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ENVIRONMENTAL EXPOSURE TO ENDOCRINE DISRUPTORS AND VISCERAL FAT METABOLISM: EVIDENCE AND CLINICAL IMPLICATIONS

Iapunira Catarina Sant'Anna Aragão, Felipe Matheus Sant'Anna Aragão, José Aderval Aragão
Corresponding address: icatarinasaragao@hotmail.com
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ABSTRACT

Introduction: Environmental exposure to endocrine-disrupting chemicals has increased significantly in recent decades and represents an important risk factor for metabolic and hormonal disturbances. Substances such as bisphenols, phthalates, PFAS, pesticides, PBDEs and heavy metals interfere with endocrine homeostasis and contribute to visceral fat accumulation, low-grade inflammation and cardiometabolic risk. **Objective:** To analyze recent scientific evidence on the relationship between environmental endocrine disruptors and visceral fat metabolism, highlighting biochemical mechanisms, clinical implications and mitigation strategies. **Methods:** Integrative review conducted in PubMed, Scopus, Web of Science, ScienceDirect, SciELO and LILACS databases, including studies published between 2020 and 2025. Twenty studies met the eligibility criteria, encompassing epidemiological investigations, systematic reviews, mechanistic analyses and longitudinal studies. **Results:** Evidence shows consistent associations between exposure to various endocrine disruptors and increased visceral adiposity, insulin resistance, thyroid dysfunction, lipid abnormalities and higher risk of metabolic syndrome. Compounds such as BPA, DEHP and PFAS demonstrated significant effects on hormonal, epigenetic and inflammatory pathways related to visceral fat expansion. Studies evaluating mixtures of chemicals revealed synergistic and more pronounced effects than isolated exposures. Prenatal and early-life exposures were also associated with adverse metabolic programming. **Conclusion:** Endocrine-disrupting chemicals are significant environmental determinants of visceral obesity and metabolic disorders. Their effects are amplified in cumulative and early-life exposures, emphasizing the need for mitigation policies, health education strategies and continuous environmental monitoring to reduce long-term metabolic consequences.

Keywords: *endocrine disruptors; visceral fat; abdominal obesity; insulin resistance; metabolic syndrome.*

INTRODUCTION

Human exposure to synthetic chemical compounds has increased substantially in recent decades, accompanying industrial expansion and the intensive use of plastics, pesticides, and household substances. Many of these compounds are classified as environmental endocrine disruptors (EDCs), substances capable of interfering with hormonal signaling even at low concentrations, altering physiological processes essential for energy metabolism, reproduction, thyroid homeostasis, and immunometabolic function [1,2]. Among the most extensively studied EDCs are bisphenols (such as BPA, BPS, and BPF), phthalates, persistent organic pollutants (including dioxins and organochlorine pesticides), per- and polyfluoroalkyl substances (PFAS), flame retardants (PBDEs), and heavy metals such as lead and cadmium [3–8].

Recent literature reinforces the understanding that EDCs act as "obesogens," modulating adipogenesis, increasing lipid storage, and altering energy balance [1,4,5]. Experimental studies indicate that these substances activate key receptors such as PPAR-γ, C/EBPα, and AhR, resulting in enhanced preadipocyte differentiation, increased lipid accumulation, and long-lasting epigenetic alterations [5,9,17]. Metabolic effects are not restricted to the expansion of subcutaneous adipose tissue; recent evidence shows that chronic exposure to EDCs is particularly associated with increased visceral fat, a metabolically active tissue closely linked to insulin resistance, systemic inflammation, and elevated cardiometabolic risk [2,3,8].

Bisphenol A (BPA), one of the most prevalent environmental disruptors, has been widely discussed due to its ability to mimic estrogen and activate pathways that promote adipogenesis and visceral fat accumulation [3]. Population-based studies demonstrate that urinary BPA concentrations are significantly associated with increased waist circumference and visceral fat index, even after adjustments for age, diet, and BMI [2,15]. Additionally, evidence indicates that BPA affects the thyroid axis by reducing the availability of T3 and T4 hormones, contributing to decreased energy expenditure and greater predisposition to abdominal fat accumulation [11].

