

OLEJNIK-CHLEWICKA, Klaudia, PUCHALSKI, Konrad, LISZKA, Paweł, PEREDIATKIEWICZ, Jakub, ZASIADŁA, Marta, PATRZYKAT, Klaudia Martyna, URBAŃSKI, Wojciech, LUCZAK, Paweł Mateusz, BRODOWSKI, Jakub and OGÓREK, Agata. Prenatal and Early-Life Exposure to Parabens: Sources, Reduction Strategies, and Developmental Health Effects - A literature review. Quality in Sport. 2026;50:68042. eISSN 2450-3118.

<https://doi.org/10.12775/QS.2026.50.68042>

<https://apcz.umk.pl/QS/article/view/68042>

The journal has been awarded 20 points in the parametric evaluation by the Ministry of Higher Education and Science of Poland. This is according to the Annex to the announcement of the Minister of Higher Education and Science dated 05.01.2024, No. 32553. The journal has a Unique Identifier: 201398. Scientific disciplines assigned: Economics and Finance (Field of Social Sciences); Management and Quality Sciences (Field of Social Sciences).

Punkty Ministerialne z 2019 - aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych). © The Authors 2026.

This article is published with open access under the License Open Journal Systems of Nicolaus Copernicus University in Toruń, Poland. Open Access: This article is distributed under the terms of the Creative Commons Attribution Noncommercial License, which permits any noncommercial use, distribution, and reproduction in any medium, provided the original author(s) and source are credited. This is an open access article licensed under the terms of the Creative Commons Attribution Non-commercial Share Alike License (<http://creativecommons.org/licenses/by-nc-sa/4.0/>), which permits unrestricted, non-commercial use, distribution, and reproduction in any medium, provided the work is properly cited.

The authors declare that there is no conflict of interest regarding the publication of this paper.

Received: 07.01.2026. Revised: 24.01.2026. Accepted: 24.01.2026. Published: 30.01.2026.

## **Prenatal and Early-Life Exposure to Parabens: Sources, Reduction Strategies, and Developmental Health Effects- A literature review**

Authors:

Klaudia Olejnik-Chlewicka [KO-C], ORCID <https://orcid.org/0009-0005-9360-3752>

[olejnikklaudia98@gmail.com](mailto:olejnikklaudia98@gmail.com)

Provincial Integrated Hospital in Kielce, Grunwaldzka 45, 25-736 Kielce, Poland

Konrad Puchalski[KP], ORCID <https://orcid.org/0009-0002-0452-4904>

[konodor42@gmail.com](mailto:konodor42@gmail.com)

Voivodeship Specialist Hospital in Wrocław, Kamińskiego 73a, 51-124 Wrocław, Poland

Paweł Liszka[PL], ORCID <https://orcid.org/0009-0003-5465-3656>

[liszkapawel99@gmail.com](mailto:liszkapawel99@gmail.com)

Jan Mikulicz-Radecki University Clinical Hospital, Borowska 213, 50-556 Wrocław, Poland

Jakub Perediatkiewicz[JP], ORCID <https://orcid.org/0009-0006-7727-3199>

[jakub.perediatkiewicz@gmail.com](mailto:jakub.perediatkiewicz@gmail.com)

New Hospital in Olkusz, Aleja Tysiąclecia 13, 32-300 Olkusz, Poland

Marta Zasiadła[MZ], ORCID <https://orcid.org/0009-0007-0171-926X>

[martazasiadla@gmail.com](mailto:martazasiadla@gmail.com)

Tadeusz Sokołowski University Clinical Hospital No. 1 PUM in Szczecin, Unii Lubelskiej Street 1, 71-252 Szczecin, Poland

Klaudia Martyna Patrzykat[K-MP], ORCID <https://orcid.org/0009-0000-9440-5444>

[patrzykat.klaudia@gmail.com](mailto:patrzykat.klaudia@gmail.com)

109 Military Hospital with Policlinic in Szczecin, Piotra Skargi 9-11, 70-965 Szczecin, Poland

Wojciech Urbański[WU], ORCID <https://orcid.org/0009-0008-6559-7510>  
[wojciech.urbanski04@gmail.com](mailto:wojciech.urbanski04@gmail.com)

Jan Mikulicz-Radecki University Clinical Hospital, Borowska 213, 50-556 Wrocław, Poland

Paweł Mateusz Łuczak[PŁ], ORCID <https://orcid.org/0009-0002-9119-8499>  
[pawelluczak.mail@gmail.com](mailto:pawelluczak.mail@gmail.com)

Independent Public Healthcare Complex- Hospital in Iłża, Bodzentyńska 17, 27-100 Iłża, Poland

Jakub Brodowski[JB], ORCID <https://orcid.org/0009-0001-5911-4841>  
[lek.jakubbrodowski@gmail.com](mailto:lek.jakubbrodowski@gmail.com)

