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