Dear Editor,

Polychlorinated aromatic hydrocarbons (PAHs) include polychlorinated dibenzofurans (PCDFs), polychlorinated biphenyls (PCBs), and polychlorinated dibenzo-p-dioxins (PCDDs). These endocrine-disrupting compounds (EDCs) are stored in animal and human tissues, causing a hormonal disturbance and developmental defects. In addition, they have the ability to cross the placenta producing several toxic effects on the placental tissue, and fetal/neonatal development. These toxic effects might be associated with variations in thyroid functions which are necessary throughout the regular development. Administration of PCB 95 at 32 mg/kg/d to newborn rats caused hypothyroidism and thyroid dysgenesis (DNA fragmentation) at postnatal day 18. Furthermore, hypothyroidism was observed in children living near PCB-contaminated sites. This may cause several adverse developmental outcomes. These disturbances might be attributed to the activation of the aryl hydrocarbon receptor (AHR). In addition, EDCs suppress the production of growth hormone (GH), insulin growth factors (IGFs), and their kinases signaling mechanisms. These alterations were further explained by other mechanisms as follows: (1) Disrupting the pituitary/hypothalamus axis, particularly hypothalamic-pituitary-thyroid axis (HPTA), hypothalamic-pituitary-ovarian axis (HPOA), and hypothalamic gene expression. (2) Activating mitogen-activated protein kinase (MAPK) cascades, cytochrome P450 (CYP) enzymes, and oxidative stress in the placental tissues. (3) Stimulating the phospholipase A2 (PLA), Ca2+ influx, protein kinase C (PKC), and caspase cascade as apoptosis instigation. (4) Inducing inflammation and oxidative stress through the aryl hydrocarbon receptors (AHRs), peroxisome proliferator activated receptors (PPAR-α), pregnane xenoobiotic receptor (PXR), constitutive androstane receptor (CAR), and androgen (AR) and estrogen receptor (ER).

Furthermore, exposure to EDCs causes dysmetabolic effects through their communication with transcription factors such as AHR, PXR, and CAR. On the basis of these results, it can be inferred that exposure to EDCs may probably induce uteroplacental dysfunctions and contribute to some developmental thyroid problems. Their toxicity can be attributed to the concentration and period of EDCs exposure, developmental period, and sex type of the species involved. Further investigations are required to elucidate the potential associations with human health. Additional information is desired to determine neurotoxic effects of EDCs during the prenatal and postnatal periods (fetuses/neonates).

Ethical Approval
Not applicable.

Competing Interests
Author declares that he has no potential conflict of interests.

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