

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  $13.7 \pm 15.8$  % fat mass loss ( $P < 0.05$ ) and a  $14.3 \pm 7.8$  % fat-free mass gain ( $P = 0.08$ ) after 14 weeks. Furthermore,  $VO_{2max}$  increased on average by  $14.3 \pm 7.8$  % ( $P < 0.01$ ), 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.

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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  $\mu$ moles 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<sup>-1</sup>) 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 $\pm$ 8.6	59.8 $\pm$ 4.6	14.7 $\pm$ 0.9	13.5 $\pm$ 1.5	81.7 $\pm$ 5.9	85.0 $\pm$ 5.0
Post-prandial FSR	88.6 $\pm$ 5.7*	92.1 $\pm$ 9.5*	15.0 $\pm$ 1.5	16.7 $\pm$ 1.5*	99.9 $\pm$ 8.3**	111 $\pm$ 12*

Means  $\pm$  SD ;  $n = 10$ ; two-way ANOVA: \* post-prandial versus post-absorptive ( $P < 0.01$ ); \*\* 20 versus 10 % ( $P < 0.05$ )

3) In the small intestine, as in liver, post-prandial FSR increase is observed with both diets, and as in muscle, a high protein diet increases FSR only during the fed state.

**Lack of recovery of muscle proteins lost after food deprivation in old rats despite a stimulation of muscle protein synthesis.**

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Sarcopenia could be the result of an inability of elderly people to recover muscle proteins lost during catabolic periods. To test this hypothesis, we deprived 12- and 24-month-old rats of food for 10 days and compared their capacity to recover after 5 days of refeeding. We measured gastrocnemius muscle protein synthesis rates in vivo using the flooding dose method ( $^{13}\text{C}$ -valine), and calculated degradation rates. Results ( $\pm$  SE) were analysed by the Duncan test

( $P \leq 5\%$ ; <sup>a,b</sup> nutrition effect per age; \* age effect):

Muscle protein mass, reduced after fasting, increased during refeeding in adults. In old rats, mobilization was similar but there was no recovery. During fasting, ribosomal capacity was maintained, whereas ribosomal efficiency, fractional and absolute synthesis rates were decreased and increased back to control values during refeeding, whatever the age. Protein degradation rates were significantly reduced during fasting in adults and increased slightly after refeeding. They were unchanged in the old rats, being equal in fed and refed animals.

Conclusions: 1) The capacities to recover muscle mass lost after a stress decreased with age. 2) This lower capacity does not seem to be related to a lack of stimulation of protein synthesis. 3) It could result from an alteration in the inhibition of proteolysis, which remained high during refeeding.

	12 months			24 months		
	Fed (6)	Unfed (6)	Refed (5)	Fed (6)	Unfed (7)	Refed (6)
Total protein (mg)	522 $\pm$ 15 <sup>a</sup>	424 $\pm$ 12 <sup>b</sup>	462 $\pm$ 20 <sup>ab</sup>	345 $\pm$ 44 <sup>a*</sup>	273 $\pm$ 23 <sup>a*</sup>	266 $\pm$ 21 <sup>a</sup>
Rib capacity (mg RNA/g prot)	5.7 $\pm$ 0.1 <sup>a</sup>	5.2 $\pm$ 0.4 <sup>a</sup>	5 $\pm$ 0.1 <sup>a</sup>	5.9 $\pm$ 0.4 <sup>a</sup>	5.8 $\pm$ 0.4 <sup>a</sup>	6.7 $\pm$ 0.5 <sup>a*</sup>
Fractional synth. rates (%/d)	4.9 $\pm$ 0.2 <sup>a</sup>	2.7 $\pm$ 0.4 <sup>b</sup>	5.2 $\pm$ 0.2 <sup>a</sup>	7.8 $\pm$ 0.5 <sup>ab*</sup>	6.5 $\pm$ 0.6 <sup>b*</sup>	9 $\pm$ 0.5 <sup>a*</sup>
Absolute synth. rates (mg prot/d)	26 $\pm$ 2 <sup>a</sup>	11 $\pm$ 2 <sup>b</sup>	24 $\pm$ 1 <sup>a</sup>	26 $\pm$ 3 <sup>a</sup>	17 $\pm$ 1 <sup>b*</sup>	23 $\pm$ 1 <sup>a</sup>
Rib effic. (mg prot/ (mg ARN.d))	9 $\pm$ 1 <sup>a</sup>	5 $\pm$ 1 <sup>b</sup>	10 $\pm$ 0.4 <sup>a</sup>	13 $\pm$ 1 <sup>a*</sup>	12 $\pm$ 1 <sup>a*</sup>	13 $\pm$ 0.4 <sup>a*</sup>
Degradation rates (%/d)	5 $\pm$ 0.2 <sup>a</sup>	3.4 $\pm$ 0.5 <sup>b</sup>	4 $\pm$ 0.1 <sup>ab</sup>	7.7 $\pm$ 0.5 <sup>a*</sup>	8 $\pm$ 0.8 <sup>a*</sup>	7.7 $\pm$ 0.4 <sup>a*</sup>