting junctional proteins or by exploiting active cellular transport mechanisms, thereby gaining entry into brain tissue [26]. However, the BBB gradually matures and becomes more selective; microplastics can still cross this barrier via the placental pathway and enter the brain parenchyma [27, 28]. Beyond crossing brain endothelial cells to penetrate the blood-brain barrier [26], microglial phagocytosis and subsequent activation may represent an alternative route for microplastic infiltration and the induction of neurotoxicity within the fetal brain [26, 29]. The formation and homeostatic regulation of amniotic fluid are closely associated with placental function. In addition to the transplacental-hematogenous route, theoretical evidence suggests that amniotic fluid ingestion may constitute another potential pathway of fetal exposure [30, 31]. The fetus regularly swallows amniotic fluid during gestation, and the documented presence of microplastics in this compartment raises the possibility of direct gastrointestinal uptake and subsequent systemic distribution. However, the quantitative contribution of this route relative to placental transfer remains undefined. Furthermore, while postnatal models have demonstrated that microplastics can reach the brain via the nasal-olfactory pathway [32] or the gut-brain axis [33], the extent to which these alternative routes operate in the fetus following amniotic fluid ingestion remains speculative. The anatomical and physiological differences between the fetal and postnatal nasal cavity and gastrointestinal tract, combined with the unique intrauterine environment, preclude direct extrapolation, necessitating further investigation to clarify the relative importance of each exposure pathway across different gestational stages.

2.3 Disruption of Placental Homeostasis by Microplastics and Subsequent Fetal Neurotoxicity

In addition to the direct impact of microplastics penetrating the brain, the disruption of the placental internal environment (de Sousa et al., 2024) should not be overlooked in its effects on fetal brain development. Microplastic exposure can directly interfere with normal placental physiological functions and induce placental vascular hypoplasia, characterized by shortened placental villous blood vessels, reduced vascular branching, and decreased vascular density. Such pathological changes directly lead to insufficient placental blood perfusion, failing to provide sufficient nutrients and oxygen for fetal brain development. Consequently, the blood and oxygen supply to the fetal brain is deficient, impeding the normal development of the nervous system [10, 34]. Microplastic exposure can trigger placental immune disorders and the release of abundant cytokines and inflammatory mediators, including IL-2, IL-4, IL-6, IL-10, TNF-α, and IFN-γ, It also induces oxidative stress and elevates the levels of related products, like MDA, CAT, MOP and H_2O_2 [35, 36], meanwhile the dysfunction of the placental barrier may allow inflammatory factors, oxidative stress products, or metabolically abnormal substances to enter the fetal circulation, thereby affecting nervous system development. This indirect

mechanism may act synergistically with microplastics' direct neurotoxicity. In addition, Placental endocrine dysfunction and the resultant hormonal imbalance can impair fetal brain maturation and disrupt neurodevelopmental rhythms, predisposing offspring to neurodevelopmental deficits. Excess glucocorticoids suppress hippocampal neurogenesis and trigger neuroinflammation, whereas oxytocin deficiency exacerbates microglial activation and myelination defects. Reduced sex steroids compromise neurite outgrowth, synaptogenesis, and myelination, while diminished IGF-I signaling attenuates synaptic plasticity. Thyroid hormone insufficiency further disrupts overall brain maturation, collectively increasing susceptibility to neurodevelopmental deficits [37].

3. Effects and Mechanisms of Prenatal Microplastic Exposure-Induced Fetal Neurodevelopmental Impairment

During pregnancy, the fetal nervous system is highly susceptible to environmental pollutants. Microplastics can cross the placental barrier and directly expose the developing brain during critical gestational windows. Experimental evidence has detected disruptions in neurogenesis, neuronal migration, synaptogenesis, and myelination. Such neurodevelopmental deficits arise from the convergence of multiple pathogenic mechanisms, including neuroinflammation, lipid metabolism disorders, endocrine disruption, and oxidative stress.

