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.

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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; β1), clenbuterol (Clen; β2), metaproterenol (Met; β1 and β2) 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 produce a significant lipolytic effect were higher in adipocytes from obese subjects than controls (Dob: 5 x 10⁻⁵ vs 10⁻⁵ M; Clen: ND vs 5 x 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.

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