groups S-U, L-U, S-R and L-R, respectively), whereas the cyclophilin signal remained unchanged. Our results suggest that both photoperiod (at a given food intake) and nutritional status regulate the expression of the gene encoding leptin in ovine adipose tissue, at least in part through pretranslational mechanisms.

Nutritional regulation of lipoprotein lipase activity and its messenger RNAs in ewe adipose tissue and heart. M. Bonnet[a], J.F. Hocquette[b], Y. Faulconnier[a], J. Fléchet[a], F. Bocquier[a], Y. Chilliard[a] ([a]Laboratoire sous-nutrition des ruminants; [b]Laboratoire croissance et métabolismes des herbivores, Inra, 63122 Saint-Genès-Champanelle, France).

The regulation of circulating triacylglycerol (TG) uptake by adipose tissue (AT) or by muscle is a part of an animal's adaptation to fluctuations in their nutritional or physiological status. It was thus interesting to obtain a better knowledge of the factors involved in TG partitioning between these two tissues. This is regulated, at least partly, by the lipoprotein lipase (LPL) activity. LPL activity and the levels of its mRNAs were assayed in perirenal AT and cardiac muscle (CM) of ten adult, dry and non-pregnant ewes. All animals were restricted to 25 % of their maintenance energy requirement (MER) for 7 days, then half of them ($n = 5$) were refed to 200 % MER for 14 days before slaughter. Refeeding increased the LPL activity (expressed per gram of tissue) in both AT (+357 %; $P < 0.001$) and CM (+45 %; $P < 0.05$). Similar trends were observed when the LPL activity was expressed either by whole tissue or by cell. Thus, contrary to previous observations in the rat, refeeding regulated ovine CM LPL activity in the same way as AT LPL activity, although with a smaller effect than in CM. Moreover, northern-blot analyses using an ovine LPL cDNA revealed an increase in LPL mRNA levels after refeeding, both in AT (lack of signals for all the restricted ewes versus strong signals for all the refed ewes) and CM (+140 %; $P < 0.02$). In conclusion, nutritional regulation of LPL gene expression seems to be carried out in the same way in ewe perirenal AT and CM, and, at least partly, by pretranslational mechanisms. The different regulation of CM LPL between ewes and rats probably arises from the peculiarities of nutrient digestion and absorption, and liver lipogenesis, in ruminant species.

Effects of the infusion of β-, β2- or β3-adrenergic agonists or epinephrine on in situ lipolysis in ewe subcutaneous adipose tissue. A. Ferlay[a], C. Charret[a], J. Galitzky[b], M. Berlan[b], Y. Chilliard[a] ([a]Laboratoire sous-nutrition des ruminants, Inra, 63122 Saint-Genès-Champanelle; [b]Inserm U317, Faculté de médecine, Toulouse, France).

An in vivo study of lipolysis is puzzling because changes in plasma glycerol concentrations depend both on the lipolytic activity of adipose tissues and on its utilization by non-adipose tissues. The microdialysis technique makes it possible to study in situ the regulation of lipolysis, which has been rarely investigated in ruminants. Twelve Lacaune ewes (body weight 78 kg and body condition score 3.9 on a 0–5 scale) were underfed at 60 % of their energy requirement for maintenance for 4 days, before the insertion of four probes (Carnegie, 0.5 × 20 mm) in the rump subcutaneous adipose tissue of each animal. The probes were perfused at 5 μL/min for 120 min with 3 β-adrenergic (β-A) agonists: isoproterenol (ISO, non-selective β-A), terbutaline (TER, β2-A), CL316243 (β3-A, Wyeth-Ayerst, USA) or epinephrine (EPI) at 10^{-6}, 10^{-5} and 10^{-4}

M. The glycerol in the dialysate (collected during 15-min periods) was determined by the radiometric method. The data were analysed using the Proc. mixed procedure of SAS (1996). At 10^{-4} M, ISO and EPI had a response area (RA) higher ($P < 0.05$) than TER (9.4, 9.7 and 3.6 mM.min, respectively). The RA during ISO infusion at 10^{-5} M was higher ($P < 0.05$) than the RA during EPI infusion, which was greater than the RA during TER infusion (10.1, 7.7 and 4.9 mM.min, respectively). The different β-A agonists had no significant lipolytic effect at 10^{-6} M, nor did the β3-A agonist at 10^{-5} or 10^{-4} M (RA of 1.0 or 0.7 mM.min, respectively). The marked lipolytic effect of EPI suggests a slight antilipolytic role of the α2-adrenoceptors. The weak lipolytic effect of TER suggests a slight lipolytic role or a desensitization of the β2-adrenoceptors. The lipolytic effect of ISO is probably not due to the stimulation of the β3-adrenoceptor in the ewe.

ADAPTATION TO UNDERNUTRITION

Nutritional recovery after fasting: restoration of body lipids and proteins versus level of depletion. F. Decrock, J.-P. Robin, E. Mioskowski, R. Seyller, A. Koch, H. Karmann, G. Herzberg, R. Groscolas, A.C. Bach (Centre d'écologie et physiologie énergétiques, CNRS, 67087 Strasbourg cedex 2, France).

What is the effect of the level of energy reserve depletion on body lipid and protein restoration during refeeding? Adult rats were fasted for 0 (controls), 5 (phase II of fasting; P2) or 8–16 (phase III of fasting; P3) days, and then refed ad libitum (standard diet) for 0 (R0), 3 (R3), 7 (R7) days, or until reaching (RT) initial body mass (in 6–8 days, P2RT; in 10–13 days, P3RT) and killed. Body mass, body composition, and food intake were determined.

During fasting, lipid and protein loss were 43 and 8 % (P2RO) or 81 and 18 % (P3RO). At the end of refeeding, restoration was total for proteins but partial for lipids (40 %, P2RT; 76 %, P3RT). After a massive depletion of energy reserves (P3), lipid restoration was rapid and energy efficiency high. After a moderate depletion (P2), protein restoration was rapid and energy efficiency was lower.

To conclude, the relative rate of body lipid and protein reserve restoration depends on the level of their previous loss. Lipids are preferentially restored after having been nearly exhausted.

Groups ($9 \leq n \leq 12$)	P2R0	P2R3	P2RT	P3R0	P3R3	P3R7	P3RT
Lipid loss/gain (g)	-19.6^{b}	$+1.2^{\delta}$	$+7.8^{\gamma}$	-36.1^{a}	$+5.7^{\gamma}$	$+15.6^{\beta}$	$+27.6^{\alpha}$
Protein loss/gain (g)	-8.0^{b}	$+5.7^{\gamma}$	$+9.8^{\beta}$	-17.9^{a}	$+2.5^{\delta}$	$+9.1^{\beta}$	$+19.0^{\alpha}$
Energy efficiency (%)	—	14.1^{β}	$19.1^{\alpha\beta}$	—	29.9^{α}	30.8^{α}	31.5^{α}

Values = means; loss (–) versus controls; gain (+) versus P2R0 or P3R0; efficiency = stored energy/ingested energy; a, b (loss) or α...δ (loss, efficiency), significant difference ($P < 0.05$; multiple comparisons).