Jan Mikulicz-Radecki University Clinical Hospital, Borowska 213, 50-556 Wrocław, Poland

Agata Ogórek[AO], ORCID <https://orcid.org/0009-0000-2916-5368>  
[agata.ogorek@dr.com](mailto:agata.ogorek@dr.com)

Lower Silesian Center of Oncology, Pulmonology and Hematology, Wrocław, pl. Hirszfelda 12, 53-413 Wrocław, Poland

Corresponding Author

Klaudia Olejnik-Chlewicka; [olejnikklaudia98@gmail.com](mailto:olejnikklaudia98@gmail.com)

## **Abstract:**

**Informations and purpose:** Parabens are widely used preservatives found in cosmetics, pharmaceuticals, foods and household products. Increasing evidence shows that women of reproductive age, including pregnant women, are consistently exposed to these endocrine-disrupting chemicals, which may influence fetal and early-life development. Understanding exposure levels and potential health consequences is important for both public health and clinical practice.

**Methods:** A systematic literature search was performed to identify studies evaluating paraben exposure during pregnancy and its effects on fetal and early childhood health. The literature search included studies published from 2019-2024 and contained searches of PubMed, Scopus, Science Direct and open access databases.

**Results:** Parabens are widely detected in maternal and fetal matrices. Exposure to parabens in utero has been shown to result in changes to fetal size, metabolic and hormone-related factors and an increased risk of developing atopic and respiratory diseases in early childhood. Reported mechanisms involve endocrine disruption (specifically acting like estrogens and anti-androgens), oxidative stress, immune modulation and possible epigenetic effects.

**Conclusion:** Current evidence indicates that parabens may influence child development through several biological pathways, with effects observable from fetal life into early childhood. Although causality remains to be fully established, reducing paraben exposure in pregnancy and early life—particularly from cosmetic and dermal sources—appears advisable. Public health measures promoting informed consumer choices may further mitigate exposure.

**Keywords:** Parabens; Pregnancy; Fetal development; Early childhood; Endocrine disruptors

## 1.Introduction

Parabens are a group of organic chemical compounds widely used in consumer products. Chemically, they are esters of p-hydroxybenzoic acid (PHBA), differing in the structure of their functional side chains. The differences in length of the alkyl chain affect their biological and toxicological characteristics. Parabens have shown to be fungistatic and antibacterial; however, the bacteriostatic effect is greater against Gram-positive compared to Gram-negative bacteria. Their bacteriostatic efficacy increases when using longer alkyl chains. Therefore, it is common practice to utilize combinations of several parabens, such as methyl-, ethyl-, propyl-, and butylparaben, in order to create a wide-spectrum preservation mechanism for consumer goods.

Recently, there has been increasing concern about the safety of parabens, especially among pregnant women. There is a large amount of evidence suggesting that parabens function as endocrine disrupting chemicals (EDCs); display estrogenic and anti-androgenic characteristics; and are able to alter gene expression through epigenetic mechanisms [18]. According to Health Canada's toxicological evaluation, parabens are able to alter hormone balance through either direct receptor interaction or through generating oxidative stress, inflammation, and altering lipid and glucose metabolic pathways [11].

According to biomonitoring results from the European HBM4EU project, parabens are found in the biological samples of nearly all pregnant women [12]. Similar findings have been reported by researchers investigating women intending to become pregnant or undergoing fertility treatments. Biomarkers of parabens were identified in virtually all biological samples collected from these women, highlighting the significance of exposure during the preconception period [16]. Moreover, many of these studies have noted that pregnant women are often exposed to multiple classes of chemical compounds, including parabens, phthalates and phenols, which may interact synergistically or additively to enhance the risk of adverse effects associated with prenatal exposures [24].

Prenatal exposure raises particular concern because pregnancy represents one of the most sensitive developmental windows. Both the World Health Organization (WHO) and the United Nations Environment Program (UNEP) characterize EDCs as a major public health problem and emphasize that the disruption of the hormonal environment during fetal development could have serious long-lasting health implications for the offspring. It is essential to evaluate the magnitude and frequency of paraben exposure in pregnant women. It is also important to understand how parabens work and what short- and long-term health risks they may pose to the developing fetus. Finally, effective clinical and preventative strategies must be developed to reduce paraben exposure during pregnancy.

### Objective of the Study

This study aims to provide a comprehensive overview of the available literature concerning paraben exposure in pregnant women. It further seeks to detail the mechanisms through which these chemicals are thought to exert their effects and to discuss potential long-term health impacts of paraben exposure at different stages of the child's development. Finally, this study will discuss practical measures to minimize exposure to parabens, relevant to public health and environmental protection.

