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Operation Damage Control: Exercise Training to Prevent Metabolic Damage from High-Fat Feeding

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Adipose tissue has recently been recognized as a participant in the metabolically favorable effects of exercise (1, 2). In a new study by Snook et al. (3), published in this issue of *Obesity*, C57BL/6J mice were exercise-trained for 4 weeks and then placed on a 60% high-fat diet (HFD) for 4 days. The primary goal was to determine whether prior exercise training results in altered beta-adrenergic and lipolytic signaling in adipose tissue following an HFD challenge. In addition, the study includes an analysis of both subcutaneous inguinal fat and epididymal adipose tissue, which is similar to visceral fat in humans (3, 4). *Ex vivo* lipolysis assays using CL 316,243 and epididymal fat from sedentary and endurance-trained mice demonstrated increased phosphorylation of hormone-sensitive lipase, as well as increased nonesterified fatty acids. These results support the authors' conclusion that previous exercise training can protect against some of the effects of short-term high-fat feeding.

Previous studies from the same group have demonstrated that exercise training also prevents HFD-induced weight gain and improves glucose tolerance and insulin signaling in skeletal muscle (5). The protective effects of exercise have been associated with skeletal muscle adaptations, such as increased mitochondria density/function (6) and adenosine monophosphate-activated protein kinase activation in adipose tissue (7). However, the role of adipose tissue and skeletal muscle should be considered from different perspectives. On one hand, adipose tissue should be highly sensitive to adrenergic stimulation during exercise to facilitate a release of high amounts of free fatty acids that can be oxidized in the skeletal muscle. Yet, during postprandial metabolism, adipose tissue should be sensitive to insulin to dramatically reduce lipolysis, therefore allowing skeletal muscle to take up glucose. In this study, insulin sensitivity was examined by Akt activation in adipose tissue and shown not to change. However, this observation does not rule out the ability of insulin to have altered antilipolytic effects under the conditions studied. Overall, the data suggest that prior exercise training alters the ability of a mouse to respond to a nutrient challenge of short-term high-fat feeding, showing that different adipose tissue depots have varied responses. The protective effects of exercise training on lipolytic signaling could have clinical implications, considering that human patients with obesity demonstrate blunted beta-adrenergic-stimulated lipolysis in subcutaneous adipose tissue (8).

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The use of dual-tracer methodology has recently revealed that exercise training promotes oxidation of labeled dietary fat but also decreases the trafficking of dietary fat to adipose tissue (9). Future studies that utilize similar labeling techniques, in addition to high-resolution imaging, can hopefully be designed to examine the fates of fatty acids that are derived from dietary fat, as well as from visceral and subcutaneous depots, in response to acute bouts of exercise and exercise training. The results from this article (3) demonstrate that exercise training offers a short-term protective effect from a metabolically damaging diet. It is also apparent that adipose tissue adapts to be more metabolically fit by increasing the release of free fatty acids to protect against a harmful diet and maintain metabolic resiliency.

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