

S29-02

THYROID HORMONES AND THE PSYCHOMOTOR DEVELOPMENT OF THE NEWBORN

S. Ares¹, J. Quero¹, G. Morreale de Escobar²

¹Neonatology Department, University Hospital LA PAZ, ²Instituto de Investigaciones Biomédicas, Autonomus University of Madrid, Madrid, Spain

Iodine is a trace element which is essential for the synthesis of thyroid hormones. If maternal iodine deficiency in pregnancy is severe, fetal brain damage will occur. This damage is irreversible after birth. Mild/moderate iodine deficiency during pregnancy and early postnatal life is associated with neuro/psycho-intellectual deficits in infants and children. The severity is not only related to the degree of iodine deficiency, but also to the developmental phase during which it is suffered, the most severe being the consequence of iodine deficiency during the first two trimesters of pregnancy. The close involvement between human brain development and thyroid hormones is widely accepted. The effects of T₃ on the central nervous system are mediated by the regulation of the expression of genes that synthesize proteins implicated in cerebral neurogenesis, neuronal migration and differentiation, axonal outgrowth, dendritic ontogeny, and synaptogenesis. They are also necessary for cerebellar neurogenesis (predominantly during early postnatal life), gliogenesis (predominantly during late fetal life to 6 months postnatally), and myelogenesis (during the second trimester of gestation to 2 years of postnatal life). From clinical studies on the effect of iodine deficiency of both mother and fetus it becomes clear that T₄ is required for brain development during gestation.

Low T₄ levels during neonatal life could be a negative factor contributing to the neurodevelopmental problems of very preterm infants. Indeed, retrospective studies have shown a relationship between hypothyroxinemia and developmental delay and an increased risk of disabling cerebral palsy.