3.1 Disruption of Fetal Neurodevelopment by Prenatal Microplastic Exposure Across Critical Gestational Windows

Tian’s experiments confirmed that pregnancy is a neurodevelopmentally sensitive period [7]. Emerging evidence suggests that exposure to microplastics may disrupt fetal neurodevelopment at all gestational stages. In a chick embryo model, exposure to polystyrene PS-NPs induced neural tube defects in offspring, which form in the early stages of embryonic brain development [38]. Moreover, several studies have shown that Prenatal microplastic exposure affects neurogenesis and neuronal migration in offspring mice, including neural stem cell maintenance, self-renewal, and proliferation, leading to a reduced neuronal population and compromised differentiation capacity [7, 39, 40], Concomitantly, cortical migration is severely disrupted, manifested specifically as impaired radial migration of neurons from superficial to deeper cortical layers [7]. Additionally, Microglia, as the resident immune cells of the central nervous system, colonize the fetal brain during early embryonic development [41], rendering them a critical cellular target for prenatal microplastic exposure. In exposed mouse models, marked microglial activation and massive proliferation indicate that neuroinflammatory responses are initiated during this vulnerable developmental window, potentially causing persistent damage to fetal brain tissue [29, 40], when synaptogenesis and myelination represent the dominant neurodevelopmental events [42] maternal nanoplastic exposure causes significant synaptic structural damage. This is characterized by widened synaptic clefts, thinning of the postsynaptic density (PSD), and downregulation of PSD95 protein expression [7]. Mechanistically, the reduction in PSD95—a key scaffolding protein that anchors NMDA and AMPA receptors to the postsynaptic membrane, the reduction of which may impair postsynaptic receptor anchoring and excitatory synaptic Transmission. Transcriptomics has also revealed molecular-level alterations. RNA sequencing analyses have demonstrated changes in synapse-related genes, perturbed neuroactive ligand-receptor interaction (NLRI) signaling pathways, reduced secretion of glutamate and GABA, and indicate that oligodendrocytes undergo molecular changes under microplastic exposure [8, 43]. In Zhang's experiment, it was demonstrated that prenatal microplastic exposure causes cerebellar myelination disorders [44]. PS-NP exposure activates the extrinsic apoptotic pathway, as evidenced by elevated Caspase-8 and Caspase-3 expression, leading to extensive oligodendrocyte apoptosis and reduced expression of myelin structural proteins, including MBP, MOG, and MAG. Consequently, myelin sheath thickness is decreased, ultimately impairing motor coordination in offspring. More importantly, PS-NPs exposure in offspring mice leads to severe neurotransmitter disorder and altered ligand-receptor pathways, thereby disrupting neural circuit formation. For instance, NLRI-associated signaling pathways involving neuroactive ligands and their receptors are perturbed, along with reduced secretion of glutamate and GABA following maternal PS-NP exposure. In addition to glutamate and GABA dysregulation, the study by Tian also reported alterations in cortical monoamine neurotransmitters, including dopamine and serotonin, suggesting a broader impact on neurotransmitter systems [7, 8, 40, 43]

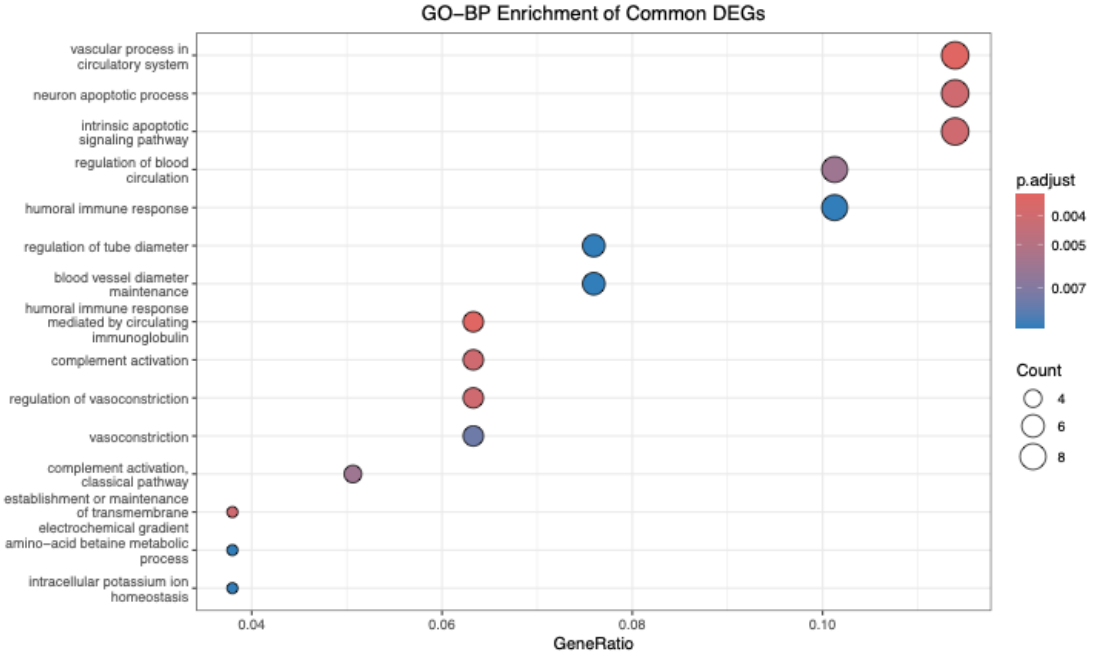
3.2 Multifaceted Mechanisms Underlying Microplastic-Induced Fetal Neurotoxicity

