**CD36 PROMOTES ADIPOCYTE DIFFERENTIATION AND ADIPOGENESIS**

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A potential functional role of the scavenger receptor CD36 was investigated in in vitro adipocyte differentiation and in vivo adipogenesis. Differentiation of murine embryonic fibroblasts deficient in CD36 into mature adipocytes was impaired as compared to wild-type controls. During differentiation of 3T3-F442A preadipocytes, expression of CD36 was upregulated and CD36 gene silencing resulted in impaired differentiation, as monitored by Oil Red O staining and expression of adipogenic markers. De novo fat pad formation in NUDE mice following injection of preadipocytes was significantly reduced upon CD36 gene silencing as compared to control. This was associated with marked adipocyte hypotrophy and reduced adipose tissue adipocyte content. Macrophage infiltration in de novo fat tissues derived from preadipocytes with CD36 gene silencing was not significantly different from controls. Collagen content was significantly higher in de novo fat with CD36 gene silencing. In a nutritionally induced obesity model, total body weight as well as subcutaneous and gonadal adipose tissue mass were significantly lower in CD36 deficient mice as compared to wild-type littermates, whereas food intake was comparable. CD36 deficiency did not significantly affect adipocyte or blood vessel size or density. On the high fat diet, CD36 deficient mice had higher levels of total and HDL cholesterol as compared to wild-type controls. Thus, our data support a functional role of CD36 in promoting adipogenesis in vitro as well as in vivo.