REVIEW ARTICLE

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ENDOCRINE DISRUPTING CHEMICALS AND POLYCYSTIC OVARY SYNDROME: A COMPREHENSIVE NARRATIVE REVIEW

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ABSTRACT

Background: Polycystic ovary syndrome (PCOS) affects 8-13% of reproductive-age women globally and represents a leading cause of infertility and metabolic dysfunction. While genetic factors contribute to PCOS susceptibility, the rising prevalence suggests environmental influences, particularly endocrine-disrupting chemicals (EDCs), play a significant role in disease pathogenesis. **Objective:** This narrative review synthesizes current evidence on the relationship between EDC exposure and PCOS, examining epidemiological associations, experimental data, and mechanistic insights to elucidate how environmental chemicals may contribute to PCOS development and progression. **Materials and Method:** We conducted a comprehensive literature search of PubMed, Embase, and Web of Science databases for studies published through December 2024, focusing on EDCs (bisphenol A, phthalates, persistent organic pollutants, pesticides, heavy metals) and PCOS outcomes. We included human epidemiological studies, animal models, and in vitro mechanistic investigations. **Results:** Epidemiological evidence demonstrates consistent associations between elevated EDC levels and PCOS prevalence, with bisphenol A and phthalates showing the strongest correlations. Women with PCOS exhibit significantly higher serum and urinary concentrations of multiple EDCs compared to controls. Animal studies reveal that prenatal EDC exposure induces PCOS-like phenotypes including hyperandrogenism, ovulatory dysfunction, and metabolic abnormalities, often persisting across generations. Mechanistic investigations identify multiple pathways through which EDCs disrupt reproductive function: interference with steroidogenesis, disruption of hypothalamic-pituitary-gonadal axis signaling, induction of insulin resistance, promotion of chronic inflammation, and epigenetic programming effects. **Conclusion:** Substantial evidence supports EDCs as significant environmental contributors to PCOS pathogenesis. Clinical implications include the need for exposure assessment in PCOS evaluation and patient counseling on EDC reduction strategies. Public health measures to minimize population-level EDC exposure may represent an important preventive approach for reducing PCOS burden.

Keywords: Endocrine disrupting chemicals, Polycystic ovary syndrome, Environmental exposure, Reproductive health, Bisphenol A.

INTRODUCTION

Polycystic Ovary Syndrome: Clinical Significance and Burden

Polycystic ovary syndrome (PCOS) stands as the most prevalent endocrine disorder among women of reproductive age, affecting an estimated 116 million women worldwide[1]. The syndrome manifests through a heterogeneous constellation of reproductive, metabolic, and psychological features that significantly impair quality of life and long-term health outcomes.

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According to the Rotterdam consensus criteria, PCOS diagnosis requires two of three features: oligo-ovulation or anovulation, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology on ultrasound[2]. This diagnostic framework, while widely adopted, captures considerable phenotypic variability, contributing to ongoing debates regarding PCOS classification and optimal management approaches. Beyond reproductive dysfunction, PCOS associates with substantial metabolic morbidity. Insulin resistance affects 50-70% of PCOS patients, predisposing to type 2 diabetes mellitus, with conversion rates 5-10 times higher than age-matched controls[3].

Dyslipidemia, hypertension, and non-alcoholic fatty liver disease occur with increased frequency, collectively elevating cardiovascular disease risk. Psychological impacts include higher rates of anxiety, depression, and eating disorders, further compromising wellbeing[4]. The syndrome's multisystem involvement necessitates lifelong management and surveillance, imposing considerable healthcare costs estimated at billions of dollars annually in the United States alone[5].

Environmental Etiology: Beyond Genetic Determinism

Traditional etiological frameworks have emphasized PCOS as a genetic disorder with familial clustering patterns suggesting heritability estimates of 70%[6]. Large-scale genome-wide association studies have identified numerous susceptibility loci, implicating genes involved in steroidogenesis, insulin signaling, and gonadotropin regulation[7]. However, these genetic variants collectively explain less than 10% of PCOS heritability, highlighting the 'missing heritability' problem common to complex diseases. More critically, the dramatic rise in PCOS prevalence observed over recent decades—particularly pronounced in industrialized nations—cannot be attributed to genetic evolution occurring on such rapid timescales[8].

