Effect from digested cod liver oil of different quality on oxidation, energy metabolism, proteome in yeast (*Saccharomyces cerevisiae*)

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It has lately been recognized that the conditions of the gastrointestinal (GI) tract appear to be pro-oxidative. Thus, PUFA-containing food items may oxidize not only during storage, but also during the GI-passage. We have recently shown that in vitro GI digestion of cod liver oil stimulated TBA-reactive substances (TBARS) formation in both the gastric and intestinal steps, while levels of lipid hydroperoxides remained nearly constant. Preformed oxidation products in the cod liver oil resulted in further elevated TBARS levels during the digestion. The aim of this study was to investigate how in vitro digested fresh and slightly rancid cod liver oils affected metabolic activity, intracellular oxidation and proteome in yeast (*Saccharomyces cerevisiae*) *cells.*

Cod liver oils with two initial levels of TBARS (3.8 µmol/kg, fresh and 21.8 µmol/kg, slightly rancid) were subjected to a static in vitro digestion model. Yeast in the stationary growth phase was then exposed to the digested oils at a concentration of 1.7 mg/ml. After 2 h incubation, the effect of both digests was studied at a cellular level by measuring cell energy metabolic activity, intracellular oxidation and proteome. The latter was done by analyzing mitochondrial proteins with 2-D electrophoresis. Differentially expressed proteins were identified by mass spectrometry.

Results showed that TBARS values increased to 71 and 273 µmol/kg lipid during digestions of the fresh and slightly rancid cod liver oils, respectively. Both digests increased intracellular oxidation and cell energy metabolic activity compared to untreated cells. No differences between digested rancid and fresh oils were measured. At the proteome level mostly down-regulation of proteins was observed and was more intensive for digested rancid oil compared to digested fresh oil. Among the down-regulated proteins were enzymes involved in organic acid metabolism, in regulation of intracellular acetyl-CoA pool and in regulation of the metabolic flux through the citric acid cycle. The enzymes might be inhibited by high cell energy charge resulting from the increased intracellular oxidation and energy metabolism.