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**RESISTANCE TRAINING ENHANCES COMPONENTS OF THE INSULIN-SIGNALING CASCADE IN NORMAL AND INSULIN-RESISTANT SKELETAL MUSCLE**

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We recently demonstrated that chronic resistance training improves insulin-stimulated glucose transport in normal rodent skeletal muscle, in part, to an increased glucose transporter (GLUT4) protein concentration (Acta Physiol. Scand. 175:315-23, 2002). However, it remained to be determined 1) if these improvements were also accounted for by alterations in components of the insulin-signaling cascade and 2) whether resistance training could improve skeletal muscle insulin resistance. Thirty two male Sprague Dawley rats were assigned to one of four groups: control diet, sedentary (CON-Sed, n=8), control diet, resistance trained (CON-RT, n=8), high-fat diet, sedentary (HF-Sed, n=8) or high-fat diet, resistance trained (HF-RT, n=8). Control animals received a normal diet and high-fat animals consumed a high-fat diet for 9 wk. The RT animals then performed 12 wk of resistance training (3 sets of 10 repetitions at 75% 1-RM, 3x/week). During the 12 week training period all animals remained on their respective diets. Following the training period animals were subjected to hind limb perfusion to assess rates of insulin-stimulated glucose transport in red (RG) and white (WG) gastrocnemius, and red (RQ) and white (WQ) quadriceps. Rates of glucose transport were decreased in RG and RQ of the HF compared to CON animals. Glucose transport in RG and RQ of the CON-TR was greater than in CON animals and normalized in HF-TR animals. Insulin-stimulated IRS-1 associated PI-3 kinase activity followed the same pattern to that of glucose transport. Of particular interest, aPKC-ζ/λ activity was reduced in the HF animals and normalized in the HF-RT animals. In addition, resistance training increased GLUT4 protein concentration in the RG and RQ of the CON-RT and HF-RT animals. Collectively, these findings suggest that resistance training increases carbohydrate metabolism in normal muscle and improves high fat diet-induced skeletal muscle insulin resistance by enhancing components of both the insulin-signaling cascade and glucose transporter effector system.