This temporal pattern strongly implicates environmental factors as major contributors to PCOS pathogenesis. Lifestyle elements including diet, physical activity, and obesity undoubtedly influence PCOS risk and severity[9]. However, these factors alone inadequately explain the population-level trends, particularly observations of increasing PCOS prevalence among normal-weight women. Emerging evidence points toward environmental chemical exposures, specifically endocrine disrupting chemicals (EDCs), as critical yet underappreciated contributors to the PCOS epidemic. The ubiquitous presence of EDCs in modern environments, combined with their capacity to interfere with hormonal signaling at environmentally relevant doses, positions these substances as plausible etiological agents warranting intensive investigation[10].

Endocrine Disrupting Chemicals: Definition and Scope

The Endocrine Society defines endocrine disrupting chemicals as 'exogenous chemical, or mixture of chemicals, that can interfere with any aspect of hormone action[11]. This broad definition encompasses diverse substances sharing the common property of perturbing endocrine homeostasis through multiple mechanisms: mimicking natural hormones by binding to hormone receptors (agonism), blocking hormone binding (antagonism), interfering with hormone synthesis or metabolism, or modulating hormone receptor expression and signaling. EDCs operate through non-traditional dose-response relationships, often exhibiting greater potency at lower doses (non-monotonic dose responses) and producing effects during critical developmental windows that manifest later in life (developmental programming)[12].

Major EDC categories of concern for reproductive health include: (1) industrial chemicals such as bisphenol A (BPA) used in plastics and epoxy resins, and phthalates employed as

plasticizers; (2) persistent organic pollutants including polychlorinated biphenyls (PCBs), dioxins, and organochlorine pesticides like DDT; (3) currently-used pesticides including organophosphates, pyrethroids, and herbicides; (4) heavy metals such as lead, cadmium, and mercury; and (5) personal care product ingredients including parabens, triclosan, and UV filters (Table 1)[13]. Human exposure occurs primarily through diet (contaminated food and water), dermal absorption (cosmetics, consumer products), and inhalation (indoor air, dust). Biomonitoring studies demonstrate near-universal exposure to multiple EDCs simultaneously, with detection rates exceeding 95% for many compounds in general populations of industrialized nations[14].

Table 1. Major Categories of Endocrine Disrupting Chemicals Relevant to PCOS

EDC Category	Representative Compounds	Primary Exposure Sources	Endocrine Mechanisms
Bisphenols	Bisphenol A (BPA), Bisphenol S (BPS), Bisphenol F (BPF)	Polycarbonate plastics, epoxy resin linings in food/beverage cans, thermal paper receipts, dental sealants	ERα/ERβ agonism, androgen receptor antagonism, altered steroidogenic enzyme expression
Phthalates	DEHP, DBP, BBzP, DEP, DiBP, DiNP	Personal care products, PVC plastics, medical devices, building materials, food packaging	Anti-androgenic activity, PPARγ activation, disruption of steroidogenesis, insulin resistance promotion
Persistent Organic Pollutants	PCBs, Dioxins, DDT, DDE, PBDEs	Contaminated fish, meat, dairy products; legacy environmental contamination; bioaccumulation in food chain	AhR pathway activation, thyroid hormone disruption, estrogenic/anti-estrogenic effects
Pesticides	Organophosphates, Pyrethroids, Atrazine, Glyphosate	Agricultural produce, contaminated drinking water, occupational exposure, residential use	Aromatase inhibition, altered gonadotropin signaling, oxidative stress induction
Heavy Metals	Cadmium, Lead, Mercury, Arsenic	Contaminated soil, water, food; cigarette smoke; industrial emissions; legacy paint	Estrogenic activity, oxidative stress, steroidogenic enzyme disruption, epigenetic modifications
Personal Care Chemicals	Parabens, Triclosan, UV filters (BP-3, 4-MBC)	Cosmetics, shampoos, lotions, sunscreens, antibacterial products, fragrances	Weak estrogenic activity, anti-androgenic effects, thyroid hormone disruption

