Cholesterol, a cell size-dependent signal that regulates glucose metabolism and gene expression in adipocytes.

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Résumé en anglais	Enlarged fat cells exhibit modified metabolic capacities, which could be involved in the metabolic complications of obesity at the whole body level. We show here that sterol regulatory element-binding protein 2 (SREBP-2) and its target genes are induced in the adipose tissue of several models of rodent obesity, suggesting cholesterol imbalance in enlarged adipocytes. Within a particular fat pad, larger adipocytes have reduced membrane cholesterol concentrations compared with smaller fat cells, demonstrating that altered cholesterol distribution is characteristic of adipocyte hypertrophy per se. We show that treatment with methyl-beta-cyclodextrin, which mimics the membrane cholesterol reduction of hypertrophied adipocytes, induces insulin resistance. We also produced cholesterol depletion by mevastatin treatment, which activates SREBP-2 and its target genes. The analysis of 40 adipocyte genes showed that the response to cholesterol depletion implicated genes involved in cholesterol traffic (caveolin 2, scavenger receptor BI, and ATP binding cassette 1 genes) but also adipocyte-derived secretion products (tumor necrosis factor alpha, angiotensinogen, and interleukin-6) and proteins involved in energy metabolism (fatty acid synthase, GLUT 4, and UCP3). These data demonstrate that altering cholesterol balance profoundly modifies adipocyte metabolism in a way resembling that seen in hypertrophied fat cells from obese rodents or humans. This is the first evidence that intracellular cholesterol might serve as a link between fat cell size and adipocyte metabolic activity.
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Liens

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