Caveolin-1 expression and cavin stability regulate caveolae dynamics in adipocyte lipid store fluctuation

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Résumé en anglais	Adipocytes specialized in the storage of energy as fat are among the most caveolae-enriched cell types. Loss of caveolae produces lipodystrophic diabetes in humans, which cannot be reversed by endothelial rescue of caveolin expression in mice, indicating major importance of adipocyte caveolae. However, how caveolae participate in fat cell functions is poorly understood. We investigated dynamic conditions of lipid store fluctuations and demonstrate reciprocal regulation of caveolae density and fat cell lipid droplet storage. We identified caveolin-1 expression as a crucial step in adipose cell lines and in mice to raise the density of caveolae, to increase adipocyte ability to accommodate larger lipid droplets, and to promote cell expansion by increased glucose utilization. In human subjects enrolled in a trial of 8 weeks of overfeeding to promote fattening, adipocyte expansion response correlated with initial caveolin-1 expression. Conversely, lipid mobilization in cultured adipocytes to induce lipid droplet shrinkage led to biphasic response of cavin-1 with ultimate loss of expression of cavin-1 and -3 and EHD2 by protein degradation, coincident with caveolae disassembly. We have identified the key steps in cavin/caveolin interplay regulating adipocyte caveolae dynamics. Our data establish that caveolae participate in a unique cell response connected to lipid store fluctuation, suggesting lipid-induced mechanotension in adipocytes.
Notes	Cet article contient des données supplémentaires à l'adresse suivante : http://diabetes.diabetesjournals.org/lookup/suppl/doi:10.2337/db13-1961/-/DC1. [31]
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