Abbreviations: ERα = estrogen receptor alpha; ERβ = estrogen receptor beta; DEHP = di(2-ethylhexyl) phthalate; DBP = dibutyl phthalate; BBzP = benzyl butyl phthalate; DEP = diethyl phthalate; DiBP = diisobutyl phthalate; DiNP = diisononyl phthalate; PPARγ = peroxisome proliferator-activated receptor gamma; PCBs = polychlorinated biphenyls; DDT = dichlorodiphenyltrichloroethane; DDE = dichlorodiphenyldichloroethylene; PBDEs = polybrominated diphenyl ethers; AhR = aryl hydrocarbon receptor; BP-3 = benzophenone-3; 4-MBC = 4-methylbenzylidene camphor.

Rationale for Examining the EDC-PCOS Connection

Several lines of reasoning support investigating EDC exposure as a contributor to PCOS pathogenesis. First, mechanistic plausibility: EDCs interfere with steroidogenesis, gonadotropin regulation, insulin signaling, and inflammatory pathways—all central to PCOS pathophysiology (Figure 1). Second, temporal concordance: the increase in environmental EDC burden parallels rising PCOS prevalence. Third, developmental sensitivity: PCOS may originate from prenatal programming, and EDCs exert profound effects during fetal development. Fourth, epidemiological associations: emerging studies demonstrate elevated EDC levels in PCOS patients. Fifth, experimental evidence: animal models show that EDC exposure induces PCOS-like phenotypes[15].

