



Applied nutritional investigation

Low energy and carbohydrate intake associated with higher total antioxidant capacity in apparently healthy adults



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ABSTRACT

Objectives: The aim of this study was to investigate the associations between plasma total antioxidant capacity (TAC) and anthropometric, biochemical, clinical, and dietary measurements in young and apparently healthy individuals.

Methods: We evaluated 156 individuals (91 women and 65 men; ages 23.1 ± 3.5 y; body mass index 22 ± 2.9 kg/m²) for anthropometrics, biochemical markers, clinical, dietary, and some components of the antioxidant defense system, including the plasma TAC. Statistical analyses were performed to detect differences between individuals with TAC higher and lower than the mean value and to screen the associations between TAC and variables of interest. A linear regression model was fitted to identify TAC predictors.

Results: Daily caloric intake and macronutrient consumption were lower in individuals who exhibited the highest TAC values ($P < 0.05$). Linear regression analysis showed that daily calories and carbohydrate intake was a possible negative TAC predictor ($P < 0.05$). Nevertheless, there was no difference in the values of oxidized low-density lipoprotein in the individuals separated by means of TAC. In contrast, individuals whose plasma TAC values were above the mean showed higher low-density lipoprotein cholesterol concentrations, total cholesterol/high-density lipoprotein cholesterol values, and selenium in nails ($P < 0.05$).

Conclusions: In physiological conditions, the caloric intake level seems to be an important factor to act in the modulation of plasma TAC, before establishing anthropometric impairments of body or metabolic composition, or both. Additionally, the plasma TAC increase may be able to act as a compensatory mechanism.

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Introduction

The continuous production of reactive oxygen species (ROS) during metabolic processes culminates in the activation of antioxidant defense mechanisms [1]. These protective mechanisms include some enzymes such as superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx); macromolecules such as ceruloplasmin; and other compounds such as the ascorbic acid, α -tocopherol, β -carotene, uric acid, selenium, copper, and zinc [2]. The total antioxidant capacity (TAC)

includes consideration of the occurrence of a synergic action of all antioxidants present in organic fluids, providing an integrative system between such compounds [3]. Thus, TAC has a higher predictive capacity and biological relevance when compared with the activity of a single antioxidant.

Several previous studies have shown that increasing the intake of antioxidant nutrients (e.g., vitamins C and E) or foods (e.g., tea, fruits, and vegetables) may have cardioprotective effects by reducing inflammation, improving vascular reactivity, and lowering oxidative stress (OS) [4–6]. However, the extent to which dietary compounds can have a significant influence on OS remains controversial [7].

In vitro studies have shown the capacity of diet antioxidant in removing the ROS or inhibiting their deleterious action [8,9]. However, in vivo studies have revealed divergent results regarding the effect of dietary antioxidants on TAC [10,11]. In this context, it is assumed that the complex interactions existing between several antioxidants limit the extension of evidences originating from in vitro studies.

Prior studies demonstrated a significant TAC reduction by several pathologic conditions associated with OS [12,13]. This reduction may be seen in obese individuals [14,15]; especially, those with increased visceral adiposity [16]. Moreover, there seem to be no doubts as for such participation in important alterations associated with metabolic syndrome [17–19], suggesting that OS precedes chronic diseases in some cases [20].

The antioxidant defense system capacity is a determining factor in health maintenance in prevention of diseases. Furthermore, the role of the diet and some nutrients on OS and its related diseases remains unclear [21,22]. Therefore, the present study investigated the possible existing associations between plasma TAC and anthropometrical, biochemical, clinical, and dietary measurements in young and apparently healthy individuals. Moreover, our study analyzed associations between plasma TAC and some endogenous (serum levels of uric acid and ceruloplasmin, GPx enzymatic activity) and exogenous (concentration of antioxidant minerals in nails: copper, zinc, and selenium) components of the antioxidant defense system and dietary intake of antioxidant compounds (vitamins C and A, zinc, and copper). Additionally, the TAC association with circulating levels of oxidized low-density lipoprotein (ox-LDL), another marker of OS, was investigated.

Material and methods

Participants

We recruited 156 individuals between the ages of 18 and 35 y to participate in the study (91 women and 65 men; ages 23.1 ± 3.5 y; and body mass index [BMI] 22 ± 2.9 kg/m²). Initial screening excluded individuals with evidence of any disease related to OS; chronic inflammation, hydric balance disorders, changes in body composition and nutrient absorption or metabolism. Other exclusion criteria were as follows: drug or nutritional treatment that affects energy balance, dietary intake, lipid profile, insulin levels, or glucose metabolism, contraceptive use up to 2 mo before participation in the study and weight loss diet follow-up or unstable weight in the past 6 mo. In agreement with the principles of the Helsinki Declaration and following a clear explanation of the study protocol, each participant signed a written informed consent form. The study was approved by the Human Research Ethics Committee of the Federal University of Viçosa, Brazil (protocol no. 009/2006). Participant recruitment occurred between January 2009 and February 2010.

