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Intramuscular Heat Shock Protein 72 and Heme Oxygenase-1 mRNA are Reduced in Patients with Type 2 Diabetes Evidence That Insulin Resistance is Associated with a Disturbed Antioxidant Defense Mechanism

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Abstract : To examine whether genes associated with cellular defense against oxidative stress are associated with insulin sensitivity, patients with type 2 diabetes (n=7) and age-matched (n=5) and young (n=9) control subjects underwent a euglycemic-hyperinsulinemic clamp for 120 min. Muscle samples were obtained before and after the clamp and analyzed for heat shock protein (HSP)72 and heme oxygenase (HO)-1 mRNA, intramuscular triglyceride content, and the maximal activities of β-hyroxyacyl-CoA dehydrogenase (β-HAD) and citrate synthase (CS). Basal expression of both HSP72 and HO-1 mRNA were lower ($P < 0.05$) by 33 and 55%, respectively, when comparing diabetic patients with age-matched and young control subjects, with no differences between the latter groups. Both basal HSP72 ($r = 0.75$, $P < 0.001$) and HO-1 ($r = 0.50$,$P < 0.05$) mRNA expression correlated with the glucose infusion rate during the clamp. Significant correlations were also observed between HSP72 mRNA and both β-HAD ($r = 0.61$, $P < 0.01$) and CS ($r = 0.65$, $P < 0.01$). HSP72 mRNA was induced ($P < 0.05$) by the clamp in all groups. Although HO-1 mRNA was unaffected by the clamp in both the young and age-matched control subjects, it was increased ($P < 0.05$) ~70-fold in the diabetic patients after the clamp. These data demonstrate that genes involved in providing cellular protection against oxidative stress are defective in patients with type 2 diabetes and correlate with insulin-stimulated glucose disposal and markers of muscle oxidative capacity. The data provide new evidence that the pathogenesis of type 2 diabetes involves perturbations to the antioxidant defense mechanism within skeletal muscle.

Keywords : euglycemic-hyperinsulinemic, HSP72, mRNA, diabete
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