INDIRECT CALORIMETRY DEMONSTRATES THAT RESTING ENERGY EXPENDITURE IS INCREASED IN PATIENTS WITH POORLY CONTROLLED DIABETES AND IS NORMALIZED BY INSULIN BOLUS

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It has been suggested that an increase in energy expenditure may promote the body weight reduction which is usually observed in diabetic patients with poor metabolic control.

Therefore, the resting energy expenditure (REE) was measured using a ventilated hood system of indirect calorimetry (Quark RMR; Cosmed, Roma, Italy) in 20 patients (8 males, 12 females) with poorly controlled type 2 diabetes (body mass index -BMI-: $34.3 \pm 2.1 \text{ kg/m}^2$; fasting plasma glucose -FPG-: $11.1 \pm 0.5 \text{ mmol/l}$), treated with oral hypoglycemic agents (n= 14) or nutritional treatment alone (n= 6). A group of non-diabetics (n= 14, 8 males and 6 females) with similar age and body size to that of the diabetic group (BMI: $35.2 \pm 1.9 \text{ kg/m}^2$, P= 0.76; FPG: $4.8 \pm 0.2 \text{ mmol/l}$, P< 0.001) was included as control group.

The diabetic group exhibited a REE normalized for the fat-free mass size (FFM, bioelectrical impedance; BIA-103, RJL, Detroit, MI, USA/Akern, Florence, Italy) higher by 6.8% (+123 kcal/24h, P = 0.04) than that of non diabetics.

Furthermore, the value of normalized REE for FFM was significantly correlated to the value of FPG (r= 0:58, P= 0.04) in diabetic patients, suggesting that the higher REE is dependent on glycemic control. As known, the value of FPG is strongly influenced by the gluconeogenesis, an energetically wasteful metabolic process. Therefore, the high REE observed in diabetic patients might be in consequence of the increased gluconeogenetic metabolism. In order to verify this hypothesis, it was administered an IV bolus of regular insulin (0.2 IU kg body weight; Actrapid ®, NovoNordisk, Denmark) in 5 diabetic participants. Following the insulin bolus a progressive reduction of REE was observed as follows (REE change): 10 min: -11.6%; 20 min: -17.4%; 30 min: -19.9%; 45 min: -22.0%; 60 min: -20.1%, P= 0.04), similar reductions of blood glucose and lactate concentrations were observed. In conclusion, diabetic patients with poor metabolic control have a higher energy expenditure probably in consequence of a significantly higher activity of gluconeogenesis. This study may contribute, at least in part, to recognize the nature of body weight reduction that occurs in concomitance with poorly controlled diabetes and of body weight gain as that commonly observed when the hypoglycemic treatment with, in particular, sulphonylureas and insulin is started.

References

- Gougeon R. Thermic and metabolic responses to oral glucose in obese subjects with non-insulindependent diabetes mellitus treated with insulin or a very-low-energy diet. Am J Clin Nutr 1996:64:78-86.
- 2) Weyer C, Bogardus C, Pratley RE. Metabolic factors contributing to increased resting metabolic rate and decreased insulin-induced thermogenesis during the development of type 2 diabetes. Diabetes 1999;48:1607-1614.
- 3) Gougeon R, Lamarche M, Yale JF, Venuta T. The prediction of resting energy expenditure in type 2 diabetes mellitus is improved by factoring for glycemia. Int J Obes Relat Metab Disord 2002;26:1547-1552.
- 4) Martin K, Wallace P, Rust PF, Garvey WT. Estimation of resting energy expenditure considering effects of race and diabetes status. Diabetes Care 2004;27:1405-1411.
- 5) Ryan M, Sallé A, Guilloteau G, Genaitay M, Livingstone MB, Ritz P. Resting energy expenditure is not increased in mildly hyperglycaemic obese diabetic patients. Br J Nutr 2006;96:945-948.
- 6) Tan TM, Field BC, McCullough KA, Troke RC, Chambers ES, Salem V, et al. Coadministration of glucagon-like peptide-1 during glucagon infusion in humans results in increased energy expenditure and amelioration of hyperglycemia. Diabetes 2013;62:1131-1138.

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