

tolerance. This may reflect the survival of low amounts of immunoreactive proteins along the gut (Tukur *et al* (1993), *op cit*) and their passage in the blood. In addition, calf Ab titres at weeks 4 and 7 were significantly correlated ( $r = +0.64$  and  $+0.62$  respectively,  $P < 0.05$ ) with those in the dams 2 months after calving. These limited results suggest direct (genetic) and/or indirect (colostral) influences of cows on the subsequent systemic Ab response of their offsprings to dietary antigens.

In conclusion, both young and adult ruminants appear to produce dependent but rather moderate levels of plasma antisoya Abs.

**$\alpha$ -Galactosides are poorly digested by germ-free chickens.** B Carré<sup>1</sup>, A Brée<sup>2</sup>, J Gomez<sup>1</sup> (<sup>1</sup> INRA, Station de Recherches Avicoles; <sup>2</sup> INRA, Station de Pathologie Aviaire et de Parasitologie, 37380 Nouzilly, France)

$\alpha$ -Galactosides were previously found to be readily digested by chickens [Carré and Lacassagne (1992) Proc 1st Eur Conf Grain Legumes, Angers, France, Assoc Eur Protéagineux, Paris, 481-482]. However, the origin of this digestion (from endogenous enzyme activity or microbial fermentation) is not known. The current study, conducted with germ-free chickens, was carried out to investigate the origin of  $\alpha$ -galactoside digestion in chickens.

Seven white Leghorn chickens (PA 12 strain) were hatched in a germ-free isolator and reared in a cage with *ad libitum* feed and drink, in germ-free conditions [Schellenberg and Maillard (1973) In: Journées Rech Avicoles Cunicoles INRA-ITAVI-WPSA, ITAVI, Paris, 283-285]. They were fed a diet sterilized by irradiation (50 kGy), and formulated to contain 21.1% protein, 2 790 kcal/kg, 1.18% calcium and 0.66% phosphorus (Extral 1M, Extralabo, Ets Pietrement, Société Colombe, France). At 5 weeks of age, a sterilized plastic foil was put under the cage, all excreta were collected for 1 d, put in a sealed sterilized box, immediately frozen at  $-20^{\circ}\text{C}$  and then freeze-dried.

A sample of excreta was incubated and revealed no contaminating bacteria.

Polysaccharide xylose (from cereals, soyabean and sunflower meals occurring in diet), considered here an undegraded and unabsorbed marker of feed, was measured in both diet and excreta, using GLC analysis of acid hydrolysates

[Carré *et al* (1990) *Poult Sci* 69, 623-633]. Polysaccharidic xylose was measured in 6 replicates of feed and excreta.

Oligosaccharides were extracted in methanol/water 50:50 under reflux and determined by GLC analysis of trimethylsilyl derivatives [Sweeley and Walker (1964) *Anal Chem* 36, 1461-1466] using a capillary column (BP1, SGE, Australia), with melezitose as internal standard. Analyses of oligosaccharides were done on 8 replicates of feed and excreta.

Raffinose and stachyose, mainly originating from the soyabean meal in the diet, amounted to 0.39 and 0.79% dietary level, respectively. Their excreta recoveries were  $0.86 \pm 0.133$  (SD) and  $0.94 \pm 0.131$  (SD), respectively. The latter high values show a nearly complete lack of endogenous  $\alpha$ -galactosidase activity in the chicken digestive tract. Accordingly, the high digestibility values (0.80-1.0) previously found in chickens for  $\alpha$ -galactosides [Carré and Lacassagne (1992), *op cit*] were due to microbial degradation.

**Effects of resistant starch supplementation on postprandial metabolism in healthy subjects.** N Faisant<sup>1</sup>, M Champ<sup>1</sup>, S Ranganathan<sup>2</sup>, C Azoulay<sup>3</sup>, MF Kergeris<sup>3</sup>, M Krempf<sup>2</sup> (<sup>1</sup> INRA, Laboratoire de Technologie Appliquée à la Nutrition; <sup>2</sup> Centre de Recherche sur Volontaire Sain, CHU Nord; <sup>3</sup> Laboratoire de Pharmacologie, Hôtel-Dieu, 44000 Nantes, France)

Resistant starch (RS) is defined as the fraction of starch not absorbed in the small intestine of healthy individuals. These structures reach the colon and are generally fermented by the microflora. The digestive fate of RS is thus comparable to that of dietary fibers and their physiological effects should be considered in that way. When RS was substituted for digestible starch, postprandial glucose and insulin [Behall *et al* (1988) *Am J Clin Nutr* 55, 81-88] and fasting triglycerides and total cholesterol after several weeks were decreased [Behall *et al* (1989) *Am J Clin Nutr* 49, 337-344]. However, the role of RS added to a meal has not been investigated. Knowing the consequences of a supplementation with some dietary fibers on postprandial glycemia [Wolever and Jenkins (1982) *J Plant Foods* 4, 127-138] and lipemia [Cara *et al* (1992) *Am J Clin*

*Nutr* 55, 81-88], we tested the postprandial 'fiber effect' of the supplementation with RS.

