food distribution). Protein synthesis was measured by L-[U-^{14}C]-phenylalanine incorporation in muscle proteins, whereas protein degradation was deduced from release of tyrosine into the incubation medium. Results were analysed by a two-way variance analysis. 1) Muscle protein mass was lower in old rats (fasted: 2.945 ± 0.284 and fed: 2.859 ± 0.125 mg.100 g rat^{-1}) than in mature rats (fasted: 3.099 ± 0.128 and fed: 3.722 ± 0.283, $P = 0.03$). 2) No effect of age ($P = 0.28$) or nutritional state was detected ($P = 0.71$) on protein synthesis (mature fasted: 0.169 ± 0.006 versus mature fed: 0.165 ± 0.015 and old fasted: 0.157 ± 0.004 versus old fed: 0.151 ± 0.014 nmoles phe.h^{-1}.mg protein^{-1}). 3) Feeding induced an inhibition of muscle proteolysis in mature rats (fasted 0.768 ± 0.036 versus fed 0.546 ± 0.050, –29 %, nmoles tyr.h^{-1}.mg protein^{-1}, $P = 0.003$) but not in old rats (fasted 0.694 ± 0.049 versus fed 0.654 ± 0.074 nmoles tyr.h^{-1}.mg protein^{-1}, –5 %, $P = 0.62$).

Conclusion: A lack of inhibition of muscle proteolysis in the fed state could be involved in the loss of muscle protein mass during ageing.

Whole body proteolysis is insulin resistant in elderly humans. Y. Boirie, P. Gachon, N. Cordat, L. Morin, M. Genest, P. Rousset, B. Beaufrère (Human Nutrition Laboratory, CRNH, Clermont-Ferrand, BP 321, 63009 Clermont-Ferrand cedex 1, France).

Inhibition of whole body proteolysis (C) by insulin was investigated using isotopic dilution of L[1-13C]leucine in 21 young subjects (Y) (23.4 ± 0.6 years, 20.4 ± 0.38 kg/m^2, m ± SEM) and 17 elderly subjects (E) (68.9 ± 0.56 years, 25.1 ± 0.8 kg/m^2) in two different experimental protocols:

1) during an euglycemic euaminoacidemic clamp with two different insulin infusion rates (CL1 and CL2, with infusion rates of 0.2 and 0.5 mU/kg.min, respectively),

2) during a 4-h continuous meal (M).

During the clamp study with an identical insulin infusion rate between the two groups, plasma insulin was higher in E than in Y, suggesting a reduced insulin clearance in E. Despite this higher insulinemia, C is decreased less in E than in Y at CL1 but not at CL2, implying that higher plasma insulin levels do compensate for an insulin resistant state. During feeding, C in Y and E were reduced more than during the clamp study, suggesting that the additional effect of the meal induced hyperaminoacidemia. However, C was less inhibited in E than in Y at the same insulin level. In conclusion, C was insulin resistant in E, which is a potential mechanism for the age-related protein loss.

	n		Plasma insulin (μU/ml)		Plasma leucine (% basal)		Proteolysis (% versus basal)	
	Y	E	Y	E	Y	E	Y	E
CL1	14	12	12.5 ± 0.6^a	17.1 ± 1.0^b	–4	7	–14 ± 1^a	–9 ± 1^b
CL2	14	12	27.4 ± 1.4^c	35.7 ± 1.3^d	–13	–8	–22 ± 1^c	–19 ± 2^{ac}
M	7	5	25.9 ± 2.3^c	26.9 ± 3.4^c	36	30	–40 ± 2^d	–26 ± 4^{ce}

Numbers with different letters are significantly different at $P < 0.05$.