Poster 13

Trans-palmitoleic acid arises endogenously from dietary vaccenic acid

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Trans-palmitoleic acid (C16:1*t*9, alternatively named *trans*-16:1n-7), a *trans* fatty acid (*t*FA) that is assumed to be exclusively diet-derived, has been linked to the beneficial metabolic effects of dairy fat consumption. Recently, plasma phospholipid C16:1*t*9 was cross-sectionally associated with improved plasma triglycerides and lower fasting insulin, and prospectively with lower incidence of type 2 diabetes in elderly [1]. In the present work, we assessed the putative endogenous and intracellular conversion of supplemented vaccenic acid (C18:1*t*11), naturally occurring in dairy fat, to C16:1*t*9. For this purpose, we re-evaluated fatty acid data 1) obtained from human serum following ingestion of C18:1*t*11 and 2) from human peripheral blood mononuclear cells (PBMC) after incubation with C18:1*t*11, respectively. Both studies have been previously published in their entirety [2, 3].

In the human study, the participants consumed a ruminant-fat free diet supplemented with 2.9 g/d C18:1*t*11 and 2.9 g/d C18:1*t*12, or a C18:1*c*9-rich control-supplement, daily over six weeks. In the *in-vitro* approach, PBMC were incubated with 11 µM C18:1*t*11 for 24 h. Serum and PBMC fatty acid distribution including *t*FA were analysed by combining two GC methods, (i) for total fatty acid methyl esters (FAME) (column: DB-225 MS: 60 m × 0.25 mm i.d. 0.25 µm film thickness; Agilent Technologies, USA) and (ii) for hexa- and octadecenoic acid methylesters with *cis*- and *trans*-configuration (column: CP-select: 200 m × 0.25 mm i.d. 0.25 µm film thickness; Varian, Netherlands).

Ingestion of C18:1*t*11 resulted in 8-fold elevated serum levels of C18:1*t*11, compared to both baseline and control group after intervention ($p < 0.001$ each). This increase was accompanied by a significant increase in C16:1*t*9 (5-fold, $p < 0.001$ each). Since the diet was free of C16:1*t*9, and a strong correlation was observed between both fatty acids ($R2 = 0.808$,

$p < 0.001$), it is most likely that C16:1t9 arose from C18:1*t*11, due to chain shortening by two C-atoms. The conversion rate of C18:1*t*11 to C16:1*t*9 was, on average, 17% (range 10% to 30%). Likewise, C18:1*t*12 and the respective C16:1*t*10 showed up in serum, what supports the assumption of an endogenous partial ß-oxidation of the supplemented fatty acids.

In PBMC, the percentage of C18:1*t*11 increased within the cellular lipids from 0.12 ± 0.02% to 17.1 ± 3.7% of total FAME ($p = 0.006$ compared with DMSO-ctrl.). In parallel, C16:1*t*9 increased 25-fold, from 0.01 ± 0.01% to 0.27 ± 0.04% ($p < 0.001$).

We conclude that endogenous C16:1*t*9 is not exclusively diet-derived but may also be produced by partial (peroxisomal) ß-oxidation of dietary C18:1*t*11.

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