contents were 69.7 and 71.9% in the subcutaneous fat and 1.74 and 1.82% in the longissimus dorsi muscle for LV and LC, respectively.

Lipogenic enzyme activities of the backfat were lower in LV than in LC: acetyl CoA carboxylase: 0.09 and 0.14 nmol HCO_3^-/min/mg protein ($p < 0.07$); malic enzyme: 7.95 and 8.96 mmol NADPH/min/mg protein ($p < 0.08$); and glucose-6-phosphodehydrogenase: 1.34 and 1.51 mmol NADPH/min/mg protein ($p < 0.11$). For the intramuscular fat from the longissimus dorsi muscle, activities were identical between the 2 groups. The increase of lipid synthesis obtained could be explain by the amount of short-chain fatty acids which are in a higher proportion in the goats' than in the cows' milk, without any modification in the rate of lipid deposition. However, the data presented did not take account for C/4 which is high in goats' milk; this supports our hypothesis.

The effect of dietary fat on the fatty-acid composition of the adipose tissue has been demonstrated further. In addition, the fat tissues have a satisfactory technological fitness because the rates of C18:2 and C16: 0 were 7% and 30% for the 2 diets respectively and lead to a lower risk of oxidation of the unsaturated fatty acids. Thus, the milk fat could be included in pig diet improve the qualities of the meat without any great change of body fat.

Effect of dietary linoleic acid on pig carcass composition and lipogenesis.

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Fatty-acid composition of pig adipose tissue is affected by dietary fat. The energy value of the diet is frequently increased by supplement of dietary lipid to improve the feed–conversion ratio. Highly unsaturated vegetable fats are used in the diet for economic reasons. A high level of unsaturated fatty acids may lead to increased risks of lipid oxidation and rancid meat. It appears that high levels of unsaturated fat in the diet can also increase carcass fatness.

The aim of the present study was to investigate the effect of dietary linoleic acid on lipogenesis activity. Thirty-six Large White castrated male pigs (35 and 100 kg liveweight) were fed diets containing 4% total lipids inclucing 3 levels of pure linoleic acid, 1,5%, 2.0% and 2.5 %. The elevation in dietary linoleic acid increases carcass fatness ($P < 0.01$). At slaughter, the C18:2 content of backfat increased with the level of dietary linoleic acid ($P < 0.001$).

Table I. Effect of dietary linoleic on acetyl CoA carboxylase activities (nmol HCO_3^-/ min/g tissue). (J Mourot *et al*)

	Acetyl CoA carboxylase activity (nmol HCO_3^-)		
	Backfat	*Leaf fat*	*Ham*
C18:2 (%)			
1.5	2.21	4.07	2.87
2.0	3.16	4.84	4.13
2.5	3.69	5.34	4.69
RDS [1]	0.94	1.52	1.86
Effect	$P < 0.03$	NS	$P < 0.06$

Lipogenesis activity was stimulated by dietary linoleic acid. Acetyl-coenzyme A carboxylase activity increased significantly in subcutaneous adipose tissues (table I). Malic enzyme and glucose-6-phosphate dehydrogenase activities increased significantly in subcutaneous adipose tissues and leaf fat, whereas no significant effect was observed in the intramuscular fat. The elevation of fat synthesis activity can explain the increased carcass fatness of pigs fed diets containing high levels of unsaturated fat.

Thus excess unsaturated fatty acids compares with the needs of the animal increases the carcass fatness in perhaps 2 ways: a direct deposit and/or a stimulation of the lipid synthesis. Dietary linoleic acid also decreases the technological qualities of fatty tissues. This utilization in the diet is negated in all attempts to control and decrease the development of subcutaneous adipose tissue.

Low-protein diets containing hydrogenated coconut or salmon oils affect hepatic and very low density lipoprotein fatty-acid compositions in growing rats.

