Thyroid hormone activates rat liver adenosine 5-monophosphate-activated protein kinase: Relation to CaMKK?, TAK1, and LKB1 expression and energy status

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AMP-activated protein kinase (AMPK) is a sensor of energy status supporting cellular energy homeostasis that may represent the metabolic basis for 3,3?,5-triiodo-L-thyronine (T3) liver preconditioning. Functionally transient hyperthyroid state induced by T3 (single dose of 0.1 mg/kg) in fed rats led to upregulation of mRNA expression (RT-PCR) and protein phosphorylation (Western blot) of hepatic AMPK at 8 to 36 h after treatment. AMPK Thr 172 phosphorylation induced by T3 is associated with enhanced mRNA expression of the upstream kinases Ca2+- calmodulin-dependent protein kinase kinase-? (CaMKK?) and transforming growth-factor-?-activated kinase-1 (TAK1), with increased protein levels of CaMKK? and higher TAK1 phosphorylation, without changes in those of the liver kinase B1 (LKB1) signaling pathway. Liver contents of AMP and ADP were augmented by 291% and 44% by T3 compared to control values ($p<0.05$), respectively, whereas those of ATP decreased by 64% ($p<0.05$), with no significant ch