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Endocrine disrupting chemicals and their impact on child development: mechanisms, vulnerability windows, and health consequences

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ABSTRACT

Endocrine-disrupting chemicals (EDCs) are a group of exogenous substances that can interfere with hormonal signaling pathways, metabolism, neurodevelopment, and reproductive maturation. They are ubiquitous in all aspects of our environment, both natural and manufactured, which makes it virtually impossible to avoid exposure. Children represent the most vulnerable group due to their rapid growth, constant hormonal and neurodevelopmental changes, and their immature detoxification systems. The objective of this review is to provide an insight into how EDCs exert their biological effects and their clinical manifestations at critical periods like fetal life, infancy, and puberty by summarizing recent literature on their biological mechanisms of action. We also wanted to emphasize emerging data regarding micro- and nanoplastics (MNPs), epigenetic reprogramming, mixture effects, and transgenerational consequences. Although there are significant research gaps, evidence increasingly supports the role of EDCs as key factors in disrupted development that affect not only the particular stage of life but can also be passed on to later generations.

Keywords: children; endocrine disruptors; neurodevelopment; obesity; diabetes

1. INTRODUCTION

During the 1990s, growing scientific evidence indicated that various chemically distinct molecules, such as pesticides, plasticizers, phenols, flame retardants, and heavy metals, could interfere with natural hormonal pathways. These substances were termed EDCs. According to the WHO definition, EDCs are any foreign substances or combinations of substances that alter endocrine function and cause adverse effects in an individual or their offspring (Lamb et al., 2014). Within the last couple of decades, there has been a worrying increase in the environmental presence of a range of chemical substances due to human activities (Kabir et al., 2015; Kumar et al., 2020).

Child population is especially susceptible to EDCs because of their high exposure levels. This occurs because of bigger ratio between body surface and the

environment which results in higher consumption of resources. Another aspect is immaturity, which is the most significant in the context of the skin and blood-brain barrier, because it enables the EDCs to penetrate deeper tissues (Predieri et al., 2022). Considering that young organisms are constantly developing this high exposure and consecutive imbalance can disrupt organ growth, brain development, and metabolic processes, which later connects to the developmental origins of diseases.

Main groups of EDCs associated with children include bisphenols, phthalates, per- and polyfluoroalkyl substances (PFAS), organochlorine pesticides, dioxins, flame retardants, heavy metals, polycyclic aromatic hydrocarbons (PAHs), and MNPs. Even though they differ in structure and mechanisms of action, most EDCs share a common property: they interfere with hormones by either mimicking or blocking their receptors.

Mechanisms of Action of EDCs

There are two main ways in which the systems in the organism are connected – genomic signaling and non-genomic one. Unfortunately, the EDCs can interfere with both through a variety of interactions. The most classic one is modulation of nuclear hormones. Many EDCs, such as BPA, dioxins, as well as metabolites of dichlorodiphenyltrichloroethane (DDT), act through binding to various nuclear hormone receptors like estrogen receptors (ERα and ERβ), androgen receptors (AR), thyroid hormone receptors (TR), glucocorticoid receptors, and PPARs (Kabir et al., 2015). Based on their degree of diversity, EDCs act either as partial or complete agonists, antagonists, or selective regulators of receptors controlling transcriptional function, chromatin accessibility, and endocrine feedback regulation. Therefore, through these roles, EDCs are able to exert influence on a number of physiological processes such as sexual differentiation, metabolism, thyroid, and nervous systems. Along with the regular genomic signaling systems, another system, the non-genomic signaling system, exists through which EDCs can act. For example, BPA and genistein show great affinity for G protein-coupled estrogen receptors (GPER/GPR30) (Di Pietro et al., 2023; Patisaul, 2021a). The receptor GPER/GPR30 activates second messenger pathways, such as cAMP, PI3K/AKT, and MAPK pathways; thus, proliferation, mitochondrial function, apoptosis, and neuroendocrine secretions are affected. This type of non-genomic pathway can be stimulated even with a low level of exposure to EDCs, where the levels are significantly lower than what is used in the typical dose-response study design.

