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REVIEW PAPER

Metabolic disruption in early life: the role of endocrine disruptors in pediatric obesity

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ABSTRACT

Child and adolescent obesity is a significant and increasingly prevalent public health challenge. Among the contributing factors are endocrine-disrupting chemicals (EDC), which interfere with pathways regulating adipogenesis and metabolic homeostasis. The mechanism of EDC action includes epigenetic modifications, interactions with hormonal receptors, and disruption of key hormones such as leptin, insulin, and thyroid hormones. Exposure during critical developmental periods – specifically in utero and in early childhood – poses a substantial risk, promoting long-term metabolic disorders. This review presents current epidemiological data and discusses potential mechanisms underlying obesity development due to EDC. It also underscores the importance of preventive strategies, including reduced plastic usage, promotion of organic diets, and strengthened regulatory measures, in mitigating the global burden of pediatric obesity.

KEY WORDS:
endocrine disruptors, obesogens, childhood obesity, pediatric endocrinology.

INTRODUCTION

Obesity represents a form of malnutrition characterized by an imbalance between energy intake and nutrient quality. Suboptimal dietary patterns result in micronutrient deficiencies that, in concert with environmental factors such as exposure to endocrine-disrupting chemicals (EDC), contribute to the pathogenesis of metabolic complications [1]. It affects low-, middle- and high-income countries, and its incidence continues to rise. Childhood obesity often persists into adulthood and is associated with a range of health consequences. As demonstrated in the /existing literature, obesity has been shown to be a contributing factor to the development of a number of serious health conditions, including metabolic syndrome (MetS) and cardiovascular disease. Childhood-onset type 2 diabetes mellitus is also associated with obesity, and this can lead to a range of retinal and renal complications, as well as non-alcoholic fatty liver disease. Obstructive sleep apnea, premature menarche, polycystic ovary syndrome, infertility, asthma, orthopedic complications, psychiatric disorders, and increased rates of malignancies are further health consequences of obesity [2, 3].

According to a 2019 prediction by the World Obesity Federation, by 2025, up to 206 million children and adolescents aged 5–19 /would be affected by obesity, with this number potentially rising to 254 million by 2030. Among the countries with the highest obesity rates in children, the top five are China, followed by India, the USA, Indonesia, and Brazil. Statistics indicate that 42 countries will have over 1 million obese children, with only 7 of these 42 nations being classified as high-income countries [2].

The endocrine system has a significant impact on regulating body weight and metabolic processes. Many hormones, including thyroid hormones, insulin, androgens, and estrogens, affect pathways that control the size and

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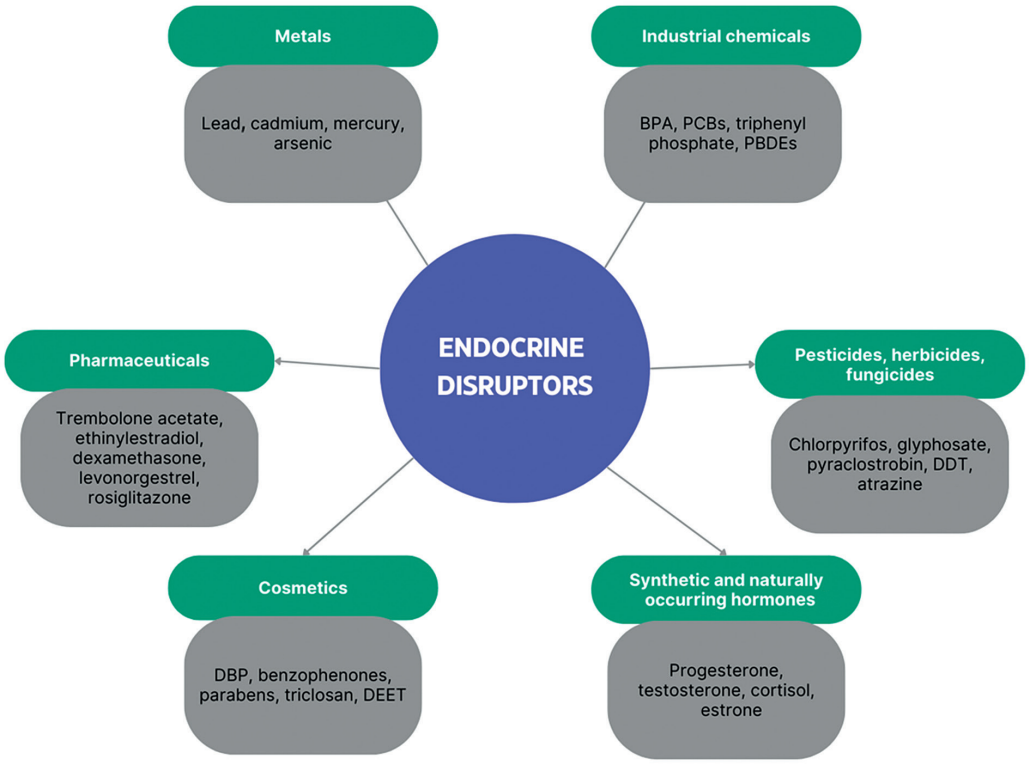

