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Bisphenol an Exposure and Its Multifaceted Effects on Child Health: A Comprehensive Review

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ABSTRACT:

Bisphenol A (BPA) is a widely used chemical in the plastic industry. Owing to its toxicity, several concerns have been raised over the years. BPA is an endocrine disruptor. Early life exposure to BPA may be higher than adult exposure due to differences in physiology, diet, and behavior. Therefore, there is considerable cause for concern over the detrimental effects of exposure to these substances throughout pregnancy and the early years of life. The significant and intricate impacts of BPA exposure on children's health are highlighted in this review. Being an endocrine disruptor, BPA affects thyroid function, immunological responses, metabolic health, prenatal growth, and neurodevelopment, among other essential physiological processes. Exposure to BPA has been associated with a number of adverse consequences, including low birth weight, obesity, behavioral and cognitive problems, and asthma, this highlights the potential for long-term health complications. To reduce these hazards, stricter laws governing the use of BPA, greater public knowledge, and improvements in biomonitoring methods are crucial.

1. Introduction

Bisphenol A (BPA) is used mainly as a monomer for the synthesis of polymeric materials, mostly polycarbonate plastic and epoxy resins, which additionally serve as raw materials for various products, such as flame retardants [1]. Polycarbonates can be found in food contact products such as storage containers, tableware, infant feeding bottles, and reusable bottles, whereas epoxy resins can be found in protective linings of containers and vats for food and beverages [2].

BPA, which was first created in 1891, has been known to have endocrine-disrupting effects since 1936 [2], with anti-thyroid, anti-androgenic, estrogenic, and anti-estrogenic properties [3]. Additionally, exposure to BPA has been linked to multiple adverse effects, including cardiovascular disease, insulin resistance, obesity, neurobehavioral disorders, and reproductive disorders [4]. Food is the primary source of BPA exposure since it becomes contaminated when BPA migrates from packaging during processing or storage [2], inhalation, or dermal contact [3]. Following exposure through food, BPA is absorbed by the gastrointestinal tract and moves to the liver, where it is conjugated primarily into BPA-glucuronide and partly into BPA-sulphate, after which more than 90% is eliminated via the kidney and colon

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within six hours of ingestion [4]. Furthermore, dust particles in indoor areas and personal grooming products such as lotions, shampoos, and cleaning products can also expose individuals to BPA [5].

BPA exposure can be greater in early life than in adulthood since there are differences in physiology, diet, and behavior. As a result, there is a greater level of concern about the adverse effects of exposure to these substances during fetal life and early childhood [6]. Crucially, research has shown that hepatic glucuronidation rates differ between fetuses and adults, which indicates that children and adolescents have higher concentrations of BPA than adults do. According to the US National Toxicology Programme, the lowest observed adverse effect level (LOAEL) in standard toxicological tests is 50,000 µg/kg/day BPA [4]. Additionally, BPA was detected in nearly all urine samples from the "German Environmental Survey for Children and Adolescents" conducted between 2014 and 2017 [7]. Hence, this review aims to provide an overview of the current literature on the effects of BPA on child health.

2. Methods

Two reviewers individually searched for full-length papers in PubMed, Embase and Web of Science published between 2019 and 2024 that assessed the effects of bisphenol A on child health. The search included only clinical studies. The search terms used were bisphenol A, BPA, effects of BPA, effects of bisphenol A, child, children, and pediatrics.

3. Discussion

1. Impact on Fetal Growth and Development

BPA is a widely used chemical in the plastic industry. Owing to its toxicity, several concerns have been raised over the years. BPA is an endocrine disruptor. It mimics estrogen and stimulates cellular responses at lower doses. At higher doses, BPA binds to estrogen receptors [8]. Bisphenol F (BPF), bisphenol S (BPS), and bisphenol AF (BPAF) are some bisphenol analogs that are being used instead of BPA. However, owing to their structural similarities, bisphenol analogs carry the same potential toxicity as BPA. BPA exposure during pregnancy raises the risk of low birth weight, premature labor, and intrauterine growth restriction [9-11]. BPA analogs can also have harmful effects on fetal growth outcomes when exposed during pregnancy. This could possibly be due to disruption in placental development and function [12]. Multiple studies have investigated the relationships between fetal growth and development and prenatal exposure to BPA and its replacements, BPF and BPS. In utero exposure to these chemicals was associated with reduced length of the babies, low birth weight and a low ponderal index. One of the studies concluded that female babies were more vulnerable to bisphenol B exposure [13]. However, in another study, increasing BPF levels in mothers were associated with low birth weights and decreased ponderal indices in male babies [14]. The toxic effects of bisphenols on fetal growth vary with different periods of gestation. In a study conducted by Hu et al., during the first and second trimesters, higher urinary concentrations of BPF and BPS were noted in the mothers. This was significantly associated with decreased length and weight at birth, and the ponderal index. However, prenatal BPA exposure in this study showed null associations. This suggests that BPA analogs are more toxic and potent for fetal growth restriction [15]. In another study, maternal urine levels of BPA, but not its substitutes (BPF or BPS), were linked to notable alterations in head circumference at birth [16].

