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Paper-filtered coffee increases cholesterol and inflammation biomarkers independent of roasting degree: A clinical trial

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ABSTRACT

Objective: The aim of this study was to compare the effects of medium light roast (MLR) and medium roast (MR) paper-filtered coffee on cardiovascular risk factors in healthy volunteers.
Methods: This randomized crossover trial compared the effects of consuming three or four cups (150 mL) of MLR or MR coffee per day for 4 wk in 20 healthy volunteers. Plasma lipids, lipoprotein(a) (Lp[a]), total homocysteine, and endothelial dysfunction–related inflammation biomarkers, serum glycemic biomarkers, and blood pressure were measured at baseline and after each intervention.
Results: Both roasts increased plasma total cholesterol, low-density lipoprotein-cholesterol, and soluble vascular cell adhesion molecule-1 (sVCAM-1) concentrations (10%, 12%, and 18% for MLR; 12%, 14%, and 14% for MR, respectively) ($P < 0.05$). MR also increased high-density lipoportein-cholesterol concentration by 7% ($P = 0.003$). Plasma fibrinogen concentration increased 8% after MR intake ($P = 0.01$), and soluble E-selectin increased 12% after MLR intake ($P = 0.02$). No changes were observed for Lp(a), total homocysteine, glycemic biomarkers, and blood pressure.
Conclusion: Moderate paper-filtered coffee consumption may have an undesirable effect on plasma cholesterol and inflammation biomarkers in healthy individuals regardless of its antioxidant content.

Introduction

Dyslipidemia, hypertension, diabetes or glucose intolerance, and obesity are associated with a marked increase in vascular reactive oxygen species (ROS) production, which play important roles in atherosclerosis, diabetes, and cardiovascular diseases (CVD). It should be highlighted that ROS are involved in endothelial dysfunction, monocyte migration, low-density lipoprotein-cholesterol (LDL-C) oxidation, and vascular smooth muscle cell growth. ROS induce the expression of vascular cell adhesion molecule-1 (VCAM-1) and monocyte chemoattractant protein-1 (MCP-1) on the endothelial cell surface.

Coffee is consumed as a beverage worldwide; however, its effect as a cardiovascular risk factor is still controversial [1–3]. Roasted coffee contains naturally present antioxidants and others that are formed during the roasting process [4]. Chlorogenic acids (CGA) and caffeine have been extensively studied because they may play a role in the inhibition of lipid peroxidation, free radical scavenging, metal chelation, and anti-inflammatory activity. They also may reduce the risk for development and progression of atherosclerosis [5] and insulin resistance [6,7], and they may decrease blood pressure (BP) [8,9]. Coffee contains diterpenes, cafestol, and kahweol, which have a cholesterol-raising effect. However, most of them are retained by the paper filter, which substantially reduces the cholesterol-raising effects potentially associated with coffee [10,11].

Epidemiologic studies have indicated that regular coffee consumption is associated with a lower risk for CVD [3,12,13]. However, the coffee compounds responsible for the suggestive

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protective effects are still unknown [1]. The data related to the effect of coffee on inflammation are conflicting, showing that coffee consumption by healthy individuals can be either directly [14], inversely [4], or not associated with proinflammatory biomarkers [15]. Therefore, we compared the effects of ingesting medium light roast (MLR) or medium roast (MR) filtered coffee in plasma lipids, total homocysteine (tHcy), endothelial dysfunction–related inflammation biomarkers, glycemic biomarkers, and BP in healthy volunteers.

Participants and methods

Participants

Twenty-two healthy and habitual coffee drinkers were recruited and gave informed consent. This study was approved by the School of Public Health of São Paulo University Review Board and registered as a clinical trial (ACTRN12609001064291). Eligibility criteria were age 20 y to 65 y, plasma cholesterol <6.21 mmol/L, blood glucose <5.56 mmol/L, nonsmoker or former smoker (>2 y), alcohol consumption less than one drink per day, absence of chronic diseases, and no use of regular medication. Two subjects declined participation, one could not attend the meetings, and one consumed coffee during the washout period. Twenty healthy participants (14 women) were evaluated (Fig. 1).

Study design

This randomized, crossover clinical trial lasted 9 wk. After a 1-week run-in, participants consumed MLR or MR paper-filtered coffee for 4 wk and then switched to the other roast for an additional 4 wk.

Participants were asked to make no changes in their diet or lifestyle. We questioned physical activity of the participants in the baseline interview and after each intervention period. A 3-d food diary (2 d during the week and 1 d on the weekend) was collected before baseline and during each intervention to control for possible confounding factors and check for compliance with dietary instructions.

Coffee samples and beverage preparation

Two commercially available blends (80% *Coffea arabica* L. cv. Bourbon and 20% *C. canephora* cv. Robusta) of caffeinated, roasted, ground coffee were used in the study. Both coffees were cultivated in the same geographic region. They were vacuum packed in 500-g aluminized bags. Roasting degree classification was done according to the "Roast Color Classification System" (Agtron/SCAA, Reno, NV, 1995). Coffee packages and paper filters (Classic n° 102) were provided by Melitta do Brasil Indústria e Comércio Ltda, São Paulo, Brazil. Coffee was distributed to participants in 500-g packages at the beginning of each intervention period. Subjects were instructed on how to prepare the beverage (15 g of coffee/150 mL cup) in a household coffee maker by filtering through paper, and to consume the total daily amount in three of four separate cups without a fixed schedule.

