Endocrine Disruptors and Human Target Cells

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Endocrine disrupting chemicals (EDCs) are compounds in the external environment that mimic or block endogenous hormones they behave as biological signals and, as such, can be misinterpreted by the organism. To date, the vast majority of known EDCs are those that activate the parts of the endocrine system associated with the steroid/retinoid/thyroid super-family of receptors, most often receptors related to the hormone estrogens that are found in all vertebrates. Endocrine-disrupting chemicals (EDCs) in the environment have been linked to human health and disease. Fetal exposure to environmental endocrine disruptors may contribute to the increased incidence of male genital tract malformations, decreased sperm quality, several neoplasms, and altered body weight. Exposure to EDCs early in life can increase risk levels of compromised physical and mental health. Epigenetic mechanisms have been implicated in this process. The ability of an environmental factor (i.e. endocrine disruptor) to promote an epigenetic trans-generational phenotype impacts the potential hazards of environmental toxins, mechanisms of disease, etiology, and evolutionary biology.

There are many problems hindering the study of endocrine disruption (reductionist stance, technically driven research biases, and study of single end points, chemicals and exposure periods).

Time exposure may be short or throughout the individual's lifetime. To understand what effect they might have, the individual's social and biological environment, namely its ecology, must be considered. This ecology includes interactions between and within species; for example, symbiotic signaling, predatory- prey interactions, males and females during mating, mother-embryo interactions, and, in mammals, even embryo-embryo interactions.

Environmental chemicals with hormone-like activity can disrupt the programming of endocrine signalling pathways that are established during perinatal life and result in adverse consequences that may not be apparent until much later in life.

Some data suggest new targets (i.e. adipocyte differentiation and mechanisms involved in weight homeostasis) of abnormal programming by EDCs, and provide evidence that support the scientific term 'the developmental origins of adult disease'. The emerging idea of an association of EDCs and obesity expands the focus on obesity from intervention and treatment to include prevention and avoidance of these chemical modifiers. Advanced molecular epidemiological designs based on markers of molecular and epigenetic damages will consistently improve knowledge on the impact on human health of exposure to EDCs.