

# Effects of nutritional fat on the growth of intestinal *E. coli*

## Projekt: 544

*Benjamin Misselwitz<sup>1</sup>, Wolf-Dietrich Hardt<sup>2</sup>, <sup>1</sup>Klinik für Viszerale Chirurgie und Medizin, Inselspital Bern, <sup>2</sup>Institut für Mikrobiologie, ETH Zürich*

Nutrition and diet significantly impact all physiological processes in the gastrointestinal tract and also determine the composition of the intestinal bacterial population (microbiota). Characteristics of a «Western» diet include high concentrations of fat and a Western diet is an established risk factor for obesity, diabetes mellitus, and atherosclerosis. Mouse experiments in the laboratory of Prof. Wolf-Dietrich Hardt suggested that a high-fat diet can also increase the risk for bacterial intestinal infections with *Salmonella* (*Salmonella typhimurium*, *S. Typhimurium*). This could be explained by higher concentrations of bile acids in the mouse intestine upon exposure to the high-fat diet. However, whether these observations also apply to human patients remains unclear.

In this project, we tested, whether a high-fat diet can increase the colonization of the human intestine with the bacterium *Escherichia coli* (*E. coli*). This bacterium can be applied safely as an approved drug (Mutaflor®). *E. coli* are closely related to *S. Typhimurium* and results with one bacterium can likely be transferred to the other bacterium.

We recruited 8 healthy volunteers; these participants consumed either a high-fat diet (target:  $\geq 150\text{g}$  fat per day) for 4 days or a low-fat diet (target:  $\leq 20\text{g}$  fat per day). After a wash-out period of two weeks with normal nutrition, participants consumed the other diet. On the third day of each diet phase, participants ingested a defined dose *E. coli* (Mutaflor®). Participants sampled and froze their stool every day and stool samples were analyzed at the end of the study.

During the study, participants complied with dietary restrictions and increased or decreased the fat content of their diet accordingly. Interestingly, during all dietary phases, in the stool, the amount of the bacteria of interest did not change significantly. Bacteria ingested during the study (*E. coli*) could be found in stool; however, during the phase with high-fat nutrition, lower *E. coli* concentrations were found than during low-fat nutrition. While in mouse experiments bile acid concentrations significantly increased upon exposure to the high-fat diet, these results could not be confirmed with human participants and bile acid concentrations were indistinguishable during both dietary phases. Therefore, our study with human volunteers could not confirm the mouse experiments. These results hint at differential regulation of bile acid production and secretion in humans and mice.