Upregulation of liver inducible nitric oxide synthase following thyroid hormone preconditioning: Suppression by N-acetylcysteine

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3,3,5-L-Triiodothyronine (T 3) exerts significant protection against ischemia-reperfusion (IR) liver injury in rats. Considering that the underlying mechanisms are unknown, the aim of this study was to assess the involvement of inducible nitric oxide synthase (iNOS) expression and oxidative stress in T 3 preconditioning (PC). Male Sprague-Dawley rats given a single dose of 0.1 mg of T 3/kg were subjected to 1-hour ischemia followed by 20 hours reperfusion, in groups of animals pretreated with 0.5 g of N-acetylcysteine (NAC)/kg 0.5-hour prior to T 3 or with the respective control vehicles. At the end of the reperfusion period, liver samples were taken for analysis of iNOS mRNA levels (RT-PCR), liver NOS activity, and hepatic histology. T3 protected against hepatic IR injury, with 119% enhancement in liver iNOS mRNA/18S rRNA ratios ($p<0.05$) and 12.7-fold increase ($p<0.05$) in NOS activity in T3-treated animals subjected to IR over values in controlsham operated rats, with a net 7.7-fold e