

## THE ADIPOSE CELL

**Effects of dietary MCT on lipid storage and thermogenesis.** E. Simon<sup>a</sup>, A.S. Del Barrio<sup>a</sup>, F. Serra<sup>b</sup>, A. Palou<sup>b</sup>, M.P. Portillo<sup>a</sup> (<sup>a</sup>Department of Nutrition, Faculty of Pharmacy, University of País Vasco, Paseo de la Universidad, 7, 01006 Vitoria; <sup>b</sup>Department of Biochemistry and Molecular Biology, Faculty of Sciences, University of Illes Balears, Palma de Mallorca, Spain).

Thermogenesis is one of the metabolic processes that control energy expenditure and therefore lipid storage. It has been suggested that the fatty acid chain length may modify thermogenic activity. In this context, the usefulness of medium chain fatty acid triglycerides for obesity treatment (MCT) has been proposed. The aim of this work was to study the effects of dietary MCT on the fat accretion and brown adipose tissue thermogenic activity of overweight rats. Female Wistar rats were divided into two groups ( $n = 8$ ) and fed ad libitum either an olive oil diet or a MCT diet, for 23 days. Both diets were isoenergetic and 12 % of their energy was provided by fat.

At the end of the experimental period, white adipose tissue from several anatomical locations and interscapular brown adipose tissue were dissected and weighed. The UCP contents were measured by immunoblotting. The student's *t*-test was used for statistical analysis. In the MCT group, body weight ( $-10$  %;  $P < 0.05$ ) and adipose depot size ( $-10$  %;  $P < 0.05$ ) were reduced. There were no differences in UCP content (arbitrary units/g tissue) between both groups ( $3371 \pm 671$  versus  $3192 \pm 394$ ;  $P = 0.414$ ).

It can be concluded that MCT induced a body weight reduction, which was related to a decrease in adiposity. These results cannot be attributed to a modification of thermogenesis.

**Lipolytic response from subcutaneous adipose tissue in rats fed diets providing different fat sources.** A.I. Tueros, D. Durán, B. Rodríguez, M.T. Macarulla, M.P. Portillo (Department of Nutrition, Faculty of Pharmacy, University of País Vasco, Paseo de la Universidad, 7, 01006 Vitoria, Spain).

Beta-adrenergic agonists induce adenylate-cyclase activation and then lipid mobilization by coupling with  $\beta$ -adrenoceptors. These receptors are placed in the adipocyte membrane. There are some factors, however, which are able to modify the phospholipid fatty acid profile of the adipocyte membrane. In this context, it has been proposed that factors affecting membrane fatty acids could induce changes in lipolytic response. The aim of this work was to study the effects of dietary fatty acids on different steps of the adenylate-cyclase cascade and so, on lipid mobilization.

For this purpose three lipid sources were used: olive oil (1), safflower oil (2) and palm oil (3). After 4 weeks of dietary treatment, animals were killed and subcutaneous adipose tissue was dissected. Adipocytes were isolated by collagenase digestion. For statistical analysis, the ANOVA and Duncan test were used.

Lipolysis was stimulated by several drugs: isoproterenol (a beta-adrenergic agonist) forskolin (an activator of adenylate-cyclase), dibutyryl-cAMP and isobutyl-methyl-xantine (products that allow the study of adenylate-cyclase activity) and adenosine deaminase (product that produces the inactivation of the adenosine released by adipocytes). Although it has been published that dietary lipids can induce some changes in membrane fatty acids, following our experimental design, those changes were not related to modifications in the lipid mobilization capacity in rat subcutaneous adipose tissue.