Endocrine Disruptive Chemicals: Silent Poison for Human Health

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Human exposure to environmental endocrine disrupting compounds (EDCs) have received increased attention in the past two decades due to the increase in documentation of widespread exposure of these compounds to humans and their effects on human health. An EDC is a foreign substance that cause adverse health effects in an organism, and/or its progeny as a result of changes in endocrine system. The normal physiological function of the endocrine system is dependent on hormones which act as chemical messengers to regulate normal functions of our body [1]. However, EDC’s can interfere in the normal function of the action of hormones at different steps of intracellular signal transduction pathways. The endocrine disruptive agents include but not limited to pesticides, polychlorinated biphenyls (PCBs), polybrominated biphenyls (PBBs), dioxins phthalates, bisphenol A (BPA), polybrominated diphenyl ethers (PBDEs), air pollutants (specifically six major air pollutants recognized by WHO), phytoestrogens, DDT, Di(2-ethylhexyl) phthalate (DEHP) among others.

Concerns about environmental endocrine disruptors began in 1962 after Rachel Carson published the monumental book called “Silent Spring” [2]. She claimed that the unselective use of chemicals, especially pesticides, badly affects the environment. Dichlorodiphenyltrichloroethane (DDT) was developed to prevent malaria epidemic by controlling mosquitoes but was later found to cause cancer and other diseases. The United States Environmental Protection Agency and the United Nations Environment Programme (UNEP) were established in 1970 and 1972, respectively, to pay closer attention to the impact of environmental chemicals on human health. United Nations now controls “Persistent Organic Pollutants (POPs),” demarcated as “chemical substances that persist in the environment, accumulate in our body through the food chain, and pose a risk on by adversely affecting human health and the environment,” by establishing Stockholm Convention (http://chm.pops.int/TheConvention/ThePOPs/tabid/673/Default.aspx).

Endocrine system is one of the most delicate communication systems of the human body which influences all aspects of human health and well-being, including reproductive potential, cognitive functions, thyroid and metabolism, digestion and hormonal balance. Lately there has been a lot of progress in the research that has been focused on the potential relationship between environment contaminants and cellular endocrine function. These endocrine disruptors mimic naturally occurring hormones in the human body such as thyroid hormones, androgens and estrogens. These chemicals bind to the receptor within the cell and block the endogenous hormone from binding, which leads to the interference or blockage of natural hormones to their receptors specifically by altering their metabolism in the liver. Furthermore, the epidemiologic data on the environmental EDCs advocate a clear speculation/association between exposure and adverse health outcomes in human systems [3].

Lately, the prevalence of obesity around the world has increased dramatically. A strong evidence has emerged over the last decades that human exposure to numerous endocrine disrupting chemicals (EDCs) may be a contributing causal factor in obesity and obesity-related metabolic diseases. Since many EDCs are manmade chemicals that are released into the environment, there is no endogenous mechanism in the human body system by which these xenobiotics get cleared out of the body. As a result, these xenobiotics buildup in the body and generate toxic effects. Also, we know EDCs are exogenous compounds that interfere with hormonal regulation and normal endocrine
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systems, thereby affecting the health of animals and humans. Furthermore, obesity and obesity-related metabolic diseases commence with fat cell development which is driven and modulated by nuclear hormone receptor signaling [4-7]. EDCs that activate or inhibit these hormone pathways may be involved in promoting modulation of fat cell development, hormonal control of appetite and satiety, basal metabolic rate, energy homeostasis and brain circuitry controlling food intake and energy expenditure and ultimately contributing to the development of metabolic syndrome [3]. However, we have very limited human data, and in many cases inconsistent data across studies, which highlight the need for systematic research on these chemicals. Future longitudinal molecular epidemiology studies with appropriately designed exposure assessments are needed to determine potential causal relationships, to identify the most important time gaps/life stages of exposure, and to define individual susceptibility factors for adverse effects on human’s health in response to exposure.

Recent, studies on the epigenetic mechanisms involved in mediating the effects of EDCs on female reproductive system are gathering momentum. There is large plethora of literature documentation that describe developmental processes that are susceptible to EDC exposures in female reproductive system, with a special emphasis on the ovary. Also, there are studies with select EDCs that have been shown to have physiological and correlated epigenetic effects in the ovary, neuroendocrine system, and uterus. Importantly, EDCs that can directly target the ovary can alter epigenetic mechanisms in the oocyte, thus leading to transgenerational epigenetic effects [8]. Epigenetic markers are also connected with depression symptoms. There are number of reports on association between depression and environmental estrogenic disruptors (EED’s) exposure, such as PCBs, BPA, arsenic and phthalates. Importantly, EDCs that can directly target the ovary can alter epigenetic mechanisms in the oocyte, leading to transgenerational epigenetic effects [9,10]. There are emerging data from animal studies that have shown that perinatal EDC exposure can cause epigenetic alterations, and these can be modified by hormone stimulation. However, more studies need to be conducted to get more clarifying insight on how these effects can be remedied to further improve public health.

Bibliography