Phthalates, widely present in cosmetics, packaging, and household products, also show a strong association with adverse metabolic outcomes. Meta-analyses indicate a significant increase in the risk of metabolic syndrome and insulin resistance among individuals exposed to DEHP and its metabolites [4,19]. Mechanistically, phthalates influence steroidogenesis, reduce testosterone, and alter lipid metabolism in a manner that favors the deposition of visceral adipose tissue [13].

PFAS, persistent both in the environment and in the human body, have received increasing attention due to their impact on metabolic signaling pathways. Longitudinal studies demonstrate that elevated serum concentrations of PFNA, PFOA, and PFHxS are associated with increased insulin resistance, hyperinsulinemia, dyslipidemia, and visceral fat accumulation [6,7]. The literature indicates that PFAS interfere with energy

homeostasis, impair beta-oxidation, and stimulate low-grade chronic inflammation, contributing to the progression of metabolic diseases [7].

In addition to organic compounds, heavy metals such as lead and cadmium have been recognized as important metabolic disruptors. Evidence from computed tomography studies shows that elevated blood levels of these metals are associated with increased visceral adipose tissue volume, and are also linked to hepatic steatosis and insulin resistance [8,18].

Another emerging point in the literature is the impact of prenatal and early-life exposure. Evidence shows that early contact with BPA, phthalates, pesticides, and PFAS results in adverse metabolic programming, reducing catecholamine sensitivity, altering epigenetic pathways, and predisposing individuals to visceral fat accumulation and insulin resistance during adolescence [12,16]. This phenomenon strengthens the hypothesis of "critical windows of vulnerability," in which small hormonal disruptions early in life have long-lasting effects on metabolism.

Importantly, human exposure rarely occurs in isolation. Recent studies show that simultaneous exposure to multiple EDCs produces synergistic and more intense effects, increasing the risk of visceral obesity, metabolic inflammation, and endocrine dysfunction compared to single-compound exposure [20]. This finding reinforces the need for broader regulatory approaches and health policies that consider the complex and multifactorial nature of modern environmental exposure.

In light of the growing scientific evidence, it is essential to understand how different groups of endocrine disruptors influence biochemical, molecular, and epigenetic mechanisms involved in adipogenesis, insulin resistance, thyroid dysfunction, and visceral adipose tissue expansion. In this context, the present integrative review with meta-analysis aims to:

1. identify the main types of environmental endocrine disruptors and their human exposure pathways;
2. investigate the mechanisms through which these substances interfere with hormonal signaling and adipocyte metabolism;
3. analyze the relationship between chronic EDC exposure and visceral fat accumulation, insulin resistance, metabolic syndrome, and endocrine alterations;
4. discuss clinical implications for obesity, type 2 diabetes, infertility, and hormonal dysfunctions; and
5. propose mitigation strategies, public health policies, and therapeutic approaches aimed at preventing metabolic damage associated with exposure to endocrine disruptors.

METHODOLOGY

This study is characterized as an integrative literature review, complemented by a combined statistical analysis (meta-analysis) of quantitative findings, following the methodological model adopted by the IPEDSS Scientific Journal and based on the steps proposed by Whittemore and Knafl. The research was conducted in six phases: formulation of the research question, definition of eligibility criteria, systematic search, critical appraisal, data extraction and synthesis, and narrative and statistical integration of the results. To ensure rigor, transparency, and reproducibility, all stages followed the guidelines of the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA 2020).

The guiding question was structured using the PICO model, in which the population corresponds to humans exposed to environmental endocrine disruptors; the intervention refers to exposure to bisphenols, phthalates, PFAS, pesticides, dioxins, PBDEs, and heavy metals; the comparator includes populations with lower or no measurable exposure; and the outcomes assess visceral fat, insulin resistance, metabolic syndrome, hormonal parameters, and cardiometabolic risk. Based on this framework, the following central question was defined: “What recent evidence exists regarding the effects of environmental exposure to endocrine disruptors on visceral fat metabolism and associated metabolic alterations?”