## 2.Methods

A narrative literature review was conducted to summarize current evidence on maternal exposure to parabens, mechanisms of action and potential health effects across fetal, neonatal and early childhood stages. For the purposes of this review, the literature searches will be limited to peer-reviewed publications from 2019 – 2024. A single older (2002) toxicology study is being included to assist in understanding how the endocrine pathways may be impacted as a result of paraben exposure. Literature searches were carried out in PubMed, PubMed Central, ScienceDirect and Scopus, complemented by official reports from Health Canada and the HBM4EU initiative. Search terms included combinations of “*parabens*,” “*pregnancy*,” “*prenatal exposure*,” “*biomonitoring*,” “*placental transfer*,” “*neonatal health*,” and “*child development*.”

The initial search identified 482 records. After removing duplicates, 326 publications were screened by title and abstract, leading to the exclusion of studies unrelated to pregnancy, pediatric populations or biomarker-based exposure assessment. Full texts of 78 articles were reviewed in detail, evaluating methodological quality, exposure assessment (urine, serum, placenta, cord blood), timing of developmental windows and adjustment for confounders.

Studies lacking sufficient methodological rigor or not reporting paraben-specific outcomes were excluded. Ultimately, 25 publications were included: cohort and cross-sectional studies, systematic reviews, mechanistic analyses, multi-cohort mixture studies and regulatory assessments.

Data extraction focused on exposure levels, biological matrices, critical windows of susceptibility, placental transfer, endocrine and metabolic endpoints, and health outcomes in fetuses, neonates and children. Due to heterogeneity in study design, exposure metrics and reported outcomes, a qualitative synthesis was applied, emphasizing consistency of findings, biological plausibility and relevance to clinical and public health practice.

### **3.Results**

#### **3.1 Exposure sources and biomonitoring evidences**

Parabens continue to be one of the most common active ingredients in consumer products as a result of their high antimicrobial activity, chemical stability, and relatively low cost of production. Due to their use in all types of consumer products, the exposure levels of parabens have been shown to be present ubiquitously in the environment of women of childbearing potential and pregnant women [11,12,23]. Parabens may enter the body through multiple exposure pathways—including dermal absorption, ingestion, and inhalation—further compounded by the fact that individuals are typically exposed to these chemicals from several concurrent sources [23].

Cosmetics constitute the dominant source of paraben exposure, contributing an estimated 85–95% of total intake [11,17]. Parabens can be found in a variety of cosmetic products as either a preservative or a fragrance ingredient, including—but not limited to—moisturizers, makeup, toothpaste, shampoo, and hair dye. According to the RIVM [17], the highest levels of exposure were detected in women who used multiple leave-on cosmetic products. Therefore, it is evident that the majority of paraben exposure occurs via dermal absorption.

Dietary intake is the second most important exposure pathway. Parabens are commonly used in the food industry for preservation purposes as additives (E214–E219), extending the shelf-life of a variety of products. These include, but are not limited to, marinades, processed vegetable and sauce products, long-lasting baked goods, and candy/confections. According to Health Canada, in addition to their direct use as food additives, parabens may also enter food

indirectly through migration from packaging materials, including paper and cardboard containers, jar linings, and bottle cap sealants [11].

Parabens are also present in pharmaceutical products, particularly in topical formulations such as creams, ointments, and liquid preparations. Iacobelli et al. [6] demonstrated that medications can be a significant source of internal exposure, especially in neonatal intensive care units, where several paraben-containing preparations—such as anti-reflux syrups, laxatives, oral vitamin solutions, electrolyte products, and antibiotic suspensions—were identified.

Environmental exposure is an often underestimated contributor to overall paraben intake. Parabens found in indoor dust and household surfaces can lead to inhalation exposures. Toxicity assessments show that parabens are highly resistant to degradation in the environment, making them persistent in drinking water and other sources, thereby exposing people during daily routines such as bathing and handwashing [11]. Occupational exposure also exists health care workers may be exposed to disinfectants containing parabens for extended periods [11]. Concerns about infants and young children have also been raised the RIVM report [17] identified parabens in a number of child-care products, specifically lotions, oils, barrier creams, and most importantly wet wipes, which constitute a major source of exposure due to frequent application on sensitive skin areas. According to the report, infants receive proportionally higher internal doses of parabens because their skin barrier is not yet fully developed and their body surface area relative to body weight is considerably larger than in adults [17]. Popa et al. [18] also point out that small amounts of parabens may be present in products labeled as “paraben-free” due to contamination during production or migration from packaging.