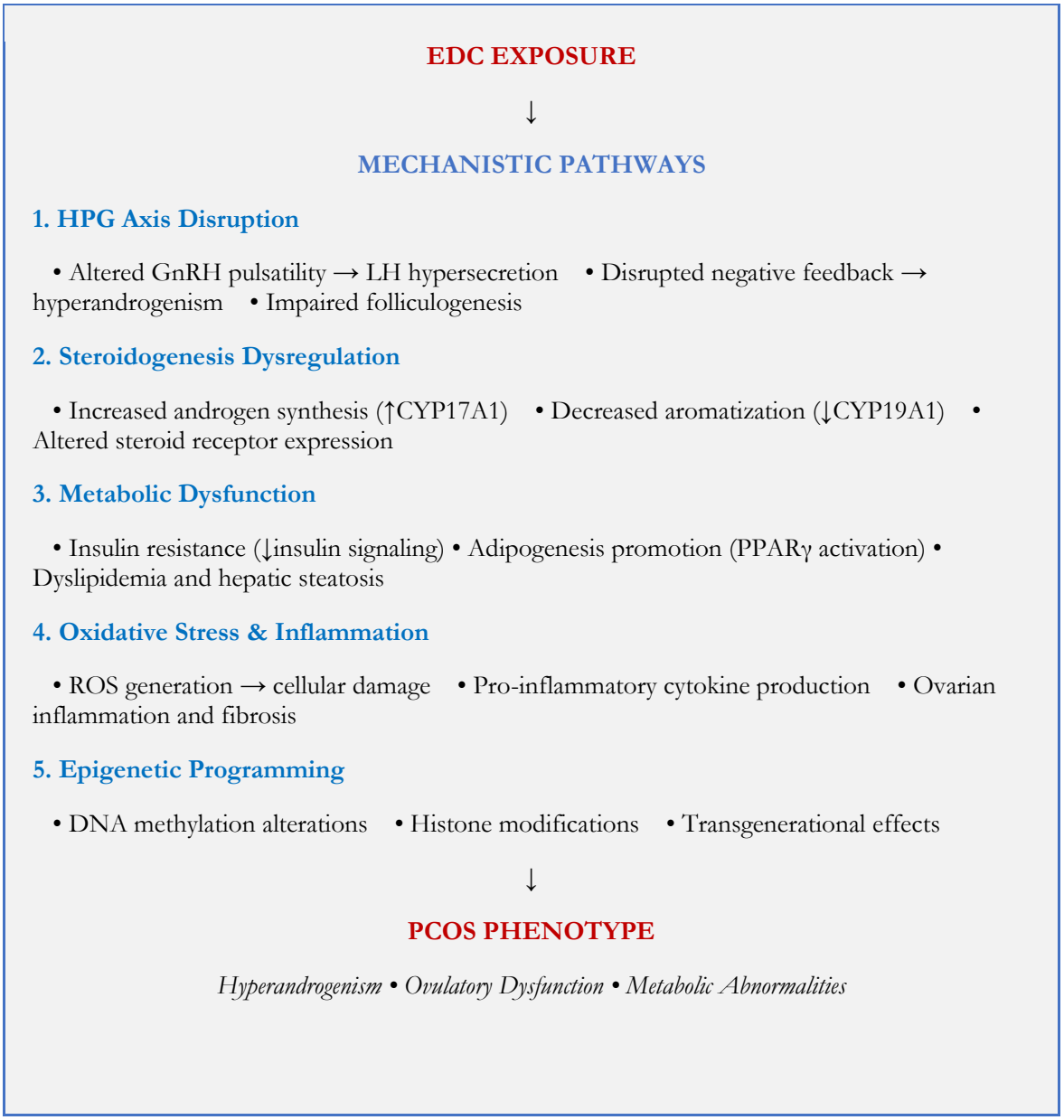

Figure 1. Proposed Mechanisms Linking EDC Exposure to PCOS Pathogenesis. This schematic illustrates the multiple biological pathways through which EDC exposure may contribute to PCOS development, emphasizing the multifactorial nature of environmental contributions to disease pathogenesis.

Abbreviations: HPG = hypothalamic-pituitary-gonadal; GnRH = gonadotropin-releasing hormone; LH = luteinizing hormone; CYP17A1 = 17α-hydroxylase; CYP19A1 = aromatase; PPARγ = peroxisome proliferator-activated receptor gamma; ROS = reactive oxygen species.

Understanding whether and how EDCs contribute to PCOS carries profound implications. Clinically, it may inform patient assessment, counseling, and potentially therapeutic strategies targeting exposure reduction. From a public health perspective, establishing causality could justify regulatory actions to minimize population exposures and prevent disease. This narrative review synthesizes current evidence examining the EDC-PCOS relationship, critically evaluating epidemiological data, experimental findings, and mechanistic insights to provide a comprehensive assessment of this emerging field and identify priorities for future research.

DISCUSSION

Epidemiological Evidence Linking EDCs to PCOS

Multiple case-control studies have documented elevated EDC biomarker levels in women with PCOS compared to age-matched controls. For bisphenol A, meta-analyses synthesizing data from over 1,500 participants demonstrate significantly higher serum and urinary BPA concentrations in PCOS patients, with pooled standardized mean differences of 0.89 (95% CI: 0.54-1.24)[16].Importantly, several studies reveal dose-response relationships, with women in the highest BPA exposure quartiles exhibiting 2-3 fold increased PCOS odds compared to lowest quartile[17]. Phthalate metabolite associations show chemical-specific patterns: DEHP and DBP metabolites correlate positively with hyperandrogenism and insulin resistance markers, while low-molecular-weight phthalates demonstrate weaker or null associations[18].

Persistent organic pollutants including PCBs and organochlorine pesticides also associate with PCOS in multiple populations, though effect estimates vary across studies, possibly reflecting differences in exposure profiles and phenotypic classification[19]. Critical limitations include the cross-sectional design of most investigations, which precludes causal inference, and the potential for reverse causation if PCOS-associated metabolic abnormalities alter EDC metabolism or excretion. Prospective cohort studies tracking exposures from before disease onset are urgently needed.

Experimental Animal Models and Mechanistic Insights

Animal models provide compelling evidence for EDC involvement in PCOS pathogenesis. Prenatal BPA exposure in sheep—a well-established PCOS model given sheep's long gestational period and reproductive physiology similar to humans—produces female offspring exhibiting all hallmark PCOS features: multifollicular ovaries, elevated anti-Müllerian hormone, LH hypersecretion, hyperandrogenism, and insulin resistance[20]. These phenotypes persist into adulthood and, remarkably, transmit to subsequent generations despite no direct F2 or F3 generation exposure, indicating epigenetic programming mechanisms[21].

