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Dietary Conjugated Linoleic Acid Alters Adipose Phenotype in a Depot-Specific Manner in Mice

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The trans-10, cis-12 (t10c12-CLA) isomer of conjugated linoleic acid (CLA) reduces adipose mass and body weight gain in mice and humans. Numerous mechanisms contribute to adipose delipidation by t10c12-CLA, including inhibition of preadipocyte differentiation and increased adipocyte apoptosis. Our group has observed that in epididymal adipose of mice, dietary t10c12-CLA increases uncoupling protein-1 (UCP1) expression, a marker of brown adipose tissue (BAT). To further characterize this observation, six-week old CD2F1 male mice were fed a control (CON) or 0.12% t10c12-CLA containing semi-purified diet for 18 days. No differences in body weight or food efficiency ratios were observed between groups, but as expected, CLA reduced epididymal and inguinal adipose mass. In epididymal but not inguinal adipose tissue, CLA increased mRNA levels of UCP1, cell death-inducing DFFA-like effector a (CIDEA), elongation of very long chain fatty acids-3 (ELOVL3), and carnitine palmitoyltransferase 1b (CPT1b), all of which are highly expressed in BAT. Furthermore, the expression of peroxisome proliferator-activated receptor coactivator-1a (PGC1a), a marker of mitochondrial biogenesis, was up regulated in epididymal but not inguinal adipose. Immunohistochemistry for UCP1 indicated evidence of localized multilocular adipocytes in the epididymal adipose depot with CLA treatment, although PR domain containing 16 (PRDM16) mRNA, a marker of BAT differentiation, was decreased. Previous findings from our laboratory indicate that CLA does not increase beta-3 adrenergic signaling in white adipose tissue (WAT), so it remains unclear if CLA is inducing UCP1 expression in white or brown adipocytes. Further studies are needed to determine if CLA is inducing the emergence of brown adipocytes in epididymal WAT.