FIGURE 1. Examples of endocrine-disrupting chemicals classified by source

BPA – bisphenol A, DBP – dibutyl phthalate, DDT – dichlorodiphenyltrichloroethane, DEET – N,N-diethyl-meta-toluamide, PBDEs – polybrominated diphenyl ethers, PCBs – polychlorinated biphenyls

number of adipocytes. Excess body fat is associated with a number of endocrine changes, of which insulin resistance is one of the most prominent. Obesity can also affect the hypothalamic-pituitary hormonal axis. This can result in growth hormone deficiency, hypogonadism, Cushing's disease, or thyroid hormone deficiency. There is a bidirectional relationship between obesity and hormonal dysregulation in obese patients, where obesity can be both a cause and a consequence of endocrine disruption. Another major threat to the endocrine system, that can also promote obesity, is EDC [4–6].

CHARACTERISTICS OF ENDOCRINE-DISRUPTING CHEMICALS

DEFINITION AND CLASSIFICATION OF ENDOCRINE-DISRUPTING CHEMICALS

Endocrine-disrupting chemicals are defined by the Endocrine Society as exogenous substances or mixtures that can interfere with any component of endocrine activity. Endocrine-disrupting chemicals are now widespread environmental contaminants and human exposure can come from a variety of sources, including pesticides, food, pharmaceuticals, personal care products, medical devices, and even toys. Epidemiological studies have linked exposure to EDC to adverse effects on the endocrine system, contributing to neurological, metabolic, and reproductive disorders [4].

Endocrine-disrupting chemicals can be classified according to their origin. The first group includes substances of industrial origin, such as dioxins, polychlorinated biphenyls (PCB), and alkylphenols. Another group includes agricultural substances such as pesticides, insecticides, herbicides, phytoestrogens, and fungicides. Another two groups are household compounds such as phthalates, polybrominated biphenyls, bisphenol, and those of pharmaceutical origin – parabens. There are also heavy metals such as cadmium, lead, mercury, and arsenic [7] (Figure 1).

SOURCES AND ROUTES OF EXPOSURE

Endocrine-disrupting chemicals affect the human body through the respiratory and digestive systems and through direct contact with the skin. Ingestion and inhalation of EDC are associated with the highest levels of absorption. However, due to the action of liver enzymes, substances absorbed in this way are better excreted and detoxified than those that interact through the skin. Food can be contaminated with more than one EDC, which can have a more complex effect on the body. What is more, EDC bioaccumulate in the body's fatty tissue [7, 8].

