

INTRODUCTION AND AIM OF THE WORK

Tuberculosis is a chronic infectious disease caused by *Mycobacteria* of the “tuberculosis complex” mainly *Mycobacterium tuberculosis* (*Stead et al., 1990*).

Nearly 170.000 children die of TB every years. But TB's indirect impact on children is even more chilling than this annual number of childhood deaths. The TB epidemic also endangers millions of other children whose parents and grandparents become ill with the disease. WHO estimates that in 1995, of all children under 15 years of age, at least 180 million were infected with TB (*WHO, 1996*).

Non thyroid illness (NTI) induces several characteristic alterations in thyroid function tests which comprise the euthyroid sick syndrome (*Schussler, 1986*).

Serum concentrations of total and free triiodothyronine (T3) are decreased due to diminished extrathyroid 5-deiodination of thyroxine (T4). Circulating inhibitors weaken the binding of thyroid hormones to serum proteins, further lowering T3 and causing some decline in T4 without affecting free hormone concentrations (*Liewandah et al., 1987*).

Thyrotropin (TSH) concentrations are usually normal in NTI but, depending on the severity and stage of the illness, may be depressed or moderately elevated (*Hamblin et al., 1986*).

Since serum thyroxin-binding globulin (TBG) is increased in some form of inflammatory liver diseases, they hypothesized that the hepatic

effect of antituberculous drugs might increase serum thyroid hormone binding (*Schussler et al., 1978 and Gangadharam, 1986*).

Despite the world-wide importance of T.B data concerning thyroid hormones in this disease remain sparse and conflicting (*Christensen et al., 1989*).

Aim of the work

The aim of this study is to measure serum thyroid hormones T3-T4, thyrotropin (TSH) and thyroxin binding globulin (TBG) in children suffering from tuberculosis in order to determine the effect of the disease and its treatment on thyroid functions.