### **3.2 Mechanism of action of parabens**

#### **3.2.1 Endocrine disruption mechanisms (combined estrogenic, anti-androgenic, HPT/HPA effects)**

Studies indicate that some types of parabens (especially those with longer carbon chains) may interfere with the hypothalamus–pituitary–gonad (HPG) axis as well as the hypothalamus–pituitary–thyroid (HPT) axis. Examples of interference with the HPG axis include a number of epidemiological studies. Jurewicz et al. [16] indicated that greater exposure to propylparaben was found to be related to fewer positive indicators for ovarian reserve, such as a lower antral follicle count and changes in hormone profile, providing indirect support for potential effects

on hormonal regulation of reproduction. Regulatory assessments conducted by Health Canada and the HBM4EU Initiative further provided evidence for the potential ability of parabens to modify thyroid hormone metabolism [11,12]. Laboratory studies of rats and mice administered propylparaben and butylparaben demonstrated inhibition of the enzyme deiodinase responsible for converting thyroxine (T4) into its active form, triiodothyronine (T3) [25]. Therefore, even when serum T4 concentrations fall within the reference range, there may be reduced tissue availability of T3. Additionally, Boberg et al. determined that parabens could also reduce the amount of thyroid hormone-binding proteins (such as TBG and albumin), which could lead to a disrupted equilibrium between free and bound hormone fractions [25].

Parabens belong to a group of compounds with weak, yet biologically relevant estrogenic activity. According to regulatory assessments by Health Canada and the HBM4EU initiative, these substances are capable of binding to estrogen receptors ER $\alpha$  and ER $\beta$  [11,12]. Animal studies provide further evidence supporting the estrogenic properties of parabens. Boberg et al. [25] demonstrated that administration of propylparaben and butylparaben to rats resulted in increased uterine weight and histological changes characteristic of estrogenic stimulation, with stronger effects observed for parabens with longer alkyl chains, indicating a structure-activity relationship.

Parabens have also been shown to act as anti-androgens based on numerous laboratory experiments. Boberg et al. [25] found that when male rats were exposed to propylparaben and butylparaben, their testosterone levels were lower than expected, and the function of Leydig cells was disrupted. The researchers concluded that, in addition to disrupting the androgen signaling pathway via a receptor-mediated mechanism, parabens could reduce the production of androgens by interfering with enzymatic processes necessary for their synthesis (i.e., 3 $\beta$ -HSD and 17 $\beta$ -HSD), providing a potential explanation for their anti-androgenic effects.

### **3.2.2 Indirect mechanisms: oxidative stress, inflammation, epigenetic regulation**

Research indicates that parabens could possibly affect biological processes indirectly via a variety of mechanisms, specifically epigenetic changes, oxidative stress, and inflammatory responses [12,18,11,10].

#### **Epigenetic modifications.**

Reviews conducted by the HBM4EU consortium [12] and Popa et al. [18] suggest that parabens are capable of altering gene expression through disruption of DNA methylation and



modification of chromatin-modifying proteins. In particular, it has been reported that propylparaben and butylparaben can modulate development-related genes, genes involved in hormone regulation, and genes involved in lipid metabolism [18].

### **Oxidative stress.**

Health Canada's toxicology evaluation indicates that parabens—primarily propylparaben and butylparaben—can disrupt the cellular redox state by interfering with mitochondrial function and reducing antioxidant enzyme activity [11]. It is well-documented that oxidative stress negatively impacts both endocrine and immune regulatory functions [11,18].

### **Activation of inflammatory pathways.**

Irizarry et al. [10] found that inflammatory biomarker levels were elevated in pregnant women with higher urinary paraben levels. Of particular relevance to endocrine regulation, chronic low-level inflammation can influence hormonal homeostasis, especially the function of the thyroid axis [10,11].

### **3.2.3 Mixture effects and cumulative toxicity**

In real-world settings, parabens co-occur with various other environmental chemicals, including phthalates, phenols, pesticides, and components of plastics. Berger et al. [21] evaluated combined prenatal exposures to phthalates, parabens, and phenols, demonstrating that the cumulative mixture was more strongly associated with increased adiposity in children than any paraben examined individually. Similarly, Ghosal et al. [22] reported that combined exposure to parabens and other environmental contaminants was associated with a higher risk of allergic and respiratory disorders in early childhood, suggesting that mixture-specific interactions may contribute to these effects.

Available evidence indicates that combined exposures may produce stronger endocrine, metabolic, and immune-related effects than parabens acting alone. These mixture effects may be additive and, in some cases, synergistic, amplifying biological responses even when individual chemicals are present at low concentrations [21,22].



### **3.3 Consequences of exposure to parabens according to the child's developmental stage**

#### **3.3.1 Prenatal period**

There is clear evidence that parabens can cross the placental barrier. Both parent compounds and their metabolites have been detected across multiple maternal–fetal biological matrices, including placental tissue and umbilical cord blood. It is evident that maternal exposure to parabens results in fetal exposure, and that the placenta does not completely protect against paraben transfer [8,5,19,24]. Due to limited metabolic capacity, the developing fetus may be more vulnerable to the toxic effects of these substances [24].