Six volunteers tested the control meal as a breakfast (providing 2 600 kJ, 20% as proteins, 40% as lipids, 40% as carbohydrates) and the supplemented meal (control + 26 g dry raw potato starch (RPS) providing 22 g RS) at one-week intervals, in a random order. Vitamin A (100 000 UI) was added to each meal, since postprandial plasma vitamin A ester was used as a marker for exogenous lipid metabolism. Blood samples were collected every 30 min for 7 h from the beginning of the meal. Glucose, insulin, triglycerides (TG), total cholesterol and vitamin A ester concentrations were measured in plasma. Chylomicron fraction was isolated from the plasma and TG and cholesterol were analyzed.

The supplementation with 30 g RPS did not modify postprandial glycaemia and insulinemia. While RPS provided a small fraction of digestible starch (5 g), this was not perceptible on glycaemia. In the same way, no significant effects on mean plasma and chylomicron TG and cholesterol responses were observed. However, 2 out of the 6 subjects, with high basal TG levels (1.8 and 1.9 mmol/l) had their chylomicron TG and cholesterol decreased by the supplementation. Plasma vitamin A ester response decreased from  $0.42 \pm 0.10$  g/l to  $0.29 \pm 0.08$  g/l over 7 h (mean  $\pm$  SEM,  $n = 6$ ), meaning that RS supplementation altered intestinal lipid absorption. While the supplementation of a normal meal by RS had little or no effects on postprandial blood parameters of normal subjects, the intestinal absorption of lipids appeared to be impaired and chylomicron remnant clearance improved in the case of 2 subjects with high fasting TG levels. These findings should be confirmed on hypertriglyceridemic patients.

### First characterization of lipid emulsification in the human stomach.

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Fat emulsification is a key step in fat digestion. For instance, the available lipid droplet surface is a

rate-limiting step on lipase action, as recently shown *in vitro* [Armand *et al* (1992) *J Nutr Biochem* 3, 333-341; Borel *et al* (1994) *J Nutr Biochem* 5, 124-133]. Though believed to be important, fat emulsification has never been studied in the human digestive tract. Thus, this study aims to determine the extent of fat emulsification in the stomach of healthy subjects, and the influence of lipolysis on gastric lipase.

Each subject was given a coarsely emulsified test meal (droplet median diameter: 52.9  $\mu$ m) through a naso-gastric tube. The meal contained commercial olive oil, 1 raw whole egg, 1 egg white and sucrose and was brought to a total volume of 400 ml with 0.15 mmol/L NaCl. The liquid meal provided 960 kcal (4010 kJ) with 29.5% carbohydrate, 65.5% fat and 5.0% protein. The meal also contained a reasonable amount of cholesterol (about 250 mg) and the triglycerides/phospholipides ratio (w/w) was close to 40:1 which is representative of usual western diets.

Gastric contents were collected 1, 2, 3 and 4 h after intubation of the test meal. Gastric lipase activity was determined using a pH-Stat titrator, the different lipid classes (free fatty acid, monoglycerides, diglycerides, triglycerides, and cholesterol) were analysed by thin-layer chromatography and video-densitometry, and fat globule size was measured using a particle-size analyser (Capa 700).

Gastric lipase activity was about 25 000 U/L 1 h after meal intake and steadily returned to higher levels close to value measured in the fasting state after 4 h (56 000 U/L). The extent of olive oil triglyceride lipolysis was in the range of 9-12%.

As regards fat globule size, we observed that during intra-gastric digestion, non-emulsified fat (droplet diameter  $\geq 100$   $\mu$ m) represented a minor fraction. A significant amount of large 70-100  $\mu$ m lipid droplets present in the test meal disappeared and fine 1-10  $\mu$ m droplets were generated. Thus, the emulsion median diameter decreased (21.9 vs 52.9  $\mu$ m) and consequently emulsion surface area increased (1.56 vs 0.58 m<sup>2</sup>/g), indicating a significant increase of dietary lipid emulsification.

In conclusion, the present data demonstrate for the first time that in human stomach most dietary lipids are present in the form of emulsified droplets, in the range of 20-40  $\mu$ m, and that gastric lipolysis (12%) can play an important role in fat digestion by facilitating fat emulsification.