

**Diversion of bile and pancreatic juice and oleic intestinal absorption in the rat.** Y Mathieu, A Bernard, H Carlier (*ENS-BANA, Université de Bourgogne, Laboratoire de Physiologie de la Nutrition, Campus Universitaire, 1 Esplanade Erasme, 21000 Dijon, France*)

A decrease of 30% in  $^{14}\text{C}$  oleic acid lymphatic intestinal absorption was observed in bile and pancreatic juice diverted rats (unpublished results). Some authors suggested a failure in the mucosal esterification of long-chain fatty acids and an altered route of lipid transport in the absence of bile (Hyun *et al*, 1967; Kayden and Medick, 1969; Tso *et al*, 1981). To elucidate these hypotheses,  $^{14}\text{C}$  oleic acid portal absorption and mucosal biochemical processes were investigated in bile and pancreatic juice diverted rats.

In experimental groups, 1 d before the experiments, the common bile duct was cannulated under ether anaesthesia. In control and experimental groups vascular perfusion was performed under ethyl ether anaesthesia and a 15-cm long intestinal loop was isolated *in situ* between 2 metallic cannulae (Bernard and Carlier, 1981). Mesenteric portal venous blood was continuously collected at 5-min intervals for 60 min after the intraduodenal infusion of 90  $\mu\text{mol}$  of an equimolar emulsion of  $^{14}\text{C}$  oleic acid (5  $\mu\text{Ci}$ ) palmitic acid and monopalmitin. At the end of the experiments, the intestine was rinsed, immediately removed and opened longitudinally for scraping. Lipids were extracted from the luminal contents, scraped mucosa and from aliquots of blood samples.

In 2 other groups of rats, 1 control (without diversion of bile and pancreatic juice), and 1 experimental (bile and pancreatic juice diverted), the same lipid infusate was intraduodenally infused in *in situ* isolated intestinal loops. Six h later, lipids were extracted from the luminal contents and from the scraped mucosa. Vascular perfusion technique failed to demonstrate an in-

crease in the  $^{14}\text{C}$  oleic acid blood transport during the hour following its intraduodenal administration in bile and pancreatic juice diverted rats. However, a significant simultaneous impairment of  $^{14}\text{C}$  oleic acid lymph intestinal absorption was observed (the radioactive lymph recovery represented 38.4% of the administered radioactivity in the experimental group vs 65.2% in the control group), particularly during the second 30 min after administration. One h after intraduodenal infusion, a significant decrease in triglyceride esterification occurred in the mucosa, but without significant increase in mucosal labelled lipids.

Six h after the outset of administration of the lipid emulsion, the amounts of radioactive lipids taken up by intestine did not differ significantly for the 2 groups of rats. However, in the experimental groups a significant load of radioactive lipids in the mucosa was noted (6-fold higher than in controls). Simultaneously, a failure of the enterocyte esterification processes was observed in the experimental group in comparison with the control group. Thus 3-fold higher amounts of labelled mucosal lipids were recovered in the fatty acid class in the experimental group compared to the control group.

These data corroborate previous studies on a decrease in long-chain fatty acid lymph absorption in the absence of bile and pancreatic juice. But despite a demonstrated failure in mucosal esterification processes and the existence of an overload of exogenous lipids in the mucosa, compensating portal blood absorption of labelled oleic acid was not detected during the postprandial period.

### References

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