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**In vitro study of the intestinal conversion of linoleic and  $\alpha$  linolenic acids.** JP Blond<sup>1</sup>, A Bernard<sup>2</sup>, C Caselli<sup>2</sup>, JP Poisson<sup>1</sup> (*Département de biologie appliquée, IUT de Dijon et <sup>1</sup> Unité de nutrition cellulaire et métabolique, EA DRED 1867, UFR Sciences de la Vie; <sup>2</sup> Département de nutrition EA DRED 580, ENSBANA, université de Bourgogne, France*).

We have investigated the bioconversion of the two essential polyunsaturated fatty acids (EFA), linoleic acid (18:2,  $n = 6$ ) and  $\alpha$  linolenic acid (18:3,  $n = 3$ ) by homogenates or microsomes of rat intestinal mucosa and by mouse jejunal explants.

Eight milligrams of homogenate proteins or 5 mg of microsomal proteins were incubated with 20 or 120 nmol of each <sup>14</sup>C labelled substrate, under the conditions used for rat liver desaturations. Desaturase activities were calculated from the conversion percentage measured by HPLC.

Desaturase activities 3.5 to 7-fold lower for the intestine than for the liver and 18:2,  $n = 6$  was more desaturated than 18:3,  $n = 3$ .

In the mouse intestinal explants incubated with the same substrates, the conversion percentage of 18:2,  $n = 6$  to desaturation-elongation products was 3.5%, whereas it was 2.1% with 18:3,  $n = 3$  by homogenates. Arachidonic (20:4,  $n = 6$ ) and docosahexaenoic (DHA, 22:6,  $n = 3$ ) acids, the main biosynthesized essential fatty acids represented 20 and 90% of the fatty acids derived from 18:2,  $n = 6$  and 18:3,  $n = 3$ , respectively.

These results confirm our previous studies performed on in vivo fatty acid absorption by rat intestine and confirm a bioconversion of the two studied EFA during the first step of their absorption in the intestine.

However, in the absorptive intestinal cells, only a few of each fatty acid was metabolized by desaturation-elongation. The differences between linoleic acid and  $\alpha$  linoleic acid are probably related to a dif-

ference in the acylation rates into phospholipids and triacylglycerols synthesized during the absorption.

**Gestational diabetes: influence of food intake on birth weight, HbA1c, and maternal weight gain.** MC Nuttens, O Verier-Mine, S Biaisque, A Wambergue, M Romon (*DIAGEST group*).

There is still some controversy about nutritional management of gestational diabetes (GD). The aim of this work was to study the influence of energy and macronutrient intake on infant birth weight, maternal weight gain and glycemic control in women with GD undergoing intensive management. It was realised among 80 women (age  $30.2 \pm 5.3$  years, BMI  $25.2 \pm 5.2$  kg/m<sup>2</sup>), with GD or mild carbohydrate intolerance according to the criteria of Carpenter and Coustan. Management began between 24 and 34 weeks of gestation. Pre-management food intake (0) was assessed by a diet history. The energy content of the prescribed diet was based on the pre-pregnancy BMI and pregnancy weight gain, a carbohydrate intake of at least 50% of energy intake was recommended, insulin was prescribed if postprandial glycemia was higher than 1.2g/L.

Results: Follow-up weight gain was correlated to fat intake ( $r = 0.25$ ,  $P < 0.05$ ), hb A1c to energy ( $r = 0.29$ ,  $P < 0.01$ ), fat ( $r = 0.38$ ,  $P < 0.001$ ), and protein intake ( $r = 0.24$ ,  $P < 0.05$ ). Birth weight was correlated to gestational duration ( $r = 0.36$ ,  $P < 0.01$ ) and negatively to smoking ( $P < 0.01$ ) and carbohydrate intake ( $r = -0.27$ ,  $P = 0.02$ ). For carbohydrate intake higher than 210 g/day, there are no macrosomes and percentage of low birth weight was the same. Predictors of birth weight were examined by a forward stepwise regression analysis. Three parameters were significant: gestation duration ( $\beta = 0.35$ ,  $P = 0.02$ ), Carbohydrate intake ( $\beta = -0.24$ ,  $P = 0.02$ )