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Dietary glycemic index/load and peripheral adipokines and inflammatory markers in elderly subjects at high cardiovascular risk

M. Bulló [a,b,*], R. Casas [b,c], M.P. Portillo [d], J. Basora [a,b], R. Estruch [b,c], A. García-Arellano [e], A. Lasa [d], M. Juanola-Falgarona [a,b], F. Arós [f], J. Salas-Salvadó [a,b]

[a] *Human Nutrition Unit, Hospital Universitari de Sant Joan, Facultat de Medicina i Ciències de la Salut, IISPV, URV, Spain*
[b] *CIBERobn-Fisiopatología de la Obesidad y Nutrición, ISCIII, Spain*
[c] *Department of Internal Medicine, IDIBAPS, Hospital Clínic, Spain*
[d] *Department of Nutrition and Food Science, UPV, Spain*
[e] *Department of Preventive Medicine and Public Health, UNAV, Spain*
[f] *Department of Cardiology, Hospital Txangorritxu, Spain*

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Abstract *Background and Aims:* Epidemiological and clinical studies suggest that low-glycemic index diets could protect against weight gain. However, the relationship between these diets and adipokines or inflammatory markers is unclear. In the present study we examine how the dietary glycemic index (GI) and dietary glycemic load (GL) are associated with several adipokines and related metabolic risk markers of obesity and diabetes in a cross-sectional and longitudinal manner.
Methods and Results: 511 elderly community-dwelling men and women at high cardiovascular risk were recruited for the PREDIMED trial. Dietary data were collected at baseline and after 1 year of follow-up. The GI and GL were calculated. Plasma leptin, adiponectin and other metabolic risk markers were measured at baseline and after 1 year. At baseline, subjects in the highest quartiles of GI showed significantly higher levels of TNF and IL-6 than those in the lowest quartiles. Dietary GI index was negatively related to plasma leptin and adiponectin levels. After 1 year of follow-up, subjects with a higher increase in dietary GI or GL showed a greater reduction in leptin and adiponectin plasma levels. There was no association between GI or GL and the other metabolic markers measured.

* Corresponding author. Human Nutrition Unit, Faculty of Medicine and Healthy Sciences, Universitat Rovira i Virgili, C/Sant Llorenç 21, 43201 Reus, Spain. Tel.: +34 977759312; fax: +34 977759322.
E-mail address: monica.bullo@urv.cat (M. Bulló).

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Conclusion: Our results suggest that the consumption of high-GI or high-GL diets may modulate plasma concentrations of leptin and adiponectin, both adipostatic molecules implicated in energy balance and cardiometabolic risk.

Introduction

Chronic low-grade inflammation associated with increased adipocytokine production from adipose tissue is recognized as a central mechanism underlying obesity and its comorbidities. Two major adipocytokines, leptin and adiponectin, are involved in the regulation of energy balance and cardiovascular homeostasis. Leptin acts on the hypothalamus and regulates satiety, food intake and energy expenditure. Leptin also induces insulin resistance through its role in the phosphorylation of the insulin receptor [1]. Adiponectin increases fat oxidation, which reduces peripheral levels of fatty acids and increases insulin sensitivity [2]. The adiponectin/leptin ratio is suggested to be a useful parameter for assessing insulin resistance and atherogenic risk, and is even more sensitive and reliable than the homeostasis model assessment-insulin resistance (HOMAIR).

Since diet is the first line of intervention for preventing and treating obesity and cardiovascular risk factors, in the last years there has been growing interest in the role that different types of carbohydrates play in the modulation of postprandial glucose/insulin response, inflammatory markers and related molecules. The consumption of diets containing high amounts of whole grains and/or dietary fiber has been associated with low serum inflammatory markers. However, the many types of carbohydrates and fiber, have a different effect on postprandial glucose and insulin responses. In this regard, Jenkins introduced the concept of the glycemic index (GI), which ranks carbohydrate-rich foods in accordance with how much they raise blood glucose levels in comparison to standard foods [3]. The concept of glycemic load (GL) was subsequently developed to take into account the amount of food consumed [4]. Hypothetically, repeated postprandial hyperglycemia and hyperinsulinemia induced by foods with a high-glycemic index may cause insulin resistance, beta cell dysfunction and inflammation by many mechanisms. However, the results of epidemiological and interventional studies on this issue have been controversial. Although epidemiological studies have linked dietary GI and GL with obesity, type 2 diabetes and high risk of cardiovascular disease [5—8], they have not been able to consistently link them with inflammatory biomarkers [9—12]. Nevertheless, the few clinical trials that have evaluated the effect of dietary GI or GL on inflammation have reported an inconsistent reduction in circulating protein-C reactive levels and no effect on tumor necrosis factor (TNF) or interleukin-6 (IL-6) [13—15].

The scarcity and the inconsistency of the evidence available on the effect of dietary GI and GL on adipokines led us to examine the changes in dietary GI and GL and changes in several adipokines and related metabolic risk markers of obesity and diabetes in a cohort of elderly subjects at high cardiovascular risk.

Methods

Study population

We assessed 568 consecutively admitted participants recruited from the PREDIMED trial centers in Reus and Barcelona. The PREDIMED study is a large, parallel group, multicenter, controlled, randomized, 6-year clinical trial designed to evaluate the effects of the Mediterranean diet (MeDiet) on the primary prevention of cardiovascular disease. Candidates were community-dwelling men and women aged 55—80 years and 60—80 years, respectively, who had no previously documented cardiovascular disease and met at least one of the two following criteria: type 2 diabetes mellitus, or three or more cardiovascular risk factors [current smoking, hypertension (blood pressure $\geq$ 140/90 mmHg or treatment with antihypertensive drugs), low-density lipoprotein cholesterol level $\geq$ 160 mg/dL (or treatment with hypolipidemic drugs), high-density lipoprotein cholesterol level $\leq$ 40 mg/dL, BMI $\geq$ 25 kg/m^2, or family history of premature cardiovascular disease]. Exclusion criteria included any severe chronic illness, drug or alcohol addiction, history of allergy or intolerance to olive oil or nuts, or a low predicted likelihood of changing dietary habits according to the Proschaska and DiClemente stages-of-change model. Participants were randomly assigned to three interventions: MeDiet with virgin olive oil (VOO), MeDiet with mixed nuts and control group (low-fat diet). Both MeDiet groups received intensive education to follow the MeDiet and VOO or mixed nuts (walnuts, hazelnuts, almonds) were provided by the study. In the control group, participants were given advice to follow a low-fat diet. Full details of the study protocol have been published elsewhere [16]. The protocol was approved by the institutional review board of both Institutions and all participants provided written informed consent.

Dietary assessment

At baseline (before randomization) and after 1 year of follow-up participants were assessed by means of a 137-item FFQ to estimate average daily nutrient intake over the previous 12-month period. Detailed information regarding the development of FFQ and the reproducibility and validity of the questionnaire has been previously reported [17]. We estimated energy and nutrient intakes by multiplying the frequency of consumption of each food by the nutrient content estimated using Spanish food composition tables. The GI was determined using the Brand-Miller tables [18].