sured in microsomes extracted from hepatic and adipose tissues of the pig. The activity was determined by measuring the conversion of [^{14}C]stearic acid to [^{14}C]oleic acid as described earlier (Kouba et al., Comp. Biochem. Physiol. 118 (1998) 509–514).

A first experiment compared Δ9-desaturase activity in subcutaneous adipose tissue (SAT) and the liver of Large White and Meishan pigs from 20 to 60 kg body weight (BW). The results confirmed that adipose tissue is the main site of Δ9-desaturation in the pig, the activity being 6- to 7-fold higher than in the liver. The activity of Δ9-desaturase did not vary significantly with BW, neither in SAT nor in the liver. The activity was slightly higher in the liver of Meishan than in that of Large White pigs (4.6 versus 3.9 nmol oleic acid formed/h·mg^{-1} protein). It was much higher in SAT of Large White than in Meishan pigs ($P < 0.001$), even though the percentage of oleate in total fatty acids was higher in SAT of the latter (47 versus 45 %). These results suggest that the higher oleate percentage in Meishan adipose tissue could result from a higher Δ9-desaturase activity at a younger age.

A second experiment compared a high linoleic acid diet (diet M, containing 4 % maize oil) with a high saturated fatty acid diet (diet T, containing 4 % beef tallow) for Δ9-desaturase activity in SAT of 100 kg BW pigs. Animals were allocated to diet M or T from 40 to 100 kg BW. The M diet led to a decrease in Δ9-desaturase activity (20.9 versus 28.7 nmol oleic acid formed/h·mg^{-1} protein) and to a lower monounsaturated fatty acid percentage in SAT (37 versus 46 %). These results suggest that Δ9-desaturase could be involved in the regulation of deposition of oleic acid in SAT of pigs given an n-6 PUFA-rich diet and hence could influence the quality of pig adipose tissues.

Further research is needed to study fatty acid desaturation processes during the early growth of Large White and Meishan pigs and the different factors involved in the regulation of the Δ9-desaturase activity.

Communication no. 16

Relationships between de novo lipogenesis and tissue lipid content in muscle, perirenal fat and liver in the rabbit; effects of age and dietary fatty acids.

F. Gondret[a,b], J. Mourot[b], M. Bonneau[b] ([a] Station de recherches cunicoles, Inra, Toulouse, France; [b] Station de recherches porcines, Inra, Saint-Gilles, France)

Rabbit meat is widely consumed in France (2.7 kg/year/inhab) and Poland (0.5 kg/year/inhab). However, its development is currently hampered because rabbit meat is often considered to be insufficiently tasty and juicy owing to a low fat content in the muscles. A better understanding of lipid partitioning between the different tissues is needed for a better control of lipid deposition in muscle. Lipid deposition proceeds from fatty acids that are either obtained from circulating triglycerides or synthesised de novo. The aim of this study was to investigate the relationships between de novo lipogenesis and final lipid content in *longissimus lumborum* muscle, as compared with perirenal fat and liver. In the first experiment, the effect of age was studied in 49 rabbits fed a commercial diet and slaughtered at different stages, from weaning to sexual maturity ($n = 7$ in each stage). In the second experiment, the effect of nutrition was studied in two groups of fifteen 11-week-old rabbits fed fat-supplemented diets (3.6 % dry matter) containing either medium-chain fatty acids (C10:0 + coconut oil) or long-chain fatty acids (sunflower oil). De novo lipogenesis was assessed by in vitro measurements of specific activities of selected lipogenic enzymes. In both experiments, the activity of acetyl-CoA-carboxylase, which is considered as the rate-limiting enzyme for lipogenesis, was related to the tissue lipid content, i.e. very low in muscle, intermediate in liver and high in perirenal fat. Furthermore, the age-related increase in intramuscular lipids was closely related ($P < 0.001$) to that of the lipogenic

enzyme activities in muscle (experiment 1). This suggests that the fatty acids deposited in muscle during growth were, at least partly, synthesised in situ. With various dietary fat sources (experiment 2), no relationship was observed, however, between diet-induced changes in enzyme activities and responses in lipid content of muscle, perirenal fat or liver. Rather, the fatty acid composition of tissue lipids mainly reflected that of dietary fat. This suggests that most of the tissue lipids were obtained from circulating triglycerides when rabbits were fed high fat diets. Thus, further investigations are needed to quantify the relative importance of lipogenic enzymes and lipoprotein lipase in the lipid deposition in the rabbit.

