

rutin (2 mg/day/animal) were administered directly into the stomach by an intragastric tube. Rats were divided into seven groups ($n = 12$): C – control; Drec – 5 days DEX followed by 5 days of post-DEX recovery; D – 5 days DEX alone; DR – 5 days DEX followed by 10 days RUT; RD – 10 days RUT followed by 5 days DEX; R+D – RUT and DEX together for 10 days; and R – 10 days RUT. After that time, rats were killed and brains were isolated. Reduced glutathione (GSH) was assayed by the reaction with 5,5'-dithio-bis (2-nitrobenzoic acid). TBARS were expressed as a sum of the substances reacting with tiobarbituric acid, CD were expressed as the amount of doubled bonds which had a maximum absorbance at 234 nm. The TBARS and CD were also assayed in brain homogenates during lipid peroxidation induced by AAPH. Administration of dexamethasone caused the highest increase in TBARS and CD concentration which was accompanied by the highest decrease in GSH level in brain homogenates in the D-group. In contrast, TBARS and CD concentrations were the lowest and the level of GSH was highest in the R-group. Administration of rutin and dexamethasone (regardless of the treatment) resulted in TBARS and CD concentrations which were lower than in the C-group and a GSH level higher than in the C-group. Changes in TBARS and CD concentrations in experimental groups during 48 h of AAPH-induced lipid peroxidation were consistent with the results obtained from non-AAPH-peroxidated brain homogenates. The TBARS and CD concentrations both at 0 and at 48 h of peroxidation were lowest in the R-group. In conclusion, rutin appears to be a potent inhibitor of lipid peroxidation during dexamethasone-induced oxidative stress.

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Effect of various phytoestrogens on lipid metabolism of isolated rat adipocytes.

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There are over 300 species of plants containing substances performing estrogenic-like action (soy, alfalfa). They are called phytoestrogens because of their origin. Some of them are also produced by *Fusaria*, which develop on plant seeds during poor storage conditions. Phytoestrogens ingested by animals may cause hyperestrogenic effects. They may also play a significant role in the etiology of some cancers or may reduce rates of some cancers and cardiovascular diseases. It is known that they can influence many biochemical events, e.g. lead to disorders of the oxidative chain or disturb corticosteroid synthesis. It is quite possible that phytoestrogens can also affect lipid metabolism in the same way that estrogens do. We used two phytoestrogens (genistein, zearalenone) to investigate their direct effect on lipid metabolism in isolated fat cells. Isolation of adipocytes was performed according to Rodbell (Rodbell, *J. Biol. Chem.* 239 (1964) 375–380). Cells from epididymal fat tissue of male Wistar rats (160 ± 5 g) were incubated for 90 min in a buffer containing phytoestrogens in the absence or presence of epinephrine (10^{-6} mol·L $^{-1}$) and then lipolysis was determined as the amount of glycerol released. The effect of phytoestrogens on lipogenesis was ascertained as [U - 14 C] glucose conversion to lipids in adipocytes in the absence and presence of insulin (10^{-9} mol·L $^{-1}$). Differences between groups were statistically evaluated using one-way analysis of variance. We found that phytoestrogens exert a significant effect on lipid metabolism. Generally,

* Values with different letters were significantly different at $P \leq 0.05$.

they enhance lipolysis in both the basal – and epinephrine – stimulated states and suppress basal – and insulin-stimulated lipogenesis. The most effective phytoestrogen was genistein. At concentrations of 10^{-3} and 10^{-4} mol·L⁻¹ in the medium it enhanced basal (respectively, $1.21 \pm 0.08^{a*}$ and 0.58 ± 0.01^b versus 0.37 ± 0.02^c $\mu\text{mol}/10^6$ cells/90 min) and stimulated lipolysis (respectively, 2.79 ± 0.05^a and 2.70 ± 0.07^a at concentrations of 10^{-4} and 10^{-5} mol·L⁻¹ versus 2.37 ± 0.04^b $\mu\text{mol}/10^6$ cells/90 min). Lipogenesis was inhibited at concentrations of 10^{-3} , 3×10^{-4} and 6×10^{-4} mol·L⁻¹, respectively, in both basal (139 ± 5^a , 349 ± 5^b and 227 ± 5^c versus 580 ± 11^d nmol/10⁶ cells/90 min) and stimulated (181 ± 5^a , 427 ± 3^b and 280 ± 6^c versus 667 ± 12^d nmol/10⁶ cells/90 min) states. Zearalenone had a similar effect on lipogenesis but inhibited stimulated lipolysis.

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Effects of a diet rich in medium-chain fatty acid on lipoprotein lipase (LPL) and carnitine palmitoyltransferase I (CPT I) activities in the heart from preruminant calves. C. Piot^a, J.F. Hocquette^a, P. Herpin^b, D. Bauchart^a (^aLaboratoire croissance et métabolismes des herbivores, Inra, centre de recherches de Clermont-Ferrand/Theix 63122 Saint-Genès-Champanelle, France; ^bStation de recherches porcines, Inra, 35590 Saint-Gilles, France)

In bovines, triglycerides (TG) are energy-yielding substrates of prime importance for growth and physical activity of muscles and of the heart. Their metabolic utilisation is controlled by the level of activity of several enzymes such as LPL and CPT I which are both considered to be rate-limiting for fatty acid (FA) uptake and catabolism in muscles. The objectives of this study were to compare the effects of the incorporation of coconut oil (CN), rich in medium-chain fatty acids (MCFA, C12:0 and C14:0) or tallow (TA), rich in long-chain fatty acids (C16:0

and C18:1) in a milk diet on the LPL and CPT I activities in calf heart.

The experiments were performed using two groups of five 1-month-old preruminant Holstein-Friesian male calves following adaptation for 19 days to a high fat milk replacer (22.4 % diet DM) containing CN or TA but the same amount of carbohydrate. Heart LPL activity was assayed with intralipid into which [³H]triolein had been incorporated (Peterson et al., *Biochim. Biophys. Acta* 837 (1985) 262–270). Heart TG contents were determined from total lipid extracts as described previously (Leplaix-Charlat et al., *J. Dairy Sci.* 79 (1996) 1826–1835). Heart CPT I activity was assayed by a radioactive method on fresh isolated intermyofibrillar mitochondria and malonyl-CoA concentration in homogenates was determined by reversed-phase HPLC (Schmidt, Herpin P., *J. Nutr.* 128 (1998) 886–893).

The higher heart LPL activity (+27 %, $P < 0.05$) measured in calves fed the CN diet suggested a stimulation of LPL activity by MCFA, and hence an increase in FA uptake. On the contrary, CPT I activity and malonyl-CoA concentrations were not significantly affected by the source of FA in the diet (1.68 versus 1.46 nmol of palmitoylcarnitine formed/min/mg mitochondrial protein and 1.26 versus 1.94 nmol of malonyl-CoA/g tissue for the TA and CN groups, respectively). This indicated that, in our conditions, CPT I activity in the heart was probably not rate-limiting for FA catabolism. Since a lower TG concentration (–42 %, NS) was found in the heart of calves fed the CN diet, we can hypothesise that MCFA from the diet were probably mainly oxidised either by the mitochondrial and/or the peroxisomal pathways. In conclusion, this study clearly showed, for the first time, that the LPL activity in calf hearts could be stimulated by coconut oil rich in MCFA, favouring a higher uptake of dietary FA without any increase in TG storage.