Effects in the rat of gestation and lactation on the cytosolic liver fatty acid binding protein (L-FAPc) in the liver. A Mallordy, P Besnard, C Berges, A Bernard, H Carlier *(ENSBANA, Université de Bourgogne, Laboratoire de Physiologie de la Nutrition, Campus Universitaire, 1 Esplanade Erasme, 21000 Dijon, France)*

In the liver, among the proteins involved in the lipid metabolism, the liver fatty acid binding protein (L-FABPc) is an abundant cytosolic polypeptide that has a high affinity for saturated and unsaturated long-chain fatty acids. Regulation of the biosynthesis of this binding protein is at present poorly understood. Studies on the effects of feeding diets rich in fats suggest that L-FABPc may be diet-regulated (Bass, 1988). Nevertheless, the use of high-fat diets containing between 30–45% lipids raises the question of the physiological relevance of these data.

Gestation and lactation are natural situations where a physiological enhancement of nutritional needs in general, and lipidic needs in particular, occurs. This phenomenon is more marked in species with large litters. The aim of this study was to investigate the expression of L-FABPc in the rat during these 2 physiological situations.

L-FABPc mRNA levels were studied in the liver of pregnant and lactating rats by Northern and dot–blot hybridization* and compared to those in virgin females of the same age. The L-FABPc specific probe is a 383 base pair (bp) *Pvu*II/*Acc*I restriction fragment isolated from the recombinant plasmid pJG418 (Gordon *et al*, 1983) which encompasses 93% of the coding sequence of rat L-FABPc mRNA.

In pregnant rats on d 22 of gestation, despite a significant increase in food intake (24.5 ± 1.8 g/day *vs* 15.5 ± 1.5 g/d in controls, $n = 8$, $P = 0.001$), a 3.5-fold decrease in L-FABPc mRNA levels detected by Northern blot on total RNA pooled from 4 animals occurred in the liver.

This dramatic decrease was systematically found in each pregnant female when the L-FABPc messengers were assayed by dot–blot hybridization. By this technic, the decrease, quantified by a spectrodensitometer, had a mean of –60%.

In females on d 14 of lactation, food intake was greatly increased (51.5 ± 5.0 g/d *vs* 15.5 ± 1.5 g/d in controls, $n = 8$, $P = 0.001$). In this group, only a slight decrease in L-FABPc mRNA levels was observed.

Our results suggest that a physiological increase in food intake does not trigger an induction of L-FABPc expression in liver, contrary to what is observed in high fat diets. The mechanism by which the messengers coding for L-FABPc respond to gestational status is unknown. Possibilities include hormonally-mediated inhibitor effects on the biosynthesis of this binding protein.

References

Bass NM (1988) *Int Rev Cytol* 3, 143-184

Gordon JI, Alpers DH, Ockner RK, Strauss AW (1983) *J Biol Chem* 258, 3356-3363

* The cDNA probe was extracted from the recombinant plasmid pJG418 generously provided by JI Gordon (Washington University, St Louis, MO 63110, USA).