Another important action of EDCs is the disruption of hormone synthesis, metabolism, and transport in the body. Many insecticides affect aromatase enzyme activity, altering the estrogen-to-testosterone ratio during critical developmental stages. PFAS, heavy metals, and phenolic compounds may affect thyroid hormone synthesis, iodine absorption, iodine conversion, and thyroid hormone transport via transthyretin. In addition, BPA and phthalates affect Leydig cells, reducing fetal testosterone synthesis. (Nanjappa et al., 2012).

Epigenetic programming represents one of the key areas of research at present, providing the common mechanism that connects early exposure to disease susceptibility (Patisaul, 2021a). Epigenetic changes, such as DNA methylation, histone modifications, and microRNA regulation, occur as a result of EDC exposure during pregnancy and the neonatal stage in somatic cells and germ line tissues (Di Pietro et al., 2023). These epigenetic modifications have long-term effects and lead to disorders in metabolism, neurobehavioural development, and reproduction. The most appropriate example of the intergenerational effects of EDCs is diethylstilbestrol (DES), with the generations having reproductive problems and increased probability of developing cancer.

Lastly, EDCs contribute to oxidative stress, mitochondrial dysfunction, and chronic low-grade inflammation, which all lead to increased endocrine disruption (Patisaul, 2021b). The effect of non-receptor-mediated signaling pathway increases the metabolic dysfunction, neurotoxicity, and apoptosis of pancreatic β-cells which are linked with obesity, insulin resistance, type 2 diabetes mellitus, and neurodevelopmental diseases. It is also important to mention that interaction of the receptor-mediated and non-receptor-mediated signaling pathway gives mixture effects whereby chemical mixtures show synergy, thereby causing toxicity at high levels than individual chemicals.

2. REVIEW METHODS

To identify prior research on this topic, we used the PubMed and ScienceDirect databases to search for our literature. In our research we used a variety of keywords: Children, Endocrine Disruptors, Diabetes, Obesity, Neurodevelopment, ADHD, Puberty, BPA, Bisphenol-A, Phthalates, Prenatal Exposure, and Fetal Health.

Regarding time limits, the focus will be on studies conducted within the past 15 years. However, studies conducted within the last five years should be prioritized since they contain more recent results and a broader perspective on the matter. Articles found in the bibliographies of relevant sources were screened for inclusion to capture any eligible article which may not have been found in the database search.

Only studies that evaluated exposure of children to endocrine-disrupting compounds before birth or shortly after birth in relation to metabolic, developmental, or reproductive health effects were included in this review. We especially focused on studies that investigated links between exposure to BPA and phthalates and their connection to conditions such as obesity, diabetes, puberty changes, neurodevelopmental conditions, and fetal growth.

Any paper that was concerned only with adults, written in any language except English, or had been published before 2009, was omitted from consideration. Eventually, the authors selected articles that provided the strongest evidence on this topic (Figure 1).