Before food reaches the consumer, it goes through many stages of production and processing. Endocrine-disrupting chemicals can enter food at any stage of this journey and may even be present in intermediate products from the outset. With this in mind, EDC can

be divided into the following categories. The first group includes EDC that can bioaccumulate in adipose tissue. They are present in zoonotic products such as milk and other products containing fatty acids of animal origin. This category includes, for example, digoxin, polybrominated diphenyl ethers (PBDE), perfluorinated compounds, or PCB. The second group consists of substances used in food production, i.e. compounds such as pesticides or food additives. The third group includes EDC that come from tools and packaging used in the storage, transport, or other industrial processes necessary to produce a food product. These include, for example, bisphenol, the use of which in food packaging has been restricted in the European Union (EU) since 2011. The last group is made up of EDC, which have hormone-like effects. These include substances such as heavy metals, phytoestrogens, iodine, and quercetin [8].

MECHANISM OF ACTION OF ENDOCRINE-DISRUPTING CHEMICALS

Endocrine-disrupting chemicals, once they enter the body, bind to receptors. They can act as agonists or antagonists by affecting a particular hormonal pathway. Most EDC bind to androgen or estrogen receptors. Some EDC can act through multiple mechanisms and have mixed properties. For example, they may have simultaneous estrogenic and antiandrogenic effects, or act simultaneously with estrogen and progesterone. In addition, the effects of EDC metabolites are often different from those of the original substance. Endocrine-disrupting chemicals also act through genomic and non-genomic mechanisms. Non-genomic responses are very rapid, with effects seen immediately, a few minutes after exposure, whereas genomic responses take longer, often several hours. The action of EDC is based on effects on the metabolism of endogenous steroid hormones, but also on modulation of nuclear receptor coactivators. They also act by degrading endogenous hormones by targeting the proteasome. The mechanism of action of EDC involves different hormonal pathways. Both estrogen and androgen receptors are mentioned, but EDC can also affect thyroid receptors (TR) and peroxisome proliferator-activated receptor γ (PPARγ) and retinoids. By binding to the receptors in question, EDC mimic the action of hormones. However, they should not be confused with endocrine modulators, which are compounds that affect the endocrine system without disrupting its function [9–11].

Endocrine-disrupting chemicals can adversely affect the functioning of the whole body through their effects on the endocrine system. At the same time, the same exposure can cause more serious effects in children than in adults. This is due to a number of factors, including the greater permeability of young children's skin or the immaturity of some organs and a less efficient detoxification process. This results in an increased risk of developing diseases in childhood such as obesity, liver dysfunction, or cardiometabolic disease [8].

MECHANISMS OF ENDOCRINE-DISRUPTING CHEMICALS' INFLUENCE ON THE DEVELOPMENT OF OBESITY IN CHILDREN

DISRUPTION OF ADIPOGENESIS AND LIPID METABOLISM

A 2022 study examined the effects of an environmentally relevant mixture of 14 EDC on human mesenchymal stem cells *in vitro*. A mixture of 14 EDC significantly promoted adipogenesis and altered DNA methylation at genes involved in metabolic regulation. Adipogenic effects were consistent across donors. DNA methylation analysis revealed 713 differentially methylated positions, mostly hypomethylated, especially in intergenic regions potentially linked to regulatory functions. Six key differentially methylated regions overlapped genes associated with adipogenesis, obesity, or birth weight, including *PM20D1*, *HOXA5*, and *RPL28*. Gene ontology analysis showed enrichment of metabolic and endocrine pathways [12]. Another research suggests that exposure to EDC can induce epigenetic changes affecting adipogenesis and lipid metabolism, with effects persisting across multiple generations. These transgenerational effects are mediated primarily through nuclear receptors like PPARγ and estrogen receptor α, which influence gene expression by altering DNA and histone methylation. Studies in both cell models and animal experiments have demonstrated increased adipocyte differentiation and obesity-related gene expression linked to hypomethylation in promoter regions. Evidence from human and animal studies supports the role of early-life exposure to EDC – such as tributyltin (TBT), bisphenol A (BPA), and dichlorodiphenyltrichloroethane – in programming obesity risk in offspring *via* stable epigenetic inheritance through both maternal and paternal germlines [13].