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Fatty liver and the similarity between skin lesions observed in kwashiorkor and those described in experimental essential fatty-acid (EFA) deficiency have led to the hypothesis that the effects of chronic malnutrition may result from combined protein and EFA deficiencies. The relationships of serum VLDL to hepatic lipid composition were studied after 28 d of protein depletion to determine the interactions between dietary protein levels and dietary EFA availability. Rats weighing 80 ± 5 g, were fed purified diets containing 20% or 2% casein and 5% fat as either salmon oil, rich in eicosapentaenoic (EPA) and docosahexaenoic (DHA) acids, or hydrogenated coconut oil, poor in EFA. All diets were isoenergetic (cornstarch replaced lacking casein). Animals were divided into 4 groups, SAC (20% casein + 5% salmon oil), SAd (2% casein + 5% salmon oil), COC (20% casein + 5% hydrogenated coconut oil), and COd (2% casein + 5% hydrogenated coconut oil). The procedures for chemical analysis utilized were similar to those described in detail in previous studies [Bouziane *et al* (1992) *J Nutr* 122, 2037-2046].

After 28 d the protein-deficient rats presented a fatty liver (mainly due to triacylglycerols) and reduced liver and VLDL phospholipid contents ($P < 0.001$). The VLDL apolipoprotein (apo) contents were diminished with protein deficiency and this decrease was mainly due to the reduced syntheses of apo B_{48} + apo B_{100}, whereas apo AI content was not affected. Triacylglycerol and phospholipid fatty acid compositions in both liver and VLDL, showed a depressed unsaturated to saturated fatty-acid ratio with protein deficiency, but more when hydrogenated coconut oil was associated. Furthermore, protein deficiency decreased linoleic, α-linolenic and arachidonic acids in VLDL phospholipids. Thus, feeding protein-deficient diets might enhance EFA requirements. Besides, in the phospholipid fractions with protein deficiency, EPA was diminished (SAd *vs* SAC, 2.7 *vs* 16.6 and 9.1 *vs* 13.0% of total fatty acids, $P < 0.001$, for liver and VLDL, respectively), and DHA was increased (SAd *vs* SAC, 16.8 *vs* 12.0 and 15.8 *vs* 11.2% of total fatty acids, $P < 0.001$, for liver and VLDL, respectively). Protein deficiency seems to accelerate the conversion of EPA to DHA with salmon oil diets. A similar effect has been observed in a previous study on phospholipid microsomes [Ulman *et al* (1992) *J Nutr Biochem* 3, 188-193]. Conversely, when protein-deficient diets were poor in both EFA and long-chain n–3 fatty acids such as hydrogenated coconut oil diets, there was more 22:5 (n–6) and less 20:4 (n–6) in VLDL lipids. Both eicosapentaenoic and arachidonic acids are the precursors of eicosanoids and their diminution may be related to some clinical symptoms observed in infants suffering from kwashiorkor.

Postprandial changes in the hepatic metabolism of lipoproteins in the preruminant calf, *Bos* spp.

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In spite of a low hepatic lipogenesis, the uptake, recycling and secretion of lipids (as lipoproteins) are major metabolic activities of the liver in the growing or lactating cattle [Bauchart (1993) *J Dairy Sci* 76, 3864-3882]. The hepatic metabolism of lipoproteins has been mainly studied in the high-producing dairy cow developing intense lipid infiltration of the liver in early lactation in situations of energy deficit [Mazur *et al* (1992) *Diabète et Métabolisme*, 18, 145-149]. Less information is available in non-pathological situations, except for the starving calf [Bauchart *et al* (1989) *J Lipid Res* 30, 1499-1514].

This work examines the postprandial changes in the hepatic metabolism of lipoproteins in four 3-week-old preruminant male calves (body weight: 46.0 ± 2.0 kg). Animals were given a conventional milk replacer containing tallow (23% diet DM) in 2 equal meals per day (9 h and 16 h). They were equipped with catheters and electromagnetic flow probes for estimation of hepatic lipoprotein fluxes.

Blood samples were collected 2 (T2), 7 (T7) and 15 h (T 15) after the morning meal. Plasma lipoproteins were isolated by density (d) gradient ultracentrifugation at 39,000 rpm for 46 h at 15°C and analyzed as described previously [Bauchart *et al* (1989) *op cit*].

Compared to the mean values determined during a 24 h period, plasma concentrations amounted to 250, 80 and 60% for insulin and 25, 180 and 160% for triglycerides (TG) at 2 (T2), 7 (T7) and 15 h (T15) after the meal, respectively. Net hepatic uptake (mg/min/kg BW) of very low density lipoproteins was noted at T2 (–0.20; NS)