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 (T0–T30) 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 T300, the patients received a prime-continuous infusion of [5,5,5-2H3] leucine (Fi = 0.04 μmol.kg^-1.min^-1). The AA solution (14.4 mmoles Leu) was enriched with [1-13C]-leucine (3.5 MPE). VCO2 was measured using indirect calorimetry.

Calculations: rates of total (Ra, Rd) and of exogenous (peritoneal) (Raexo, Rdexo) Leu appearance and disappearance calculated with pV = 0.125 l.kg^-1 (Boirie et al.). Rate of endogenous Leu appearance (proteolysis) Raendo = Ra–Raexo–Fi. Oxidation of exogenous Leu (Leuox) was calculated from VCO2 and 13CO2. Non-oxidative disposal of exogenous Leu (contribution to protein synthesis) NOLD = Rdox–Leuox.

Statistics: ANOVA and paired Student’s t-test.

Results (moy ± SEM): insulinaemia: +800 % and VCO2: +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 T90 to T300 during II versus I (P < 0.05). Raexo was similar during the two tests (0.99 ± 0.21 and 0.92 ± 0.12 μmol.kg^-1.min^-1) at T60 (I and II). NOLD was similar during the two tests (0.87 ± 0.18 versus 0.74 ± 0.14 μmol.kg^-1.min^-1) at T60 (I and II). Leuox was similar during the two tests (0.18 ± 0.04 versus 0.21 ± 0.04 μmol.kg^-1.min^-1) at T120 (I and II). Raendo 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.


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