GLYCEROL METABOLISM BY THE HUMAN COLONIC MICROBIOTA

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INTRODUCTION & AIM: Upon ingestion of dietary lipids, significant amounts of glycerol may reach the colonic microbiota intact. In an anaerobic environment glycerol is typically transformed into 1,3-propanediol, with 3-hydroxypropanal (3-HPA) as intermediate. In solution, 3-HPA is part of the HPA-system, also known as reuterin. Reuterin may have significant health-modulating effects that range from a broad antimicrobial activity to (geno)toxicity as it can chemically bind biological molecules in the gastrointestinal tract. Only a few species are known to have the tools for this fermentation process. Although all of them are numerously present in the gastrointestinal tract, the glycerol metabolism has barely been studied in mixed cultures of the human colonic microbiota.

MATERIALS & METHODS: Faecal samples were obtained from 10 healthy volunteers. Upon homogenization, yeast extract (7 g/L) was added to the inoculum resulting in a final dilution of 1 to 50. All faecal samples were either incubated without and in the presence of 140 mM glycerol and samples were collected regularly for quantitative analysis of SCFA, lactate, glycerol, HPA and 1,3-PDO. Qualitative changes in the total bacterial community were investigated using PCR-DGGE. For integrated data analysis Principal Component Analysis (PCA) was performed on metabolic parameters, microbial community parameters and on the combination of both.

RESULTS & DISCUSSION: For all data sets, PCA resulted in a clear separation of the treated and untreated incubations, demonstrating that glycerol addition significantly altered the faecal microbial metabolism and community composition. On the one hand, glycerol treatment resulted in a decreased concentration of branched SCFA, demonstrating that a more saccharolytic metabolism took place. On the other hand, microbial PCA showed a shift in the total bacterial community caused by glycerol addition. More specifically, while untreated samples were scattered on the plot, glycerol treated samples were grouped close together, implying a directional effect of glycerol to certain bacterial species in all faecal samples.
Among the treated incubations a variable metabolic response to the addition of glycerol was found. Metabolic PCA allowed to identify three groups of treated samples. One group was formed due to their common fast glycerol consumption, high 1,3-PDO yield and high acetate production. In contrast, a second group was characterized by very slow glycerol consumption, a low 1,3-PDO yield and low acetate production. The remaining five incubations had intermediate glycerol consumption and 1,3-PDO production. Remarkably, faecal samples with a slower glycerol consumption displayed an increased propionic acid and/or butyric acid concentration. We therefore hypothesized that glycerol reduction to 1,3-PDO competes with other hydrogen consuming reactions, such as propionate and butyrate production. A slow glycerol consumption rate can then be considered health promoting because of i) cholesterol lowering effects from propionate and ii) butyrate inducing apoptosis of colon cancer cells. The rate of glycerol consumption could thus play a role in the occurrence of obesity, associated cardiovascular diseases and colon cancer, implying that rapid glycerol fermentative persons should minimize their glycerol consumption.