2. Epigenetic Changes and Low Birth Weight

Most researched epigenetic marker is DNA methylation. It is a naturally occurring modification in the early phases of fetal development and embryogenesis. It is crucial during X chromosome inactivation and genomic imprinting [17, 18]. Huang et al. Huang et al. carried out one of the first studies linking utero BPA exposure and low birth weight with an epigenetic marker. The association between BPA exposure in the second trimester and low birth weight was significant. Also, a significant association was between DHRS9 methylation in cord blood and low birth weight [19]. Prenatal exposure to BPA, particularly in the second trimester, and low birth weight have also been noted in a few other studies [20, 21]. Nahar et al. reported a positive association between BPA levels and hypermethylation in 2nd trimester placental tissue [22].

3. Disruption of Thyroid Hormone Regulation

Children are more vulnerable to harmful environmental contaminants than adults. The exposure of children to BPA is a growing concern. One of the main ways to be exposed to BPAs is through diet, particularly through

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seafood consumption [23, 24]. BPAs are known to disrupt thyroid hormone functions. Thyroid hormones are vital regulators of growth and brain development in children. Several studies have concluded that the underlying mechanisms by which BPAs interfere with thyroid hormone functions involve decreasing thyroid gland volume, obstructing the uptake of iodide in thyroid cells, and functioning as antagonists to impair thyroid function [25-28]. Disruptions in thyroid hormone levels, especially during early life, can lead to cognitive deficits, delayed growth, and metabolic irregularities. In a study conducted by C. Guo et al., a higher serum BPA concentration was noted in hyperthyroid children than in euthyroid and nonhyperthyroid thyroid dysfunction patients. In euthyroid children, a significant inverse correlation between TSH and BPA was reported. However, a significant positive correlation in hyperthyroid children was noted [1]. Hyperthyroidism is treated by blockade of the thyroid gland and BPA works as an antagonist to disrupt thyroid function [28, 29]. Thus, BPA can be considered an endocrine-disrupting chemical that acts on the hypothalamic–pituitary–thyroid axis.

4. Childhood Obesity

BPA exposure can have adverse effects on the body including insulin resistance and obesity. BPA exposure is a concern during the early developmental period. It can disturb the normal hormonal regulation and can have long-lasting adverse effects in children. Childhood obesity is an emerging public health issue. Very few studies on BPA exposure leading to childhood obesity are available. BPA can mimic estrogen and thus it disturbs the normal hormonal balance. It interferes with insulin signaling, stimulates the preadipocytes differentiation, alters the microbiome of gut, induces inflammation, and affects energy metabolism [30-33]. Recent studies have suggested that exposure to BPA may predispose childhood obesity, possibly by triggering epigenetic modifications of genes involved in metabolism and energy balance [34]. Guo et al. investigated the connections between children's body composition metrics and BPA exposure. High BPA levels in young children were linked to higher levels of obesity-related body composition metrics, including BMI, BFM, FFM, and PBF [5]. A study conducted in Spain including peripubertal boys concluded that higher the BPA exposure, higher the BMI z scores, greater the risk of overweight and or obesity, higher waist–height ratios, and a greater cardiometabolic risk but not a higher body fat mass percentage [35].

5. Long-term cardiometabolic risks

Adolescence is a critical period during which several hormonal changes take place. Thus, the action of endocrine disruptors such as BPA during this period can be very significant. Studies have shown that soft drinks are one of the main contributors to BPA in adolescent groups [35, 36]. V. Magalhães et al. conducted research on BPA exposure among adolescents. They found a substantial correlation between the cardiometabolic risk pattern and BPA exposure. Adolescents who were exposed to more BPA had higher blood triglyceride and insulin levels, a higher BMI, and an increase in body fat and waist circumference [2]. In children, exposure to bisphenols leads to oxidative stress, insulin resistance, and endothelial dysfunction [37]. Studies conducted in Spain have shown that girls with increased dietary exposure to BPA are overweight and/or obese and have greater cardiometabolic risk than girls with a low BMI. This could be due to higher energy consumption and processed food by overweight girls [38]. Another study in Spain concluded that prenatal exposure to phenol metabolites along with phthalates had weak association with cardiometabolic health in children. A study involving Chinese children revealed higher blood pressure in those girls whose mothers had higher prenatal levels of BPA. Additionally, boys whose mothers had medium levels of prenatal BPA exposure had higher serum glucose levels [39].

