which was probably related to the maintenance of lean mass. This stabilization contributed to the weight loss between 3 and 6 months. A determination of TEE when the body weight stabilizes will allow the quantification of the energy intake.

**Modification of the β-adrenergic lipolytic response in obese human adipocytes.**
AS del Barrio [1], MP Portillo [1], E Campo [2], J Errasti [2], MA Garcia [1], C Martinez [2], AM Rocandio [1] *([1] Nutrición, Univ País Vasco; [2] Cirugía, Hospital de Txagorritxu, Vitoria, Spain)*

The mobilization of stored triglycerides is under the control of different subtypes of β-adrenergic receptors, which can be modified under several physiopathological situations, such as obesity (Hellmer et al [1992] *J Clin Endocrinol Metabol* 75, 15). This investigation focused on the study of the lipolytic response induced by β-adrenergic agonists in adipocytes from obese and nonobese patients.

Omental adipose tissues were obtained during different surgical situations and the adipocytes were isolated by incubation with collagenase in a KRBA buffer (pH 7.4). A control (body mass index [BMI] < 25; $n$ = 17) and an obese (BMI ≥ 25; $n$ = 21) group were formed. The in vitro lipolytic response was induced by dobutamine (Dob; $\beta_1$), clenbuterol (Clen; $\beta_2$), metaproterenol (Met; $\beta_1$ and $\beta_2$) in a range of concentrations of $10^{-8}$ to $10^{-4}$ M.

The adipocytes from the obese group showed a higher basal lipolysis (0.18 ± 0.01 µmol glycerol/100 mg lipids) than controls (0.16 ± 0.06 µmol glycerol/100 mg lipids; $P < 0.05$). The minimum concentrations of the β-agonists needed to produced a significant lipolytic effect were higher in adipocytes from obese subjets than controls (Dob: $5 \times 10^{-5}$ vs $10^{-5}$ M; Clen: ND vs $5 \times 10^{-7}$ M; Met: $10^{-6}$ vs $10^{-7}$ M). In relation to maximal effect, a statistically significant reduction was only observed for the lipolytic response to Met in the obese group (139.5 vs 220.6% of basal lipolysis).

It can be concluded that obesity alters the metabolic pathways relating to the lipolytic capacity controlled by β-adrenergic agonists.

The financial support by UPV/EHU 101.123-EA 142-94 is acknowledged.