Salatrim™, a short-chained fatty acid based low-caloric fat alters adipose and hepatic gene-expression and ectopic lipid deposition in obese mice on a weight-loss program.

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In vitro, short-chain fatty acids (SCFA) increase the expression of the nuclear receptor PPAR-α and reduce expression of the inflammatory transcription-factor NFκB. Thus, dietary SCFA could have a positive impact on fatty acid oxidation and inflammatory responses, and therefore accelerate the re-establishment of a lean metabolic state during a weight-loss program. This hypothesis was tested in obese mice by combining weight-loss with intake of LC n-3 PUFA and Salatrim, a SCFA based low-caloric fat, either alone or in combination. **Method:** Obese mice were transferred to a low-fat diet (2.5 mass% fat), where the fat fraction constituted a control fat (C) or fish-oil (FO). In two groups, 25 mass% of the fat was exchanged with Salatrim. After four weeks on the low-fat diets animals were sacrificed. **Results:** Salatrim intake caused a 5% increase in weight-loss ($p=0.02$ for Salatrim effect). This was also manifested as a reduction in fasting insulin ($p=0.02$). In adipose tissue, Salatrim altered expression of 14 out of 41 assayed genes related to inflammation and lipid metabolism. This was not only an effect of the weight-loss, since only 7 of the genes were altered in the direction of the lean reference group. In the liver, only 4 out of the 41 assayed genes were significantly changed. FO increased the weight-loss induced reduction in hepatic TAG levels ($p<0.01$), but no synergistic effects of Salatrim and fish oil was seen on TAG in either the heart or the liver. Combined with the control-fat, Salatrim caused a substantial increase in both cardiac and hepatic free fatty acids (FFA), while it induced a decrease in FFA concentration when combined with the FO-diet. Thus, Salatrim caused systemic alterations in the gene-expression and tissue FFA-levels. The latter effect was modulated by the presence of LC n-3 PUFA in the diet.