An additional aspect of fetal vulnerability relates to critical windows of susceptibility. An extensive multi-cohort study conducted by Oskar et al. [7] indicated that there are fluctuations in paraben exposure during the course of pregnancy, but that parabens have an especially detrimental effect during early gestation (9–20 weeks). Parabens were significantly associated with lower CRL measurements, reduced rates of fetal weight gain, and lower measurements of head and abdominal circumference. Across the included cohorts, researchers also observed reduced long-bone growth velocity and altered development of hormone-sensitive tissues, such as abdominal adipose tissue. These findings suggest that early pregnancy may represent a period during which parabens interfere with essential processes of organogenesis, including development of the endocrine, nervous, and immune systems [7].

Exposure during the second trimester was associated with altered production of placental hormones (e.g., progesterone, hCG) and changes in the activity of placental membrane transporters involved in maternal–fetal exchange. Although the third trimester is considered less sensitive from a structural developmental perspective, paraben exposure was still linked to relevant perinatal outcomes, including increased neonatal adiposity and subtle disturbances in glucose metabolism [7].

#### **3.3.2 Neonatal and infant period**

Early infancy is an especially vulnerable time for individuals to experience the impact of the environment because many of the body's regulatory systems have not yet developed. The use of parabens by individuals in the early infancy stage typically occurs through two primary pathways: first, the transfer of parabens from the mother, and second, the introduction of new

products, particularly in hospital settings where such products are routinely used. Among the most susceptible groups to parabens in early infancy are newborns, especially those who are premature and receiving care in Neonatal Intensive Care Units (NICUs). According to a prospective study conducted by Iacobelli et al. [6], nearly all infants in the study received at least one medication or care product containing parabens, and premature infants typically received multiple paraben-containing products. Given the greater skin permeability and immature hepatic metabolism in premature infants, it has been suggested that typical therapeutic doses may result in substantially greater systemic absorption, heightening concerns regarding potential endocrine-disrupting effects during this critical developmental window [6,18].

Research also suggests that the effects of paraben exposure experienced in early childhood may begin to manifest shortly after birth. Reimann et al. [8] measured paraben concentrations in placental tissue along with metabolic biomarkers in cord blood. The results indicated that higher concentrations of parabens, especially propylparaben, were associated with elevated leptin and reduced adiponectin levels in newborns—a pattern consistent with early metabolic disturbance. Furthermore, Reimann et al. [8] demonstrated that children with higher prenatal paraben exposure more frequently exhibited accelerated weight gain and higher Body Mass Index (BMI) during follow-up, suggesting that metabolic alterations linked to prenatal exposure may emerge before long-term metabolic patterns are established.

In addition to prenatal exposure, maternal physiological factors may influence newborn health. Research by Irizarry et al. [10] demonstrated that higher maternal urinary paraben concentrations were associated with elevated levels of pro-inflammatory cytokines such as interleukin-6 (IL-6) and C-reactive protein (CRP). Although this study focused on pregnant women rather than infants, these findings have important implications for neonatal health. Chronic maternal inflammation is known to alter placental function and may predispose newborns to early disturbances in metabolic and immune regulation [10,11].

### **3.3.3 Childhood effects**

The earliest stages of childhood represent a period during which the immune and endocrine systems mature rapidly. Many longitudinal studies have shown that children exposed prenatally to parabens are more frequently identified as having alterations in metabolic function,

immune responsiveness, and body composition, indicating that exposure during this stage may have long-lasting effects on physiological regulatory systems [8,21].

One of the key concerns identified in the literature relates to atopic diseases. There is evidence suggesting that parabens may influence the differentiation of epidermal cells, contributing to the development of atopic disease in early infancy. Thürmann et al. [20] conducted a study assessing the relationship between prenatal paraben exposure and the likelihood of developing atopic dermatitis (AD) early in childhood. Children were followed until two years of age, and those whose mothers had the highest urinary concentrations of parabens—particularly propylparaben—were more likely to develop AD. The association was strongest when exposure occurred during the second trimester of pregnancy, a critical period for both skin barrier maturation and the initiation of immune function.

Children exposed to parabens also demonstrated poorer respiratory health. Quiros-Alcalá et al. [13] found that children with higher urinary concentrations of methylparaben and propylparaben experienced more episodes of asthma, more frequent wheezing, and higher rates of chronic cough. Importantly, these associations remained significant after adjusting for age, weight, socioeconomic status, and exposure to tobacco smoke, indicating that parabens may contribute to respiratory health problems through immunomodulatory and pro-inflammatory mechanisms.

Another example of the potential effects of parabens on the immune system comes from the study by Ghozal et al. [22], who demonstrated that children exposed to higher levels of parabens during pregnancy had a higher occurrence of allergic conditions, particularly atopy and allergic rhinitis. The highest risk occurred when paraben exposure coincided with exposure to other chemicals such as phthalates and phenols, indicating that fetal exposure to multiple chemicals may result in additive or even synergistic effects.