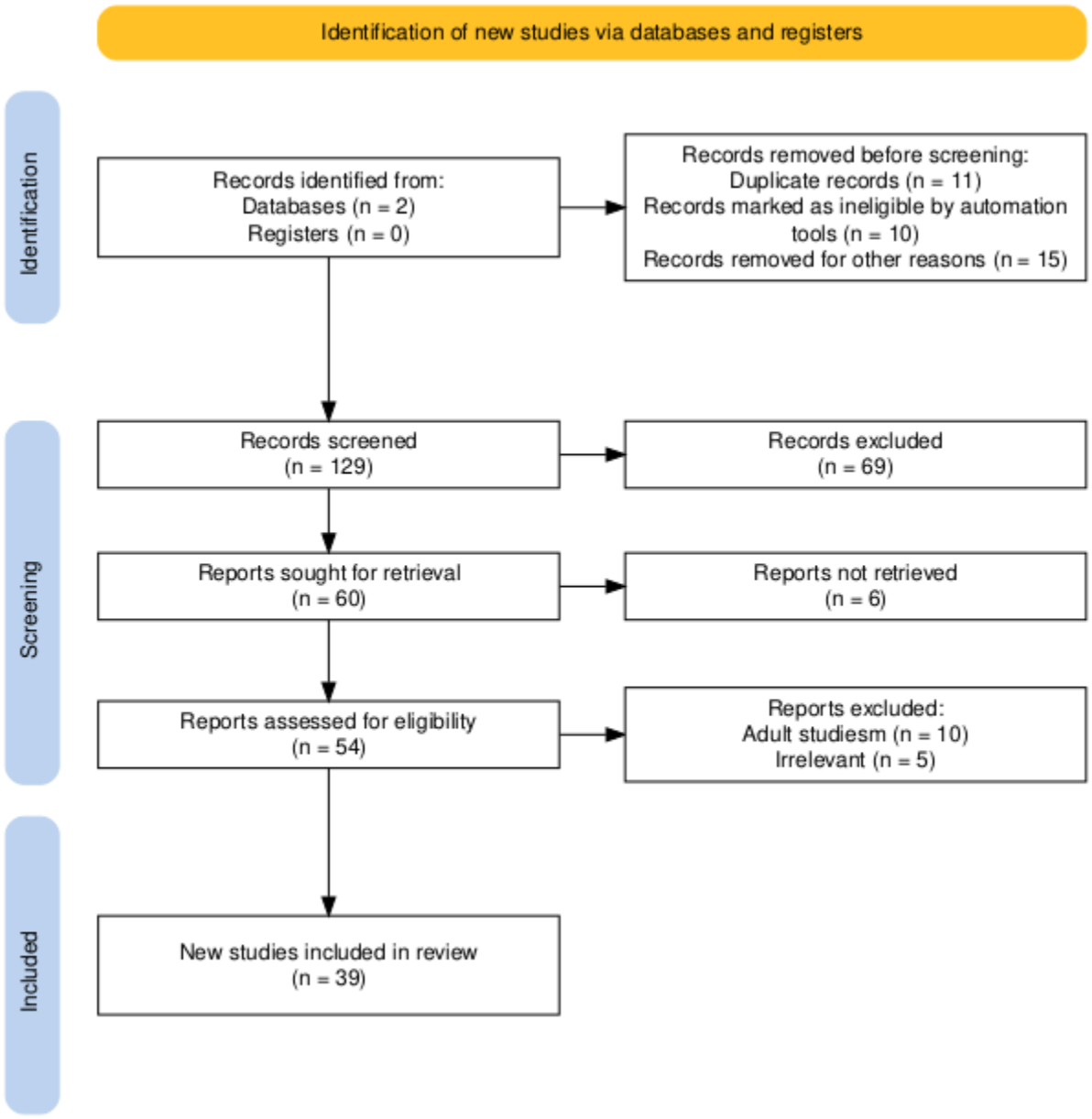

Figure 1. Process of identification of studies

3. RESULTS AND DISCUSSION

Fetal Development, Placenta Transfer and Reproductive Maturation

Placental Transfer and In Utero Endocrine Programming

The role of the placenta as both a selective barrier and an active endocrine gland allows significant permeability of EDCs. Owing to their lipophilic nature, small size, and the ability to diffuse passively or actively, many EDCs have been identified in human placenta,

cord blood, amniotic fluids, and meconium. The potential for such effects at critical developmental stages is highly worrisome, given that placental malfunctions caused by EDCs appear to contribute significantly their effect on overall health.

EDCs affect critical functions of the placenta, including hormone synthesis (hCG, progesterone, and placental lactogens), nutrient transport (especially glucose and amino acids), and vascular development. Each of these physiological mechanisms has been linked to poor fetal developmental consequences (growth restriction and prematurity) and increased vulnerability to metabolic disorders in the future (DiVall, 2013).

Among those especially vulnerable to EDCs are insulin-like growth factors, which are key modulators of fetal growth and placental development. Dysregulated expression of insulin-like growth factor 1 (IGF1), insulin-like growth factor 2 (IGF2), and IGFBPs due to EDC exposure, particularly BPA and dioxins, has been noted (Street and Bernasconi, 2020). Because the IGF pathway regulates proliferation, nutrient transport, and the growth trajectory of the developing fetus, it can be considered a mechanism linked to fetal programming of metabolic or endocrine disease.