6. Asthma and Allergic Diseases

Over the years, an increasing trend in asthma and other allergic disorders in children has been reported. This coincides with the increasing use of harmful chemicals. BPA, an endocrine-disrupting chemical, is associated with an increased risk of asthma and other allergic diseases in children. BPA influences gene expression by triggering a number of epigenetic processes, including DNA methylation, histone changes, and noncoding

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Table 1: Summary of the studies showing impact of bisphenol A on child health

Study	Year	Place	Type of study	Sample size	Parameters used to study BPA	Conclusion
Yang et al.	2020	Taiwan	Cross-sectional	228	Urine, serum	High BPA exposure resulted in decreased methylation of MAPK1 gene which is associated with higher risk of developing asthma in children.
Hansen et al.	2021	Odense, Denmark	Cohort	1085	Urine	Prenatal BPA exposure may raise the risk of ASD symptoms, which may be a predictor of later social abilities.
Z Huang et al.	2021	Anhui Province, China	Cohort	1783	Serum	Demonstrated the effects of prenatal BPA exposure on pre-schooler's cognitive and behavioral development, both sex and age specific.
YF Huang et al.	2021	Taiwan	Longitudinal	162	Urine, serum	Prenatal BPA exposure and hypermethylation of DHRS9 gene in cord blood is associated with low birth weight.
Guo et al.	2021	Dalian, China	Case-control	345	Serum	In the hyperthyroid group, the BPA concentration was found to be higher, and a significantly positive correlation was noted between TSH and BPA.
Guo et al.	2023	Guangzhou, China	Cross-sectional	200	Urine	The body composition parameters in children and BPA exposure showed a positive correlation, which highlights the possible risks connected to obesity in young children.
Eatman et al.	2023	Atlanta, Georgia	Cohort	247	Urine	Increased prenatal exposure to phthalates and BPA was associated with low birth weight.
Magalhaes et al.	2024	Porto, Portugal	Cross sectional	2386	Urine	Adolescents with higher levels of BPA exposure had higher cardiometabolic risk pattern.

RNAs [40-42]. BPA mediates alterations in DNA methylation which triggers macrophages in the airway to express MAPK1. This leads to activation of inflammatory responses. MAPK signaling pathways are known to be involved in inflammation of the airways and regulation of immune cells, which are the classical features of asthma [43]. Yang et al. reported that BPA exposure leads to the asthma in children. Alterations in DNA methylation, particularly MAPK1 5'CGI methylation acts as an epigenetic biomarker for the development of asthma in children [44]. Wang, I.J. et al. reported high IgE levels in children exposed to BPA [45]. Donohue et al. reported an association between BPA and childhood asthma [46]. Prenatal and postnatal exposure to BPA reportedly increases the risk of developing asthma and other allergic disorders in children [47].

7. Neurodevelopmental and Behavioral Disorders

Neurodevelopment is a crucial process and begins in the early embryonic stage. This can be easily affected by

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various factors, including exposure to environmental chemicals. Any disruption in cell proliferation, neural migration and differentiation can have adverse effects on the developing fetal brain. This results in neurobehavioral disorders in children. BPA is an endocrine disrupter and possesses estrogenic, antiestrogenic, antiandrogenic and antithyroid properties [48]. Exposure to BPA disturbs the thyroid as well as estrogenic pathways. BPA alters brain-derived neurotrophic factor (BDNF) levels. As a result, BPA exposure can lead to abnormal neurodevelopment [49]. It is observed that blood BDNF levels are elevated in children with ASD [50]. Prenatal BPA exposure affects the white matter microstructure development. This can induce behavioral problems in children. One of the studies reported an association between delayed language development and prenatal BPA exposure [51]. Hansen et al. reported that low-concentration prenatal BPA exposure can increase the risk of ASD symptoms in children [3]. ASD is more frequently reported in boys than in girls. A Canadian study on BPA exposure suggested a significant association between prenatal BPA exposure and poor reciprocal social behaviors in children, most commonly in boys [52]. However, Hansen et al. reported stronger associations in girls. Huang et al reported that higher prenatal BPA exposure can increase the incidence of ADHD among children. Their results on impact of prenatal BPA exposure on neurodevelopment was age and sex specific. Increased risks of peer relationship problems, poor ability to regulate emotions and poor control on behavioral responses were reported more among girls. However, both sexes had equal risk of developing conduct problems following high prenatal BPA exposure [53].

4. Conclusion

The reviewed research highlights the substantial and complex effects of exposure to BPA on the health of children. As an endocrine disruptor, BPA interferes with vital physiological functions, affecting immunological responses, thyroid function, metabolic health, fetal growth, and neurodevelopment. Exposure to BPA has been associated with a number of adverse consequences, including low birth weight, obesity, behavioral and cognitive problems, and asthma, this highlights the potential for long-term health complications. Recent findings also imply that BPA substitutes such as BPS and BPF can have similar, if not more pronounced, toxicological consequences.

Children are particularly vulnerable because of their slower metabolic detoxification and developmental sensitivity, which emphasizes the need for focused interventions. To reduce these hazards, stricter laws governing the use of BPA, greater public knowledge, and improvements in biomonitoring methods are crucial. Long-term studies should be given priority in future research in order to comprehend cumulative exposures and the possible epigenetic consequences of BPA and its analogues. Public health initiatives can more effectively protect children's development and health by filling in these gaps.

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