ABSTRACT

Experimental studies were carried out on pregnant rats using radiolabelled-thyroxine (^{125}I-thyroxine) to determine whether thyroxine is taken up by placentae and fetuses during pregnancy, particularly before the onset of fetal thyroid gland function.

Gross uptake of 1.09 $\pm$ 0.6 was seen from the 14th day of gestation, and this increased throughout the gestation period under investigation. A similar pattern was observed in the fetal brain and liver. The placentae had very high uptake rates of 22.1 $\pm$ 5.7 at day 14 which increased to 30.2 $\pm$ 2.6 on the 20th day of gestation. The placenta was observed to have an uptake rate of over 5 times that of the maternal thyroid and 15-20 times that of other maternal organs.

Thyroxine uptake by individual tissues were measured by using radiolabelled iodide (^{131}Iodide) as an internal control to equate transcapillary flow rates (trans-membrane flux) of the iodide molecules. These results showed a net uptake and utilisation (1.26 $\pm$ 0.43) of thyroxine at day 14 (i.e. before the onset of fetal thyroid function) but not afterwards ($p \leq 1.0$). This indicated that maternal thyroxine is utilised by

the fetus for its developmental requirements when the fetal thyroid has not begun its own hormone secretion.

A study *in vitro* of 5'- monodeiodinase activity in the placentae and fetuses was done to estimate the metabolism of injected ^{125}I-thyroxine in those tissues. The fetuses had a high deiodination rate of 258 $\pm$ 78.5 on day 14 and a sharp reduction to 62.8 $\pm$ 17.4 on day 15. It reduced further thereafter. The deiodination rates in the placentae were much higher, increasing from 500.5 $\pm$ 212.6 at day 13 to a peak of 964.2 $\pm$ 439.9 on day 15. These results suggest a change in the utilisation pattern of the injected ^{125}I-thyroxine. High 5'- monodeiodination rates upto day 15 suggest the placental transfer and fetal utilisation of active hormone, whereas after onset of fetal thyroid gland function on or about day 16, thyroxine is metabolised by the placenta into inactive rT_3 and molecular iodine.

These results indicate that maternal thyroxine has an important role to play in fetal development before the onset of fetal thyroid gland function, and also is instrumental in the supply of iodine by its metabolism in the placenta. This additional source of iodine, for which the fetus does not have to compete with other maternal organs, may be of critical significance to fetuses carried by mothers living in low-iodine

environments. This has serious epidemiological implications in the iodine-deficient regions of the world where mental retardation and other less apparent neurological deficits due to fetal deficiency of thyroxine or iodine or both, are a grave threat to the health and the quality of life of millions of people who inhabit these regions.