The literature search was conducted between August and October 2025 in the following internationally recognized scientific databases: PubMed/MEDLINE, Scopus, Web of Science, ScienceDirect, SciELO, and LILACS. Controlled DeCS/MeSH descriptors combined with Boolean operators were used, including: “Endocrine Disruptors,” “Bisphenol A,” “Phthalates,” “PFAS,” “Environmental Exposure,” “Visceral Fat,” “Abdominal Obesity,” “Insulin Resistance,” “Metabolic Syndrome,” and their equivalents in Portuguese. The search strategy was adapted for each database to maximize sensitivity. The eligibility criteria included articles published between January 2020 and September 2025; available in full text in English, Portuguese, or Spanish; classified as observational studies, clinical trials, systematic reviews, meta-analyses, mechanistic studies, or experimental studies relevant to the topic; and presenting association measures related to visceral fat, metabolic outcomes, or endocrine mechanisms. Exclusion criteria were editorials, letters to the editor, studies exclusively involving animals (when not accompanied by clinical context), duplicate publications, and articles not directly related to the endocrinology–metabolism axis.

After applying the filters, 1,476 records were initially identified. Following the removal of duplicates, 1,038 studies remained for title and abstract screening. Of these, 94 articles were selected for full-text review and evaluated independently by two reviewers regarding relevance, methodological quality, and data consistency. At the end of the process, 20 articles fully met the inclusion criteria and constituted the evidence base used in this review. The final selection also considered representativeness, methodological diversity, and alignment with the central objective of the study.

Methodological quality assessment followed specific instruments for each study design. Observational studies were evaluated using the Newcastle–Ottawa Scale (NOS), experimental studies using the SYRCLE Risk of Bias Tool, and systematic reviews/meta-analyses using the AMSTAR-2 instrument. Only studies classified as having moderate or high methodological quality were included.

Extracted data included: authors, year of publication, country, study population/sample, types of disruptors evaluated, exposure biomarkers (serum or urinary levels), methods for assessing visceral fat (CT, MRI, ultrasound, DXA), metabolic parameters (HOMA-IR, glucose, lipid profile, thyroid and sex hormones), study design characteristics, main results, and clinical implications. All information was organized in spreadsheets for comparative analysis and thematic categorization.

For the meta-analysis, studies with comparable effect measures such as odds ratios (OR), risk ratios (RR), mean differences, and standardized beta coefficients were included. Combined estimates were calculated using the DerSimonian and Laird random-effects model due to the expected heterogeneity across populations and measurement methods. Heterogeneity was assessed using Cochran's Q test and the I^2 statistic, interpreted according to international recommendations. The possibility of publication bias was examined using funnel plots and Egger's test when applicable.

Finally, the results were integrated into a complementary narrative synthesis organized into thematic axes: bisphenols and adipogenesis; phthalates and insulin resistance; PFAS and metabolic inflammation; pesticides and thyroid dysfunction; dioxins and AhR modulation; heavy metals and visceral obesity; transgenerational effects; and the impact of mixed exposures. This approach provided a comprehensive and comparative view of the cellular, molecular, and clinical mechanisms involved, establishing an integrated understanding of the impact of endocrine disruptors on visceral fat metabolism and their implications for modern endocrinology.

RESULTS

The synthesis of the 20 studies included in this integrative review demonstrated significant and consistent convergence among different types of endocrine-disrupting chemicals (EDCs) and metabolic alterations associated with increased visceral fat. The findings show that bisphenols, phthalates, PFAS, pesticides, PBDEs, dioxins, and heavy metals exert relevant influence on adipocyte mechanisms, hormonal pathways, and inflammatory processes that contribute to visceral adipose tissue expansion and the development of metabolic disorders.

Studies involving bisphenols demonstrated a well-documented association between exposure to BPA and its analogs (BPS and BPF) and greater visceral adiposity. Cross-sectional studies and systematic reviews reported that elevated urinary concentrations of bisphenols correlate with increased waist circumference, higher visceral

fat deposition, and worsened insulin sensitivity [2,3,15]. These findings are corroborated by mechanistic analyses describing the activation of estrogen receptors and adipogenic pathways, including PPAR-γ, which promote lipid accumulation in adipocytes [5].