Rodent studies demonstrate similar outcomes with prenatal or peripubertal exposure to BPA, phthalates, or pesticides inducing PCOS-like reproductive and metabolic abnormalities[22]. At the molecular level, EDCs disrupt multiple pathways central to PCOS: (1) altered steroidogenesis through modulation of CYP17A1, CYP19A1, and other steroidogenic enzymes; (2) disrupted gonadotropin regulation via effects on hypothalamic GnRH neurons and pituitary gonadotrophs; (3) induction of ovarian and systemic insulin resistance through interference with insulin receptor signaling and glucose transporter expression; (4) promotion of chronic low-grade inflammation via activation of NF-κB and production of pro-inflammatory cytokines; (5) epigenetic modifications including altered DNA methylation patterns in genes regulating reproduction and metabolism[23].

Clinical Implications and Knowledge Gaps

The EDC-PCOS evidence base carries important clinical implications despite remaining uncertainties. Patient counseling should address modifiable exposure sources, emphasizing pragmatic strategies to

reduce EDC burden: minimizing plastic food contact, selecting EDC-free personal care products, consuming organic produce when feasible, and filtering drinking water. For women planning pregnancy, preconception exposure reduction may protect offspring from developmental programming effects[24]. However, several critical gaps limit translation to clinical practice. First, biomarker utility: current evidence does not support routine EDC biomonitoring in individual patients, as single-timepoint measurements poorly reflect chronic exposure and lack validated clinical decision thresholds[25]. Second, intervention efficacy: no randomized trials have examined whether exposure reduction improves PCOS outcomes. Third, mixture effects: real-world exposures involve complex chemical mixtures with potentially synergistic or antagonistic interactions, yet research predominantly examines single chemicals. Fourth, phenotype specificity: whether EDCs associate differentially with PCOS phenotypes (reproductive vs. metabolic predominance) remains unclear. Addressing these gaps requires long-term prospective cohort studies, intervention trials, and mechanistic investigations examining chemical mixtures and gene-environment interactions[26].

CONCLUSION

This comprehensive narrative review demonstrates substantial and growing evidence that endocrine-disrupting chemicals contribute to polycystic ovary syndrome pathogenesis through multiple biological mechanisms. Epidemiological investigations consistently document elevated EDC biomarker levels in PCOS populations, with dose-response relationships supporting causality. Experimental animal models unequivocally demonstrate that EDC exposure, particularly during critical developmental windows, induces PCOS-like phenotypes that persist into adulthood and transmit across generations. Mechanistic studies elucidate biologically plausible pathways through which EDCs disrupt reproductive and metabolic homeostasis, interfering with steroidogenesis, gonadotropin regulation, insulin signaling, and inflammatory processes central to PCOS pathophysiology.

Despite this progress, significant knowledge gaps necessitate continued investigation. Prospective cohort studies with comprehensive exposure assessment from preconception through reproductive maturity are essential for establishing temporal relationships and identifying critical vulnerability windows. Intervention trials examining whether exposure reduction improves PCOS outcomes would provide crucial evidence for clinical translation. Research examining EDC mixture effects, gene-environment interactions, and phenotype-specific associations will refine understanding of susceptibility factors and disease heterogeneity. From a clinical perspective, while routine biomonitoring currently lacks justification, patient education regarding exposure reduction represents a prudent, low-risk intervention consistent with precautionary principles.

The EDC-PCOS connection underscores broader implications for reproductive health in an era of ubiquitous environmental chemical exposure. Public health strategies to minimize population-level EDC burden—through regulatory actions, consumer product reformulation, and awareness campaigns—may represent important preventive approaches for reducing PCOS incidence and associated morbidity. As evidence continues accumulating, integrating environmental exposure considerations into PCOS research, clinical practice, and public health policy will be essential for comprehensive disease prevention and management. The stakes are high: with PCOS affecting over 100 million women worldwide, even modest reductions in environmentally-attributable disease burden could yield substantial population health benefits.

RECOMMENDATIONS

For Clinical Practice

1. Incorporate environmental exposure history into PCOS patient assessment, including occupational exposures, residential environment, dietary patterns, and personal care product use.
2. Provide evidence-based patient counseling on practical EDC exposure reduction strategies, emphasizing modifiable sources: minimizing plastic food contact materials, selecting phthalate- and paraben-free personal care products, consuming organic produce when feasible, filtering drinking water, and avoiding thermal paper receipt handling.
3. Implement preconception counseling for women with PCOS planning pregnancy, addressing EDC exposure reduction to potentially minimize offspring developmental programming effects.

