Hepatic glutathione biosynthetic capacity in hyperthyroid rats

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The influence of hyperthyroidism on the capacity of the liver to synthesize glutathine (GSH) was evaluated as a possible mechanism of depletion of the tripeptide. For this purpose, the effect of daily doses of 0.1 mg 3,3',5-triiodothyronine (T3/kg for 3 consecutive days on hepatic GSH biosynthetic capacity was assessed by a combined assay measuring gamma-glutamylcysteinyl synthase and GSH synthase simultaneously. T3 treatment induced a significant 56% depletion of liver GSH in parallel with an increase in the rate of GSH synthesis, the latter effect being completely abolished by L-buthionine sulfoximine. According to these data, the fractional rate of hepatic GSH turnover exhibited a 3.2-fold enhancement in hyperthyroid rats compared to control animals. It is concluded that the enhanced GSH utilization in the liver of hyperthyroid rats previously observed [Fernandez et al., Endocrinology 129, 85-91, 1991], is accompanied by an increment in GSH synthesis that is insufficient to sustain