Intestinal metabolism of lineoleic acid during its intestinal absorption in the rat. A Bernard [1], C Caselli [1], JP Blond [2], H Carlier [1] *([1] ENSBANA, Laboratoire de Physiologie de la Nutrition; [2] Université de Bourgogne; Unité de Recherches Nutrition Cellulaire et Métabolique, 21000 Dijon, France)*

A desaturation and elongation process of ^{14}C linoleic acid into ^{14}C arachidonic acid was detected at the peak of absorption of ^{14}C linoleic acid infused intraduodenally in the rat (Bernard *et al,* 1991). Although the intestine possesses a $\Delta 6$ desaturase activity (Garg *et al,* 1988), arachidonic acid of liver origin was generally suspected (Hjelte *et al,* 1990). To help to clarify this point, we investigated to what extent the intraduodenally infused ^{14}C linoleate taken up from the portal blood by the liver in its initial chemical form or converted in the liver to radiolabelled arachidonate was secreted in bile.

For this purpose, taking into account previous results (Bernard *et al,* 1991), the bile of 9 adult male Wistar rats was diverted. Twenty-four h later, 90 µmol of an equimolar lipid mixture (oleic acid, monopalmitin, linoleic acid added with 5 µCi 1–^{14}C linoleic acid 30/30/30 mol/ mol/ mol) emulsified with 1 ml bile and 0.5 ml Ringer's solution were infused intraduodenally as a single dose. In a first group of 6 animals, the bile was harvested at 1-h intervals for 6 h after the onset of lipid infusion.

Two h after the onset of lipid emulsion administration, *ie* just before the lymphatic peak of ^{14}C lineolic absorption (Bernard *et al,* 1991), the mucosa of a second group of animals (3 rats) was scraped and intestinal walls removed. The radioactivity of lipid extracts from bile samples, from the rest of the infused lipid emulsions, from the mucosa and the intestinal walls was measured by liquid scintillation counting. The labelled lipid extract fatty acids were analyzed by reverse phase liquid chromatography (Narce *et al,* 1988).

The appearance of labelled arachidonic acid in the lymph (4–6% of the radioactivity) at the absorption peak (from the 2nd to the 4th h of lymph collection) seemed to be a consequence of the simultaneous appearance of labelled arachidonic acid detected in the mucosa and the intestinal wall (from 0.4–1.25% the radioactivity). Thus, the significant quantity of labelled arachidonic acid in the lymph preceded the significant radioactivity peak observed in the bile which occurred only 4 h after lipid administration. Moreover, in the lymph the radioactivity recovered of the arachidonic acid (400 nmol) during the 6 h of collection was significantly higher than the total radioactivity recovered in the bile (110 equivalent nmol of either linoleic or arachidonic acid) during the same time period. The labelled arachidonic acid detected in the intestine revealed an enrichment from the mucosa to the intestinal wall for each animal. Dividing lymph samples throughout the absorption process allowed the concentration in lymph samples of labelled arachidonic acid synthesized from labelled linoleic acid in the mucosa. Indeed, the biotransformation of linoleic acid to arachidonic acid in the mucosa was masked by a dilution which took place due to the luminal uptake of labelled linoleic acid by the enterocytes and the simultaneous secretion of lipoproteins containing esterified labelled arachidonic acid.

Thus, under our experimental conditions, at the peak of labelled linoleic acid absorption where a concentration of exogenous lipids occurred in their chemical initial form or as their metabolites, the enterocytes ability to convert linoleic acid to arachidonic acid was demonstrated.

References

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