In addition to the hormonal effects during pregnancy, EDCs have been shown to cause epigenetic modifications in placental tissues (Predieri et al., 2022). Modifications resulting from DNA methylation, histone modifications, and microRNA expression can permanently affect the fetus's physiology.

Mini-Puberty and Early Postnatal Hormonal Vulnerability

The occurrence referred to as mini-puberty has been described as the transient reactivation of hypothalamic-pituitary-gonadal (HPG) activity during the first 6 months of life, an important period when reproductive tissue differentiation occurs rapidly. The stage concerns steroidogenic cell types, such as Leydig and Sertoli cells in males, early follicular action in females, and sexual differentiation in the brain. Mini-puberty is highly susceptible to EDC disturbance, because it is a rapidly developing stage that is very dependent on hormones.

Anti-androgenic phthalates reduce the production of testosterone during fetal and early neonatal stages, which may result in abnormal male reproductive organ formation, such as cryptorchidism (Goodyer et al., 2017), hypospadias, small anogenital distance, and poor masculinization of the reproductive system (Gaudriault et al., 2017; DiVall., 2013). On the other hand, exposure to BPA affects the female reproduction systems causing premature ovarian follicle maturation and improper development of the mammary glands and uterus (Fernández et al., 2010). These alterations may predispose individuals to impaired fertility, altered gonadal function, and hormone-dependent pathology later in life.

As evidence shows the influence of such developmental programming is not only affecting the earliest stages of life, but may also affect the onset of puberty, ovarian reserve, testis volume, and reproductive hormone levels further disrupting the physiological human development (Salami and Rotimi, 2024; Neblett et al., 2020).

Pubertal Development and Endocrine Disruption

The start of puberty is one of the best coordinated and most complex processes that occur in our organisms in which all the signaling systems are involved. There is increasing evidence that EDCs might play a role in global changes in the age of puberty. The association between the presence of BPA, phthalates, phenols, and parabens in the body and premature thelarche, early menarche, premature pubarche, and delayed puberty has been demonstrated in numerous epidemiological studies (Wolff et al., 2015).

The way the disruption of puberty occurs due to EDCs can be described through the following mechanisms:

- Inhibiting the GnRH release (Fernández et al., 2010),
- Altered sensitivity of the pituitary gland (Lee et al., 2019),
- Changes in estrogenic and androgenic signaling
- Altering pathways of thyroid hormone maturation,
- Epigenetic changes in genes of HPG-axis regulation (Lee et al., 2019).

The PBDEs and PCBs may alter puberty by interfering with hormonal balance or altering thyroid hormone levels, which are important in the process of puberty induction (Den Hond et al., 2011). Interference with the endocrine system would thus produce either precocious or delayed gonadal maturation, depending on the EDC class used, the duration of exposure, and the individual's sensitivity. Taken together, these findings suggest that the impact of EDCs on the HPG axis contributes to the observed effects.

Neurodevelopmental and Neurocognitive Effects of Exposure to EDCs

Neurocognitive diseases have attracted significant scientific attention in recent years, inducing rapid progress in diagnostic approaches. Despite all the advances in neuroscience there is still insufficient knowledge about underlying factors for some neurocognitive diseases (Eubig et al., 2010). The brain is an extremely sensitive organ, regulated by various biochemical mediators, including steroid hormones, neuropeptides, and thyroid hormones, all of which can be altered by EDCs (Yesildemir and Celik, 2024). This sensitivity is particularly noticeable in childhood due to the blood-brain barrier's lack of selectivity.

Recent research has shown that EDCs exert neurodevelopmental effects via multiple pathways. There is evidence that EDCs may provoke neuroinflammation, interfere with neuronal proliferation and migration, impair synaptogenesis, and impair myelination (Yu et al., 2016). It has been shown that phthalates alter hippocampal structure and impair neurogenesis (Harley et al., 2013). In addition, data suggest that this impact may be mediated, at least in part, by microplastics, which act as vectors of EDCs. Disruption of thyroid hormone function is a common effect of EDCs, as these hormones participate in the development of the cortex and neurons.

