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Opinion

Gestational Dioxin Acts as Developing Neuroendocrine-Disruptor

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A higher transplacental passage of thyroid hormones (THs) from pregnant to their fetuses is necessary for the fetal and neonatal development [1-47]. 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) belongs to polychlorinated aromatic hydrocarbons (PAHs) [1,48,49], and accumulates in the human food chain [50-55]. In addition, TCDD can pass placenta to alter thyroid functions [1,56-58], and cause a perinatal hypothyroidism [1,51,59-62], growth retardation [63,64], permanent brain damage [65], neonatal toxicity [1,50,51] and persistent effects during childhood [66]. In addition, Hattori., *et al.* [67] reported that maternal TCDD can decrease the expression of growth hormone (GH) and the levels of glucocorticoid in pregnant dams and their newborns. Numerous studies have been shown that the maternal TCDD can disrupt the fetal pituitary-gonad axis and the neonatal sexual behaviors [68] by disrupting the thyroid hormone receptors (TRs; α and β), mineralocorticoid receptor, glucocorticoid receptor (GR), progesterone receptor (PR), androgen receptor (AR), and steroid hormone receptors [51,69]. In addition, these alterations may be mediated by inducing the aryl hydrocarbon receptor (AhR) [1,51,70-79] and the uridine diphosphoglucuronosyl transferase (UDPGT), a TH metabolizing enzyme, in liver to stimulate the removal of T4 [50,51,60]. In general, when animals are exposed to PAHs, they show neural disorders comparable to those present in cretinism both in humans and animals [80-82]. Thus, TH-disrupting chemicals can cause apoptosis in the fetal neural cells and deteriorate the normal neural circuit [83]. This suggests that TCDD can cause developmental neuroendocrine disorders.

It should be noted that the maternal TCDD may act as fetal/neonatal neuroendocrine-disrupting actions. This disruption may be depending on the concentration and period of TCDD exposure and the species involved. Further studies are essential to recognize the numerous neuro-molecular mechanisms of TCDD, mainly the AhRs. There are a lot extra evidences required to explore the link between the molecular and toxicological/epidemiological information.

Conflict of Interest

The author declares that no competing financial interests exist.

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