P 4-3 Vaccenic acid-mediated reduction in cytokine production is independent of cis-9,trans-11-CLA in human T-helper cells

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The ruminant trans fatty acid vaccenic acid (VA) favourably alters markers of inflammation. However, it is not yet clear whether these effects are attributed to its endogenous conversion to c9,t11-CLA, which is known to possess anti-inflammatory properties. We compared the cytokine reducing potential of VA to c9,t11-CLA in human T-helper (Th) cells as a main source of cytokine production during inflammation. Secondly, we assessed whether a bioconversion of VA to c9,t11-CLA via stearoyl CoA desaturase (SCD) encoded activity takes place in peripheral blood mononuclear cells in order to relate the outcomes of intracellular cytokine measurement to the degree of conversion. VA reduced the percentage of both IL-2 and TNF-α expressing Th cells significantly, but to a lesser extent compared to c9,t11-CLA, as determined by flow cytometry after stimulation of PBMC with PMA+ionomycin. Pre-treatment with the selective PPARγ antagonist T0070907 largely re-established the IL-2 and TNF-α positive Th cell population in both VA and c9,t11-CLA treated cultures. Interestingly, while the portion of VA increased dose-dependently within the cellular lipid fraction, only marginal amounts of c9,t11-CLA were detectable with levels similar to the control. However, SCD mRNA although abundantly expressed in PBMC was not regulated by VA. Conclusively, these results suggest that the cytokine reducing effect of VA in human T cells is independent of c9,t11-CLA, since no bioconversion occurred. Moreover, the data provide evidence that VA mechanistically acts in a manner similar to c9,t11-CLA.

P 4-4 The supplementation of alpha-linolenic acid in humans increased endogenously synthesized eicosapentaenoic acid in cheek cells similar to plasma and cellular blood lipids

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Background: For the analysis of the fatty acid (FA) status in humans, test materials of choice are blood fractions such as plasma, erythrocytes and peripheral mononuclear cells. For the subjects the invasive sample removal is an unpleasant incident in human intervention studies. By contrast, sampling of cheek cells can be considered as a non-invasive alternative.

Aim: To investigate the expressiveness of FA of oral cheek cells as marker for dietary FA intake, cheek cells were collected during a intervention study with the supplementation of either alpha-linolenic acid (ALA; C18:3 n-3) containing oil or oleic acid (OA)-rich (C18:1 n-9) oil.

Methods: The collection of the cheek cells was performed by scraping the inside of a cheek with a brush from 38 subjects. Over an 8 week period the test group (n = 23; Ø 29 y) received ALA by a linseed/sunflower oil mixture, while the control group (n = 15; Ø 39 y) received an n-3 PUFA-free olive oil. Cheek cells and blood were collected on day 0, 7 and 56 of the study. All subjects consumed a diet free of fish, n-3 PUFA-rich oils and linseeds.

Results: The ALA portion in cheek cells was increased from baseline 0.27 % FAME to 0.38 % FAME after 7 days and to 0.51 % FAME after 56 days of ALA supplementation. Additionally, endogenously converted LC-n-3 metabolites like eicosatetraenoic acid (C20:4 n-3) and eicosapentaenoic acid (C20:5 n-3) increased significantly. Olive oil did not affect the n-3 PUFA level in cheek cells while OA increased. The docosahexaenoic acid (C22:6 n-3) in cheek cells remained unchanged during both treatments. Significant correlations between cheek cell FA and FA of plasma and blood cells were found.

Conclusion: The increase of LC-n-3 PUFA in cheek cells reflects that a regular use of ALA-rich oil can improve the n-3 PUFA status. In general, FA of cheek cells can be considered as adequate non-invasive biomarkers for the analysis of FA status after a supplementation with linseed oil mixture and olive oil.