A recent meta-analysis (Xu et al., 2025) on the environmental EDC-ADHD association in children found an important association between exposure to multiple chemicals, mainly BPA, and ADHD diagnosis risk (Harley et al., 2013). Indeed, BPA readily crosses the placenta, where it accumulates, making it a neurotoxic compound that interferes with brain development during the earliest period of gestation. BPA's mechanism of action is known to induce changes in glutamatergic neurotransmission, as well as to alter neuronal morphology and synaptogenesis, resulting in an anxiety/depressive phenotype (Perera et al., 2012).

Prenatal and postnatal exposure to BPA has been associated with a risk for developing ADHD in children (Xu et al., 2025), nevertheless, some studies have shown a possible difference in sex-specific susceptibility to ADHD, depending on the amount of BPA in the child's urine samples (Sagiv et al., 2012a; Sagiv et al., 2012b). In particular, boys might show greater sensitivity because of their higher BPA levels, suggesting a possible sex hormone-related pathway.

Phthalates represent yet another group of EDCs contributing to the formation of ADHD-related behavioral profiles (Shoaff et al., 2020). Most of the existing evidence indicates the connection between the prenatal exposure of an individual to phthalates and their attention and behavior disorders; at the same time, postnatal exposure to these chemicals does not seem to carry similar risks. Notably, some studies suggest that phthalates can affect the manifestation of behavioral abnormalities even if the individual does not have ADHD (Shoaff et al., 2020). Moreover, these behavioral effects differ between boys and girls; boys tend to exhibit greater withdrawal and internalizing behaviors, while hyperactivity is more frequently observed in girls (Daniel et al., 2020).

Data interpretation from currently available information is complicated by the heterogeneity of the phthalate mixture itself, the short biological half-lives of the phthalate compounds involved, and developmental periods with critical phases of exposure that disproportionately affect certain developmental events.

Neurodevelopmental effects of EDCs on children are not restricted to the development of ADHD. Studies show that prenatal exposure to pesticides is linked to lower intelligence quotients (IQ), shorter attention spans, and problems with executive functions. In addition, recent studies have reported a potential association between DDT metabolites and ASD-related behavioral phenotypes (Yesildemir and Celik, 2024). Children whose mothers were found to have increased levels of these metabolites had significantly more ASD-related characteristics, specifically lack of social interactions and attention problems (Sagiv et al., 2012a; Sioen et al., 2013). Yet, this link remains inconclusive because the compounds are rapidly broken down in the body.

Metabolic and Endocrine Effects of Exposure to Endocrine-Disrupting Chemicals

One of the most studied health consequences of exposure to EDCs relates to the onset of obesity along with its associated metabolic abnormalities (DiVall, 2013). It is now thought that EDCs are metabolic disrupters, as they affect pathways involved in adipocyte differentiation, glucose balance, energy expenditure, endocrine pancreas functions, and inflammation (Bergman et al., 2012). EDC-induced disruption of hormone-regulated metabolism may negatively affect for organisms, especially during early embryonic stages and postnatal period, as differentiation and metabolism remain highly plastic.

EDCs and the Promotion of Adipogenesis

Another mechanism by which EDCs cause obesity is the induction of adipogenesis (DiVall, 2013; Predieri et al., 2022). Some EDCs, such as bisphenols and phthalates, exhibit agonistic effects on estrogen receptors; in addition, some can act on nuclear receptors, such as PPARγ, which take part in the process of adipocyte differentiation (Diamanti-Kandarakis et al., 2009). Consequently, lipid synthesis becomes more efficient, while glucose metabolism is disturbed, leading to the onset of insulin resistance. As it was observed in the

laboratory research, EDCs can elevate plasma free fatty acid levels, which promotes the maturation of the adipocyte. Those changes lead to adipose tissue expansion further disrupting the glucose metabolism and its tolerance (Mohd Efendy Goon et al., 2024).

Central regulatory mechanisms have also been found to be disrupted following EDC exposure (Patisaul, 2021b). This is exemplified by changes of the hypothalamic pathways regulating hunger and satiety through leptin, ghrelin, and neuropeptide Y signaling (Mohd Efendy Goon et al., 2024). In addition, alterations in gut microbiota composition caused by exposure to various EDCs seem to contribute to increased fat deposition. These alterations occur regardless of energy consumption, suggesting that EDC-induced dysbiosis may amplify metabolic disturbances. This could be achieved by inhibiting transcription factors which are active during fasting, leading to excessive fat deposition and fatty acid accumulation in adipocytes.

