It has been demonstrated that rats could acquire an aversion for an amino acid devoid diet (DEV) [Booth and Simson (1971), *Quart J Exp Psy* 23, 135-149]. When given ad libitum a DEV for several days, a lot of deleterious metabolic consequences have been observed, eg, fatty liver infiltration, organs atrophy, etc.

We have shown previously that (i) essential amino acid deficient rats acquire an aversion for the familiar deficient food which induces a preference (neophilia) for a novel food when available and (ii) that a 4-day delay is necessary in order to reverse the initial choice in favour of a novel deficient food and to prefer the now corrected familiar version (COR) [Fromentin et al (1996), *Br J Nutr*].

This experiment was undertaken to determine the influence of the length of pre-feeding a threonine devoid diet (THR-DEV) on the strength of aversion, as assessed by both phenomena previously mentioned (i and ii). Group 1: 12 male Wistar rats (220-255 g) were pre-fed ad libitum a THR-DEV(8 days), and given a choice between a COR and a protein free diet (PO%) during the following 6 days. Group 2-(n = 12) were pre-fed ad libitum a chow diet (8 days), then a THR-DEV(3 g during 15 min). After a 5 h delay, they were given the same choice as group 1. The outcome variable was the daily PO% preference ratio, calculated as the grams of daily PO% diet intake over total daily food intake.

Whatever the group and as soon as the first choice day, a daily PO% preference was measured (> 0.5). No between group difference was observed when we measured the daily PO% preference or the 4 days delay necessary to reverse the initial choice in favour of a novel deficient food (PO%) and to prefer the COR.

As soon as the first meal of THR-DEV (group 1) and after only a delay of a few hours, the amino acid induced aversion acts as an alarm signal indicating to the rat to avoid this deficient diet if available or to depress its food intake in order to diminish the deleterious consequences. Therefore the strengh of aversion is not reinforced by the real deleterious consequences dramatically increased by a repeated THR-DEV intake.

Effects of a threonine-devoid diet on plasma aminoacids, energy metabolism and feeding. V Rolland [1], S Feurte [1], S Roseau [1], G Fromentin [1], S Mahe [2], S Nicolaïdis [1], D Tomé [2], PC Even [1] *([1] CNRS UPR 9054, Collège de France, 75005 Paris; [2] GER Nutrition humaine, Ina-PG, 75005 Paris, France).*

The mechanism of rapid recognition of an aminoacid-(AA) deficient diet and of its avoidance is still unknown. It is usually assumed that anorexia is induced, first by an AA imbalance at the level of plasma that subsequently results in neurochemical changes which constitute in fine the active agents at the level of feeding centers. Alternatively, the plasma AA imbalance could, by itself, alter the overall oxidative metabolism and thus generate another signal that can be used for building up anorexia. The aim of this study was to reveal the existence of AA imbalance, of alterations of energy metabolism and of concomitant anorexia following a calibrated threonine-devoid diet and to see whether changes in substrate-utilisation could precede or not the first symptoms of anorexia.

Thirteen male Wister rats raised on a standard stock diet were housed in a calorimetric chamber and given either a threonine-corrected (TC) meal (55 kJ) containing 6 g/kg of threonine (control, n = 7) or a threonine-devoid (TD) meal (n = 6). Calorimetric measurements were performed from 2 h before to 10 h after feeding.

The extra energy production induced by feeding (thermic effect of feeding) did not differ between the TD and the TC diet. The

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 Wister 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 begining 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-Achagiotis [1], N Reith [1], PC Even [2] *([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 Wister 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 $m.min^{-1}$ 2 $h.day^{-1}$ (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 $m.min^{-1}$ 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.