INTERFERENCE WITH HORMONES REGULATING BODY WEIGHT

Leptin

Appetite and energy balance are regulated by leptin. A hormone secreted predominantly by adipocytes, signaling satiety to the ventromedial hypothalamic nucleus and the lateral hypothalamic nuclei. Moreover, leptin controls appetite and metabolism by suppressing the production and secretion of neuropeptide Y within the arcuate nucleus. In pediatric patients, proper leptin signaling is essential for maintaining energy homeostasis during growth and development. Endocrine-disrupting chemicals can interfere with leptin's regulatory function in multiple ways. Early-life exposure to EDC such as

BPA, phthalates, and perfluoroalkyl substances (PFAS) may disrupt leptin production and receptor sensitivity, leading to leptin resistance. This resistance impairs satiety signaling, promoting hyperphagia and subsequent weight gain. For instance, PCB promotes pre-proinsulin expression by activating aryl hydrocarbon receptor and suppressing the transcription of nuclear factor erythroid 2-related factor 2, while perfluorooctanoic acid markedly elevates the proinsulin-to-insulin ratio. Endocrine-disrupting chemicals-induced inflammation may further alter leptin's intracellular signaling pathways, compounding the effects on energy balance and promoting obesity from the early stages of life [14]. Leptin production may be altered by EDC by modification of genes involved in its production. Exposure to specific phthalates has been associated with increased adipogenesis, potentially elevating leptin production and contributing to leptin resistance. Some EDC can bind to or modify leptin receptors, impairing their normal function. This interference hampers leptin's ability to signal satiety, potentially leading to overeating and weight gain. Endocrine-disrupting chemicals may affect the hypothalamus, leading to impaired appetite regulation and energy homeostasis [15].

Insulin

Insulin is critical for glucose metabolism and is intricately linked to lipid storage and energy regulation. In children, disruption of insulin signaling contributes to insulin resistance and obesity-related metabolic dysfunction. Endocrine-disrupting chemicals influence insulin function by various mechanisms: they can impair β-cell development and insulin secretion, as seen with BPA and TBT, or induce insulin resistance through chronic inflammation and oxidative stress. Several EDC interfere with insulin signaling *via* phosphorylation of insulin receptor substrates, reducing glucose uptake in peripheral tissues. Furthermore, EDC such as dioxins and PCB induce inflammatory pathways like nuclear factor-κB and c-Jun N-terminal kinase, which reinforce insulin resistance and contribute to a prodiabetogenic environment. These disruptions, particularly if occurring in utero or in early life, may predispose pediatric patients to metabolic syndrome, hyperinsulinemia, and increased adiposity [14]. Exposure to certain EDC has been shown to impair β-cell function, leading to inadequate insulin production. For example, BPA exposure has been linked to altered insulin secretion and β-cell apoptosis, contributing to glucose intolerance and increased diabetes risk. Endocrine-disrupting chemicals can promote insulin resistance by interfering with insulin signaling pathways. This disruption necessitates higher insulin levels to maintain glucose homeostasis, increasing the risk of developing type 2 diabetes. Some EDC affect the expression or function of glucose transporter proteins, such as glucose transporter type 4, impairing glucose uptake into cells and contributing to hyperglycemia and insulin resistance [16–18].

Thyroid hormones

Thyroid hormones are essential regulators of basal metabolic rate and thermogenesis. During childhood, they are crucial not only for energy metabolism but also for normal growth and neurodevelopment. Endocrine-disrupting chemicals disrupt thyroid homeostasis at multiple levels of the hypothalamus-pituitary-thyroid (HPT) axis, including thyroid-stimulating hormone (TSH) regulation, T4/T3 synthesis, transport, metabolism, and receptor binding. Perchlorate, nitrates, and thiocyanates inhibit the sodium/iodide symporter, reducing iodide uptake and thyroid hormone synthesis, particularly critical in pregnancy and infancy. Phthalate exposure correlates with decreased serum free and total T4 and increased TSH, suggesting impaired thyroid output, while BPA acts as a weak TR antagonist and alters thyroxine (T4) levels *via* nongenomic pathways. Polychlorinated biphenyls and PBDE displace thyroid hormones from transport proteins and act as hormone modulators, with prenatal exposure linked to neurodevelopmental deficits. These changes can increase hepatic clearance of T4/triiodothyronine (T3), lower circulating hormone availability, and dysregulate basal metabolic rate, predisposing to fat accumulation and obesity. Because of compensatory feedback in the HPT axis, serum TH levels may remain within reference ranges despite significant tissue-level effects, making subtle EDC-induced metabolic disruption a concern for obesity risk [19–23].