EDC's role in Insulin Resistance, and Diabetes Risk

There has always been an established link between obesity, insulin resistance, and development of T2DM, but now this link can also be drawn back to earlier stages of life. According to the epidemiological studies, early prenatal exposure to environmental pollutants affects glucose tolerance and predisposes to development of T2DM (Roth and Petriello, 2022). These findings lay the groundwork for a hypothesis that fetal exposure to environmental chemicals induces persistent metabolic changes through metabolic programming.

There is one particular compound which is especially active in this area. BPA compounds with its antagonistic relation to estrogenic pathway, can cause a diabetogenic effect in the organisms by promoting apoptosis of pancreatic β-cells (Predieri et al., 2022). As it was observed in the preclinical studies BPA disrupts the protective function of 17β-estradiol by two mechanisms: classically through estrogen receptors (ERα and ERβ) and indirectly through the G protein-coupled estrogen receptor (GPER). It was additionally confirmed when the receptor activity was inhibited, what reduced the negative impact of BPA, reducing β-cell apoptosis. Moreover, BPA increases oxidative stress, which is the pancreas which β-cells have especially low antioxidant capacity.

In addition, there is another field of EDC effects that is largely theoretical and concerns immunological disruption. This hypothesis relies on the capacity of EDCs to cross the placental barrier and accumulate in fetal organs, potentially altering immune tolerance during early development. It is supported by previously observed processes of oxidative stress in β-cells following BPA exposure, which may predispose pancreatic cells to earlier functional exhaustion and thus increase the risk of type 1 diabetes (T1DM). However, the autoimmune mechanisms underlying T1DM development are complex, and currently there are no established methods for evaluating this relationship (Bergman et al., 2012). To date, there is no clear association between EDC exposure and T1DM.

Disruption of Thyroid Hormone Homeostasis

Thyroid function is another major area of concern in which the impact of EDCs has been observed. The role of thyroid hormones is vital to the functioning of the organism, as the regulate metabolism, impact the neurodevelopment and somatic growth. Taking that into consideration, disturbances in the synthesis, transport, or binding of those hormones can have profound consequences.

Among EDCs, BPA acts as antagonist of thyroid hormone receptors (Predieri et al., 2022); it acts at the level of gene expression, inhibiting T3–mediated transcription (Diamanti-Kandarakis et al., 2009). There are also other environmental contaminants, including heavy metals such as cadmium and lead, that, in research, have presented negative effects on thyroid hormone synthesis, deiodinase activity, and peripheral metabolism, resulting in reductions in circulating free thyroid hormone concentrations (Nascimento et al., 2018; Diamanti-Kandarakis et al., 2009).

Exposure to EDCs can not only disrupt the balance of thyroid hormones but also interfere with normal organ maturation. As a result, thyroid gland hypoplasia can be observed, which is connected to impaired neurodevelopment and increased risk of ADHD – like behaviors (Predieri et al., 2022). The postulated mechanism is based on disturbances in both estrogenic and thyroid hormone pathways, which impair dopaminergic transmission and calcium signaling, the foundations of attention-regulation networks.

MNPs as Emerging Endocrine Disruptors

The influence of MNPs on our health and function has only recently been recognized as an important topic, despite their presence in our environment for years. To date, the accumulation of MNPs has been detected in the placenta, fetal tissues and multiple organs (Medley et al., 2023). It raises significant concern about the long-lasting effects of this exposure on organogenesis and metabolic programming during the earliest and most vulnerable period of human development.

Placental Accumulation and Disruption of Placental Function

MNPs in human placental tissues were first identified in 2020 and their presence was later confirmed by numerous studies examining their biological effects (Ragusa et al., 2021). In animal models, MNP exposure caused significant morphological changes in the placenta, including reduced size, disrupted vascular formation, and fewer glycogen-rich cells (Anifowoshe et al., 2025). Those changes are linked to impaired nutrient transfer and also reduce placental endocrine and immunological functions.

