

Thyroid hormone administration induces rat liver Nrf2 activation: Suppression by N-acetylcysteine pretreatment

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Background: Oxidative stress associated with 3,3',5-triiodo-L-thyronine (T3)-induced calorigenesis upregulates the hepatic expression of mediators of cytoprotective mechanisms. The aim of this study was to evaluate the hypothesis that in vivo T3 administration triggers a redox-mediated translocation of the cytoprotective nuclear transcription factor erythroid 2-related factor 2 (Nrf2) from the cytosol to the nucleus in rat liver. Such translocation of transcription factors is considered to be an activating step. **Materials and Methods:** The effect of T3 administration in the presence and absence of N-acetylcysteine (NAC) on cytosol-to-nuclear translocation of Nrf2 was evaluated, with inhibition of this process by NAC taken as evidence that the process was redox mediated. Male Sprague-Dawley rats weighing 180-200 g were given a single intraperitoneal dose of 0.1 mg T3/kg. Another group of rats were given the same dose of T3 and were also pretreated with NAC (0.5 g/kg) at 0.5 hour before