EPIGENETIC CHANGES AND LONG-TERM METABOLIC PROGRAMMING

Emerging evidence suggests that exposure to EDC during critical developmental windows can lead to epigenetic modifications that increase the risk of obesity and metabolic disorders. Toxicants such as BPA, phthalates, vinclozolin, methoxychlor, and polycyclic aromatic hydrocarbons (PAH) have been linked to heritable epigenetic changes, particularly through alterations in the germline that persist beyond fertilization. Animal studies have demonstrated that prenatal exposure to EDC can induce DNA methylation changes –particularly in regulatory regions of genes involved in adipogenesis and metabolism – resulting in increased adiposity, altered metabolic profiles, and elevated expression of key adipogenic markers such as PPAR*γ* and *CCAAT/enhancer-binding protein* α. Notably, these epigenetic and phenotypic alterations have been observed not only in the directly exposed offspring but also in subsequent generations, supporting the concept of transgenerational epigenetic inheritance. Human data also associate maternal BPA exposure with altered methylation of the *insulin-like growth factor 2 receptor* gene and higher body mass index (BMI) in female offspring. These effects may involve modulation of DNA methyltransferases and methyl donors like S-adenosyl-L-methionine [24, 25].

IMPACT OF PRENATAL AND EARLY CHILDHOOD EXPOSURE ON OBESITY RISK

Epidemiological evidence indicates that exposure to thyroid-disrupting endocrine chemicals during prenatal and early childhood windows is associated with increased obesity risk in offspring. A 2022 review of human cohort studies /showed that maternal exposure to BPA, phthalates, dioxin-like compounds, and persistent organic pollutants correlates with higher BMI and increased adiposity in children, often detectable from infancy through mid-childhood. Early exposure may alter metabolic programming *via* interference with thyroid hormone synthesis, transport, or receptor binding resulting in subtle reductions in T4 or T3 availability during critical growth periods [26]. For example, reduced thyroid hormone action in fetal life may impair energy expenditure set-points, while elevated TSH or altered deiodinase activity postnatally may shift lipid metabolism toward fat accumulation. These effects appear dose- and sex-dependent, and in many cohorts, exposures in the first and second trimesters are particularly predictive of later increases in BMI or waist circumference. Thus, prenatal thyroid hormone disruption by EDC plausibly contributes to childhood obesity, both directly *via* hormonal effects and *via* downstream modifications of metabolic regulation [27, 28]. A prospective cohort study of mother-child pairs found that higher maternal serum PFAS concentrations were associated with increased child BMI, triceps skinfold thickness, and risk of overweight/obesity at 5 years of age. Notably, non-monotonic dose-response (NMDR) relationships were observed for maternal perfluorooctanesulfonic acid and PCB 153 concentrations and child BMI. These findings align with emerging evidence suggesting that low prenatal PFAS exposure may be linked to increased adiposity in offspring, while higher exposures may exert cytotoxic effects leading to growth restriction. The study highlights the complexity of EDC-related health outcomes and underscores the need for further research into NMDR effects, epigenetic mechanisms, and long-term metabolic consequences of prenatal exposure to persistent organic pollutants [29]. Another study indicates consistent positive associations between prenatal exposure to dichlorodiphenyldichloroethylene and hexachlorobenzene (HCB) and increased childhood BMI, supporting the classification of these compounds as potential chemical obesogens [30, 31]. A recent multicenter European study reported that prenatal exposure to mixtures of persistent organic pollutants – including organochlorine pesticides, PBDE, PFAS, and metals – was associated with higher childhood MetS risk scores, while phthalate mixtures were associated with decreased risk. Notably, specific chemicals such as HCB and perfluorononanoic acid emerged as key contributors within their respective classes, aligning with previous findings from birth cohorts. Moreover, molecular profiling in children revealed that prenatal exposure to these mixtures was associated with upregulation of proinflammatory proteins like C-reactive protein and leptin, and with metabolomic changes indicative of disrupted lipid and amino acid metabolism – both consistent with increased MetS and obesity risk. Importantly, sex-specific associations were observed, with females showing greater susceptibility to PFAS and PCB exposure, underscoring the need for stratified analyses in future research [31, 32]. Other prenatal exposures, such as cigarette smoke, PAH, TBT, and BPA, have also been linked to obesity through mechanisms like hormone disruption and altered stem cell differentiation [32].

