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Long-term consumption of a green/roasted coffee blend positively affects glucose metabolism and insulin resistance in humans

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ABSTRACT

Protective health effects of coffee could have a widespread impact on public health considering the high intake of this beverage in industrialized countries. However, certain of coffee's health effects are contradictory such as those on type 2 diabetes mellitus (T2DM). Green coffee is richer in antioxidant phenols than roasted coffee, and thus it is likely to be a healthier option. This work evaluated the effects of long-term consumption of green coffee consumption, blended with roasted beans to improve palatability, on different glucose homeostasis markers as T2DM risk factors. A, randomized, controlled, crossover study was performed in 52 healthy men and women who consumed three servings/day of the green/roasted (35:65) coffee blend for 8 weeks during the intervention in comparison with not consuming coffee in the control stage. At the beginning and end of the coffee and control interventions, blood samples were collected, body weight measured, and dietary records and physical activity questionnaires completed. After the coffee intervention, fasting glucose levels and HOMA-IR values were significantly lower, whereas QUICKI values were higher showing improved insulin sensitivity. Fasting glucagon levels decreased, which may be associated with the increase in the glucagon-like peptide-1 (GLP-1), whereas C-peptide, glucose-dependent insulinotropic polypeptide (GIP), insulin, and HOMA-β were not affected. In conclusion, regularly consuming the green/roasted coffee blend may be recommended to prevent T2DM and reduce cardiovascular risk.

1. Introduction

The worldwide prevalence of type 2 diabetes mellitus (T2DM) is increasing globally and may reach 366 million people by 2030 (Ding, Bhupathiraju, Chen, van Dam, & Hu, 2014). T2DM is associated with variable degrees of insulin resistance, impaired insulin secretion, moderate to severe beta-cell apoptosis and increased hepatic glucose production. Unlike type 1 diabetes mellitus, the onset of T2DM is slow and the metabolic abnormalities that lead to hyperglycemia are established long before overt diabetes. Hepatic glucose production is the main contributor to fasting plasma glucose concentration and is regulated primarily by plasma insulin and glucagon concentrations (Abdul-Ghani, Williams, DeFronzo, & Stern, 2006). In turn, incretin hormones glucagon-like peptide 1 (GLP-1) and glucose-dependent insulinotropic polypeptide (GIP), which are secreted in response to food intake, contribute to the regulation of glucagon and glucose-dependent insulin secretion. Both incretins stimulate insulin secretion, although they exert opposing effects on glucagon, since GLP-1 suppresses and GIP enhances glucagon secretion (Yabe et al., 2015). The effects of the incretin hormones are very limited during fasting conditions, as circulating concentrations of GLP-1 and GIP are low (Nolan & Færch, 2012).

Many studies have shown that dietary components or foods affect postprandial glucose, glucagon, insulin and incretin hormones; however, less have examined the effects of regular consumption of dietary components on the fasting concentrations of the glucose metabolism related biomarkers. Particularly, studies looking into the effects of micronutrients and phytochemicals on glucose metabolism are scarce. Advances in understanding the anti-diabetic actions of dietary flavonoids have been recently reviewed by Babu, Liu, and Gilbert (2013). In a previous revision by van Dam (2006), consumption of coffee, rich in hydroxycinnamic acids and caffeine, was pointed to affect postprandial glucose metabolism rather than fasting glucose levels. Some human trials have shown that glucose tolerance is reduced shortly after ingestion of caffeine or caffeinated coffee, suggesting that short-term coffee consumption could increase the risk of diabetes (Olthof, van Dijk, Deacon, Heine, & van Dam, 2011). However, there is increasing scientific evidence that supports an inverse relationship between coffee consumption and T2DM (Akash, Rehman, & Chen, 2014), being stronger the association with decaffeinated coffee (Pereira, Parker, & Folsom, 2006); therefore, it has been suggested that the positive, T2DM protective effects of coffee are associated with non-caffeine compounds.

Coffee contains diterpens (cafestol and kahweol, up to 0.6% of final weight), and micronutrients, among which outstands magnesium

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(Ding et al., 2014), but it is also a rich source of polyphenols (up to 11% of the coffee bean). The main phenolic compounds in green coffee are hydroxycinnamic acids, mostly 3-, 4-, and 5-caffeoylquinic acids (3-, 4- and 5-CQA), 3,4-, 3,5-, and 4,5-dicaffeoylquinic acids (3,4-, 3,5-, and 4,5-DCQA), and 3-, 4-, and 5-feruloylquinic acids (3-, 4-, and 5-FQA), among others (Alonso-Salces, Serra, Reniero, & Héberger, 2009). Roasting drastically degrades and/or transforms green coffee polyphenols (Perrone, Farah, & Donangelo, 2012; Schenker et al., 2002; Somporn, Kamtuo, Theerakulpisut, & Siriamornpun, 2011) inducing the formation of Maillard reaction products and quinides. The intake of green coffee products has increased in recent years as a healthier option than roasted coffee (Kozuma, Tsuchiya, Kohori, Hase, & Tokimitsu, 2005), although its bitter taste limits green coffee acceptance. Bearing this in mind, a blend of green and roasted beans can be an alternative well accepted by coffee consumers with greater health potential than traditional roasted coffee.

