

Effect of green tea on postprandial antioxidant capacity, serum lipids, C-reactive protein and glucose levels in patients with coronary artery disease

Antonios E. Koutelidakis · Loukianos Rallidis · Katerina Koniari · Demosthenes Panagiotakos · Michael Komaitis · Antonis Zampelas · Maria Anastasiou-Nana · Maria Kapsokefalou

Received: 13 February 2013 / Accepted: 11 June 2013 / Published online: 23 June 2013
© Springer-Verlag Berlin Heidelberg 2013

Abstract

Purpose Cardiovascular risk factors have been identified in the postprandial state, particularly in patients with coronary artery disease (CAD). Tea consumption has been linked to cardiovascular risk reduction, but the beneficial effect of tea has not been investigated under postprandial conditions. The objective was to examine the effect of green tea on postprandial levels of plasma total antioxidant capacity (TAC), serum lipids, C-reactive protein (CRP) and glucose in patients with CAD.

Methods In a randomized controlled, parallel design with 2 arms, 43 patients with CAD were assigned to consume breakfast consisting of bread, butter and 330 ml water or tea (4.5 g green tea/330 ml, providing approximately 400 mg catechins). Blood samples were drawn immediately before and 1.5, 3 and 5 h after breakfast. TAC was measured in plasma with the ferric reducing antioxidant power of plasma and oxygen radical absorbance capacity assays. Total cholesterol, high-density lipoprotein-cholesterol (HDL-C), low-density lipoprotein-cholesterol (LDL-C), triglycerides, glucose, CRP, uric acid and pancreatic lipase levels were measured in serum.

Results Tested biomarkers did not differ between tea and water group at baseline, 1.5, 3 and 5 h ($P > 0.05$) postprandially. However, TAC increased 1.5 and 3 h after consumption of breakfast with tea ($P < 0.005$), but no change was observed after consumption of breakfast with water. Serum triglycerides levels significantly increased 3 h after breakfast with water ($P = 0.031$), but not after breakfast with tea. Serum uric acid decreased 1.5 h after breakfast with tea ($P = 0.038$). Pancreatic lipase, CRP, total cholesterol, HDL-C, LDL-C and glucose levels remained unchanged after breakfast with tea at any time point ($P > 0.05$).

Conclusions Tea consumption did not affect selected biomarkers at any postprandial time point in patients with CAD.

Keywords Coronary artery disease · Postprandial · Green tea · Total antioxidant capacity · Serum lipids · Glucose · CRP

Introduction

The postprandial state, following a meal providing lipids and carbohydrates, is characterized by an increase in plasma triglycerides and glucose and promotion of oxidative processes. Postprandial lipemia and oxidative stress are linked to atherosclerotic processes because they mobilize inflammation parameters, promote endothelial dysfunction, encourage low-density lipoprotein (LDL) oxidation and assist platelet activation [1–4]. Therefore, the study and/or management of the postprandial state is of high importance, especially in patients with cardiovascular disease due to the need of secondary prevention [5]. The effect of tea consumption on cardiovascular disease risk

A. E. Koutelidakis · M. Komaitis · A. Zampelas · M. Kapsokefalou (✉)
Unit of Human Nutrition, Department of Food Science and Human Nutrition, Agricultural University of Athens, Iera Odos 75, 11855 Athens, Greece
e-mail: kapsok@aua.gr

L. Rallidis · K. Koniari · M. Anastasiou-Nana
Second Department of Cardiology, University General Hospital, Attikon, Greece

D. Panagiotakos
Department of Nutrition and Dietetics, Harokopio University, Athens, Greece

factors has been studied in plethora of studies but not in the postprandial state.

Appropriate dietary choices may contribute to cardiovascular disease prevention. Consumption of foods and beverages rich in antioxidant compounds, such as tea, wine, fruits and vegetables, seems to provide benefits, evaluated through a variety of biomarkers that characterize oxidative reactions, endothelial function and the postprandial levels of lipids and glucose [4, 6, 7]. Tea, which is second in beverage consumption worldwide [8], may contribute to disease prevention [1, 9–14]. Acute studies show that tea consumption has favorable effects on management of oxidative stress [8, 11], particularly inhibition of lipoprotein oxidation [12] and reverse of endothelial dysfunction [14]. Chronic studies show that tea consumption may additionally lead to improvement of response of inflammation biomarkers as C-reactive protein (CRP) and P-selectin [13], and evasion of platelet aggregation [1, 13].

Limited knowledge is available on potential postprandial effects of tea following a meal rich in lipids and carbohydrates [4, 15]. Several studies support that tea polyphenols may affect lipid emulsification, hydrolysis and droplet size and consequently reduce intestinal absorption of lipids [16]. Moreover, tea polyphenols, specifically catechins, may affect lipid absorption via inhibition of gastric and pancreatic lipases, which are basic hydrolytic enzymes [6, 17]. Therefore, multifactorial and complicated interactions among various components of the meal and of tea, such as lipids and polyphenols, respectively [16], may lead to managing the postprandial state. It is imperative to further and fully investigate whether there are expected benefits from tea consumption in the postprandial state; this knowledge may be of use toward the objective of disease prevention at the primary level as well as at the secondary level when referring to groups such as coronary artery disease (CAD) patients.

