

initial rise in respiratory quotient (RQ) was also similar in both groups. However, in rats fed the TD diet, the increase in RQ started dropping approximately 2 h after the onset of the meal, while in rats fed the TC diet the decline in RQ was more progressive and began its decrease only 3–4 h after the onset of feeding. This result shows that the absence of threonine in the diet is sufficient to strongly modify the post-prandial overall oxidative metabolism.

In a second experiment, the same diets were given ad libitum to two groups of eight male Wistar rats. Rats fed the TD diet showed the first significant ($P < 0.05$) decrease in food intake after 120 min of feeding (corresponding to a consumption of 50 KJ or 3.3 g).

The same schedule was applied in rats chronically implanted with an intravenous catheter allowing for remote, stress-free blood sampling. Only in rats fed the threonine-devoid diet did the sample performed 90 min after the onset of feeding reveal a 47% decrease in threonine.

In conclusion, the three experiments in the present study demonstrate that the diet-induced plasma-threonine imbalance may bring about qualitative metabolic changes that coincide with the beginning of anorexia, suggesting a possible common mechanism and interaction.

Analysis of the reciprocal influences between training, diet and substrate utilisation during exercise in the rat. C Larue-Achagiots¹, N Reith¹, PC Even² (¹ CNRS URA 1294, 45, rue des Saints-Pères, 75006 Paris; ² CNRS UPR 9054, Collège de France, 75005 Paris, France).

Food intake as well as training are known to modify substrate utilisation during exercise. However, little is known about the reciprocal influences of diet, training and substrate mobilisation during exercise. The purpose of

this study was to further investigate this point.

Twenty-four Wistar rats were allowed to freely select their food from three sources of macronutrients (proteins, lipids, carbohydrates) completed with minerals and vitamins. One group ($n = 9$) was exercised daily by treadmill running, at the beginning of the night period, until the rats were able to run $20 \text{ m}\cdot\text{min}^{-1} \text{ } 2 \text{ h}\cdot\text{day}^{-1}$ (slope 0°). The other group ($n = 15$) was only habituated to run on the treadmill (5 min per day for 3 days). In all rats, glucose and lipid utilisation before, during and after 1 h running at $10 \text{ m}\cdot\text{min}^{-1}$ was investigated by indirect calorimetry.

Training increased 24 h protein intake (from 39% to 51% of total caloric intake) at the expense of carbohydrate (39.7% vs 23%); lipid intake was not modified (21.9% vs 26.5%). Glucose oxidation was comparable in trained and untrained rats at rest (60.4% vs 60.3% of total oxidation) as well as during (54.2% vs 48.9%) and after (49.1% vs 50.1%) running.

Rats who preferentially used carbohydrates to fuel resting metabolism immediately before running (average 73% vs 50% of total oxidation), continued to use more carbohydrate during (61% vs 44%) and after (59% vs 41%) treadmill running. These rats were characterized by a smaller lipid intake (16% vs 30% of daily caloric intake), but trained and untrained rats were equally distributed between the two groups.

Rats with a larger carbohydrate intake (46% vs 15% of 24 h caloric intake) were equally distributed between trained and untrained rats. In addition they showed no trend to oxidise more glucose at rest (60% vs 61% of total oxidation) as well as during (52% vs 53%) or after (49% vs 50%) treadmill running.

In conclusion, this study revealed that training modified diet selection, but this change could not be directly attributed to the energetic requirements of the exercise.

However there certainly exists some direct relationship between diet selection and substrate oxidation during exercise since glucose oxidation before and during exercise was larger in rats with the smallest lipid intake. Therefore, the relationship between diet selection, substrate oxidation and exercise training is certainly complex. A more precise assessment of protein metabolism may be important to better understand this point.

Perprandial changes in gastric wall tension control ingestion in pigs. L Lepionka, CH Malbert (*Station de recherches porcines, Inra, 35590 Saint-Gilles, France*).

Short term control of ingestion remains hypothetical because meal induced fundic relaxation cancels the possible gastric distension. Similarly, experimentally induced wall tension changes modify the sensation of a gastric distension. The aim of this study is to evaluate the characteristics of the ingestive pattern during proximal gastric distensions.

Perprandial isobaric or isovolumic fundic distensions were performed in four awake pigs by using an electronic barostat. Distension values were: 200/7, 250/11, 400/16 and 450/21 (mL/mmHg). Ingestive pattern for 500 g meal was characterized by duration of the meal, food intake rate FIR and no ingestion periods duration. These values were obtained by continuous weighing of the contents of the trough during the meal. No ingestion periods corresponded to FIR values less than $0.5 \text{ g}\cdot\text{sec}^{-1}$. The ingestion period was divided in three equal periods to study the role of gastric filling in the perprandial evolution of FIR.

Meal duration was not significantly different for isovolumic distension vs. control (no distension) (8.9 ± 0.31 vs 8.2 ± 0.45 min, 450 mL vs control). On the contrary, meal duration was significantly shorter for isobaric vs isovolumic distension at

$7/200 \text{ mmHg/mL}$ (8.0 ± 0.28 vs 8.9 ± 0.33 min). This shortening was the consequence of (i) an increased FIR during the second third of meal (57 ± 2.8 vs $51 \pm 2.2 \text{ g}\cdot\text{min}^{-1}$), and (ii) a reduced duration of no ingestion periods (194 ± 23.4 vs 263 ± 28.9 sec). A strictly inverse relationship was observed for higher pressures (10.2 ± 0.37 vs 8.7 ± 0.39 min, 11 mmHg vs 250 mL). The longer meal duration was related to only a reduced FIR during the second third of meal (47 ± 2.3 vs $55 \pm 2.1 \text{ g}\cdot\text{min}^{-1}$).

In conclusion, proprioceptive signals originating from the proximal stomach are responsible for perprandial control of ingestive behaviour in pigs. These stimuli corresponded to intraluminal pressure and wall tension informations.

Involvement of the protein network in the in vitro degradation of starch from spaghetti. A Fardet, C Hoebler, B Bouchet, F Guillon, DJ Gallant, JL Barry (*Human Nutrition Research Center, National Institute for the Agricultural Research, BP 1627, 44316 Nantes cedex 03, France*).

Among the factors responsible for the slow degradation of starch from pasta, the presence of a fine and compact protein network could be a decisive parameter. This work aimed to study by light microscopy and enzymatic treatments the involvement of the protein network in the alpha-amylase susceptibility of starch from spaghetti. Pasta were cooked 10 min, then either cut in 5 mm strands or ground. After 0 or 2 h of incubation with pepsin, spaghetti were incubated 24 h in vitro with either human salivary or pig pancreatic alpha-amylase (HSA and PPA, respectively). Compared to ground spaghetti, starch from intact strands of spaghetti was slowly degraded with both alpha-amylases. When incubated with HSA, starch was progressively removed from the protein network which remained intact.