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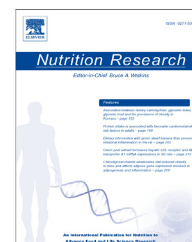

An acute intake of a walnut-enriched meal improves postprandial adiponectin response in healthy young adults

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ABSTRACT

A deficit in adiponectin plays an important causal role in insulin resistance and metabolic syndrome. We hypothesized that as seen during the fasting state, the intake of a walnut-enriched meal increased postprandial adiponectin. Twenty-one healthy white men followed a 4-week baseline diet and then consumed 3 fat-loaded meals that included 1 g fat/kg body weight (65% fat) according to a randomized crossover design: olive oil–enriched meal (22% saturated fatty acids [SFA], 38% monounsaturated fatty acids [MUFA], 4% polyunsaturated fatty acids [PUFA]), butter-enriched meal (35% SFA, 22% MUFA, 4% PUFA), and walnut-enriched meal (20% SFA, 24% MUFA, 16% PUFA, and 4% α-linolenic acid). Leptin, resistin, adiponectin, and free fatty acids were determined at 0, 3, 6, and 8.5 hours after the fat load. After the walnut-enriched meal, plasma adiponectin concentrations were higher at 3 and 6 hours ($P = .011$, $P = .046$, respectively) compared with the butter-enriched meal and higher at 6 hours compared with the olive oil–enriched meal ($P = .036$). Free fatty acid levels decreased from baseline at 3 hours after the walnut-enriched meal ($P = .001$). No differences were observed between the 3 meals for leptin and resistin responses. Our data confirmed a beneficial profile in the postprandial response to walnuts, source of omega-3 PUFA with an increased postprandial adiponectin and lower postprandial free fatty acid responses. These findings suggest that the postprandial state is important for understanding the possible cardioprotective effects associated with omega-3 PUFA dietary fat.

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1. Introduction

Adipose tissue produces a multitude of bioactive peptides, known as "adipokines" that not only affect the adipocyte function in an autocrine and paracrine manner but also participate in more than one pathway through the circulatory system of the blood. The concept of adipose tissue as an endocrine organ originated in 1995 with the discovery of leptin

Abbreviations: ALA, α-linolenic acid; ANOVA, Analysis of variance; AUC, Area under the curve; C, Cholesterol; ELISA, Enzyme-linked immunosorbent assay; FFA, Free fatty acids; HOMA-R, Homeostasis model assesment ratio; IL-6, Interleukin 6; MUFA, Monounsaturated fatty acids; n-3, Omega-3; PUFA, Polyunsaturated fatty acids; SFA, Saturated fatty acids; TG, Triglycerides; TNF-α, Tumor necrosis factor α; TRL, Triglyceride-rich lipoproteins.

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and the extent of their biological functions [1]. For instance, high levels of adiponectin have been associated with less likelihood of developing insulin resistance, type 2 diabetes mellitus [2], and coronary heart disease [3]. In addition, plasma levels of resistin have been predictive of coronary atherosclerosis in asymptomatic patients with family history of coronary heart disease [4].

Several candidate genes may affect primary insulin action, and particularly, the interaction with dietary fat is an important modulator of glucose metabolism and insulin resistance [5,6]. In this context, previous data indicate that after a fatty meal, a rise has been noted in inflammatory cytokines such as tumor necrosis factor α (TNF-α) or interleukin 6 (IL-6), which are involved in the regulation of adiponectin, by decreasing its concentrations. Moreover, chronic consumption of saturated fatty acids (SFAs) has been inversely correlated with fasting adiponectin [7,8]. Interestingly, 1 study showed an improvement in plasma adiponectin concentrations after the daily consumption of 6.5 mL of olive oil for 1 year, but this was enriched with omega-3 (n-3) polyunsaturated fatty acids (PUFA) [9]. Furthermore, an increase in circulating concentrations of adiponectin can be observed with chronic consumption of walnuts [10,11], a source of n-3 PUFA of vegetable origin, being associated with a lower risk of developing cardiovascular disease [12,13]. However, the postprandial response to an overload with high fat content has not been well established, with highly variable results, and no evidence exists about the effect of different quality of fats on it.

