

Introduction

Thyroid hormones regulate growth, development, differentiation and metabolic processes by interacting and activating thyroid hormone receptors (*Von Vliet, 2001*).

Thyroxine (T₄) is the principal hormone secreted by thyroid gland and in fact it appears to function as a prohormone for the production of the more biologically active form triiodothyronine (T₃) by 5-monodeiodination (*Valero et al., 2004*).

During fetal life , thyroid gland develops with production of thyroxine (T₄) and triiodothyronine (T₃) and secretion into the serum from about 12 weeks gestation and levels of which increase to term (*Rovet, 2004*).

Transient hypothyroxinemia was suggested as a common phenomenon, in expression of temporary hypothalamic-pituitary immaturity or a manifestation of non thyroidal illness (*Lim et al., 2005*).

Transient hypothyroxinemia characterized by low levels of serum thyroxine, is a common finding among premature infants (*Biswas et al., 2002*).

Sick term infants with low 5 minutes Apgar score have been shown to have a reduction in the thyroid hormone levels after birth (*Moster et al., 2002*).

Preterm infants with perinatal illness develop transient hypothyroxinemia during the first week of life . Term infants has been investigated less extensively (*Lim et al., 2005*).