

Maternal Hypothyroidism and Developing Hearing Loss

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SHORT COMMUNICATION

The availability of gestational thyroid hormones (THs) is significant for the development of fetus and neonates (El-bakry et al., 2010; Ahmed, 2011, 2012a,b, 2013, 2014, 2015a-c, 2016a-d, 2017a-v, 2018a-r; Ahmed and Ahmed, 2012; Ahmed et al., 2008; 2010; 2012; 2013a,b, 2014, 2015a,b, 2018a,b; Ahmed and Incerpi, 2013; Van Hercket et al., 2013; Ahmed and El-Gareib, 2014; Incerpi et al., 2014; Candelotti et al., 2015; De Vito et al., 2015; El-Ghareeb et al., 2016; Ahmed and El-Gareib, 2017), in particular the morphogenesis and development of auditory system (the inner and middle ear) (Crofton, 2004; Wasserman et al., 2008 & 2012; Dayal et al., 2016; Over cash et al., 2016; Oliveira de Andrade et al., 2017). Also, Cordas et al. (2012) reported that thyroid hormone receptors (TRs; α and β) can regulate the size of the ossicular bones, and the development and maturation of the middle ear. In addition, other factors such as Pax 2 and Pax 8 can share in the morphogenesis and innervation of the inner ear (Bouchard et al., 2010). Central auditory processes are responsible for auditory discrimination, localization/lateralization, auditory pattern recognition, and performance with competing or degraded acoustic signals (Bellis, 2003).

On the other hand, deficiency in the levels of thyroxine (T4) and 3,5,3'-triiodothyronine (T3), congenital or acquired hypothyroidism, can disrupt the central auditory pathway and the activity of cochlea (sensorineural hearing loss) (Musiek and Gollegly, 1985; Ng et al., 2004; Knipper, 2000; Crofton, 2004; Santos, et al. 2010; Dayal and Prasad, 2015; Prabhuet al., 2015; Dayal et al., 2016). Before the onset of hearing process, any deficiency in THs levels can cause permanent damage to peripheral and central auditory systems (Knipper et al.,

2000). Moreover, Weber et al. (2013) recorded that the genetic disorders in hydrogen peroxide (H_2O_2) generation in the thyroid gland can interrupt the hearing process. Group of Wasserman et al. (2008 & 2012) reported that the elevation in the concentration of autoantibody thyroid peroxidase (TPOaAb) during the third trimester of pregnancy was noteworthy and considerably connected to the neonatal sensorineural hearing loss (SNHL). Furthermore, maternal iodine exposure can increase the prevalence of the fetal goiter and neonatal hearing loss (Over cash et al., 2016). More importantly, even mild hearing loss in children may increase the risk of learning impairments, delayed speech and other overt or subtle developmental disorders (Smith et al., 2005; Bailey, 2010). This can be attributed to insufficient maternal free T4 during the early pregnancy (hypothyroid state). In both maternal hypothyroidism and hyperthyroidism states, the frequencies of neonatal hearing dysplasia were meaningfully increased (Su et al., 2011).

Thus, it is also worth declaring that THs and their receptors can regulate the development and functioning of auditory system. In addition, any dysfunctions in the activities of THs during the development may cause hearing loss. These disorders may delay the developmental cognitive behaviors and neonatal responses, cause numerous lifelong consequences and severely impact quality-of-life. The prevention with early detection and treatment of hearing loss may be amenable. Yearly audio logical evaluation to monitor hearing should be done. The results may be depending the age of hearing examination, the methods of hearing assessment, and the genetic factors in different patient populations. Further studies are required to determine the exact prevalence of hearing impairment in children with permanent congenital hypothyroidism.

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