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Abstracts

NUTRITION AND MUSCULAR FUNCTION

Effect of fatty acid infusion on muscle glycogen resynthesis after exercise in healthy subjects: ^{13}C NMR study. M.C. Delmas-Beauvieux[a,b], B. Quesson[a], E. Thiaudière[a], P. Canioni[a], H. Gin[b] ([a]RMSB UMR5536 CNRS Université Bordeaux 2, 146, rue Léo Saignat, 33076 Bordeaux cedex; [b]Service de nutrition et diabétologie, Hôpital Haut-Lévêque Pessac, France).

A tissue level competition between carbohydrate substrates and lipid substrates does not clearly explain the role of free fatty acids (FFA) in muscular metabolism and the muscular insulinoresistance (IR) observed in non-insulinodependent diabetes mellitus. FFA could impair the recovery of the glycogen depleted during an exercise, but the use of methods such as a hyperinsulinic clamp and/or muscular biopsies does not allow the determination of the exact role of FFA. Their effect on the kinetic of the glycogen pool of gastrocnemius can be non-invasively studied by carbon 13 nuclear magnetic resonance (NMR). Six male healthy subjects (40 ± 2 years, BMI 24.34 ± 1.28 kg/m^2, m ± SEM) were perfused, after informative consent, with glycerol (0.26 mg/kg/h) (GLY) or Ivélip 10 % (0.015 mL/kg/h) (IVE), with a washout period of 5 weeks. ^1H decoupled ^{13}C NMR spectra were obtained with a surface coil (50.3 MHz) on a Brucker Biospec 47/50 spectrometer (4.7 Tesla). Plantar flexions performed during 92 ± 4 min led to similar glycogen depletion whatever the substrate (GLY = 46.7 %, IVE = 47.6 % of glycogen initial value). The glycogen resynthesis was then studied for 3.5 h in a resting state. Plasmatic glucose, insulin, FFA and triglycerides were measured during the same period. During the insulinodependent phase of glycogen synthesis following the moderate exercise, the glycogen recovery was significantly higher in GLY (61 ± 3 %) than in IVE (49 ± 4 %) (P = 0.05, ANOVA). The peripheral IR obtained with the infusion of FFA led to a lower level of glycogen resynthesis demonstrating their role in the IR in the muscular effector. A lipid substrate decreases muscle glycogenogenesis in healthy subjects. The establisment of an IR is the *primum movens*.

Time-course metabolic adaptations during endurance training according to initial physical capacities of sedentary elderly people. B. Morio[a], P. Ritz[a], C. Montaurier[a], N. Fellmann[b], B. Beaufrère[a], M. Vermorel[a] ([a] Laboratoire de nutrition humaine; [b] Laboratoire de physiologie et biologie du sport, 58, rue Montalembert, BP 321, 63009 Clermont-Ferrand cedex, France).

Fast mass loss is one of the main alterations induced by endurance training in elderly people. It can only result from a negative lipid balance due to either a decreased lipid intake (which is probably not the case) or to an increased capacity to oxidize lipids. However, the latter explanation has not been studied previously. Therefore, we studied the short-term (7 weeks) and the medium-term (14 weeks) effects of a progressive endurance training programme on lipid oxidation (LIPox) over 24 h and during sleep in 13 healthy and initially sedentary subjects aged 63 ± 2 years. LIPox was determined from respiratory gaseous exchanges measured in wholebody calorimeters. Energy balance during the measurement period was calculated as the difference between daily energy expenditure and energy intake.

Analysis of results took into account the variation during the training period in maximal oxygen consumption (VO_{2max}) and in body composition, determined using the deuterium dilution based method and the skinfold thicknesses.

Even if the body weight was kept constant, training induced 13.7 ± 15.8 % fat mass loss ($P < 0.05$) and a 14.3 ± 7.8 % fat-free mass gain ($P = 0.08$) after 14 weeks. Furthermore, VO_{2max} increased on average by 14.3 ± 7.8 % ($P < 0.01$), however its increase was negatively correlated to its initial level ($r = -0.52$, $P < 0.05$). After adjustment for differences in energy balance, LIPox increased by 14 % over 24 h ($P = 0.06$) and by 28 % during sleep ($P < 0.01$) after 7 weeks of training. Thereafter it returned to its initial level after 14 weeks of training. After 7 weeks of training, the increase in LIPox was correlated positively with fat-free mass gain ($r = 0.51$, $P = 0.05$) but negatively with variations in VO_{2max} ($r = -0.70$, $P < 0.02$). In conclusion, endurance training induces transient metabolic adaptations which were partly related to the initial physical capacities of the elderly volunteers. The consequence of these adaptations is a transient increase in the capacity to oxidize lipids.

PROTEIN SYNTHESIS AND DEGRADATION

Specific post-absorptive and post-prandial responses of protein synthesis to dietary protein levels in muscle, liver and small intestine. M.A. Arnal, M.C. Valluy, P. Capitan, G. Bayle, P. Patureau Mirand (Centre de Recherche en nutrition humaine, Unité d'étude du métabolisme Azoté, Inra Clermont-Ferrand, Theix, 63122 Saint-Genès-Champanelle, France).

The effects of dietary protein level on protein mass and fractional synthesis rate (FSR) were investigated in the gastrocnemius muscle, liver and small intestine of growing rats in post-prandial or post-absorptive states.

Methods: Forty young male Sprague-Dawley rats (75 g body weight) were pair-fed either a 10 or a 20 % protein diet for 10 days. Tissue protein synthesis (FSR) was determined in vivo, in the fed (post-prandial) or 12-h-fasted (post-absorptive) states, using an intravenous flooding dose of 300 µmoles of L-1-^{13}C valine (enrichment excess 45 % mole)/100 g body weight.

Results: The higher protein mass in liver, gastrocnemius and small intestine (+23, +34 and +19 %, respectively) of rats fed with the 20 % protein diet than with the 10 % protein diet corresponded to different patterns of FSR (%.d^{-1}) responses to protein intake levels, see *table.*

Conclusion:

1) In the liver, there is a stimulation of FSR with both diets during the fed state, but no effect of dietary protein levels.

2) In gastrocnemius muscle, a low protein diet inhibits post-prandial FSR stimulation and a high protein diet increases FSR only during the fed state.

	Liver		Gastrocnemius		Small intestine	
Dietary protein level (%)	10	20	10	20	10	20
Post-absorptive FSR	55.9 ± 8.6	59.8 ± 4.6	14.7 ± 0.9	13.5 ± 1.5	81.7 ± 5.9	85.0 ± 5.0
Post-prandial FSR	88.6 ± 5.7*	92.1 ± 9.5*	15.0 ± 1.5	16.7 ± 1.5*	99.9 ± 8.3*$^{+}$	111 ± 12*

Means ± SD ; $n = 10$; two-way ANOVA: * post-prandial versus post-absorptive ($P < 0.01$); $^{+}$ 20 versus 10 % ($P < 0.05$)