HEALTH AND SOCIAL CONSEQUENCES OF PEDIATRIC OBESITY RELATED TO ENDOCRINE DISRUPTORS

Endocrine-disrupting chemicals have been increasingly recognized as significant contributors to the obesity epidemic, particularly in children. These substances interfere with hormonal homeostasis, leading to metabolic dysregulation and increasing the risk of obesity-related diseases [33].

IMPACT ON PEDIATRIC HEALTH

Numerous epidemiological studies have demonstrated a correlation between early-life exposure to EDC and an elevated risk of metabolic disorders such as obesity, type 2 diabetes, and cardiovascular disease [34]. Mechanistically, EDC affect adipogenesis, lipid metabolism, and glucose homeostasis, often through interactions with nuclear receptors such as PPARγ and estrogen receptors [35].

For instance, BPA and phthalates have been found to induce insulin resistance and pancreatic β-cell dysfunction, both of which are hallmarks of type 2 diabetes [14]. Longitudinal cohort studies have further suggested that prenatal and early postnatal exposure to EDC significantly increases the likelihood of developing childhood obesity and MetS [36].

PSYCHOLOGICAL AND SOCIAL CONSEQUENCES

Childhood obesity, irrespective of its etiology, is associated with a range of psychological challenges, including low self-esteem, depression, and social stigmatization [37]. Given the increasing evidence linking EDC to obesity, children exposed to these chemicals may not only suffer from metabolic complications but also from the societal repercussions of obesity.

Furthermore, obesity-related discrimination can have profound effects on a child's mental health, academic performance, and social relationships. Studies indicate that obese children are more likely to experience bullying, which can lead to social withdrawal and increased susceptibility to psychiatric disorders [38].

ECONOMIC BURDEN OF PEDIATRIC OBESITY

The economic costs associated with obesity, particularly when influenced by environmental factors such as EDC exposure, are substantial. A European study estimated that EDC-related obesity and diabetes contribute to healthcare expenditures exceeding €10 billion annually [36].

Indirect costs, such as decreased workforce productivity, higher absenteeism rates, and increased disability claims, significantly contribute to the economic burden on global healthcare systems. Research on major depressive disorder highlights that it leads to substantial indirect costs, including unemployment, absenteeism, and disability, extending beyond direct healthcare expenses [39]. Similarly, studies on obesity indicate that rising rates of the condition result in increased public expenditure, placing greater pressure on national healthcare budgets and affecting workforce productivity [40].

Given these economic implications, addressing EDC exposure as a public health concern is imperative for mitigating the long-term financial and societal impact of obesity.

PREVENTION STRATEGIES AND REGULATORY FRAMEWORKS FOR ENDOCRINE DISRUPTORS

Given the significant health risks associated with EDC exposure, regulatory efforts, and preventive measures are essential to mitigate their impact on pediatric obesity. Both individual-level strategies and policy interventions are crucial in addressing this public health challenge.