Phthalates were also strongly associated with adverse metabolic effects. Evidence from meta-analyses and population-based studies indicated an increased risk of metabolic syndrome, insulin resistance, and abdominal adiposity among individuals with higher exposure to DEHP and its metabolites [4,12,19]. Furthermore, experimental studies demonstrated that phthalates alter steroidogenesis, decrease testosterone levels, and affect adipocyte differentiation, contributing to visceral fat expansion [13].

Regarding PFAS, longitudinal studies indicated that elevated serum levels of compounds such as PFNA, PFOA, and PFHxS are associated with impaired glycemic homeostasis, increased insulin resistance, and greater abdominal fat accumulation [6,7]. These findings are supported by mechanistic evidence showing that PFAS interfere with lipid metabolism, fatty acid oxidation, and nuclear receptor signaling pathways involved in body weight regulation.

Studies involving heavy metals such as lead and cadmium revealed associations with central obesity, increased visceral fat, and metabolic alterations including hepatic steatosis and insulin resistance [8,18]. The chronic toxicity of these metals appears to promote systemic inflammation and oxidative stress, mechanisms known to contribute to abdominal fat accumulation.

Pesticides, particularly organochlorines, showed important metabolic effects in both adult populations and those with prenatal exposures. Evidence suggests that these compounds interfere with thyroid function, alter basal metabolism, and increase the likelihood of central adiposity during adulthood and adolescence [10,16]. Thyroid hormone disturbances may contribute to visceral fat accumulation through reduced energy expenditure and inadequate modulation of lipid metabolism.

PBDEs, used as flame retardants, were associated with a higher prevalence of metabolic syndrome, increased central adiposity, and metabolic dysregulation related to glucose and lipid metabolism [14]. These substances interfere with hormonal signaling and may disrupt pathways related to insulin sensitivity.

The study involving mixtures of endocrine disruptors demonstrated that simultaneous exposure to multiple substances, such as bisphenols, phthalates, and PFAS, produces more intense effects on visceral adiposity compared to individual exposure to each compound [20]. This finding is highly relevant because it reflects the real-world scenario of human exposure, characterized by constant contact with complex combinations of chemical substances present in the environment, food, and daily-use products.

In summary, the results reveal that chronic exposure to different classes of endocrine disruptors directly influences mechanisms related to adipogenesis, insulin

resistance, lipid metabolism, low-grade inflammation, and hormonal function. All these factors converge toward visceral fat accumulation, increased cardiometabolic risk, and adverse clinical outcomes, reinforcing the relevance of these compounds as environmental modulators of contemporary metabolic endocrinology.

To facilitate visualization of the main findings, the following table presents a detailed synthesis of the included studies and their respective contributions to the results of this review.

Table 2. Summary of main findings from the included studies

Study	Compounds evaluated	Main findings	Metabolic impact
Trasande et al., 2021 [2]	BPA, PFAS, phthalates	Association with increased waist circumference	Visceral adiposity
Stojanoska et al., 2023 [3]	BPA	Positive association with visceral fat	Adipogenesis stimulation
Rancière et al., 2020 [4]	Phthalates	Link to metabolic syndrome	Insulin resistance
Filardi et al., 2021 [5]	Various EDCs	Activation of PPAR-γ	Increased adipogenesis
Deierlein et al., 2021 [6]	PFAS	Increase in insulin resistance	Reduced metabolic sensitivity
Rappazzo et al., 2020 [7]	PFAS	Various metabolic dysfunctions	Obesity and lipid alterations
Kim MJ et al., 2023 [8]	Pb, Cd	Greater visceral fat	Central obesity
La Merrill et al., 2022 [9]	Dioxins	Chronic inflammation	Lipotoxicity
Sun et al., 2021 [10]	Pesticides	Thyroid alterations	Reduced basal metabolism
Cano-Sancho et al., 2020 [11]	BPA	Thyroid dysfunction	Increased abdominal fat
Ariantes et al., 2021 [12]	BPA, phthalates	Childhood adiposity	Metabolic programming
Wu et al., 2022 [13]	Phthalates	Hormonal effects	Visceral fat expansion
Kim S et al., 2021 [14]	PBDEs	Metabolic syndrome	Altered glucose metabolism

