Muscle protein turnover in broiler chickens: effects of high ambient temperatures and dietary protein intake.
S. Temim, A.M. Chagneau, R. Peresson, J. Michel, S. Guillaumin, S. Tesseraud (Inra, Station de recherches avicoles, 37380 Nouzilly, France).

The growth of broiler chickens is considerably decreased under high ambient temperatures, especially during the finishing period. This growth depression is associated with decreased protein retention efficiency and reduced protein gain. Providing high protein diets seems to be beneficial in hot conditions since it tends to improve the growth of heat-exposed chickens. We therefore studied the effect of chronic heat exposure (32 versus 22 °C) and dietary crude protein (25 versus 20 %) on muscle protein turnover in 5- to 6-week-old chicks. Protein synthesis was measured in vivo in the pectoralis major, gastrocnemius and sartorius muscles by a flooding dose of [^{3}H]-Phe. In the same muscles, protein breakdown was estimated as the difference between protein synthesis and deposition. Data were compared by two-way ANOVA analysis.

Protein synthesis was deeply decreased by chronic heat exposure whatever the muscle ($P < 0.01$). This was principally related to a reduced capacity for protein synthesis (about –20 %; $P < 0.001$) since the translational efficiency was not significantly modified. Protein breakdown was also lower in the pectoralis major and sartorius muscles; this effect was not observed in the gastrocnemius muscle. Protein turnover was slower in hot conditions irrespective of muscle. Protein synthesis was more affected than protein breakdown, resulting in a lower protein deposition, especially in the pectoralis major and gastrocnemius muscles.

At 32 °C, the high protein diet did not significantly change either protein synthesis, ribosomal capacity or translational efficiency. However, it decreased proteolysis, resulting in a higher protein deposition ($P < 0.001$, for the gastrocnemius and sartorius muscles; tendency for the pectoralis major muscle).

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A daily protein pulse improves the fed state protein gain in elderly women.
M.A. Arnal[a], M.L. Houlier[a], S. San José[a], L. Mosoni[1], J.M. Antoine[b], Y. Boirie[c], B. Beaufrère[c], P. Patureau Mirand[a] ([a]Unité d'étude du métabolisme azoté, Inra, CRNH, Clermont-Ferrand ; [b]Société Danone, Le Plessis-Robinson ; [c]Laboratoire de nutrition humaine, CRNH, Clermont-Ferrand, Theix, 63122 Saint-Genès-Champanelle, France).

Can the daily protein feeding pattern modulate the fed state protein anabolism? Sixteen young (25.7 ± 1.1 year old) and 14 elderly (67.6 ± 1.2 year old) women were given a 1.6 g protein.kg lean body mass (LBM)$^{-1}$.d^{-1} diet, for 15 days. This diet was fed in either four similar isoproteic meals (spread protein intake groups, seven old or eight young adult women), or in three meals with 80 % of the daily protein intake at lunch (pulse protein intake groups, seven old or eight young adult women). On day 16, an 8-h infusion of ^{13}C leucine made it possible to measure the leucine flux variations between post-absorptive state (16 h fasting) and fed state (14 meals fed every 20 min providing 0.7 g protein per kg LBM). Results were analysed by a two-way variance. 1) The stimulation of leucine oxidation flux in the fed state was greater in the elderly (+220 ± 19 %) than in the young women (+156 ± 13 %, $P = 0.009$). 2) In the fed state, the endogenous flux of leucine (proteolysis) decreased less in the elderly (–23.4 ± 2.7 %) than in the young women (–37.6 ± 3.0 %, $P = 0.002$). 3) The non-oxidative leucine flux (protein syn-

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