

Results: The mean total quantities of SCFA in the portal blood were, respectively, for PoS, HS and RHS: 131.4 ± 25.8 , 85.0 ± 13.3 and 163.0 ± 35.5 mmol with HS statistically different from PoS and RHS. The mean total quantities of nC4 measured in the portal vein varied significantly between starches: PoS (15.2 ± 2.8 mmol) versus HS (0.2 ± 0.2 mmol) and RHS (3.1 ± 1.1 mmol).

Conclusion: The mean total quantities of SCFA measured in the portal vein agree with previous studies. The nC4 produced from HS and RHS fermentation seems to be completely used up by the colonic mucosa. On the other hand, after consumption of the PoS diet, a part of the nC4 appears in the portal blood. This result could indicate a saturation of its utilization by the colonocytes when a large amount of nC4 is produced, as previously observed *in vitro*.

Measurement of the colonic acetate production after lactulose ingestion in humans – using isotopic dilution.

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Lactulose is a disaccharide which is not digested or entirely degraded by colonic fermentation. This results in an exogenous acetate supply. Our aim was to evaluate quantitatively this colonic acetate production following lactulose ingestion. Six healthy volunteers (24–38 years, 21.7 ± 1.5 kg·m⁻²) were given a low fiber diet for 3 days (< 5 g·d⁻¹). They then received a prime of [¹⁻¹³C] acetate (33 ± 10 μmol·kg⁻¹) intravenously and were infused with the same tracer (1.0 ± 0.1 μmol·kg⁻¹·min⁻¹) for 7 h. At $t = 60$ min, the subjects received an oral dose of 20 g of lactulose. Breath samples and arterial blood samples were collected every 15

min throughout the study. The isotopic enrichment levels and the concentrations were measured by gas chromatography/mass spectrometry. The acetate turnover was calculated, after being validated, with the steady state equation at each sample point. During the initial period, the hydrogen and methane levels were 7 ± 2 and 10 ± 4 ppm, respectively. The concentration and the acetate turnover were 141 ± 14 μmol·L⁻¹ and 6.0 ± 0.7 μmol·kg⁻¹·min⁻¹, respectively. At $t = 195$ min, the hydrogen and acetate concentrations reached 63 ± 15 ppm and 313 ± 25 μmol·L⁻¹, the methane level remained unchanged. Simultaneously, the whole body acetate turnover increased to 9.8 ± 1.5 μmol·kg⁻¹·min⁻¹ and then decreased, to close to the initial value, at the end of the study. The area under the curve of the whole body acetate turnover variations minus the constant initial turnover, supposed to be endogenous, represents the colonic exogenous acetate supply. It was 140 ± 12 mmol, which was 86 % of the expected stoichiometric amount. This approach is unique. It made it possible to quantitatively estimate the colonic acetate production and thus the colonic fermentation, *in vivo*, in humans.

In vitro fermentation of Acacia gum by human fecal flora: effects on the bacterial populations and on the production of short-chain fatty acids.

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Several carbohydrates have been identified as prebiotics. They are not digested in the small intestine but are highly fermented in the colon where they specifically promote the growth of lactic acid bacteria. Although prebiotics are mainly oligosaccharides, high molecular weight polymers are likely to exert similar effects. This study aimed to determine the effects

of Acacia gum polymers (GA) on both human fecal flora composition and metabolism in vitro.

Methods: GA, together with short chain fructo-oligosaccharides (FOS), were incubated with human fecal bacteria in a continuous fermentor for 10 days. The culture was operated at 0.1 h⁻¹ dilution rate, under an anaerobic atmosphere and the pH was controlled at 6.5. After eight bacterial population turn-overs, the short-chain fatty acids (SCFA) concentrations ($n = 5$) and the numbers of bacteria from the main intestinal genera ($n = 3$) were determined from culture effluents. Results were statistically (ANOVA) compared.

Results: SCFA concentration was significantly ($P < 0.05$) increased by both GA and scFOS fermentation (81.2 ± 2.8 and 73.3 ± 3.5 versus 40.1 ± 2.3 mM for GA, scFOS and control, respectively). The molar proportion of butyrate was significantly ($P < 0.05$) higher from GA (17.1 ± 0.1 %) compared to scFOS (12.9 ± 0.9 %) metabolisms. Both GA and scFOS increased *Lactobacillus* sp. members compared to the control (7.8 ± 0.1 and 7.6 ± 0.2 versus 6.1 ± 0.1 log CFU.mL⁻¹ for GA, scFOS and control, respectively). Conversely, none of these carbohydrates induced changes in total anaerobes nor in total aerobic bacterial cells.

Conclusion: In vitro, GA and scFOS exhibit similar effects both on SCFA production and bacterial composition of human fecal microbiota. Thus, GA could exert a prebiotic effect similar to that of scFOS.

In vivo enhancing effect of Propionibacteria on the growth of fecal Bifidobacteria. Relationship with colonic transit time. D. Bouglé, N. Roland, F. Lebeurier, J.L. Maubois, P. Arhan (Laboratoire de physiologie digestive et nutritionnelle, CHU, Avenue Général Clémentineau, 14033 Caen cedex; Stand-Industrie, Caen; Inra, Rennes, France).

While several reports suggest that probiotics are liable to modulate digestive transit, they do not provide details concerning the changes in the digestive flora and their functional consequences.

Colonic transit time was compared with fecal flora during supplementation of healthy subjects ($n = 19$) with Propionibacteria, regularly used for cheese making. Five 10¹⁰ CFU of two freeze dried strains (equivalent to the ingestion of 100 g Emmental) were ingested daily for 2 weeks. They came from strains known to stimulate in vitro the growth of bifidobacteria.

Segmental colonic transit time was measured by counting radiopaque markers on a plain X-rays of the abdomen before and at the end of the first week of supplementation. Viable Propionibacteria and Bifidobacteria were counted in feces collected weekly, twice before (D-8; D-1), twice during (D7; D14), and twice after the end of supplementation (D21; D28).

Results (table below):

Transit time in right and left colons, and rectosigmoid were 17.4 ± 8.1 , 7.0 ± 5.0 , 12.8 ± 8.5 h before and 17.3 ± 8.3 (NS), 11.9 ± 9.4 ($P = 0.02$), 13.3 ± 10.2 (NS) during supplementation. A significant correlation was found between tran-

	Before 1	Before 2	During 1	During 2	After 1	After 2
Propioni.	< 5*	< 5	5.6 ± 0.6	6.5 ± 0.6	< 5	< 5
Bifido.	7.6 ± 0.7	8.2 ± 0.6	8.3 ± 1.0^a	8.2 ± 1.2^a	8.5 ± 1.3^a	7.7 ± 1.6^b

* log CFU/mL feces; mean \pm 1SD; ^adifferent from Before 1 ($P < 0.05$); ^bdifferent from After 1 ($P < 0.05$).