It is important to note that MNPs also have been found in fetal tissues, which is alarming particularly in context of long-term effects. In rodents MNPs have been identified in a variety of tissues including hearts and brains, confirming their ability to cross placental barriers and distribute systemically. They have been also detected in human meconium and amniotic fluid, which raises even greater concern considering yet unknown effects of such early exposure.

Molecular and Cellular Effects

Some mechanisms of MNPs action that have been investigated in the human placenta model involve inhibition of angiogenesis, changes in gene expression pattern, and disruption of iron metabolism (Anifowoshe et al., 2025). This leads to impairment of fetal oxygen supply, development, and metabolic function, resulting in abnormal organ development, lipid balance, and muscle dysfunction in animal models. The most interesting effect was observed in mice, where MNP affected neural stem cells in the hippocampus decreasing the number of cells, which indicates disruption of neurogenesis and cognitive development.

Additionally, some studies have found that the abnormalities in organ size and metabolism continue into adulthood, consequently to the reduced birth weight, and therefore indicate programming effects rather than temporary effects on the fetus.

Limited Understanding and Resources for Assessment

However, despite these startling results, there is still much more to learn about the potential health impacts of MNPs due to the absence of appropriate methodology. To begin with, the available tools for mechanism studies are not sufficient to trace the molecular mechanisms involved in the toxicity of MNPs and thus give us an insight into the link between exposure and its impact on biological processes. Furthermore, in the case of nanoplastics, it is difficult to conduct exposure assessment since the measurement techniques employed in research are not sufficiently sensitive.

There is also a significant discrepancy between the controlled experimental conditions of study and real- world exposure scenarios. Every day, humans are exposed to various kinds of particles, which are absorbed and accumulated simultaneously, whereas in studies, chemical compounds are tested individually (Zurub et al., 2023). There is a real possibility of synergistic interactions between compounds themselves or between particles and adsorbed chemicals, which may produce effects greater than those of each component in isolation.

One more point to make about this subject is the fact that the levels at which various animal models were exposed are counted in millions of particles per kilogram of body weight. This prominent level of exposure can be hardly observed in any real-life conditions, making the findings less relevant (Zurub et al., 2023).

Another aspect that must be acknowledged is that MNP research is relatively new, and the long-term effects of MNP exposure across the lifespan have not yet been fully characterized. Early evidence indicates the possibility of lifelong consequences, but direct evidence remains lacking.

In summary, current studies indicate that MNPs may be able to cross the placental barrier, accumulate in fetal tissues, and interfere with endocrine signaling and balance. More research is needed to further explore the issue of MNPs' effects on humans.

The "Cocktail Effect"

As mentioned earlier, it is important to remember that in real-life environments humans are constantly exposed to various EDCs in mixtures so complex that they are impossible to recreate in experimental trials. Although our knowledge of EDCs and their individual mechanisms continue to expand, current methods are insufficient to adequately measure and analyze the combined biological effects of chemical mixtures. This combined exposure represents one of the biggest challenges in modern environmental health research and is known as the "cocktail effect."

All current toxicological models are ineffective at simulating the complex, dynamic nature and low-dose exposures found in natural environments. In controlled experiments, higher dosages have typically been used to discover the underlying mechanism, yet such studies do not provide predictive power for chronic low-dose exposures in living organisms. It is reasonable to assume that the toxicity

profiles of EDC mixtures cannot be predicted based solely on single EDC due to non-linear and non-monotonic dose-response relationships characteristic to these compounds.

A growing body of evidence indicates that a mixture effect may be additive, synergistic or even potentiating, meaning that exposure to a mixture of chemicals can produce effects exceeding those observed for individual chemicals. The chemical variability of the environmental EDC mixture, encompassing lipids, volatiles, metals, persistent organic pollutants, and microplastics, increases the likelihood of chemical interactions across biological scales, namely molecular, cellular, and systemic.

To further complicate matters, there are considerable variations in sensitivity among individuals to EDCs, owing to genetic differences, hormonal differences between males and females, age, nutrition, gut bacteria, and exposure to other chemicals. In such a situation, it is impossible at the moment to find a "safe dose" for complex mixtures of endocrine disruptors. Besides, measurement problems related to nanoplastics and short-lived metabolites make it difficult to conduct risk assessment.

