

Iodine and thyroid hormones during pregnancy and postpartum

Faustino R. Pérez-lópez

Abstract

Iodine is a trace element essential for synthesis of the thyroid hormones, triiodothyronine and thyroxine. These hormones play a vital role in the early growth and development stages of most organs, especially the brain. The World Health Organization (WHO) has declared that, after famine, iodine deficiency is the most avoidable cause of cerebral lesions including different degrees of mental retardation and cerebral paralysis. The main function of iodine in vertebrates is to interact with the thyroid hormones. During pregnancy sufficient quantities of iodine are required to prevent the appearance of hypothyroidism, trophoblastic and embryonic or fetal disorders, neonatal and maternal hypothyroidism, and permanent sequelae in infants. Thyroid hormone receptors and iodothyronine deiodinases are present in placenta and central nervous tissue of the fetus. A number of environmental factors influence the epidemiology of thyroid disorders, and even relatively small abnormalities and differences in the level of iodine intake in a population have profound effects on the occurrence of thyroid abnormalities. The prevalence of disorders related to iodine deficit during pregnancy and postpartum has increased. Iodine supplementation is an effective measure in the case of pregnant and lactating women. However, it is not implemented and the problem is still present even in societies with theoretically advanced health systems. During pregnancy and postpartum, the WHO recommends iodine intake be increased to at least 200 µg/day. Side-effects provoked by iodine supplementation are rare during pregnancy at the recommended doses.

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giner@unizar.es

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