STRATEGIES TO REDUCE EXPOSURE TO ENDOCRINE DISRUPTORS

Preventing EDC exposure requires a multi-faceted approach that includes dietary modifications, lifestyle changes, and consumer awareness. Strategies such as reducing plastic use, avoiding processed foods containing high levels of chemical additives, and choosing BPA-free products are recommended to limit direct exposure to obesogens [41]. Educational campaigns targeting parents and caregivers can further enhance awareness regarding the potential sources of EDC and encourage informed decision-making regarding food packaging, household products, and personal care items [42]. Promoting organic diets is considered an effective method for reducing exposure to pesticide residues, which are known to have endocrine-disrupting properties.

According to a review article published in Frontiers in Public Health pod numerem 43 jest inny tytuł, organic food production is strictly regulated, leading to lower use of synthetic pesticides and, consequently, reduced exposure to their residues [43].

REGULATORY APPROACHES IN THE EUROPEAN UNION AND WORLDWIDE

Regulatory approaches in the EU to address EDC focus on assessing and managing the risks associated with chemical exposures. The European Union's REACH (Registration, Evaluation, Authorization, and Restriction of Chemicals) regulation plays a critical role in this effort. REACH aims to ensure that chemicals used in consumer products are safe for human health and the environment by evaluating their risks and restricting the use of harmful substances. This regulatory framework is essential in mitigating the impact of EDC, particularly those that pose long-term health risks, such as endocrine disruption.

In addition to REACH, the EU has implemented several other regulations to monitor and control the use of chemicals that can affect human health, emphasizing the need for evidence-based strategies to protect public well-being [44]. The European Union has developed a comprehensive approach to addressing EDC, with the establishment of clear scientific criteria for their identification and regulation. Specifically, the EU's Endocrine Disruptor Strategy includes guidelines for identifying EDC in pesticides, biocides, and other chemicals used in consumer products. These criteria aim to ensure that harmful substances are systematically evaluated and restricted, with a focus on protecting public health and the environment. The European Union's regulatory framework also emphasizes the need for evidence-based decision-making to reduce exposure to EDC in food, cosmetics, and industrial chemicals [44, 45].

Internationally, organizations such as the World Health Organization (WHO) and the United Nations Environment Programme (UNEP) advocate for stricter controls on EDC. In their joint report, WHO and UNEP highlight the need for globally harmonized regulations and increased research funding to better understand the long-term effects of these chemicals. Despite these efforts, regulatory gaps persist, particularly in non-EU countries where policies regarding EDC exposure remain less stringent. The disparity in global regulations underscores the necessity for international collaboration and unified standards to ensure comprehensive protection against endocrine disruptors [44].

FUTURE DIRECTIONS AND POLICY RECOMMENDATIONS

Moving forward, stricter regulatory measures, improved biomonitoring systems, and increased public education initiatives are essential to mitigating the impact of EDC on childhood obesity. Policymakers must prioritize research funding for longitudinal studies that assess the long-term consequences of early-life EDC exposure [46].

Additionally, the development of safer chemical alternatives and the implementation of "green chemistry" principles can contribute to reducing environmental and human exposure to harmful endocrine disruptors [47].

CONCLUSIONS

Childhood obesity continues to be a major global health issue, impacting various socioeconomic groups and leading to serious physical, mental, and financial consequences. Endocrine-disrupting chemicals play a crucial role in the rising number of pediatric obesity incidents by interfering with key hormonal pathways involved in adipogenesis, lipid metabolism, insulin regulation, and overall energy homeostasis. Endocrine-disrupting chemicals exposure during critical developmental windows – especially prenatal and early childhood – further increases susceptibility to obesity through mechanisms such as hormonal dysregulation and transgenerational epigenetic changes. Various comprehensive strategies aimed at reducing EDC exposure – such as dietary modifications, consumer education, and regulatory measures – are essential to address the public health impacts of pediatric obesity. Enhanced international collaboration, stringent regulatory frameworks, improved biomonitoring, and continued research into safer chemical alternatives represent necessary and effective responses to mitigate the adverse health effects of endocrine disruptors.

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