The Maternal Thyroid Gland as A Sentinel Organ for A Development: Signs of The Possible Harm of Air Pollution in Development

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COMMENTARY

The systematic modifications in the maternal thyroid hormones (THs) levels during pregnancy are needed to get a regular fetal and neonatal development (El-bakry et al., 2010; Ahmed, 2011, 2013, 2015, 2016a-c, 2017a-c & 2018a-c; Ahmed et al., 2008; 2010, 2012; 2015 a, b & 2018 a, b; Ahmed and Incerpi, 2013; Van Herck et al., 2013; Incerpi et al., 2014; Candelotti et al., 2015; De Vito et al., 2015; El-Ghareeb et al., 2016; Ahmed and El-Gareib, 2017). Air pollution includes a complicated mixture of liquids, gases, and particulate matter (PM10, 2.5 or 0.1 mm) (Brook et al., 2004; Pope and Dockery, 2006; Sun et al., 2010). The other pollutants involve polycyclic aromatic hydrocarbons (PAHs), ground-level ozone (O3), nitrogen dioxide (NO2), sulfur dioxide (SO2), nitric oxide (NO), and carbon monoxide (CO) (Zheng et al., 2016). In addition, the major sources of air pollutants are indoor cooking, motor vehicle emissions, construction and demolition actions, industrial combustions, and power plants (Lee et al., 2014). Tobacco smoking is an extra appropriate source of exposure to heavy metals includes lead (Pb) and cadmium (Cd) (Mussalo- Rauhamaa et al., 1986; Marano et al., 2012). Inhalation of the polluted air is the chief pathway of exposure (Pronczuk- Garbino, 2007). Thus, all humans are exposed to these pollutions in particular in low- and middle-income countries (Zheng et al., 2016).

There has been cumulative concern about the harmful health consequences of air pollutants. The exposure to airborne PAHs (Baccarelli et al. 2008; Abdelouahab et al. 2013), active or passive cigarette smoke (Soldin et al. 2009) and cadmium (Iijima et al. 2007) might alter the levels of 3,5,3'-triiodothyronine (T3) and thyroxine (T4) in neonates and adults. Exposure to ambient PM2.5 pollution during the gestation can increase the risk of adverse gestational outcome (Pedersen et al. 2013) including the preterm birth (Rappazzo et al. 2014) and low birth weight (Pedersen et al. 2013). More importantly, several authors (Medici et al., 2013; Korevaar et al., 2016; Janssen et al., 2017) reported that exposure women to airborne PM2.5 during pregnancy can disrupt the activities of free T4, free T3 and thyroid-stimulating-hormone (TSH) and contribute to decrease the birth weight. Moreover, PM exposure in rats can perturb the action of hypothalamic–pituitary– thyroid axis (HPTA) (Thomson et al. 2013). Exposure pregnant to urbane air might induce premature deaths (Cohen et al., 2017). These variations may be attributed to PM exposure during the gestation can increase the activity of glucocorticoid (Thomson et al. 2013) suppressing the release of TSH (Wilber and Utiger 1969). Also, the anti-inflammatory actions of glucocorticoids can induce the response of a systemic oxidative stress (Janssen et al. 2012) and elevate the placental protein-bound 3-nitrotyrosine (Saenen et al. 2016). Alternatively, PM2.5 can enter the lungs and blood circulation, where they may prompt the release of oxidative stress resulting in several neurodevelopment complications (Grahame et al., 2014). Furthermore, the disturbance in the activity of THs during pregnancy can cause several attention deficit and hyperactivity disorder (ADHD) in neonates (Modesto et al. 2015). Costa et al. (2017) and
Ye et al. (2017) observed that exposure pregnant to air pollutants during the late pregnancy or early postnatal period can increase the possibility of autism spectrum disorders and cognitive deficits in children. In general, exposure to air pollutants can cause several neurological diseases such as schizophrenia (Pedersen et al., 2004), depression (Lim et al., 2012), and dementia (Power et al., 2016; Tzivian et al., 2016; Chen et al., 2017). Moreover, exposure to air pollution can cause mortality and morbidity from cardiovascular and respiratory diseases (Pope et al., 2002 & 2007; Pelucchi et al., 2009; Newby et al., 2015).

Finally, the current short commentary proposed that air pollutants may act as developmental endocrine disruptors perturbing the actions of HPTA and may delay the development and growth. This disturbance during gestation may increase the fetal and neonatal complications including teratogenic outcomes, preterm birth, and several brain disorders. Additional investigation is required to assess possible outcomes later in life.

**CONFLICT OF INTEREST**

The author declares that no competing financial interests exist.

**REFERENCES**


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