Considering the high intake of this beverage, particularly in industrialized countries where it is the largest source of dietary antioxidants (Tunnicliffe & Shearer, 2008), the health protective effects of coffee could have a widespread impact on the population health. Moreover, coffee could be recommended to patients at risk of T2DM as a supplementary therapy in preventing the further progression of the disease or to prevent the onset in healthy and at risk adults. Therefore, the aim of this work was to evaluate the effects of long term consumption of a green/roasted (35/65) coffee blend on glucose homeostasis markers, as T2DM risk factors, in healthy adults, attempting to understand the mechanisms involved.

2. Experimental methods

2.1. Subjects

This study was conducted according to the guidelines laid down in the Declaration of Helsinki and all procedures were approved by the Clinical Research Ethics Committee of Hospital Universitario Puerta de Hierro Majadahonda in Madrid (Spain). Written informed consent was obtained from all subjects. Volunteer recruitment was carried out through placing advertisements in the Universidad Complutense campus as well as through giving short talks between lectures. The inclusion criteria were: being non-diabetic (excluded by the results of a glucose test and health questionnaire), non-vegetarian, non-smoker, non-pregnant women and men, between 18 and 55 y old, not suffering from any other chronic pathology and presenting a body mass index between 20 and 25 kg/m^2. None had taken dietary supplements, laxatives, or antibiotics six months before the start of the study.

Fifty-three subjects initially accepted to participate in the study, however 52 completed it. Baseline characteristics of the volunteers are shown in Table 1.

2.2. Study design

This was a randomized, controlled, crossover study carried out in free-living people. After a 2 week run in stage, subjects were randomly assigned to the coffee or control intervention, lasting 8 weeks each, which were separated by a 2 week washout period. During the coffee intervention, volunteers consumed three times a day the soluble green/roasted coffee blend, the first at breakfast, the second between breakfast and lunch, and the third between lunch and dinner. In the control intervention, instead of the coffee product, the study participants had water or an isotonic drink, free of sugar, polyphenols and methylxanthines. The soluble green/roasted coffee blend, which was commercialized at the time of the study, was provided by the manufacturing company in unlabelled, individual sachets containing 2 g of coffee (equivalent to two teaspoons, quantity that can reasonably be used to prepare a cup of coffee). The coffee studied contained 85.1 ± 1.6 mg/g (dry matter) of total hydroxycinnamic acids (mainly chlorogenic acid) and 20.0 ± 1.8 mg/g (dry matter) of caffeine. Therefore, volunteers daily consumed 6 g of the coffee blend which provided 510.6 and 120 mg of total hydroxycinnamic acids and caffeine, respectively. The green/roasted coffee blend was particularly interesting to study because on the one hand, it is richer in chlorogenic acid than roasted coffee and thus was expected to be healthier, and on the other hand, the blend keeps the organoleptic properties of roasted coffee (which green coffee lacks) that are much appreciated by coffee drinkers, adding to its acceptability by consumers. From the run-in stage till the end of the study, foods rich in polyphenols and methylxanthines were restricted. Hydroxycinnamic acids are abundant in a variety of fruits and vegetables, such as chard, artichoke, eggplant, broccoli, loquats, tangerines, oranges, apricot, cherries, plums, prunes, grapes, raisins, blueberries and other fruits of the forest. All these foods were constrained, as well as coffee, mate, cocoa, and tea and derived drinks. On the other hand, ferulic acid and its derivatives are the most abundant hydroxycinnamic acids found in cereals, thus whole grain products were also restricted along the study.

Table 1
Baseline characteristics of the participants in the study

	Women (n = 32)	Men (n = 20)
Age (years)	29.4 ± 9.5	29.8 ± 8.9
Body mass index (kg/m^2)	21.7 ± 2.5	24.8 ± 2.7

Data represents mean ± standard deviation of mean.

2.3. Dietary control and compliance

Subjects were asked to maintain the same dietary habits along the study. Their dietary intake was regularly evaluated to control any possible changes. Volunteers were instructed on how to fill in the dietary records before starting the study. In the run in stage and at the end of the two intervention periods, volunteers were asked to complete a 72-hour detailed food intake report, specifying the ingredients and amounts of food consumed, including serving weights (when possible) or household measurements. Compliance was controlled by counting the number of coffee sachets provided to the volunteers before and after the intervention, as well as by weekly calling the volunteers. In order to assess dietary composition, the program DIAL [Department of Nutrition and Bromathology I. School of Pharmacy. Complutense University of Madrid (UCM), Spain] was used.

2.4. Blood samples

Blood samples were drawn after 8–10 h overnight fasting at baseline and at the last day of the control and coffee intervention. Serum (without anticoagulant) and plasma (EDTA-coated tubes) were separated by centrifugation and frozen at −80 °C until analysis.

2.5. Diabetes biomarkers and related indexes

Fasting glucose was analyzed using a colorimetric kit (Sprinreact). Fasting insulin, as well as GIP, GLP-1, C-peptide, and glucagon were analyzed using the Bio-Rad Multiplex Diabetes kit on Bio-Plex MAGPIX system. Using fasting glucose and insulin data, Homeostasis Model Assessment indexes were calculated to estimate insulin resistance (HOMA-IR) and beta cell function (HOMA-β) according to the equations by Matthews et al. (1985): HOMA-IR = [Glucose (mg/dL) × Insulin (mU/L)] / 405; HOMA-β = [(Insulin (mU/L) × 360) / (Glucose (mg/dL) − 63)]. Another model to calculate beta- cell function, the insulin/glucose ratio, was also used (Meier et al., 2001). In addition, the Quantitative Insulin Sensitivity Check Index (QUICKI) was calculated according to the formula by Katz et al. (2000): QUICKI = 1 / [log Insulin (mU/L) + log Glucose (mg/dL)].