The lipoatrophic caveolin-1 deficient mouse model reveals autophagy in mature adipocytes

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Résumé en anglais	Adipose tissue lipoatrophy caused by caveolin gene deletion in mice is not linked to defective adipocyte differentiation. We show that adipose tissue development cannot be rescued by endothelial specific caveolin-1 re-expression, indicating primordial role of caveolin in mature adipocytes. Partial or total caveolin deficiency in adipocytes induced broad protein expression defects, including but not limited to previously described downregulation of insulin receptor. Global alterations in protein turnover, and accelerated degradation of long-lived proteins were found in caveolin-deficient adipocytes. Lipidation of endogenous LC3 autophagy marker and distribution of GFP-LC3 into aggregates demonstrated activated autophagy in the absence of caveolin-1 in adipocytes. Furthermore, electron microscopy revealed autophagic vacuoles in caveolin-1 deficient but not control adipocytes. Surprisingly, significant levels of lipidated LC3-II were found around lipid droplets of normal adipocytes, maintained in nutrient-rich conditions or isolated from fed mice, which do not display autophagy. Altogether, these data indicate that caveolin deficiency induce autophagy in adipocytes, a feature that is not a physiological response to fasting in normal fat cells. This likely resulted from defective insulin and lipolytic responses that converge in chronic nutrient shortage in adipocytes lacking caveolin-1. This is the first report of a pathological situation with autophagy as an adaptative response to adipocyte failure.
URL de la notice	http://okina.univ-angers.fr/publications/ua303 [33]

Liens

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