Endocrine disruptors: Ubiquitous, yet less known

Sir,

Though Endocrine disruptor chemicals (EDs) are ubiquitous in nature, and have many known ill-effects on humans, the awareness about them, unfortunately, is very limited. EDs have shown to impair hormonal balance, which can result in a variety of developmental and reproductive abnormalities. Thus EDs have played a role in increased incidence of malformations and neoplasms of the male genital tract, thereby causing decreased sperm quality; alterations in both the male and female reproduction; changes in the normal working of the neuroendocrinology system; changes in behavior; obesity; metabolic system associated conditions like type 2 diabetes, and also diseases related to the thyroid and cardiovascular endocrinology.

Prenatal phthalate exposure is associated with greater social deficits and poorer cognition, social communication and awareness. Bisphenol A (BPA) levels show significant correlation with testosterone, androstenedione and insulin resistance; and, are significantly higher in women who are lean or overweight and suffer from polycystic ovary syndrome (PCOS). Diethylstilbestrol (DES) and BPA induce persistent epigenetic changes in the developing uterus, and while the former is associated with an increased risk of breast cancer in adult women, the latter is shown to induce neoplastic changes in the mammary tissue in animal models. Treatment of human breast adenocarcinoma cell line Michigan Cancer Foundation - 7 (MCF-7) with DES or BPA led to a three and two fold increase in Histone-lysine N-methyltransferase (EZH2) messenger ribonucleic acid (mRNA) expression and EZH2 protein expression, respectively.[1] Maternal and paternal occupational exposure to EDs was associated with an increased Time to Pregnancy (TTP).

EDs like dihydrotestosterone equivalents (in smokers, heavy drinkers, and in those exposed to disinfectants or welding/soldering fumes) and 17β-estradiol equivalents (in those exposed to pesticides, disinfectants, and exhaust fumes) are more ubiquitous than we think.

EDs have also been found in bottled water due to leaching action, and the contents of the polyethylene terephthalate (PET) per bottle, the storage-temperature and the pH may influence the rate and magnitude of leaching (phthalates and antimony leach into lower pH and/or warmer products). In a study on bottled water from France, Germany, and Italy, significant estrogenic response was seen in about 60% bottled water samples, which showed relative proliferative effects 2 on MCF-7 ranging from 19.8 to 50.2%.[2] When comparing water of the same spring, estrogenic activity is three times higher in water from PET bottles than glass.

EDs like atrazine, via aromatase induction, cause an increase in the levels of estradiol and lower testosterone. Methoxychlor, apart from aromatase induction, also down-regulates hydroxysteroid (17-beta) dehydrogenase 3 (HSD17B3) and produces similar effects.[3] In the city of Rio de Aveiro, elevated EDs’ concentrations’ were observed, including alkylphenol ethoxylates (A9PEO), o-phenylphenol (PhP), nonylphenoxy ethoxy acetic acids and BPA.[4] A study showed that apart from prediction of type 2 diabetes and cardiovascular diseases, persistent organic pollutants like dichloro- 2,2-bis(p-chlorophenyl) ethylene (p,p'-DDE), consistently predicted higher body mass index (BMI) and triglyceride levels, homeostasis model assessment of insulin resistance (HOMA-IR) and lower High-density lipoprotein (HDL) cholesterol levels.[5] Although a direct and absolute causal linkage might not be feasible owing to different types of bias in some cases, strong history of exposure to EDs, especially depending upon the duration and degree of exposure, combined with behavioral/lifestyle risk factors and similar (herd) manifestations from culprit EDs, can reinforce the causality.

Avoiding household pesticides, checking for sources of food contamination, avoiding heating/storing food in plastic containers (using glass/stainless steel instead) and proper education, can help to curb some of the disastrous effects of EDs. The need for more awareness and encouragement for in-depth research into the sources and effects of EDs is also being felt.

EDs have a gargantuan role in causing a variety of disorders and there is an urgent need to wake up to their manifold effects.

Dilip Gude
Department of Internal Medicine, Medwin Hospital, Nampally, Hyderabad, Andhra Pradesh, India

Corresponding Author: Dr. Dilip Gude, AMC, 3rd Floor, Medwin Hospital, Chirag Ali lane, Nampally, Hyderabad, Andhra Pradesh – 500 001, India. E-mail: letsgo.dilip@gmail.com
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Loss of follow-up of diabetic patients: What are the reasons?

Sir,

Diabetes mellitus (DM) is a very common disease in general clinical practice. In each hospital, the DM clinic is usually crowded with many diabetic patients. An important concept in managing diabetic cases is to have a good control of blood sugar of the patients. Hence, it is no doubt that long-term follow-up of the patients is required. However, an important practical problem in general practice is the poor control of DM and an important reason is loss of follow-up. Here, the author would like to show some interesting experience on 200 diabetic patients who are regular diabetic patients in a primary care unit and had the history of loss of follow-up.

A simple question for every patient is “What is the reason for loss of follow-up?” Focusing on the answers, the following reasons are given: (a) patients think that the blood glucose is already in control (from the data in previous laboratory report) (110 cases, 55%), (b) loss of appointment card (40 cases, 20%), (c) busy on the appointment date (20 cases, 10%), (d) the appointment date is the holiday vacation period (10 cases, 5%), (e) they try to have other alternative treatment(s) (10 cases, 5%), and (f) fear of side effects of prolonged usage of antidiabetic drugs (10 cases, 5%).

Of interest, it can show that many patients who lost for follow-up have the reasons according to poor attitude and knowledge on DM. This is already described in some similar studies done previously in other settings[1-2]. Indeed, the importance of DM education for general population is recently mentioned in this journal by Pal et al[3]. The author hereby would like to add that a more specific education to the one with disease is as important.

Viroj Wiwanitkit
Wiwanitkit House, Bangkhae, Bangkok-10160, Thailand; Visiting University Hospital, Hainan Medical University, China
Corresponding Author:
Prof. Viroj Wiwanitkit,
Wiwanitkit House, Bangkhae, Bangkok-10160, Thailand.
E-mail: wviroj@yahoo.com

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