Finally, numerous studies have demonstrated that exposure to certain environmental chemicals in utero contributes to early disturbances in metabolism. Berger et al. [21] reported that children prenatally exposed to a combination of phthalates, phenols, and parabens had higher fat mass and larger waist circumference at ages three to five years. Although the specific contribution of parabens was not individually quantified, the findings indicate that prenatal paraben exposure likely contributed to the overall metabolic effects of these endocrine-disrupting chemicals. These results align with evidence showing less favorable metabolic

biomarkers in cord blood—specifically elevated leptin and decreased adiponectin—among newborns with higher prenatal paraben exposure [8]. Children displaying these early metabolic disturbances were more likely to experience rapid postnatal growth and higher BMI later in childhood. Collectively, these findings suggest that parabens may contribute to the initiation of early adipogenesis and metabolic programming, potentially acting alongside other hormonally active environmental chemicals.

### **3.4 Ways to limit exposure**

Even though parabens are found in almost all everyday products, it is possible to limit exposure to them—particularly for individuals at high risk, including pregnant women and their newborns. A review conducted by Health Canada [11] and the National Institute for Public Health and the Environment (RIVM) [17] determined that for pregnant women the largest contributor to paraben exposure is cosmetic/personal care products applied directly to the skin. The best way to limit exposure is therefore to consciously choose products that do not contain parabens. To accomplish this, it is necessary to carefully read ingredient lists, since methylparaben, propylparaben, and butylparaben are among the most commonly used in fragrances and other long-lasting products such as creams and color cosmetics. Research has shown that even partial substitution of these product types with fragrance-free versions or products with fewer ingredients can result in significant decreases in urinary paraben levels after only a short period of time.

Food products represent another relevant exposure pathway. While the amounts of parabens in food are typically lower than those in cosmetic/personal care products, they may be added as preservatives (E214–E219) and can migrate into food from packaging materials [11]. Avoiding highly processed foods, choosing products stored in glass rather than plastic or cardboard, and opting for fresh rather than pre-packaged items can help reduce dietary exposure.

Finally, pharmaceutical products should also be considered. Reports from regulatory agencies have emphasized the need to substitute alternative preservatives in medications intended for pregnant women and newborns. Additionally, these agencies stress the importance of clear labeling medications [6].

Increasing attention has been directed toward understanding how humans are exposed to parabens through the environment. Strategies such as increasing indoor ventilation, vacuuming regularly, and using fewer fragranced household products may help reduce indirect

exposure. Even though these actions may appear minor, biomonitoring studies have shown that removing even a single paraben source can substantially reduce an individual's overall paraben burden, as reflected in urinary concentrations [23].

To improve public health, a substantial body of evidence indicates that consumer education is essential. Popa et al. [18] highlight that one of the major challenges is the limited ability of consumers to recognize paraben names on product labels and the general lack of awareness of their ubiquity. Recent research has clearly shown that lack of knowledge about parabens is a primary barrier to reducing exposure [18].

Therefore, collectively, current research suggests that while parabens are extremely widespread, exposure can be reduced without requiring dramatic lifestyle changes. Implementing several simple strategies simultaneously can significantly decrease overall paraben burden, which is particularly important for protecting the health of the developing fetus and young child.

#### **4. Discussion**

The evidence from this review demonstrates that both prenatal and early life exposures to parabens occur commonly and are biologically relevant. Biomonitoring data have shown that maternal urine, placenta and cord blood all contain parabens, indicating that fetal exposure is unavoidable if mothers apply products containing parabens [5,12]. Multiple cohorts have demonstrated a relationship between prenatal exposure to parabens and alterations to fetal growth and development, including changes to metabolism and increased risk of atopic and respiratory diseases in infancy and early childhood [7,8,13,20,22].

Strengths and limitations of the evidence must be considered. Strong aspects include the consistency of biomonitoring findings across regions, the replication of associations between paraben exposure and child outcomes and the growing mechanistic support involving endocrine disruption, oxidative stress, inflammation and epigenetic alterations [10,11,18]. However, most epidemiological studies rely on single spot-urine samples, capturing short-term exposure only, and differ in exposure metrics and analytical methods, introducing heterogeneity. Residual confounding—such as concurrent exposure to phthalates and phenols—remains a challenge, as does the observational nature of available studies, which precludes firm causal inference. Similarly, this review is limited by the variability of study designs and the inability to perform quantitative synthesis due to methodological heterogeneity.

In comparison to the broader scientific literature about EDCs, the results presented in this review are generally consistent with the existing scientific consensus. Like phthalates and bisphenols, parabens appear to produce small yet significant biological effects when exposure occurs during critical developmental periods. Furthermore, mixture studies demonstrate that exposure to combinations of EDCs produces larger associations with metabolic and allergic disease outcomes than exposures to single chemicals [21,22], highlighting the need to evaluate paraben effects within the context in which they occur in the environment.