Coffee beverages were prepared as instructed to the participants for the analysis of the antioxidant content and diterpenes. CGA and caffeine were determined by high-performance liquid chromatography (HPLC) with diode-array detector (DAD) and mass spectrometer [16]. Cafestol and kahweol were determined in the unsaponified matter by HPLC with DAD [17].

Blood collection and biomarker assessment

At baseline, and at the end of each intervention, venous blood samples were taken after a 12-h overnight fast and centrifuged for plasma separation. Plasma samples were stored at −80°C for inflammation biomarker analyses.

Plasma concentrations of total cholesterol (TC), high-density lipoprotein-cholesterol (HDL-C), and triacylglycerol were measured by enzymatic assays on a Dimension RXL (Siemens Healthcare Diagnostics Deerfield, IL, USA). LDL-C was calculated according to the Friedewald formula [18]. Plasma lipoprotein(a) (Lp[a]) concentration was assessed by immunonephelometric assay on a BN II analyzer (Siemens Healthcare Diagnostics, Marburg, Germany). Plasma tHcy concentration was measured by a chemiluminescence immunoassay on an Immulite 2000 Analyzer (Diagnostic Products Corporation, Los Angeles, CA, USA).

Fasting serum glucose concentration was determined by a commercial kit (Siemens Healthcare Diagnostics, Marburg, Germany). Blood-glycated hemoglobin and frutosamine were measured by immunoturbidimetric and colorimetric assays, respectively. Fasting serum insulin concentration was determined by a chemiluminescence assay. These analyses were performed by use of an automated analyzer (Dimension RXL, Siemens Healthcare Diagnostics, Llanberis, Gwynedd, UK). Insulin resistance was estimated by Homeostasis Model Assessment for insulin resistance [19,20].

Plasma fibrinogen concentrations were measured by the Clauss method [21] on a Destiny Max (MedLab) and high-sensitivity C-reactive protein (hs-CRP) concentration by use of a high-sensitivity latex-enhanced immunonephelometric assay on BN II analyzer (Siemens Diagnostics). Interleukin (IL)-1β, IL-6, tumor necrosis factor (TNF)-α, MCP-1, soluble E-selectin (sE-selectin), sVCAM-1, soluble intercellular adhesion molecule-1 (sICAM-1), and tissue-type plasminogen activator inhibitor-1 (PAI-1) were quantified by bead-based multiplex Luminex xMAP technology assays (Millipore, Missouri, USA) on a Luminex 200 analyzer (Luminex Corporation, Austin, TX, USA).

Assessment of other measurements

Weight, height, abdominal circumference, and body fat were measured after the washout period and after each intervention. Body mass index (BMI) was calculated as weight (kg) divided by height (m^2). Body fat percentage was measured by bipolar impedance on a portable electronic scale (Plenna, São Paulo, Brazil).

BP was measured by ambulatory BP monitoring, using a 90207 portable monitor (Spacelabs Healthcare, Washington, USA), scheduled for six measurements during the day (between 0700 and 2200) and three at night (between 2200 and 0700).

Statistical analysis

The results are expressed as means ± SD. CGA, caffeine, and diterpenes content of the two coffee beverages were compared by *t* test for independent samples. Differences in biomarker concentrations were analyzed by paired *t* test (glucose biomarkers) or by repeated-measures analysis of variance (ANOVA) for comparisons of MLR coffee intake with MR and of each roast with the baseline. Variables that were not normally distributed (Lp[a], fasting glucose, diastolic BP and heart rate) were submitted to the logarithmic transformation and analyzed by repeated-measures ANOVA. Evaluation of the two groups' mean profile was carried out to measure the effects of the MLR and MR order of consumption and possible carryover effect. All analyses were performed using SPSS 18.0 (SPSS Inc., Chicago, IL, USA). A two-tailed $P < 0.05$ was considered significant.

Results

Participants

Twenty healthy individuals (49 ± 9 y, range 37–63 y) were evaluated. Table 1 shows their baseline characteristics. Most participants were women (70%) and overweight (76%).

During the first 4-wk intervention period, 45% of the participants (55.6% women) ingested MLR coffee and 55% (81.8% women) MR coffee. In the next 4-wk period, they switched to the opposite roast coffee. Coffee consumption was 482 ± 61 mL/d throughout the study. Coffee provided 334 mg of CGA per cup in MLR and 210 mg per cup in MR ($P < 0.001$) and 231 mg of caffeine per cup in MLR and 244 mg per cup in MR ($P = 0.003$). The mean concentrations of cafestol and kahweol were 5.36 mg and 0.79 mg per cup in MLR and 6.30 mg and 0.51 mg per cup in MR, respectively. MLR provided more CGA and kahweol and less caffeine, cafestol, and melanoidins ($P < 0.001$) than MR.

Self-reported diets showed that none of the participants consumed a significant amount of polyphenol-rich foods other than coffee during the study and the nutritional intake was similar before and after each intervention period for all participants ($P > 0.05$). Additionally, we found no significant differences in physical activity, body composition, and BP throughout the study (data not shown).

Cardiovascular risk biomarkers

Table 2 shows a significant increase in TC and LDL-C after intake of each coffee roast compared with the baseline. TC concentrations increased 0.46 mmol/L after MLR ($P < 0.01$) and 0.59 mmol/L after MR ($P < 0.001$). LDL-C increased 0.36 mmol/L