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Activity and regulation of mitochondrial carnitine palmitoyltransferase I are influenced by age, cold exposure and muscle type in piglet skeletal muscle.
I. Schmidt, M. Damon, P. Herpin (Station de recherches porcines, Inra, 35590 Saint-Gilles, France)

Lipid utilisation increases rapidly during the first postnatal days in pigs, especially during cold-induced muscular shivering thermogenesis. In the liver, carnitine palmitoyltransferase I (CPT I) controls the entry of long-chain fatty acids into the mitochondria and its regulation by malonyl-CoA plays a pivotal role in the regulation of fatty acid oxidation (McGarry et al., Biochem. J. 214 (1983) 21–28). This study investigates the possibility that such a regulatory system is involved in the modulation of fatty acid utilisation in pig skeletal muscle during early postnatal development.

Intermyofibrillar (IM) and subsarcolemmal (SS) mitochondria were isolated from *longissimus thoracis* (LT) and *rhomboideus* (RH) muscles from newborn (n = 10) and 5-d-old piglets. At 5 d, piglets were maintained for a 4-h period in thermoneutral (TN, 30 °C, n = 10) or cold (C, 20 °C, n = 10) conditions before being killed. CPT I activity was assayed as the formation of [^{3}H]-palmitoyl-L-carnitine from [methyl-^{3}H]-carnitine and palmitoyl-CoA according to Herbin (Herbin et al., Eur. J. Biochem. 165 (1987) 201–207). The sensitivity of CPT I to malonyl-CoA inhibition corresponded to the concentration of malonyl-CoA required for 50 % inhibition of the enzyme activity (IC_{50}) in IM mitochondria. Muscle concentration of malonyl-CoA was determined by reversed-phase HPLC on LT and RH muscles.

In SS mitochondria, CPT I activity was lower in LT than in RH muscle at birth (–40 %, $P < 0.001$) and increased by about 600 and 200 % in LT and RH muscles, respectively, within 5 days; there was no effect of cold exposure. In IM mitochondria, CPT I activity was much higher than in SS mitochondria (+620 % at birth, +170 % at 5 d, $P < 0.001$) but was not affected by age, muscle type or cold exposure. Sensitivity of CPT I to malonyl-CoA was similar in both muscles (IC_{50}: 3–6 μM) and 50–100 times lower than in rat muscle (0.035 μM, Mc Garry et al., 1983). It decreased by 47 % ($P < 0.05$) with age. After 4 h of cold exposure, sensitivity of CPT I to malonyl-CoA was unaffected in RH muscle but tended to be greater (+30 %, $P < 0.06$) in LT muscle. Malonyl-CoA concentrations decreased by 27 and 42 % ($P < 0.01$) between birth and 5 d in LT and RH muscles, respectively. Cold stress led to a 27 % ($P < 0.05$) reduction in malonyl-CoA concentration in RH muscle. In conclusion, the postnatal enhancement of CPT I activity in SS mitochondria and weakening of the inhibitory effect of malonyl-CoA upon CPT I activity in IM mitochondria support the suggestion that fatty acid oxidation increased rapidly after birth in piglet skeletal muscle. During cold stress, the muscle-specific decrease in malonyl-CoA observed in RH muscle could partly relieve CPT I inhibition and favour fatty acid oxidation. Finally, the lower sensitivity of CPT I to malonyl-CoA in piglet