Microplastics affect fetal brain development through convergent pathophysiological mechanisms rather than a single toxicological axis, including immune system dysregulation, lipid metabolism disorders, and endocrine disruption. Neuroimmune perturbation constitutes one major pathway: microglial priming and subsequent phenotypic shift toward a pro-inflammatory state disrupt blood-brain barrier integrity, establishing a feedforward loop of sustained neuroinflammation and progressive neural injury [26, 29]. Lipid homeostasis disruption represents another critical dimension. Given that cerebral lipids function as structural components of neuronal membranes and myelin sheaths, as well as mediators of synaptic transmission and intracellular signaling, their metabolic perturbation carries substantial developmental consequences [45]. Exposure to nanoplastics during gestation remodels brain lipid profiles, with particularly pronounced effects on sphingolipid pathways, lysophospholipid depletion, and plasmalogen reduction [9, 46]. These lipidomic alterations display both sex-dependent variation and regional heterogeneity across the hippocampus, cerebral cortex, cerebellum, and dorsal raphe nucleus[9] Endocrine interference provides an additional mechanistic route. Nanoplastics can directly compromise hypothalamic GnRH neurons, impeding their embryonic migration, cytoskeletal architecture, and secretory output, thereby diminishing gonadotropin-releasing hormone synthesis and disrupting reproductive neuroendocrine signaling [47]. According to Fowler, gestational micro- and nanoplastic exposure markedly disrupts the homeostasis of multiple endocrine axes in offspring, including the hypothalamic-pituitary-gonadal axis, thyroid axis, and adrenal stress axis, thereby inducing extensive neuroendocrine disorders, especially ASD-like behaviors [48]. At the molecular level, oxidative stress-mediated reactive oxygen species (ROS) overproduction, together with the consequent augmentation of apoptosis and inflammatory responses, has been experimentally validated as a core pathological mechanism of microplastic-induced neurotoxicity [43, 44, 49-51]. Overall, it mainly affects regions including the cerebellum, hippocampus, striatum, and prefrontal cortex [44] and ultimately leads to impaired spatial memory, decreased motor coordination, exacerbated anxiety-like behaviors, reduced learning ability, and the emergence of ASD and autism-like symptoms [40, 48].

4. Bioinformatic Identification of Shared Placental-Brain Pathways in Microplastic-Induced Developmental Toxicity

To further explore the underlying mechanisms by which microplastics affect fetal development via the placenta–brain axis, we conducted a bioinformatic analysis by using to screen the common regulatory pathways mediated by microplastic toxicity in the placenta and brain tissues, this study retrieved transcriptomic datasets of PS NPs-exposed ex vivo perfused human placental tissue (GSE220756) and mouse prefrontal cortex (GSE160012) from the GEO database, and identified shared regulatory pathways through differential expression analysis. For the mouse prefrontal cortex transcriptomic dataset, differentially expressed genes (DEGs) were identified using $P < 0.05$ and $|\log FC| > 1$, yielding 883 significant DEGs, which were subsequently mapped to human orthologs. Owing to the limited sample size and insufficient statistical power of the human placental perfusion dataset, no DEGs survived stringent multiple testing correction (Bonferroni/FDR); therefore, an exploratory analysis strategy was adopted for this dataset, with a threshold of raw $P < 0.05$ and $|\log FC| \geq 0.5$, identifying 3,388 DEGs. The intersection of DEGs from both datasets was then extracted for subsequent Gene Ontology (GO) functional enrichment analysis. The results showed that the common biological mechanisms identified from the two differential datasets converged on aberrant vascular development, immune-inflammatory dysregulation, and apoptotic imbalance, including Vascular process in circulatory system, neuron apoptotic process, intrinsic apoptotic signaling pathway, regulation of blood circulation, humoral immune response, regulation of tube diameter, and so on. Such observations are broadly concordant with recent studies [29, 43, 49, 51]. However, it is worth focusing on the vascular pathways. Some research found that Microplastics impair placental vascularization and significantly reduce the number and diameter of uterine arteries in pregnant mice, resulting in inadequate placental perfusion, increased reactive oxygen species (ROS) levels, and impaired umbilical blood flow [36, 52]. Meanwhile, exposure to microplastics and nanoplastics also induced a significant reduction in the middle cerebral artery pulsatility index in fetuses, indicating cerebral vasodilation and impaired retinal vascular development [53, 54]. These findings strongly confirm that angiogenesis and vascular development play a crucial role in microplastic-induced damage to the placenta-brain axis.