The shortcomings of such methodologies and the numerous inconsistencies across studies indicate the need to develop further new scientific research methods, such as mixture toxicology, systems biology, and PBPK modeling. Unless and until such development is achieved, it seems difficult to fully comprehend the concept of mixture toxicity and develop appropriate regulations around it.

In light of current knowledge, further multidisciplinary research integrating toxicology, endocrinology, epidemiology, epigenomics and environmental sciences is necessary. Table 1 summarizes areas of EDCs influence and their potential health consequences on various stages of development and on major systems, which function is disrupted by them.

Table 1. Summary of mechanisms of EDCs and their clinical consequences

Developmental stage / system	Mechanisms of EDC/MNP action	Potential health consequences	Key evidence sources
Placenta and prenatal development	Disruption of placental function (hormone synthesis, nutrient transport, vascular development), epigenetic modifications, dysregulation of the IGF axis	Fetal growth restriction, preterm birth, and developmental programming of metabolic and endocrine disorders later in life	Street & Bernasconi, 2020; Predieri et al., 2022;
Mini-puberty (first 6 months of life)	Disruption of HPG axis activity through the anti-androgenic effects of phthalates and estrogenic activity of BPA	Abnormal reproductive organ development, impaired gonadal function, and reduced fertility later in life	Goodyer et al., 2017; Gaudriault et al. 2017; DiVall, 2013; Fernández et al., 2010
Pubertal development	Altered GnRH secretion, disrupted estrogenic and androgenic signaling, thyroid hormone interference, and epigenetic regulation of the HPG axis	Precocious or delayed puberty and impaired reproductive function	Wolff et al., 2015; Lee et al., 2019; Den Hond et al., 2011
Nervous system	Neuroinflammation, impaired neurogenesis, neuronal migration, synaptogenesis, myelination, and thyroid hormone signaling	ADHD, reduced cognitive performance, lower IQ, executive function deficits, and ASD-related traits	Yesildemir & Celik, 2024; Yu et al., 2016; Xu et al., 2025; Shoaff et al., 2020
Metabolic and endocrine system	Enhanced adipogenesis (PPARγ activation), impaired glucose metabolism, oxidative stress in pancreatic β-cells, and disruption of thyroid hormone homeostasis	Obesity, insulin resistance, type 2 diabetes mellitus, and thyroid dysfunction	DiVall, 2013; Diamanti-Kandarakis et al., 2009; Mohd Efendy Goon et al., 2024
Micro- and nanoplastics (MNPs)	Placental transfer, accumulation in fetal tissues, disruption of angiogenesis, altered gene expression, and impaired iron metabolism	Impaired organ development, fetal growth restriction, and long-term metabolic and neurodevelopmental alterations	Anifowoshe et al., 2025; Zurub et al., 2023

4. CONCLUSION

In this review, we summarized the latest knowledge about the EDCs' impact on environmental health. As studies show, exposure to EDCs at an early age can cause lifelong effects on a variety of systems, especially reproductive, nervous, and endocrine. While the fundamental biological effects of EDCs are mostly understood, there remains another even broader area of epigenetic alterations, interference with signaling, and activation of oxidative stress. Further research is needed to broaden our understanding of health consequences for people, exposures to low-dose concentrations, and the interactions between multiple environmental toxins.

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Authors' Contributions

Conceptualization, all authors; methodology, O.B. and J.M.; resources (1-20), N.S., S.A. and N.B., resources (21-39) P.Z. B.W. H.M.; writing—original draft preparation, O.B. and E.M.; writing—review and editing, all authors (O.B., J.M., N.S., S.A., E.M., N.B., B.W., P.Z. H.M.). All authors have read and agreed to the published version of the manuscript.

Informed consent

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Ethical approval

Not applicable. This article does not contain any studies with human participants or animals performed by any of the authors.

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Conflict of interest

The authors declare that they have no conflicts of interest, competing financial interests or personal relationships that could have influenced the work reported in this paper.

Data and materials availability

All data associated with this study will be available based on the reasonable request to corresponding author.

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