# Lipid Digestion increases Endotoxemia in Humans and *in vitro*

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Obesity and type 2 diabetes are nutritional diseases that have become major health problem, characterized by low-grade endotoxemia and inflammation. Recent studies have demonstrate a relationship between endotoxemia, inflammation and nutrition. Some reports have postulate that high-fat diets led to the absorption of lipopolysaccharides (LPS) from Gram negative bacteria present in the intestinal microflora. This absorption would contribute to the onset and maintenance of low-grade inflammation because LPS, so-called endotoxins, are proinflammatory compounds.

In this study, we aimed to elucidate the role of dietary lipids on intestinal endotoxin absorption in humans and using Caco-2 cells. In humans, we thus measured the kinetics of endotoxemia and triacylglycerol during the digestion of a mixed meal containing 33 g of lipids. *In vitro*, Caco-2 cells were incubated with mixed lipid micelles with or without added LPS.

In humans, we observed a transient increase of postprandial endotoxemia during digestion of the meal, and increasing plasma concentration of the LPS receptor, sCD14. *In vitro*, incubation of Caco-2 cells with increased fatty acid concentrations enhanced epithelial absorption of LPS.

Our results demonstrate that dispersed lipids can increase postprandial endotoxemia in humans, which may be dose-dependent. Controlling lipid digestion can thus be a possible strategy to limit low-grade inflammation.