From a clinical and public health perspective, the implications of the evidence presented in this review are considerable. Products designed for cosmetics and personal care are the primary route through which pregnant women are exposed to parabens. From a public health standpoint, providing a clear opportunity for educating pregnant women and consumers about safe and effective alternatives to products containing parabens, as well as the need for improved product labeling. Reducing paraben exposure in pregnancy and early childhood represents a low-cost, low-risk strategy for potentially mitigating risks of metabolic, allergic and respiratory disease later in life.

Overall, although gaps in causal evidence remain, the convergence of epidemiological findings with mechanistic data supports precautionary strategies aimed at minimizing maternal and early-life exposure to parabens, especially during the most sensitive developmental windows.

## **5. Conclusion**

The information collected in this study demonstrates that, as of today, parabens continue to represent one of the most prevalent groups of chemicals in the daily environments of both pregnant women and young children. Exposure to parabens occurs via cosmetics and personal care products, food, pharmaceuticals and environmental sources, creating a multidimensional exposure profile that can be challenging to minimize or eliminate. Consistent with previous biomonitoring studies, parabens were detected in maternal urine, maternal serum, placenta and cord blood, indicating their ability to cross the placental barrier and expose the developing fetus throughout critical periods of development.

Results from numerous cohort studies indicate that prenatal exposure to parabens has been linked to smaller birth size, altered anthropometric measures and early metabolic disruptions, including elevated leptin and reduced adiponectin levels. Additionally, the evidence suggests

that prenatal paraben exposure may increase the likelihood of rapid postnatal weight gain and higher BMI during childhood. Increasingly, research also indicates that parabens may impact immune function, contributing to elevated risks of atopic and respiratory diseases in early life. Mechanistic studies provide further support by demonstrating associations between parabens and altered endocrine, inflammatory and epigenetic processes essential for proper development.

A second concern related to postnatal exposure includes the use of pharmaceuticals in hospitalized newborns. Because newborns have underdeveloped skin and metabolic systems, systemic exposure to parabens from medications may be substantially higher than in the general population.

While a direct causal relationship between parabens and adverse developmental outcomes has not yet been conclusively established, the consistent findings from epidemiological studies—combined with mechanistic evidence—support a precautionary approach. Strategies to reduce exposure include informed decision-making in cosmetics purchasing, reduced consumption of processed foods, limiting the use of fragranced products and selecting paraben-free medications when possible.

Therefore, based on the current body of scientific evidence, there is sufficient rationale to establish public health recommendations aimed at reducing paraben exposure in pregnant women and young children. Two key strategies include increasing consumer awareness of the potential risks associated with parabens and enhancing transparency in product labeling.

Declarations:

Authors Contribution:

Conceptualization:[KO-C],[KP], [PL],[JP]

Methodology: [AO],[JB],[MZ],[KP]

Formal analysis:[KO-C],[KP]

Investigation:[KO-C],[PL], [K-MP],[WU],

Writing- Rough preparation:[MZ] ,[K-MP], [WU],[PL], [KO-C]

Writing- Review and Editing:[KP],[PL],[JP],[AO], [JB]



All authors have read and agreed with the published version of the manuscript.

#### Funding Statement:

The authors declare no external funding.

#### Conflicts of Interest:

The authors declare no conflict of interest.

#### Ethical Approval:

Not applicable — narrative review

#### References:

- [1] Trasande L, Sathyanarayana S, Messerlian C, et al. Prenatal phenol and paraben exposures and adverse birth outcomes: a prospective analysis. *Environ Int.* 2023;176:108079. <https://doi.org/10.1016/j.envint.2023.108079>
- [2] Pacyga DC, Barrett ES, Sathyanarayana S, et al. Maternal diet quality moderates associations between prenatal parabens and birth size. *Environ Res.* 2022;207:112182. <https://doi.org/10.1016/j.envres.2021.112182>
- [3] Bräuner EV, Uldall Pallesen J, Andersson AM, et al. Prenatal paraben exposure and sex-specific associations with birth size. *Environ Int.* 2023;176:108073. <https://doi.org/10.1016/j.envint.2023.108073>
- [4] Shin B, Park H, Lee J, et al. Prenatal exposure to parabens and adverse birth outcomes: a prospective cohort study. *Sci Total Environ.* 2021;768:144678. <https://doi.org/10.1016/j.scitotenv.2020.144678>
- [5] Vrijens K, Tsamou M, Madhloum N, et al. Placental paraben load and its association with birth weight and neonatal anthropometry: the ENVIRONAGE birth cohort. *Environ Health.* 2020;19:75. <https://doi.org/10.1186/s12940-020-00635-5>

