Policy Statement on Environmental Endocrine Disrupting Chemicals & the Impact on Obesity and Cardiovascular Disease
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Position

The American Heart Association (AHA) recognizes that the causes of obesity are multi-factorial and complex, and therefore, must be addressed on multiple levels. Recently, endocrine disrupting chemicals (EDCs) such as diethylstilbestrol, bisphenol A, phthalates and organotins have been proposed as potential "obesogens" that contribute to a toxic chemical burden that may initiate or exacerbate the development of obesity and its related comorbitites.[1-7] EDCs are found in a variety of products including plastics, cosmetics, shampoos, soaps, lubricants, pesticides, paints and flame-retardant materials.[2, 8] Laboratory studies are still elucidating the exact mechanisms by which these substances affect weight, but current evidence suggests that they disrupt developmental and homeostatic controls over fat production and energy balance.[9-12] However, determining the link with obesity can be especially challenging because obese people might be eating more and therefore exposing themselves to more of the chemicals in food packaging. Teasing out causality can be challenging. Although limited research exists on the effect of these environmental chemicals on human populations, several epidemiological studies have found that chemical exposure, particularly during critical developmental periods, is positively correlated with increased weight, cardiovascular disease and diabetes.[8, 13-18] Additional research is needed to clarify these results and establish a causal link between exposure to EDCs and adverse health effects in humans as well as discern the physiological/cellular/metabolic impact of exposure. The AHA recommends further research before taking a proactive advocacy position.

Future Research Questions:

1. What is the overall health burden of these chemicals with long-term, cumulative exposure over a life-time, versus short-term use?
2. What proportion of susceptibility to obesity is explained by chemicals in the environment?

Background

Endocrine disrupting chemicals are defined as "compounds that mimic or interfere with the normal actions of endocrine hormones including estrogens, androgens, thyroid, hypothalamic and pituitary hormones."[2] Some EDCs, such as phytoestrogens, are found in nature, but man-made EDCs are believed to pose a more significant risk to human health.[2] Industrially-produced EDCs include diethylstilbestrol (DES), bisphenol A (BPA), phthalates and organotins. They are found in a variety of products such as plastics, cosmetics, shampoos, soaps, lubricants, pesticides, paints and flame-retardant materials [2, 8]

Because classical toxicology focuses on identifying toxic doses of chemicals that result in death, malformation or low birthweight, EDCs have long been considered "safe." Recent research, however, has demonstrated that EDCs do in fact cause subtle epigenetic changes that disrupt hormonal signaling and cell differentiation.[6] Exposure during critical periods of development, such as pregnancy, as well as chronic exposures over long periods of time may lead to increased susceptibility to disease later in life, including obesity, cardiovascular disease and diabetes. [8, 13-18]

In 2002, Ballie-Hamilton proposed that chemical toxins played a role in the development of obesity by noting that the rise in obesity has paralleled the increased use of industrial chemicals over the last 40 years.[1] Over the past decade, several studies have attempted to elucidate this relationship. In 2009, Chen postulated that EDCs influence obesity by either stimulating or inhibiting energy metabolism pathways and expression of key enzymes involved in the regulation of energy balance.[19] Several animal studies have supported this hypothesis.[3, 9, 10, 17] Phthalates were found to alter hepatic energy metabolism, suggesting a potential role in development of metabolic disorders.[9] Organotins were found to lead directly to adipocyte differentiation, potentially predisposing exposed individuals to obesity and related metabolic disorders.[3] Perinatal exposure of rats to low dose BPA increased adipogenesis in female rats at weaning, possibly by altering the "set point" for adult weight.[17]

Although evidence of the effects of EDCs on humans is currently limited, several studies have demonstrated correlations between EDC exposure and adverse health effects. High concentrations of urinary BPA was found to be associated with a diagnosis of cardiovascular disease, diabetes and liver-enzyme abnormalities.[13, 15] The presence of certain increased phthalate metabolites in urine was associated with abdominal obesity, insulin resistance, increased body mass index and higher waist circumference in males aged 20-59.[8, 20] Prenatal exposure to EDCs has also been correlated with increased weight and body mass index in adult female offspring, and may therefore contribute to obesity in women.[18] However, additional research is needed to establish conclusive, causal relationships and further elucidate adverse effects on human health.

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