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Short Communication

Maternal Alcohol Consumptions Act as Developmental Neuroendocrine-Disrupting Actions: Hard Facts and New Thinking

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Maternal thyroid hormones (THs) have vital trophic factors for the regular somatic development [1-74]. In addition to their well-known action in cellular metabolism, 3,5,3'-triiodothyronine (T3) and thyroxine (T4) have significant roles upon neural differentiation and growth [67,70]. On the other hand, the maternal alcohol consumption during gestation can influence on the maternal and fetal hormones, and the fetal and neonatal development [75-83]. In general, the gestational alcohol consumption can disrupt the functioning of several neuroendocrine axes such as the hypothalamic-pituitary-thyroid axis (HPTA), the hypothalamic-pituitary-adrenal axis (HPAA), the hypothalamic-pituitary-gonadal axis (HPGA), and the growth hormone (GH)/insulin-like growth factors [84-86]. The maternal alcohol ingestion can cause a fetal alcohol spectrum disorder [87] and decrease the levels of serum total T4 [88], free T4, free T3, and thyroid-stimulating hormone (TSH) in both pregnant and newborns (a teratogenic outcome) [75,89,90]. These disabilities can increase the risk of mental retardation, cognitive and behavioral defects that illustrate Alcohol-Related Neurodevelopmental Disorder (ARND) [83,91-93]. In the USA, about 2-5% of young children are influenced by the fetal alcohol spectrum disorder [94]. In other instance, fetal alcohol spectrum disorder in human [95] or experimental animal models [96,97] can impair the development of hippocampus and cause learning and memory disability [98-100]. This impairment can be attributed to the following: (1) abnormality in the levels of THs during the development [75]; and (2) disruption the expression of thyroid hormone receptors (TR) and genes in the hippocampus. Another explanation is that the deprivation in essential nutrients or poor maternal nutritional status due to alcohol consumption can decrease the birth weight, and cause several physical malformations, and cognitive and behavioral dysfunctions [81,101]. In human [102,103] or animal [83,104] examinations, most of the above defects due to maternal alcohol consumption can transfer to the subsequent generations. Despite the high prevalence of gestational alcohol consumption, the mechanisms through which ethanol produces these disturbances or how ethanol might disturb the future generations are unknown.

From the previous data, this mini-review assumed that the maternal alcohol consumption at any point of time during pregnancy can penetrate the placenta and disrupt the functions and interactions of maternofetal THs and TSH (alter the hormonal synthesis or release). These disorders may cause a fetal alcohol syndrome, contribute to several fetal and neonatal adverse disruptions (teratogenic outcomes and food intake imbalance), impair the development of thyroid-brain axis, and inhibit the differentiation of the neuroendocrine system. In general, gestational alcohol consumption can inhibit the embryogenesis, and impair neural organization. Thus, the gestational alcohol consumption may act as developmental neuroendocrine-disrupting actions during the prenatal and postnatal periods. This consideration is significant during thyroid-brain development and may elucidate the cognitive disorders, behavioral dysfunction, and mental retardation that are observed in the gestational alcohol consumption. These disorders may be depending on the time and quantity of alcohol consumption, and a family history of alcohol consumption. Until now, it is not clear whether the reported effects of gestational alcohol consumption on the growth of the neuroendocrine system in experimental animals might be appropriate to human health. Thus, following the activities of maternal THs during the gestational alcohol consumption should be required to avoid the above disturbances in both fetus and newborn. Additional studies are crucial to follow the clinical and pathological aspects of maternofetal alcohol consumption and developmental thyroid-brain dysfunctions.

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