ISSN NO: 2770-2936
January, 2026

Impact Of Hypothyroidism During Pregnancy On Children's Neurodevolution (Including Autism Spectrum Disorders)

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Abstract: This article provides an in-depth analysis of the impact of hypothyroidism during pregnancy on child neurodevelopment, particularly autism spectrum disorders (ASD). The effects of thyroid hormone deficiency Considering the epidemiological, mechanistic and clinical aspects, maternal thyroid dysfunction has a cumulative negative impact on fetal brain development. Based on research, risk ratios, treatment efficacy, and environmental factors are discussed, and the need for preventive measures is emphasized. The article is based on a scientific approach, covering a wide range of evidence from animal models to clinical data.
Keywords: Pregnancy, hypothyroidism, thyroid hormones, neurodevelopment, autism spectrum disorders (ASD), fetal brain development, epidemiological studies, clinical treatment, autoimmune thyroiditis, environmental factors, prevention.

Hypothyroidism during pregnancy, i.e., decreased thyroid function, is a condition that poses a serious threat to the health of the mother and fetus, and can especially negatively affect the child's neurodevelopment. Thyroid hormones, in particular thyroid hormones (thyroxine - T4 and triiodothyronine - T3), play an important role in fetal brain development, as they regulate the processes of neuronal migration, synaptogenesis, myelination, and the formation of the cerebral cortex. In the early stages of pregnancy, the fetus develops its own thyroid Until the gland begins to function (approximately 16-20 weeks in humans), maternal thyroid hormones pass through the placenta to the fetus, supporting brain development. In hypothyroidism, when maternal T4 and T3 levels are low, the fetal brain is under hormonal stress. deficiency occurs, which can lead to long-term neuropsychiatric disorders, including autism spectrum disorders (ASD). Many epidemiological studies show that hypothyroidism in pregnancy increases the risk of developing ASD in a child, but this relationship depends on the quality of treatment and the duration of the disorder. For example, large population- based studies have found that maternal hypothyroidism is associated with a diagnosis of ASD in a child, in which case the risk ratio can increase by 1.3-2.6 times. In one such study, which analyzed more than 51 thousand births, the risk of ASD in children of mothers with both chronic and gestational hypothyroidism was 2.61 times higher. This relationship is characterized by a dose-response effect: the longer the duration of hypothyroidism (one, two or three trimesters), the higher the risk, 1.69, 2.39 and 3.25 times, respectively. These results indicate a cumulative effect of thyroid hormone deficiency on fetal brain development, as early During the third trimester, brain neurons proliferate and differentiate. The effects of maternal hypothyroidism on child neurodevelopment are not limited to ASD; they can also lead to intellectual disability, attention deficit / hyperactivity disorder (ADHD), and other neuropsychiatric disorders. For example, mild hypothyroidism in early pregnancy has been associated with signs of ASD in the child, with a 1.5-fold increased risk. This association is thought to be due to decreased maternal T4 levels, which are converted to T3 in the fetal brain, which is essential for neurotransmitter synthesis and cortical development. In animal models, such as rats, hypothyroidism during pregnancy (via methimazole) has been shown to result in impaired social interaction, reduced ultrasonographic vocalizations, and reduced social play in offspring, consistent with signs of ASD. Epidemiological data suggest that the effect of maternal hypothyroidism on child ASD may be influenced by racial and ethnic factors; for example, the association is stronger among Hispanics and mixed-race people, with a hazard ratio of 1.09-1.08. This suggests an interaction between genetics and environmental factors, as thyroid dysfunction autoimmune diseases (e.g. Hashimoto's thyroiditis), the prevalence of which varies across populations. In addition to hypothyroidism, maternal thyroiditis Other forms of thyroid dysfunction, such as hyperthyroidism and hypothyroxinemia, can also affect

ISSN NO: 2770-2936
January, 2026

neurodevelopment. In the case of hyperthyroidism, the results are less consistent, but some studies have found a 1.42-fold increased risk of childhood ASD. Hypothyroxinemia (low T4 with normal TSH) is associated with reduced intellectual development in children, with meta -analyses confirming that maternal hypothyroxinemia increases the risk of childhood intellectual disability. In terms of mechanisms, thyroid hormones play a central role in brain development: they regulate gene expression regulates the expression of neuronal regulates proliferation and migration, and modulates the glutamate and GABA neurotransmitter systems. Hypothyroidism in pregnancy results in a deficiency of T3 in the fetal brain, which leads to malformation of neuronal networks. For example, in rat models, maternal hypothyroidism causes a reduction in the number of neurons in the fetal cerebral cortex and disruption of synaptic connections, which leads to structural changes in the brain (e.g., amygdala and prefrontal cortex) that are associated with ASD symptoms in humans. cortex dysfunction). In addition, thyroid hormones affect the immune system; the mother is autoimmune Thyroiditis can negatively affect a child's neurodevelopment because antithyroid antibodies Passes through the placenta and reaches the fetal thyroid gland In clinical studies, the effect of maternal hypothyroidism on child neurodevelopment has been linked to treatment: adequately treated chronic Hypothyroidism (via levothyroxine) does not increase the risk of ASD in children, but the risk increases significantly if the disorder is untreated or persistent. For example, in one study, chronic Only adequately treated hypothyroidism is not associated with ASD in the child, but the risk is 2.61 times higher when chronic and gestational hypothyroidism are combined. This is a clinical practice in which thyroid disease in pregnant women emphasizes the need for regular monitoring of maternal thyroid function, as normalization of TSH and free T4 levels may protect fetal neurodevelopment. Also, maternal thyroid function There are sex differences in the impact of thyroid dysfunction on the neurodevelopment of the child: some studies have found a 1.27-fold increased risk of ASD in boys and a 1.51-fold increased risk in girls, indicating an interaction between sex hormones and the thyroid system. In addition, prenatal Hypothyroidism also increases the risk of ASD in children, as maternal thyroid status can affect fetal development even before conception. Environmental factors, such as exposure to per- and polyfluoroalkyl substances (PFAS), can also affect maternal thyroid function. may increase thyroid dysfunction and increase the risk of ASD in the child, as these substances disrupt the transport of thyroid hormones. Epidemiological studies show that maternal thyroid disorders due to PFAS exposure are associated with neurodevelopmental disorders in the child. In general, hypothyroidism in pregnancy is a factor that negatively affects child neurodevelopment, including ASD, and to prevent it, thyroid therapy before and during pregnancy is recommended. function Screening and adequate treatment are necessary. Future studies should further explore the molecular mechanisms of this association and identify individual risk factors, as this will help improve prevention strategies. Thus, maternal thyroid health is a key factor in the neurodevelopment of the child, and its disruption can lead to long-term neuropsychiatric consequences, which requires serious attention in medicine.

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