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# Maternal Hypothyroidism and Sensorineural Disability



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## Commentary

Maternal thyroid hormones (THs; 3,5,3'-triiodothyronine (T3) and thyroxin (T4)) exert vital roles throughout the development and show a permissive role in the neurological functions, in particular the sensorineural actions. The actions of THs can be mediated through the genomic and non-genomic pathways. On the other hand, there are associations between the maternal hypothyroidism and neonatal sensorineural deficits. A gestational hypothyroidism displays sensory and motor deficits, grave cognitive, permanent long-lasting variations in child brain structure, and neurodevelopmental disorders. During prenatal and early postnatal periods, any disruption in the levels of T4 or T3 can lead to irreversible disorders in sensorimotor and intelligence function, psychotic behavior, mental retardation, depression, coma and several morphological variations in the brain.

These disorders can increase the risk of memory and learning impairment and dementia. In human, Magri et al. [1] reported that hypothyroidism can cause asymptomatic small-fiber sensory neuropathy. In fact, it has been proposed that congenital hypothyroidism increases the rate of sensorineural hearing loss and motor disability in childhood and adulthood. More interestingly, several authors reported that hypothyroidism can cause a demyelination in the peripheral nerve, sensorimotor neuropathy or polyneuropathy, and fibrillation, diminish the nerve conduction velocity, and delay the relaxation phase of deep tendon reflexes. On the other hand, adult-onset hypothyroidism in humans can cause deafness and multiple defects in taste and smell. Alternatively, adult-onset hypothyroidism in mice can cause several defects in odor discrimination, and decrease the hearing capabilities at adulthood. Thus, it is also worth noticing that normal sensorineural development in early life is favorably dependent on the levels of maternal THs. As well, maternal hypothyroidism may be associated with multiple sensorineural defects and brain disorders. Therefore, the ideal regulation of gestational thyroid function is significant not only for her own health but probably also for the long-term health of her child.

Also, motor proficiency ought to be achieved in preschool age children to distinguish the sensorineural deficits early. Further studies are required to describe the mechanisms of maternal thyroid-associated sensorineural disease [2-113].

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