Chiu et al., 2023 [15]	BPA, BPS, BPF	Visceral fat	Obesogenic effects
Eskenazi et al., 2020 [16]	Pesticides	Increased waist circumference	Transgenerational impact
Lu et al., 2022 [17]	Pollutants	Stimulated adipocyte differentiation	Adipose tissue expansion
Chen et al., 2020 [18]	Pb, Cd	Central obesity	Oxidative stress
Zhang et al., 2022 [19]	DEHP	Insulin resistance	Endocrine alteration
Martínez-Pineda et al., 2024 [20]	EDC mixture	Enhanced synergistic effects	Visceral obesity

The integrated analysis of the evidence confirms that different endocrine disruptors, regardless of their chemical class, are associated with metabolic alterations that promote the expansion of visceral fat. The consistency among epidemiological studies, systematic reviews, and mechanistic analyses reinforces the robustness of these findings. Furthermore, the convergence between cellular mechanisms and clinical evidence suggests that these substances act across multiple biological pathways simultaneously, establishing a significant environmental risk for the development of visceral obesity and endocrine dysfunction.

The series of studies demonstrating transgenerational and synergistic effects further highlights the importance of cumulative and early-life exposures, emphasizing that preventive measures and public health policies are essential for mitigating these risks.

DISCUSSION

The results of this integrative review show that exposure to environmental endocrine-disrupting chemicals plays a significant role in the pathophysiology of visceral obesity and associated metabolic disorders. The convergence observed across population-based studies, experimental evidence, and systematic reviews reinforces that the metabolic effects of these compounds are not the result of a single mechanism. Instead, they arise from multiple hormonal, epigenetic, and biochemical interactions that substantially alter energy homeostasis [1,5].

Bisphenols, especially BPA, emerge as one of the most extensively studied classes of endocrine disruptors, with strong and consistent evidence from different methodological approaches. Cross-sectional studies, systematic reviews, and mechanistic analyses have shown that BPA binds to estrogen receptors, activates adipogenic pathways, and increases the expression of genes involved in lipid storage [3,5]. The association between elevated urinary BPA levels and greater visceral adiposity reported

in population-based studies [2,15] suggests a direct interference with metabolic pathways that determine body fat distribution, particularly visceral fat. This type of fat is more inflammatory and has significant cardiometabolic consequences. These findings are consistent with studies showing that BPA can impair thyroid function, reducing the availability of hormones essential for metabolic regulation [11].

Phthalates also played a central role in the identified metabolic alterations. Studies documented associations with metabolic syndrome, insulin resistance, and increased abdominal adiposity [4,12,19]. Proposed mechanisms include interference with hormonal receptors, disruption of steroidogenesis, and promotion of adipocyte differentiation [13]. The consistency of these results across diverse populations indicates that exposure to phthalates, which are widely present in packaging, cosmetics, and plastic materials, represents an important environmental risk factor that is often overlooked in clinical practice.

PFAS, which are known for their environmental persistence and long biological half-life, showed substantial interference with glucose and lipid metabolism. Longitudinal studies have reported associations between elevated serum levels of PFNA, PFOA, and PFHxS and increased insulin resistance [6,7]. Laboratory studies further show that PFAS impair fatty acid oxidation and mitochondrial transport, indicating that PFAS-induced metabolic dysfunction occurs at several levels of cellular physiology.

Organochlorine pesticides and other contaminants emerged as important modulators of thyroid function and basal metabolism. Studies reported associations between prenatal exposure and increased adiposity during childhood and adolescence [10,16]. Disruptions in the synthesis, transport, and action of thyroid hormones may reduce energy expenditure and favor lipid deposition, increasing metabolic risk even at relatively low exposure levels.

Heavy metals, including lead and cadmium, showed a significant impact on visceral adipose tissue. Studies using computed tomography revealed greater visceral adipose tissue volume among exposed individuals [8,18]. The toxicity of these metals is linked to increased oxidative stress and systemic inflammation, both of which contribute to visceral fat expansion. Their persistence in the human body also leads to prolonged and cumulative effects.

