Zeitschrift für
Ernährungswissenschaft
Z Ernährungswiss 32:71–73 (1993)

Postprandial thermogenesis of rats with glutamate induced obesity in relation to energy intake

L. Aust, U. Frenz, R. Noack, and J. Proll

German Institute for Human Nutrition, Bergholz-Rehbrücke, FRG

Die postprandiale Thermogenese bei Ratten mit Glutamat-induzierter Adipositas in Verbindung mit der Energieaufnahme

Summary: Postprandial thermogenesis was estimated in 4-month-old male rats with glutamate induced obesity after being fed with 300, 450 and 600 kJ/kg$^{0.75}$ of a pellet diet, respectively by indirect calorimetry in computer-controlled open circuit metabolic cages over 8 h. After an intake of 600 kJ/kg$^{0.75}$ (above the maintenance energy requirement) postprandial thermogenesis was significantly reduced in the obese animals to about 40 % of control rats (12.0 versus 31.5 kJ/kg$^{0.75}$ x 8 h). It is concluded that the glutamate obese rat can be accepted as an animal model with impaired facultative thermogenesis, mainly caused by a reduction of sympathetic adrenergic activity.

Zusammenfassung: Bei 4 Monate alten Ratten mit Glutamat-induzierter Adipositas wurde die postprandiale Thermogenese über 8 h nach Fütterung von 300, 450 und 600 kJ/kg^{0,75} einer Pellet-Diät mittels indirekter Kalorimetrie in computergesteuerten Stoffwechselkäfigen mit offenem Kreislauf bestimmt. Bei den adipösen Tieren war die postprandiale Thermogenese nach Aufnahme von 600 kJ/kg^{0,75} (oberhalb des Energieerhaltungsbedarfs) signifikant auf 40 % der Thermogenese der Kontrolltiere reduziert (12,0 gegenüber 31,5 kJ/kg^{0,75} x 8 h). Es wird geschlußfolgert, daß die Ratte mit Glutamat-induzierter Adipositas als ein Tiermodell mit beeinträchtigter fakultativer Thermogenese anzusehen ist, die hauptsächlich durch eine Verminderung der sympathischen adrenergen Aktivität verursacht ist.

Key words: Rat – glutamate-induced obesity – postprandial thermogenesis

Schlüsselwörter: Ratte – Glutamat-induzierte Adipositas – postprandiale Thermogenese

Introduction

There is evidence from studies in humans (10, 11) as well as in rodents (1, 2, 4, 5), that a higher body fat content, at least in a certain proportion of obese persons and in some animal models, is associated with a lower thermogenic response after food ingestion. This is assumed to indicate an increased efficiency of energy utilization.

Marchington et al. (5) found in obese (fa/fa) Zucker rats fed a pellet diet a 50 % reduction in thermogenic response, estimated by comparing the energy expenditure 2 h pre and postprandially. About the same extent of reduction was described by Hoover-Plow and Nelson (4) in two strains of mice, differing in body fat content by about 50 %. In postnatally overfed rats, a model of a moderate alimentary induced obesity, Aust et al. (1) observed a decrease in postprandial energy expenditure to about 70 % in comparison to control animals.

To characterize the energy metabolism of rats with glutamate-induced obesity (a model with hypothalamic lesions (7)) the postprandial energy expenditure was estimated by indirect calorimetry after increasing levels of energy intake.

Methods

The experiment was carried out with male rats of the Wistar strain (Wist Shoe, Versuchstierproduktion Schönwalde near Berlin), which received from the first to the fifth day of life a subcutaneous dorsal injection of sodium glutamate (2 mg/g body mass). After weaning the animals were kept in single cages with free access to food (standard pellets type R, Versuchstierproduktion Schönwalde, 12.4 kJ/g, 20 % protein, 10 % fat, 70 % carbohydrates as metabolizable energy) and water until the age of 4 months. The average body mass and the body fat content at that time was: obese rat: 381 g and 36,5 %, control rat: 454 g and 13,0 %, respectively. The lower body mass is characteristic for this animal model of obesity (9). The animals were then transferred into metabolic cages (Simax, ČSFR), supplied with a constant stream of fresh air (60 l/h, 25 ± 1 °C, 100 % relative humidity). Air flow was measured by wet gas meters (Junkalor, Dessau). Oxygen consumption and carbon dioxid production were estimated by using a paramagnetic O_2 analyzer (Permolyt II) and an infrared CO_2 analyzer (Infralyt IV), both from Junkalor, Dessau. Urinary nitrogen excretion was determined by the Kjeldahl method. Energy expenditure was calculated according to Hoffmann and Schiemann (3).

