Maternal Thyroid Dysfunction, Intrauterine and Fetal Growth Restriction

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Letter to Editor

The bioavailability of maternal thyroid hormones (THs) during pregnancy is critical for the development and growth of fetus and neonates [1-58]. During the second half of gestation, thyroxine (T4) and 3, 5, 3'-triiodothyronine (T3) induce intrauterine growth through increase the fetal anabolic state/metabolic state and the positive action on the growth regulatory factors and other endocrine systems [59-60]. In late gestation period, the prepartum elevation in the bioavailability of T3 can mediate the maturational actions of the glucocorticoids and increase the functionality of the sympathetic nervous system [60-62]. In turn, this mediation can protect the neonate from the stress of delivery and from the new extrauterine environment [60].

On the other hand, intrauterine growth restriction or fetal growth restriction, a heterogeneous syndrome, means a fetus that cannot reach its growth potential [63-65]. These defects, impair fetal growth and compromises the neonatal adaptation to extrauterine life, can be attributed to the deficiency of THs during intrauterine development [66-68]. These data are reinforced by the results of fetal growth restriction in human and experimental animals (undernutrition and placental insufficiency) [69-72]. Also, hypothyroidism in sheep, goats, horses, and pigs induces the fetal growth restriction [60,73,74]. In epidemiological studies, the presence of anti thyroid antibodies (ATA) can increase the risk of intrauterine growth restriction or small for gestational age (SGA; infants are smaller in size and weight than normal) [65,75,76]. More interestingly, the risks of birth hypoxia, perinatal death, neonatal obstacles, neurodevelopmental and metabolic syndromes during adult life such as hypertension, type 2 diabetes, and coronary heart disease were increased during intrauterine and fetal growth restriction [77-81]. Indeed, intrauterine growth restriction is related to the gestational hypertensive disorders, under nutrition, infection, smoking, and unexplained factors [82]. Sharma [83] reported that intrauterine growth restriction is a main and silent cause of fetal and neonatal morbidity and mortality.

Conclusion

In conclusion, it is also worth stating that THs promote the general body growth and the development of fetal individual tissues and organs. In addition, any disorders in the levels of THs during intrauterine development may cause intrauterine and fetal growth restriction. These disorders may disrupt the development and growth of fetus and neonates, and cause several lifelong consequences through permanent fluctuations in most biological systems. Thus, monitoring the activity of maternal THs may prevent any undesirable pathological state during intrauterine development. Additional studies should evaluate the relation between the maternal thyroid disorders, thyroid auto antibodies, intrauterine and fetal growth restriction [84-89].

Conflict of Interest

The author declares that no competing financial interests exist.

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