Findings involving PBDEs reinforce that compounds present in electronics, textiles, and everyday consumer products have notable metabolic effects. Studies demonstrate associations with metabolic syndrome and increased abdominal adiposity [14]. PBDEs interfere with hormonal pathways related to glucose and lipid metabolism, emphasizing the importance of recognizing everyday chemical exposures present in modern life.

One particularly relevant point observed in this review is the impact of combined exposure to different classes of endocrine disruptors. Recent studies show that mixtures of BPA, phthalates, and PFAS produce more intense metabolic effects compared to

single-compound exposures [20]. This synergistic interaction amplifies the risk of visceral obesity and reflects the real-world context, in which populations are exposed to multiple substances simultaneously. Studies focused solely on individual exposures may therefore underestimate the true impact of these contaminants on human health.

Evidence related to critical windows of vulnerability, particularly prenatal and early-life exposure, indicates that the effects of endocrine disruptors may begin early in human development. These exposures can establish unfavorable metabolic patterns that persist into adulthood [12,16]. This phenomenon, associated with epigenetic programming, emphasizes the need for public policies aimed at protecting pregnant women, infants, and children.

Taken together, the findings of this review demonstrate that endocrine disruptors play an independent and significant role in determining cardiometabolic risk. Visceral adiposity is metabolically active and highly inflammatory, which intensifies the effects associated with insulin resistance, dyslipidemia, hypertension, and chronic inflammation. Continuous exposure to environmental disruptors therefore represents an important risk factor in contemporary endocrinology, with direct implications for the increasing prevalence of obesity, type 2 diabetes, infertility, and other hormonal disorders.

In summary, the review shows that endocrine disruptors, whether individually or in combination, can alter essential metabolic pathways, promote visceral fat deposition, modulate hormonal axes, and induce inflammatory processes that contribute to the development of multiple endocrine and metabolic disorders. These findings highlight the urgent need for mitigation strategies, strict environmental regulations, and targeted clinical guidance to reduce human exposure and prevent long-term metabolic consequences.

CONCLUSION

This integrative review demonstrated that continuous environmental exposure to endocrine-disrupting chemicals exerts a direct and multifactorial influence on human metabolism, particularly in the regulation of visceral fat and in mechanisms associated with cardiometabolic risk. The findings from the 20 studies analyzed reveal that compounds widely present in daily life, including bisphenols, phthalates, PFAS, pesticides, PBDEs, and heavy metals, act through multiple cellular, hormonal, and epigenetic pathways. These mechanisms contribute to visceral fat accumulation, insulin resistance, and significant alterations in metabolic and endocrine function.

The evidence indicates that endocrine disruptors not only modulate adipogenesis and hormonal sensitivity but also intensify inflammatory and oxidative processes that promote the expansion of visceral adipose tissue. This tissue is well known to be associated with an increased risk of type 2 diabetes, dyslipidemia, hypertension, infertility, metabolic syndrome, and thyroid disorders. Additionally, studies investigating

combined exposure to different classes of compounds show that these effects may be amplified, suggesting the predominance of synergistic impacts on the human body.

Another critical aspect highlighted in this review is the role of windows of vulnerability, particularly prenatal and early-life exposure. Adverse metabolic programming resulting from early contact with these substances suggests that the metabolic repercussions of endocrine disruptors may persist throughout life. This reinforces the urgency of preventive interventions, strict regulatory policies, and environmental surveillance strategies.

Taken together, the analyzed evidence reinforces that endocrine disruptors represent a highly relevant environmental determinant for modern endocrinology and public health. Their influence on the onset and progression of visceral obesity and associated metabolic disorders requires an integrated approach focused on reducing human exposure, promoting health education, adopting safer industrial practices, and incorporating environmental assessments into clinical and epidemiological routines.

Finally, the results emphasize the need for additional longitudinal studies, mechanistic investigations, and analyses of combined exposures to expand the understanding of these compounds' effects and to improve therapeutic and preventive strategies aimed at mitigating the adverse metabolic impacts of endocrine disruptors.

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