Distinct roles of endothelial and adipocyte caveolin-1 in macrophage infiltration and adipose tissue metabolic activity.

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Auteur: Briand, Nolwenn [1], Le Lay, Soazig [2], Sessa, William C [3], Ferré, Pascal [4], Dugail, Isabelle [5]

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**OBJECTIVE:** Defective caveolin-1 expression is now recognized as a cause of lipoatrophic diabetes in patients, due to primary caveolin gene mutations or secondary caveolin deficiency caused by PTRF/cavin gene defects. The goal of this study was to establish the relative contribution of endothelial cells and adipocytes, both highly expressing caveolin-1 to the lipoatrophic phenotype of mice with global caveolin-1 gene invalidation (Cav1-KO).

**RESEARCH DESIGN AND METHODS:** We compared adipose tissue development and metabolic phenotype of wild-type (WT), lipoatrophic Cav1-KO, and a murine model with specific rescue of caveolin-1 expression in endothelial cells (caveolin-1-reconstituted [Cav1-RC]).

**RESULTS:** Defective adipose tissue development, reduced adipocyte size, and global alteration in adipose tissue gene expression that characterize lipoatrophic caveolin-1 null mice were still observed in Cav1-RC, indicating a prominent role of adipocyte-derived caveolin in lipoatrophy. We also observed that Cav1-KO adipose tissue contained an increased proportion of infiltrated macrophages compared with control mice, mostly with an alternate activation M2 phenotype. In contrast with defective lipid storage and lipoatrophy, macrophage infiltration was normalized in Cav1-RC mice, pointing to caveolin-1-dependent endothelium permeability as the causing factor for adipose tissue macrophage infiltration in this model.

**CONCLUSIONS:** This is the first report of a specific role for adipocyte caveolin expression in lipid storage. Our study also shows that endothelium caveolin critically participates in the control of macrophage extravasation from the blood into adipose tissue, therefore establishing distinct roles depending on topology of caveolin expression in different cell types of adipose tissue.

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