Postprandial metabolism is the physiological state of humans in modern society and has become more important because it has been demonstrated that the postprandial phase is associated with increased inflammation and oxidation, which influences vascular function through a continuous aggression to the endothelium by atherogenic lipoprotein. In this context, adipokines play a main role in the inflammatory processes. On the other hand, dietary fat is probably the most important environmental factor that modulates postprandial lipemia and changes in postprandial metabolism take place every time we eat a meal. Based on that, we hypothesized that the acute intake of a walnut-enriched meal improves the postprandial adipokines response as previously observed during the fasting state. On this basis, we conducted a very well-controlled study including healthy white volunteers who were given 3 meals based on natural foods (butter, olive oil, and walnuts) with the same fat content, but with different fatty acid composition, to evaluate differences in the response of several postprandial adipokines levels.

2. Methods and materials

2.1. Subjects

Twenty-one males, aged 23 ± 1.5 (range, 18-30) years, were included in this study. All of them gave informed consent and underwent a comprehensive medical history, physical examination, and clinical chemistry analysis before enrollment. Subjects showed no signs of any chronic disease, and none had unusually high levels of physical activity. None of the subjects were taking medication or vitamins known to affect plasma lipids. One of the inclusion criteria was having a body mass index lower than 30 kg/m^2. The study protocol was approved by the Human Investigation Review Committee of the Reina Sofia University Hospital.

2.2. Study design

Each subject, after 4 weeks on a typical Western-style stabilization basal diet rich in saturated fat (38% fat: 16% SFAs, 16% monounsaturated fatty acids [MUFA], and 6% PUFA; 15% protein and 47% carbohydrates), underwent 3 postprandial lipemia trials, in which they consumed meals with the same fat content (1 g fat/kg body weight, 7 mg cholesterol/kg, and 40 retinol equivalents/kg body weight) but with different fatty acid compositions, following a random administration order. After fasting for 12 hours, at time 0, the subjects were provided with a fatty meal consisting of 50% to 66% of their normal daily intake of calories. The meals provided 60% fat, 15% protein, and 25% carbohydrates. The fat composition of the foods used in the postprandial lipemia studies was as follows: fatty food based on extra virgin olive oil (olive oil–enriched meal: 22% SFA, 38% MUFA, 4% PUFA, and 0.7% α-linolenic acid), fatty food based on butter (butter-enriched meal: 35% SFA, 22% MUFA, 4% PUFA, and 0.7% α-linolenic acid), and fatty food based on walnuts (*Junglans regia* L.) (walnut-enriched meal: 20% SFA, 24% MUFA, 16% PUFA, and 4% α-linolenic acid). The meals were administered according to a Latin-square design, so that all subjects received in random sequence the 3 diets on 3 different occasions separated by 1 week. Throughout the 8.5-hour duration of the study, the subjects neither performed physical activity nor consumed anything but water. Venous blood samples were collected in tubes containing 1 mg/mL EDTA 0, 3, 6, and 8.5 hours after fat food intake at each time point.

2.3. Lipoprotein separations

Blood was collected in tubes containing EDTA to give a final concentration of 0.1% EDTA. Plasma was separated by centrifugation at 1500*g* for 15 minutes at 4°C. The chylomicron fraction of triglyceride (TG)–rich lipoproteins (TRL) (large TRL) was isolated from 4-mL plasma overlain by 0.15 mol NaCl/L, 1 mmol EDTA/L (pH 7.4, density <1.006 kg/L) by a single ultracentrifugal spin (36 200*g* for 30 minutes at 4°C) in a type 50 rotor (Beckman Instruments, Fullerton, CA). Chylomicrons, contained in the top layer, were removed by aspiration after cutting the tubes, and the infranatant was centrifuged at a density of 1.019 kg/L for 24 hours at 183 000*g* in the same rotor. The nonchylomicron fraction of TRL (small TRL) was removed from the top of the tube. All operations were done in subdued light. Large and small TRL fractions were stored at −70°C.

2.4. Lipid and insulin analysis

Cholesterol (C) and TG in plasma and lipoprotein fractions were assayed by enzymatic procedures [14,15]. Apolipoprotein A-I and Apolipoprotein B were determined by turbidimetry [16]. High-density lipoprotein C was measured by analyzing the supernatant fluid obtained after precipitation of a plasma

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