Figure 1: Bubble plot showing Gene Ontology (GO) enrichment analysis of shared differentially expressed genes (DEGs) between PS-NP-exposed human placental tissue and mouse prefrontal cortex. The top 15 enriched biological processes are displayed. Bubble size represents gene count; color intensity indicates significance level based on adjusted P values (FDR-corrected). The enriched pathways converge on vascular development, immune-inflammatory response, and apoptotic processes



5. Discussion

Although numerous studies have examined microplastic exposure in pregnant mice, most tissue samples from offspring are collected after or during the lactation period [29, 40, 51]. This sampling strategy may confound the effects of in utero exposure with those of postnatal exposure through breastfeeding. Especially, Jeong's study demonstrated that lactational exposure is one of the pathways for microplastic transmission from mother to offspring [55]. Meanwhile, there remains a discrepancy between the microplastic exposure levels used in animal experiments and actual environmental exposure levels, and detecting nanoplastics remains a considerable challenge [10]. Given the limited sample size in the bioinformatic analysis of placental and mouse brain tissues and the fact that the current data are predominantly derived from animal experiments without establishing a comprehensive prenatal placental–brain axis microplastic exposure model, the findings of this study have certain limitations. Notably, our bioinformatic analysis identified vascular development as a core convergent pathway. From a mechanistic perspective, placental vascular hypoplasia and cerebrovascular abnormalities may form a vicious cycle: insufficient placental perfusion leads to chronic hypoxia and inflammatory factor release [56], which are transmitted to the fetal brain via the fetal circulation, further disrupting cerebral angiogenesis and blood-brain barrier maturation [57]. Conversely, cerebrovascular dysregulation exacerbates metabolic waste clearance deficits in brain tissue, creating a "placental-brain axis vascular injury cascade. Therefore, targeting angiogenic and vascular developmental pathways may offer novel therapeutic strategies for intervening in microplastic-induced neurodevelopmental toxicity. Given the limited accessibility and scarcity of clinical samples, future research may be pursued along the following lines: First, at the sample and model levels, efforts should be made to expand the acquisition of clinical specimens and to establish robust animal models of prenatal microplastic exposure to overcome the inherent limitations of human studies. Second, from a mechanistic perspective, multi-omics technologies—including single-cell sequencing and spatial transcriptomics—should be integrated to focus on the placenta-brain axis and precisely delineate the dynamic injury and interactive crosstalk among placental vascular endothelial cells, trophoblasts, and various neuronal subpopulations in the fetal brain following prenatal microplastic exposure. Concurrently, large-scale population-based cohort data should be leveraged to systematically validate the clinical association between microplastic exposure and placental-fetal brain developmental abnormalities. Third, at the analytical level, machine learning and deep learning algorithms could be applied to multi-omics datasets to identify key

molecular targets and early predictive signatures of microplastic-induced fetal brain developmental impairment, ultimately facilitating the construction of risk-prediction and early-warning models. [58]. Finally, as an important extension, prior experiments have revealed marked sex differences in the neurotoxic effects of prenatal microplastic exposure on offspring [9, 39, 48], suggesting that this toxic effect may be sex-dependent. This dimension merits particular attention and in-depth exploration in future investigations.

6. Conclusion

In conclusion, microplastics can cross the placental barrier and the fetal blood-brain barrier, exerting neurodevelopmental toxicity through the placenta-brain axis. On one hand, microplastics can directly invade fetal brain tissue and cause damage; on the other hand, they disrupt placental homeostasis, inducing vascular hypoplasia, oxidative stress, immune-inflammatory disorders, and endocrine imbalance, thereby indirectly inhibiting fetal nervous system development. Prenatal microplastic exposure can interfere with core developmental events, including neural tube formation, cell proliferation and differentiation, neuronal migration, synaptogenesis, and myelination, leading to offspring phenotypes such as reduced spatial memory, decreased learning ability, impaired motor coordination, and anxiety-like behaviors. Notably, our bioinformatic analysis confirmed that placental and brain injuries share common regulatory pathways characterized by aberrant vascular development, dysregulated inflammatory responses, and an imbalance in apoptosis. Angiogenic impairment, in particular, may serve as the critical node linking placental dysfunction to fetal neurotoxicity, offering a novel molecular perspective for understanding the integrated mechanisms of microplastic-induced neurodevelopmental toxicity via the placenta-brain axis. This review systematically elucidated the potential pathogenic mechanisms underlying fetal neurodevelopmental impairment caused by microplastics through the placenta-brain axis, focusing on distinct developmental windows of prenatal microplastic exposure, clarifying the critical mediating role of the placenta-brain axis in neurotoxicity, and exploring shared regulatory pathways of placental and brain injuries, thereby providing an important theoretical reference for deciphering the association between environmental exposure and neurodevelopmental disorders in offspring. However, in vivo experimental validation remains insufficient. Future studies should combine animal models with molecular experiments to more deeply validate the regulatory roles of key pathways and core genes, laying the foundation for understanding the reproductive and developmental toxicity of microplastics and for formulating prevention and control strategies for environmental pollutants during pregnancy.

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Conflicts of Interest

The authors declare no conflict of interest.

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