The oxidative profile of white adipose tissue is not related to intrinsic exercise capacity or whole-body metabolic health

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Impaired visceral white adipose tissue (WAT) metabolism has been implicated in the pathogenesis of several chronic lifestyle-related disease states (Dankel *et al.*, 2011). Indeed, diminished mitochondrial gene expression has been observed in WAT from insulin resistant humans (Dahlman *et al.*, 2006; Wang *et al.*, 2010) and rodents (Laye *et al.*, 2009; Sutherland *et al.*, 2008; Rong *et al.*, 2007). Given the importance of WAT metabolism in regulating whole-body metabolic health, it has been suggested that alterations in the oxidative profile of WAT may contribute to the development of insulin resistance. We have used genetically heterogeneous N:NIH rats that were artificially selected for high- (HCR) and low- (LCR) running capacities over 22 generations (Koch & Britton, 2001). In the absence of exercise training or diet manipulation, these divergent strains simultaneously present with divergent metabolic phenotypes (Wisloff *et al.*, 2006), including differences in skeletal muscle mitochondrial content (Stephenson *et al.*, 2012). We hypothesized that mitochondrial protein expression would be reduced in WAT from LCR compared to HCR and that endurance exercise training (6 wk of incremental treadmill running, 4 d/wk) would ameliorate this difference.

Using Western blotting techniques, we found that independent of training state, the expression of mitochondrial proteins involved in the tricarboxylic acid cycle and oxidative phosphorylation was similar in WAT from LCR and HCR rats. The abundance of the proteins PGC-1α (a transcriptional regulator involved in mitochondrial biogenesis) and the AMP-activated protein kinase was also similar in both groups. Exercise training altered the expression of several proteins involved in glucose metabolism, including: the glucose transport protein GLUT4 (increased 16% in HCR, $P<0.0001$); the β_3-adrenergic receptor (increased 16% in LCR, $P<0.05$); and the orphan nuclear receptor NOR1 (decreased 24% in LCR and 21% in HCR, $P<0.05$).

In conclusion, despite reducing total body and fat pad mass while concomitantly improving whole-body insulin sensitivity, exercise training had little effect on the oxidative profile of WAT from either LCR or HCR rats. This suggests a disconnect between mitochondrial energy metabolism and glucose homeostasis in WAT, and that such changes are independent of intrinsic exercise capacity or exercise training state.

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