Anthropometric and body composition assessments

Height was measured with a stadiometer (Seca 206 model, Hamburg, Germany) to the nearest 0.1 cm. Body weight was measured to the nearest 0.1 kg by using an electronic micro digital scale (Tanita TBF-300 A model, Tokyo, Japan). BMI was calculated by the quotient between body weight and square height (kg/m²).

Waist and hip circumference was measured with an inelastic and flexible tape to the nearest 0.1 m [23]. Triceps, biceps, subscapular and suprailiac skinfold thicknesses were measured to the nearest 1 mm by using a skinfold caliper (Lange caliper, Cambridge Scientific Industries Inc., Cambridge, MD, USA) [24]. The sum of skinfold thickness was calculated. Total body fat percentage was measured to the nearest 0.1% using a body composition analyzer (Biodynamics 310 model, Seattle, WA, USA). Body fat mass and body free fat mass were also estimated using the same body composition analyzer [25]. Truncal fat percentage was computed as the sum of subscapular and suprailiac skinfold thicknesses divided by the sum of four skinfold measurements [26].

Blood pressure assessment

Systolic and diastolic blood pressures were measured by a mercury sphygmomanometer (BIC, SP, Brazil) following World Health Organization criteria [27].

Analyses of biological samples

Blood samples were drawn by venous puncture after a 12-h overnight fast. The ethylenediaminetetraacetic acid plasma, heparin plasma, and serum samples were separated from whole blood by centrifugation at 3500g at 5°C for 15 min (Eppendorf AG, 5804 R model, Hamburg, Germany) and immediately stored at -80°C until analyzed.

Lipid and glucose profile

Serum glucose, total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), and triglyceride concentrations (mg/dL) were assessed by an automated colorimetric assay (BS-200, Shenzhen Mindray Bio-medical Electronics Co., Nanshan, China) using specific commercially available kits (Bioclin, Quibasa, Minas Gerais, Brazil). LDL cholesterol (LDL-C) data were calculated by the Friedewald equation as previously described [28] and validated [29]. The ratio of TC to HDL-C was also assessed [30]. Plasma insulin concentrations (sensitivity 2 $\mu\text{U/mL}$) were measured by an enzyme-linked immunosorbent assay kit as described by the supplier (Linco Research, St. Charles, MO, USA). Insulin resistance was evaluated by the homeostasis model assessment of insulin resistance calculated as fasting glucose (nmol/L) \times fasting insulin ($\mu\text{U/mL}$)/22.5 [31].

Antioxidant biomarkers

Plasma TAC was assessed by a colorimetric assay, which relies on the ability of antioxidants in the sample to inhibit the oxidation of 2,2'-Azino-di-(3-ethylbenzthiazoline) sulphonate (ABTS) to ABTS^{•+} by metmyoglobin. The amount of ABTS^{•+} produced was monitored by reading the absorbance at 750 nm. Under the reaction conditions used, the antioxidants in the sample caused suppression of absorbance at 750 nm to a degree, which is proportional to their concentration. The capacity of antioxidants in the sample to prevent ABTS oxidation was compared with that of 6-hydroxy-2,5,7,8-tetramethylchroman-2-carboxylic acid (Trolox), a water-soluble tocopherol analog, and quantified as millimolar Trolox equivalents (709001, Cayman Chemical, Ann Arbor, MI, USA). Plasma ox-LDL concentrations (sensitivity < 6.56 U/L) were determined by an enzyme-linked immunosorbent assay kit (Mercodia, Uppsala, Sweden). GPx activity (nmol/[mL/min]) was measured in erythrocytes by a commercially available kit (703102, Cayman Chemical). Uric acid and ceruloplasmin concentrations (mg/dL), were assessed by an automated colorimetric assay (BS-200, Shenzhen Mindray Bio-medical Electronics Co., Nanshan, China) using specific commercially available kits (Bioclin, Quibasa, Minas Gerais, Brazil).

Trace elements in the nails

Nail samples were collected at the time of interview and stored at room temperature in clean polypropylene bags. Finger- and toenail samples were treated with sub-boiling nitric acid in a high-pressure Teflon digestion vessel using a microwave digestion system (Ethos Plus, Milestone, Sorisole, Italy). A Perkin Elmer Analyst 800 atomic absorption spectrometer (Norwalk, CT, USA), equipped with transverse-heated graphite atomizer, Zeeman background corrector, and AS-800 autosampler, was used for measurement of selenium at 196.0 nm with a spectral band width of 2.0 nm [32]. An electrodeless discharge lamp (Perkin Elmer) was used as a light source operated at 280 mA. Pyrolytic-coated graphite tubes with end caps supplied by Perkin Elmer were used. Zinc and copper concentrations in digested acid solutions were analyzed by flame atomic absorption spectrophotometry (Perkin Elmer). Zinc and copper hollow cathode lamps (Perkin Elmer) provided resonance lines of 213.9 and 324.8 and were operated at 15 mA with a slit width set at 0.7 nm. The measured concentration values were adjusted for the sample weight and expressed as $\mu\text{g/g}$ of nail for copper and zinc and ng/g of nail for selenium.

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