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PROCEEDINGS

Conjugated linoleic acid down-regulates expression of resistin and adiponectin in fully differentiated 3T3-F442A cells

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Conjugated linoleic acid (CLA) is a naturally occurring derivative of linoleic acid found in dairy products [1]. Dietary supplementation of CLA is known to have several beneficial health effects as an anti-carcinogenic, anti-atherogenic, hypocholesterolemic, anti-diabetic and anti-obesity agent [2–5].

Adipose tissue produces a variety of factors, which contribute to insulin resistance characteristic of obesity and obesity-linked type 2 diabetes [6–7]. To explore the possible molecular link between CLA and its anti-diabetic effects, we studied the mRNA expression levels of several molecular markers of diabetes following exposure to CLA [8–11].

Fully differentiated 3T3-F442A cells were treated either by supplement with CLA or linoleic acid (LA, positive control), (100 μm) or untreated (negative control) for 24 h. Subsequently, total RNA was extracted and transcript levels of resistin, adiponectin and PPARγ were assayed by quantitative RT-PCR and normalized to GAPDH (house keeping gene). Supplementation of CLA significantly down-regulated the mRNA levels of resistin and adiponectin (to 20 and 30%, respectively), as compared to LA

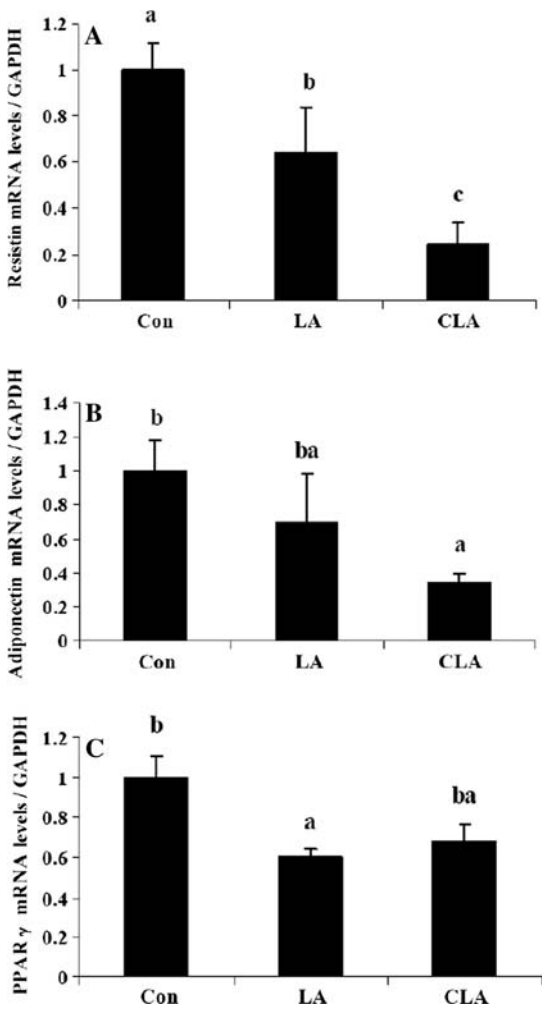

Fig. 1 Effect of linoleic and CLA treatment on resistin, adiponectin, and PPARγ mRNA levels in 3T3-F442A adipocytes. Confluent 3T3-F442A cells were induced to differentiate as described in "Materials and methods". At day 10 of differentiation the cells were treated with 100 μM of CLA and Linoleic acid (*LA*) for 24 h. For each sample, transcript levels of *Resistin* (**a**), *Adiponectin* (**b**) and *PPARγ* (**c**) were determined by QPCR and were normalized to internal house keeping control gene levels (*GAPDH*). The results are shown relative to control cells (untreated). Results expressed as mean + SE of three independent experiments ($n = 3$). *Columns* not sharing a superscript are significantly different ($P < 0.05$)

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and untreated-control adipocytes (Fig. 1a, b). PPARγ mRNA levels did not change significantly following CLA supplementation (Fig. 1c). The present results indicate that CLA supplementation down-regulates both resistin and adiponectin, adipockines known to either ameliorate or deteriorate insulin sensitivity, respectively. Further work is currently underway.

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