- [6] Iacobelli S, Bonsante F, Kermorvant-Duchemin E, et al. Paraben exposure through medications in neonatal intensive care units: a prospective study. *Front Pharmacol.* 2023;14:1200521. <https://doi.org/10.3389/fphar.2023.1200521>
- [7] Oskar SS, Strøm M, Høgh M, et al. Identifying critical windows of prenatal exposure to phenols and parabens: a multi-cohort analysis. *Environ Int.* 2024;180:108205. <https://doi.org/10.1016/j.envint.2023.108205>
- [8] Reimann B, Frederiksen H, Kyhl HB, et al. Associations between placental parabens, cord blood metabolic markers, and childhood BMI. *Environ Res.* 2021;197:111010. <https://doi.org/10.1016/j.envres.2021.111010>
- [9] Bommarito PA, Braun JM, Buckley JP. Prenatal exposure to environmental phenols and fetal development: a review of recent evidence. *Curr Environ Health Rep.* 2024;11:1–12. <https://doi.org/10.1007/s40572-023-00399-1>
- [10] Irizarry RA, Ferguson KK, Rosen EM, et al. Maternal urinary phenol and paraben concentrations and inflammation biomarkers during pregnancy. *Environ Health Perspect.* 2024;132(4):047004. <https://doi.org/10.1289/EHP12089>
- [11] Health Canada. Draft Screening Assessment – Parabens. Canadian Environmental Protection Act. Ottawa, Canada; 2020. Available from: <https://www.canada.ca/>. Accessed 2024 Oct 15.
- [12] HBM4EU Consortium. HBM4EU Parabens Scoping Document. European Human Biomonitoring Initiative. Brussels, Belgium; 2021. Available from: <https://www.hbm4eu.eu/>. Accessed 2024 Oct 15.
- [13] Quiros-Alcalá L, Hansel NN, McCormack MC, et al. Paraben exposures and asthma-related outcomes among children. *J Allergy Clin Immunol.* 2019;143(3):972–975. <https://doi.org/10.1016/j.jaci.2018.07.017>
- [14] Vrijheid M, Slama R, Robinson O, et al. The ENVIRONAGE birth cohort: design, methods and initial findings. *Environ Int.* 2020;136:105508. <https://doi.org/10.1016/j.envint.2019.105508>

- [15] Philippat C, Botton J, Calafat AM, et al. Maternal paraben exposure and overweight in early childhood. *Environ Int.* 2020;137:105523. <https://doi.org/10.1016/j.envint.2020.105523>
- [16] Jurewicz J, Radwan M, Wielgomas B, et al. Parameters of ovarian reserve in relation to urinary concentrations of parabens. *Environ Health.* 2020;19:102. <https://doi.org/10.1186/s12940-020-00580-3>
- [17] RIVM. Aggregate exposure to parabens in personal care products. National Institute for Public Health and the Environment; 2022. Report 2022–0201. Available from: <https://www.rivm.nl/>. Accessed 2024 Oct 15.
- [18] Popa DS, Camelia V, Botnariu GE. The controversies of parabens: an updated review. *Acta Pharm.* 2021;71(3):355–373. <https://doi.org/10.2478/acph-2021-0020>
- [19] Karzi V, Trikalinos A, Chalkiadaki G, et al. Determination of prenatal exposure to parabens and triclosan in maternal–fetal matrices. *Toxicol Rep.* 2021;8:544–551. <https://doi.org/10.1016/j.toxrep.2021.03.022>
- [20] Thürmann L, Rolle-Kampczyk UE, Schmitt-Kopplin P, et al. Prenatal paraben exposure and risk of atopic dermatitis in early childhood. *Allergy.* 2021;76(10):3245–3249. <https://doi.org/10.1111/all.14858>
- [21] Berger K, Eskenazi B, Balmes J, et al. Prenatal exposure to mixtures of phthalates, parabens and phenols and child adiposity. *Int J Environ Res Public Health.* 2021;18(4):1796. <https://doi.org/10.3390/ijerph18041796>
- [22] Ghosal R, Cordier S, Botton J, et al. Prenatal dietary exposure to mixtures of chemicals and allergic/respiratory diseases in early childhood. *Environ Health.* 2024;23:7. <https://doi.org/10.1186/s12940-023-01046-y>
- [23] Wojtkiewicz J, Rzymski P, Klimaszyk P. Human exposure to parabens in north-central Europe: biomonitoring study. *Sci Rep.* 2021;11:23649. <https://doi.org/10.1038/s41598-021-03152-8>
- [24] Čok I, Peterlin Mašič L, Heath E. Prenatal exposure to mixtures of phthalates, parabens and phenols: methodological and toxicological considerations. Technical Report; 2021. [Unpublished report].

[25] Boberg J, Taxvig C, Christiansen S, Hass U. Possible endocrine disrupting effects of parabens and their metabolites. *Reprod Toxicol.* 2010;30(2):301–312.  
<https://doi.org/10.1016/j.reprotox.2010.03.011>