4. Advocate for EDC-free medical devices, pharmaceuticals, and clinical supplies, particularly for reproductive-age women and pregnant patients.
5. Develop standardized patient education materials on environmental risk factors for PCOS, suitable for diverse literacy levels and cultural contexts.

For Research Priorities

6. Conduct large-scale, multicenter prospective birth cohort studies with comprehensive EDC biomonitoring (measuring multiple chemical classes) from preconception through adolescence to establish causal relationships, identify critical exposure windows, and characterize dose-response relationships.
7. Investigate EDC mixture effects using innovative analytical approaches (e.g., weighted quantile sum regression, Bayesian kernel machine regression) that reflect real-world co-exposure scenarios rather than single-chemical paradigms.
8. Examine gene-environment interactions through integration of genomic data with exposure assessment to identify genetically susceptible subpopulations who may derive greatest benefit from exposure reduction interventions.
9. Develop and validate exposure biomarkers with improved temporal stability and specificity for chronic exposure assessment, overcoming limitations of current spot measurements.
10. Conduct randomized controlled intervention trials examining whether structured exposure reduction programs improve PCOS clinical outcomes (ovulatory function, metabolic parameters, quality of life) in women with established disease.
11. Investigate phenotype-specific associations to determine whether EDC exposure patterns differ between reproductive-predominant, metabolic-predominant, and mixed PCOS phenotypes.
12. Explore epigenetic mechanisms mediating transgenerational effects using multi-generational animal models and human family-based studies with epigenomic profiling.

For Public Health Policy and Regulation

13. Implement comprehensive population biomonitoring programs to track temporal trends in EDC exposure levels and evaluate effectiveness of regulatory interventions, with stratification by demographic factors and reproductive status.
14. Strengthen regulatory oversight limiting EDC use in consumer products, food contact materials, and personal care items, with particular protection for products marketed to or frequently used by pregnant women and children.
15. Mandate transparent labeling of EDC content in consumer products to enable informed purchasing decisions and facilitate market-driven demand for safer alternatives.
16. Develop public awareness campaigns targeting women of reproductive age, healthcare providers, and policymakers regarding EDC sources, health effects, and practical exposure reduction strategies.
17. Support green chemistry research and development initiatives to create safer chemical alternatives to current EDCs used in manufacturing, agriculture, and consumer products.
18. Integrate EDC exposure considerations into clinical practice guidelines for PCOS diagnosis, management, and prevention, reflecting current evidence while acknowledging remaining uncertainties.
19. Establish interdisciplinary collaborations between reproductive endocrinology, environmental health, toxicology, epidemiology, and policy sectors to facilitate translation of research findings into actionable prevention strategies.

For Individual Risk Reduction

20. Choose glass, stainless steel, or ceramic containers for food and beverage storage, preparation, and heating rather than plastic containers, particularly avoiding plastics labeled #3 (phthalates) and #7 (may contain BPA).
21. Minimize consumption of canned foods with BPA-containing epoxy linings; select fresh, frozen, or foods packaged in glass containers when available.

22. Select personal care products, cosmetics, and household cleaning products labeled as phthalate-free, paraben-free, and fragrance-free, consulting databases such as EWG's Skin Deep for product safety ratings.
23. Reduce handling of thermal paper receipts (gas station, ATM, store receipts); decline receipts when possible or request email receipts; wash hands after handling.
24. Prioritize organic produce for items on the 'Dirty Dozen' list with highest pesticide residues; thoroughly wash all produce with water.
25. Use high-quality water filtration systems (activated carbon or reverse osmosis) to reduce drinking water contaminants including heavy metals and pesticides.
26. Maintain adequate intake of dietary fiber, antioxidants, and cruciferous vegetables which may enhance EDC metabolism and excretion.
27. Vacuum frequently and wet-mop to reduce household dust accumulation, a significant source of phthalate and flame retardant exposure, particularly in homes with young children.

CONFLICT OF INTEREST

There is no conflict of interest.

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