Hypoxia stimulates glucose transport in insulin-resistant human skeletal muscle

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Insulin and muscle contraction stimulate glucose transport into muscle cells by separate signaling pathways, and hypoxia has been shown to operate via the contraction signaling pathway. To elucidate the mechanism of insulin resistance in human skeletal muscle, strips of rectus abdominis muscle from lean (body mass index [BMI] < 25), obese (BMI > 30), and obese non-insulin-dependent diabetes mellitus (NIDDM) (BMI > 30) patients were incubated under basal and insulin-, hypoxia-, and hypoxia + insulin-stimulated conditions. Insulin significantly stimulated 2-deoxyglucose transport approximately twofold in muscle from lean ($P < 0.05$) patients, but not in muscle from obese or obese NIDDM patients. Furthermore, maximally insulin-stimulated transport rates in muscle from obese and diabetic patients were significantly lower than rates in muscle from lean patients ($P < 0.05$). Hypoxia significantly stimulated glucose transport in muscle from lean and obese patients. There were no significant differences in hypoxia-stimulated glucose transport rates among lean, obese, and obese NIDDM groups. Hypoxia + insulin significantly stimulated glucose transport in lean, obese, and diabetic muscle. The results of the present study suggest that the glucose transport effector system is intact in diabetic human muscle when stimulated by hypoxia.