The objective of this study was to examine the acute postprandial effect of a catechin enriched green tea infusion on cardiovascular biomarkers in patients with CAD.

Methods

Subjects

The study protocol was approved by the Ethics committee of “Attikon” Hospital of Athens, and the study performed in accordance with the Declaration of Helsinki. All the participants signed an informed consent form.

Forty-three patients, 39 men and 4 women, aged 45–70 years were recruited from the Department of Cardiology of “Attikon” Hospital in Athens, Greece. All patients had history of angiographically proven but

clinically stable coronary artery disease, such as myocardial infarction or stable angina or surgical revascularization more than 6 months before the enrollment. Exclusion criteria were unstable angina, arrhythmia, history of chronic illness including, liver disease and renal disease, BMI >35 and menopausal women taking hormonal medication. None of the participants had chronic inflammation; all subjects with CRP values >10 mg/l were excluded from the sampling. The patients were screened by questionnaire to exclude those who were taking antioxidant supplements, those who were smoking more than 10 cigarettes per day and those who were consuming more than 40 g alcohol per day. A food frequency questionnaire was used to record habitual consumption of tea, red wine, fruits and vegetables. Foods rich in polyphenolic compounds, namely tea, wine, coffee, apples, green leafy vegetables and chocolate, such as foods rich in fat and alcohol, were withdrawn 24 h before the experiment. Subjects were asked to abstain from any food or beverage for 12 h before the experiment.

In vitro assessment of antioxidant properties and phenolic content of tea infusion

A catechin enriched green tea (Green Linea Label, Unilever, The Netherlands) was selected for this intervention study. The tea infusion was firstly screened for antioxidant properties. Tea infusion was prepared by steeping 2 bags (4.5 g) of tea for 2 min in 330 ml boiling water. The TAC of the infusion was tested with the ferric reducing antioxidant power of plasma (FRAP) assay [18] and the oxygen radical absorbance capacity (ORAC) assay [19], while total phenolic content with the Folin–Ciocalteu assay [20].

FRAP was determined according to the method of Benzie and Strain, adapted to employ a 96-plate reader. Briefly, 20 μ l of infusion was put in each well of a 96-well plate reader, diluted with 100 μ l HCl 40 mM and the plate was read at 595 nm immediately, for the absorption at 0. Then, 20 μ l of the sample was put in each well of a 96-well plate reader, diluted with 100 μ l FRAP reagent and the plate was read at 595 nm after 30 min, for the absorption at 30 min. The plates were read in a multiwell absorbance plate reader (ELx808TM, BioTek Instruments, Inc., Vermont, USA) with temperature maintained at 37 °C. Standards were prepared from FeSO₄ in HCl 0.01 N in concentrations ranging from 100 to 1,000 μ M.

ORAC was determined according to the method of Huang [19], adapted to employ a 96 fluorescent plate reader. This method is based on the protection provided by antioxidants on the fluorescence decay of fluorescein (lag-phase) during a controlled peroxidation reaction. Briefly, 20 μ l of sample or 20 μ l of 75 mM phosphate buffer (pH 7.4) was added to 200- μ l fluorescein (8.16×10^{-5} mM) and 20 μ l of 2,2'-azo-bis (2-amidinopropane) (ABAP)

(119.4 $\mu\text{mol/ml}$) into a 96-well plate. The plate was immediately transferred to the plate reader, and the fluorescence was measured every minute for 35 min at 37 °C, by a Victor-X fluorescent plate reader spectrometer (Antisel, Greece). Final ORAC values were expressed as micromole Trolox equivalents per ml.

Total phenolics content of tea infusion was measured according to the method of Spanos and Wrolstad [20] using the Folin–Ciocalteu reagent. According to the procedure in a test tube were added 3 ml of extract, 1 ml Folin reagent and 1 ml Na_2CO_3 . After vortex, the mix was incubated for 30 min at room temperature on dark conditions. The absorbance was measured at 765 nm, using a spectrophotometer UV–vis.

All chemicals were purchased from Sigma-Aldrich (Steinheim, Germany). Double distilled, deionized water was used throughout the experiments.

Treatments

Tea was a catechin enriched green tea infusion prepared from 2 bags (4.5 g) green tea (Green tea Linea Label, Lipton, Unilever, The Netherlands) steeped for 2 min in 330 ml boiling water (called “tea” from this point on). The infusion provided 400 mg total catechins, according to the manufacturer. The white bread was prepared from 1 kg white flour, 20 g margarine, 15 g sugar, 16 g salt, 20 g yeast and 540 g water as described earlier [21]. Bread was stored at -20 °C, thawed before use and served as slices of 40 g. Butter was unsalted bars of 10 g each (President Butter, France). The content of each breakfast and infusion in selected nutrients was calculated from a food composition database [22] and information provided by the manufacturer.

