

Maternal Iodine Excess and Pregnancy Complications: Intimate Relations



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Letter to Editor

The applicable maternal Thyroid Hormones (THs; Thyroxine (T₄) and 3,5,3-Triiodothyronine (T₃)) levels during the gestation are essential for the regular fetal development [1-72]. Iodine is a crucial micronutrient for THs synthesis [62,73-93]. Also, iodide can enter the thyrocytes through the activity of the Sodium-Iodide Symporter (SIS) [65,90]. The levels of iodine remain increased in the placenta and amniotic fluid during the progress of gestation [89,92] and in breast milk during the lactation period [7,81].

On the other hand, several studies reported the association between iodine excess and thyroid disease [62-75]. In addition, the consumption of iodine excess due to widespread environmental iodine exposure including higher salt ingestion or overstated usage of kelp supplements are also associated with thyroid dysfunctions [65-79]. In particular, increase the consumption of iodine excess during the gestation and lactation periods can increase the risk of hypothyroidism, subclinical hypothyroidism or hypothyroxinemia [80-93]. These disorders can cause several fetal disorders [51,79,85] and neurocognitive dysfunctions in children [62]. In addition, excess iodine exposure can cause an acute Wolff-Chaikoff effect [63].

These disorders can be explained as the following [63-79]:

- Inhibition in the organification of iodine by an autoregulatory mechanism
- Reduction in the activity of Thyroid Peroxidase (TPO)
- Inhibition in the thyroid hormone synthesis and
- Development of hypothyroidism.

More importantly, Serrano-Nascimento et al. [84] reported that exposure male and female rats to iodine excess during the gestation and lactation periods alters the behavior of maternal Hypothalamus-Pituitary-Thyroid axis (HPTA), causes maternal hypothyroidism and disrupts iodide transfer to the milk. This state can promote deleterious actions on the development of neonates [75].

Finally, it is also worth revealing that the steady association between the dams and their fetuses depends on the stability in the actions of THs and in the concentration of iodine during pregnancy. As well, any disorders or excess in the concentration of iodine during pregnancy may cause several pregnancy disorders, hypothyroidism, and disrupt the fetal brain development. Further studies are needed to clarify the frequency as well as the temporal association between excess maternal iodine exposures, thyroid dysfunction and brain disorders (neurobehavioral development outcomes in infants and toddlers). In conclusion, the effects of maternal iodide uptake inhibitors on the maternal iodine status during pregnancy are required.

Conflict of interest

The author declares that no competing financial interests exist.

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