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Endocrine Disruptors: Bisphenol and Its Relation to Polycystic Ovary Syndrome

COMMENTARY

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Abstract

Endocrine disruptors is an issue with great importance toxicological. Among them, there is bisphenol A (BPA) and its possible contributing towards the development of polycystic ovary syndrome (PCOS). Animal studies and cross-sectional models in humans demonstrate the harmful effects from exposure to BPA and drive the need to seek environmentally safe and healthy alternative to the use of BPA.

Keywords
Endocrine Disruptors; Polycystic Ovary Syndrome; Endocrinology.

Endocrine-disrupting compounds (EDCs) comprise a wide range of natural and synthetic compounds, which exhibit a potential to elicit negative effects on endocrine systems acting as mimics or antagonists of endogenous hormones, and, thus, may cause health effects in people and wildlife. They can cause adverse effects by interfering in some way with the body's hormones and may induce a broad spectrum of toxic responses at low environmentally relevant doses. These effects are of particular environmental concern since the middle of the 1990s. EDCs include natural and synthetic (anti)estrogens and (anti)androgens, pharmaceuticals, pesticides, industrial chemicals, and heavy metals. Many of these EDCs have been shown to have the ability to bind to estrogen or androgen receptors, thus disrupting the normal endocrine functions in organisms [1-6].

One of the most studied EDCs is bisphenol A (BPA; 2,2-bis (4-hydroxyphenol) propane). BPA was first synthesized by Dianin in 1891, and its estrogenic activity was discovered in 1936. In the 1950s, it was observed that BPA could be polymerized to make polycarbonate plastic, a miraculous cheap product that is lightweight, transparent, colorable,

resistant to impact, heat, and chemicals, inalterable with time, and easy to mold and thermoform. BPA can leach from food or beverage containers and is then ingested. This is the main source of contamination, although its ubiquitous distribution leads also to contamination through the skin, especially in the case of thermal paper [7], or via inhalation of household dusts [8].

Animal studies indicate that BPA affects reproduction, however, the gene-environment interaction mechanism(s) involved in this association remains unclear. Evidence shows that BPA can interfere with endocrine function of hypothalamic-pituitary axis, such as by changing gonadotropin-releasing hormones (GnRH) secretion in hypothalamus and promoting pituitary proliferation. Such actions affect puberty, ovulation and may even result in infertility. Ovary, uterus and other reproductive organs are also targets of BPA. BPA exposure impairs the structure and functions of female reproductive system in different times of life cycle and may contribute to infertility. Both epidemiological and experimental evidences demonstrate that BPA affects reproduction-related gene expression and epigenetic modification that are closely associated with infertility. The detrimental effects on reproduction may be lifelong and transgenerational [9].

Recently, there has been interest in whether endocrine-disrupting chemicals (EDCs) in the environment, particularly Bisphenol A (BPA), may contribute to the development of polycystic ovary syndrome (PCOS), which represents a common endocrine disorder among women of reproductive age. With a lack of a clear etiology for its generative roots, PCOS is now considered to enclose genetic and environmental components [10] leading to phenotypic variability. Traditionally, women with PCOS manifest both reproductive (chronic anovulation, hyperandrogenism, polycystic ovarian morphology) and metabolic (insulin resistance, metabolic syndrome, obesity) derangements. However, there is much heterogeneity of clinical and biochemical features raising the possibility that a cluster of etiological factors synergistically contribute to the final PCOS phenotype. In animal models, exposure to BPA during the perinatal period dramatically disrupts ovarian and reproductive function in females, often at doses similar to typical levels of human exposure. BPA also appears to have obesogenic properties, disrupting normal metabolic activity and making the body prone to overweight. In humans, cross-sectional data suggest that BPA concentrations are higher in women with PCOS than in reproductively healthy women, but the direction of causality has not been established [11, 12].

Due to the already known hazards caused by the exposure to BPA and the association to the variability of phenotypes in the PCOS, it is necessary to find low-cost, environmental safe and healthy alternatives to BPA. More biomonitoring studies are required to evaluate long term effects of the exposure to BPA, as most evidences are inconclusive.

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