At 9 a.m. the rats received 300 kJ/kg$^{0.75}$ of the same standard pellet diet after 16 h fasting. On following 2 days they were offered, at the same time, 450 and 600 kJ/kg$^{0.75}$, respectively. To estimate the basal energy expenditure the rats remained in the cages for an additional day without food. Due to this feeding regimen the animals consumed the offered food within 8 h. Frequency of food intake was not different in the two animal groups.

Postprandial thermogenic response is calculated as the difference between energy expenditure 8 h after food and an equal time period without food.

Results and Discussion

The postprandial energy expenditure of rats with glutamate induced obesity related to energy intake is given in Table 1. Although the postprandial increase in energy expenditure has a high deviation, postprandial thermogenesis after an intake of 600 kJ/kg$^{0.75}$ is significantly lower in the obese animals and reaches only about 40 % of control rats (12.0 versus 31.5 kJ/kg$^{0.75}$ x 8 h). At an intake of 300 and 450 kJ/kg$^{0.75}$ differences in postprandial energy expenditure cannot be verified due to the slighter increase and high variation of postprandial response.

The significant difference after an intake of 600 kJ/kg$^{0.75}$ indicates that reliable values of postprandial thermogenesis in animal models of obesity can be obtained at an energy intake above the maintenance level. Postprandial increase of energy expenditure related to basal energy expenditure or to energy intake is likewise significantly lower in the glutamate obese rat (relative and absolute postprandial thermogenesis). For the control rats these values are of the magnitude expected (1, 8). Conclusions in this experiment are made on the basis of the chosen feeding schedule by comparing the obese animals with the control group, thus giving reliable results about the relative differences in postprandial thermogenesis. However, the absolute values could be influenced by randomizing the order of the level of energy intake. This should be elucidated in further experiments.

These data confirm earlier results, i.e., that an obese state can be accompanied by an impaired postprandial thermogenic capacity, possibly caused by an increased efficiency in energy utilization. Glutamate-induced obesity in rats leads to an impairment of

Table 1. Postprandial energy expenditure of rats with glutamate-induced obesity in dependence on energy intake

Energy intake [kJ/kg$^{0.75}$]	300		450		600	
	Control	Obese	Control	Obese	Control	Obese
n	7	7	8	7	8	7
Energy expenditure [kJ/kg$^{0.75}$ x 8 h]						
Basal	←		139 ± 9	146 ± 11	→	
Postprandial	148 ± 4	143 ± 6	161 ± 10	157 ± 12	179 ± 18	158 ± 10*
Postprandial increase	9.1 ± 9.3	-2.3 ± 6.9	22.2 ± 9.7	11.6 ± 9.7	31.5 ± 13.7	12.0 12.6*
Postprandial increase in % of basal	7.0 ± 7.2	-1.4 ± 4.5	16.1 ± 7.5	8.2 ± 6.9	22.5 ± 9.6	8.6 ± 9.4*
Postprandial increase in % of intake	3.0 ± 3.1	-1.8 ± 1.4	4.9 ± 2.1	2.6 ± 2.2	5.3 ± 2.3	2.0 ± 2.1*

Means ± SD, * $p < 0.05$

brown adipose tissue function (6), outlined by a lower GDP-binding to the mitochondrial membrane as well as by a lower blood flow through this tissue after noradrenaline stimulation. The glutamate obese rat can be accepted as an animal model with impaired facultative thermogenesis, which is mainly caused by a reduction of sympathetic adrenergic activity (2).

References

1. Aust L, Noack R, Proll J (1986) Nahrung 30:451–452
2. Himms-Hagen J (1989) Can J Physiol Pharmacol 67:394–401
3. Hoffmann L, Schiemann R (1980) Arch Tierernähr 30:733–742
4. Hoover-Plow J, Nelson B (1985) J Nutr 115:303–310
5. Marchington D, Rothwell N, Stock M, York D (1983) J Nutr 113:1395–1402
6. Noack R, Leopold F, Frenz U, Rehorek A, Remke H, Steiniger J (1989) Ernährungsforschung 34:111–116
7. Olney I (1969) Science 164:719–721
8. Porrata-Maury C, Aust L, Noack R, Eschrich H (1987) Nahrung 31:311–319
9. Remke H, Wilsdorf A (1985) Nahrung 29:619–620
10. Schutz Y, Bassard T, Jequier E (1984) Am J Clin Nutr 40:542–552
11. Steiniger J, Karst H, Noack R, Steglich HD (1987) Ann Nutr Metabol 31:117–125

Authors' address:

Dr. Lothar Aust, Deutsches Institut für Ernährungsforschung, A.-Scheunert- Allee 114–116, O-1505 Bergholz-Rehbrücke, FRG