Adipose-Specific Knockout of raptor Results in Lean Mice with Enhanced Mitochondrial Respiration

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raptor is a specific and essential component of mammalian TOR complex 1 (mTORC1), a key regulator of cell growth and metabolism. To investigate a role of adipose mTORC1 in regulation of adipose and whole-body metabolism, we generated mice with an adipose-specific knockout of raptor (raptor(ad-/-)). Compared to control littermates, raptor(ad-/-) mice had substantially less adipose tissue, were protected against diet-induced obesity and hypercholesterolemia, and exhibited improved insulin sensitivity. Leanness was in spite of reduced physical activity and unaffected caloric intake, lipolysis, and absorption of lipids from the food. White adipose tissue of raptor(ad-/-) mice displayed enhanced expression of genes encoding mitochondrial uncoupling proteins characteristic of brown fat. Leanness of the raptor(ad-/-) mice was attributed to elevated energy expenditure due to mitochondrial uncoupling. These results suggest that adipose mTORC1 is a regulator of adipose metabolism and, thereby, controls whole-body energy homeostasis.
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