thesis) was stimulated in the fed state, with the pulse protein intake (+18.0 ± 3.8 %) and not with the spread one (+2.5 ± 4.4 %, $P = 0.016$).

Conclusions: 1) In elderly women, the greater stimulation of leucine oxidation and the lower inhibition of proteolysis in the fed state are involved in the protein losses occurring during ageing. 2) A daily protein pulse intake stimulates protein synthesis in the fed state.

Leucine metabolism following a peritoneal aminoacid load. J. Delarue, Cl. Maingourd, M. Objois, M. Pinault, C. Couet, F. Lamisse (Laboratoire de nutrition, Association régionale d'aide aux urémiques du centre ouest, CHU de Tours 37044, France).

Aims: To study a) the acute changes in leucine metabolism following the peritoneal delivery of an aminoacid (AA) solution for peritoneal dialysis associated or not with the simultaneous ingestion of a CHO-fat meal.

Patients and methods: Six patients (53 ± 13 years, BMI: 23.9 ± 6.8 $kg.m^{-2}$) were studied twice (randomization) in basal state then after the peritoneal delivery (T_0–T_{30}) of a 1.1 % aminoacid peritoneal solution (Nutrineal®, Baxter) mixed with 600 mL tap water (I) or 600 kcal/600 mL (II). From T_{-150} to T_{300}, the patients received a prime-continuous infusion of [5,5,5-$^2H_3$] leucine (F_{iv} = 0.04 $\mu mol.kg^{-1}.min^{-1}$). The AA solution (14.4 mmoles Leu) was enriched with [1-^{13}C]-leucine (3.5 MPE). VCO_2 was measured using indirect calorimetry.

Calculations: rates of total (Ra, Rd) and of exogenous (peritoneal) (Ra_{exo}, Rd_{exo}) Leu appearance and disappearance calculated with pV = 0.125 $l.kg^{-1}$ (Boirie et al.). Rate of endogenous Leu appearance (proteolysis) $Ra_{endo} = Ra - Ra_{exo} - F_{iv}$. Oxidation of exogenous Leu (Leu_{ox}) was calculated from VCO_2 and $^{13}CO_2$. Non-oxidative disposal of exogenous Leu (contribution to protein synthesis) NOLD = $Rd_{ox} - Leu_{ox}$.

Statistics: ANOVA and paired Student's *t*-test.

Results (moy ± SEM): insulinaemia: +800 % and VCO_2: +13 % (II versus I, $P < 0.001$). Ra stimulated during the 2 tests: +56 % and +52 % (I and II) at T60). Ra and Rd decreased from T_{90} to T_{300} during II versus I ($P < 0.05$). Ra_{exo} was similar during the two tests (0.99 ± 0.21 and 0.92 ± 0.12 $\mu mol.kg^{-1}.min^{-1}$) at T_{60} (I and II). NOLD was similar during the two tests (0.87 ± 0.18 versus 0.74 ± 0.14 $\mu mol.kg^{-1}.min^{-1}$) at T_{60} (I and II). Leu_{ox} was similar during the two tests (0.18 ± 0.04 versus 0.21 ± 0.04 $\mu mol.kg^{-1}.min^{-1}$) at T_{120} (I and II). Ra_{endo} was unaffected during I and was 25 % inhibited during II.

Conclusion: following peritoneal AA delivery, 16 % of absorbed Leu was oxidized/5 h, 27 % was extracted by liver, 56 % contributed to protein synthesis. Proteolysis was inhibited with a concomitant meal.

The inhibition of muscle proteolysis in the post-prandial state is altered in 2-year-old rats. M.A. Arnal, M.L. Houlier, J.F. Rey, C. Sornet, D. Dardevet, P. Patureau Mirand (Unité d'étude du métabolisme azoté, Inra, CRNH, Clermont-Ferrand, Theix, 63122 Saint-Genès-Champanelle, France).

Is it possible to explain protein loss by an impairment of post-prandial protein gain? Twenty-five male Sprague-Dawley rats (13 1-year-old and 12 2-year-old rats) were given an 18 % protein diet. After 3 weeks, muscle protein turnover was measured using the epitrochlearis incubation method, both in the fasted state (12 h without food) and in the fed state (3–5 h after

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 $P < 0.05$.