

Influence of Milk Lipids on Human Colon Cells

Christian Degen, Alfred Lochner, Katrin Kuhnt, Gerhard Jahreis,
Friedrich Schiller University, Institute of Nutrition
Jena, Germany

Until now the influences of milk including dairy products on carcinogenesis remains controversially. Some ingredients like calcium, vitamin D and conjugated fatty acids are assigned to exhibit positive effects *in vitro* and *in vivo*. In terms of fat, negative or no effects are associated with saturated fatty acids (SFA), monounsaturated fatty acids (MUFA) and *trans* fatty acids (*t*FA). The aim of this study was to elucidate the impact of the origin and quality of milk lipids on the late stage of carcinogenesis *in vitro*.

Milk lipid extracts of different origin (alpine vs. conventional; MLalp vs. MLcon.) were tested in comparison to control fat (olive oil; OO). Testing of lipid compounds *in vitro* requires a solution of free fatty acids (FFA). In brief, total fat was saponified with ethanolic NaOH and neutralised to pH 7.0 following extraction of FFA with chloroform. The distribution of FFA (MLalp, MLcon, OO) was carried out after derivatisation to fatty acid methylesters by qualitative GC-FID. Some fatty acids, including palmitic acid and stearic acid, were quantified by GC-FID using an internal standard. Total FFA were adjusted to 200 mM in ethanol. Tumorigenic HT29 adenocarcinoma cells were exposed to the medium containing different types of FFA solutions (100 μ M). The cell viability and proliferation were determined subsequently after 24 h incubation (n = 3, in duplicate). The viability of the cells was assessed by measurement of the metabolic activity (CellTiterBlue®; Promega). The proliferation was determined with the DAPI-assay. Incorporation and lipid metabolism of the cells were determined after fatty acid analysis. Preliminary results showed the incorporation of the different FFA mixtures into the cell membranes. Incubation with 100 μ M yielded viability > 90 % and did not affect cell proliferation. In addition, the conversion of *t*11-C18:1 into *c*9,*t*11-CLA was observed. Incubation with MLalp leads to a significantly higher rate of *c*9,*t*11-CLA compared to MLcon.

This incubation method can help to understand mechanism and importance of anticarcinogenic compounds. In case of CLA, it could clarify the difference between the incubation of pure CLA-isomer and a complex milk lipid extract depending on origin and quality.