Study design

It was a 2-arm, parallel, single-blind, randomized and controlled acute tea intervention study. On enrollment, the patients were randomly assigned to the tea group (23 volunteers) and the control group (20 volunteers). The randomization was carried out with the method of random numbers sequence. All patients were taking antiplatelets (mainly acetylsalicylic acid), 39 statins, 31 beta-blockers and 15 angiotensin converting enzyme inhibitors. During the day of the experiment, all patients were instructed to take their medication in the afternoon, after the end of the intervention. The patients came to the clinic at 8 am, after a 12 h fast, and consumed within 20 min a breakfast consisting of 80 g bread, 20 g butter and 330 ml of green tea infusion (tea group) or 330 ml water (control group).

Ten-ml blood was drawn just before breakfast and 1.5, 3 and 5 h after ingestion from all patients into EDTA or heparin-treated tubes, for plasma or serum separation, respectively. Plasma or serum was separated by centrifugation for 10 min at 3500xg in a clinical centrifuge immediately after blood collection. Aliquots of plasma or serum were stored at -80 °C until analysis. TAC was evaluated in plasma with the FRAP and ORAC assays as described above. Total cholesterol, HDL-cholesterol, triglycerides, glucose, uric acid, CRP and pancreatic lipase were measured in serum with an automated analyzer (COBAS INTEGRA 400 plus, Roche, Switzerland). LDL-cholesterol was calculated by the formula $\text{LDL} = \text{Total Cholesterol} - (\text{HDL} + \text{TG}/5)$.

Statistical analysis

All statistical analyses were performed by SPSS (SPSS V13.0). Results are presented as mean \pm SEM, and significance was at $P < 0,005$. Differences between patients' baseline characteristics were tested with Bonferroni post-test. Differences between plasma and serum samples at 1.5, 3 and 5 h were tested for each group with repeated measurements ANOVA and Bonferroni posttest. Differences between the two groups at any time were also tested. A mixed factor repeated measures ANOVA was used to determine differences between the treatments over time. The study had >80 % power to detect a >10 % increase in total antioxidant capacity and 10 % decrease in triglyceride levels 1.5 and 3 h after ingestion of tea. Sample calculation power analysis was performed by G.Power v3.0.10. Data were not tested for potential outliers; consequently, all data were incorporated into the analyses. The variables followed a normal distribution.

Results

Baseline characteristics

Forty-three patients completed the study, while 3 patients dropped out due to non-compliance. No differences were observed between the two groups at baseline for age, weight, height and BMI ($P > 0.05$) (Table 1). There were no differences in the baseline measures of total antioxidant capacity, uric acid, total, LDL-, HDL-cholesterol, pancreatic lipase, glucose and CRP. Analysis of the food frequency questionnaires showed that all subjects were consuming less than 4 servings per day of fruits and vegetables, less than 1 glass of red wine and less than 1 cup of tea per day (data not shown).

Table 2 shows the composition of the breakfast with tea or water.

Table 1 Patients' characteristics in each group at baseline

	Tea group	Control group
Patients	23	20
Age (years)	63.2 ± 10.5a*	62 ± 8.3a
Weight (kg)	80.7 ± 15.2a	77.7 ± 12a
Height (m)	1.69 ± 0.07a	1.67 ± 0.05a
BMI (kg/m ²)	27.9 ± 4.8a	27.8 ± 4.3a
Men	19	18
Women	4	2
Bypass	7	3
Stent	12	8
Hypercholesterolemia	14	15
Diabetes	7	7
Smoking	8	6
Heredity	7	9
Postmenopausal women	2	1

* For age, weight, height and BMI values are mean ± SD. The same letters in each row represent no significant differences at $P < 0.05$

In vitro antioxidant capacity and total phenolics of the catechin enriched green tea

The catechin enriched green tea infusion used in the study (called “tea” from this point on) had total antioxidant capacity $104.98 \pm 1.45 \mu\text{mol FeSO}_4/\text{ml}$ as measured with the FRAP assay and $6.15 \pm 0.31 \mu\text{mol Trolox/ml}$ as measured with the ORAC assay. The green tea infusion also had $246.57 \pm 6.19 \mu\text{g gallic acid/ml}$ total polyphenols, as measured with the Folin method.

Plasma total antioxidant capacity and uric acid levels

Plasma TAC did not differ significantly between the two groups at any time point ($P > 0.05$). However, in the tea group, FRAP and ORAC levels increased significantly 1.5 and 3 h after the intervention ($P = 0.005$ and $P = 0.039$, respectively, for FRAP, while $P = 0.0001$ and $P = 0.045$, respectively, for ORAC). In the control group, no

differences among the four time points were observed for both FRAP and ORAC ($P > 0.05$) (Table 3).

Uric acid levels did not differ between the two groups ($P > 0.05$). In the tea group, uric acid decreased 1.5 h after the intervention ($P = 0.038$), while no differences were observed among time points in the control group ($P = 0.21$ at 1.5 h) (Table 3).

Serum lipids, pancreatic lipase, glucose and CRP levels

Among serum lipids, no differences were observed neither between the two groups nor among the time points in each group for total, HDL- and LDL-cholesterol levels ($P > 0.05$). In the control group, triglyceride levels increased 3 h after the intervention ($P = 0.031$), while in tea group, no change was observed ($P = 0.130$). Pancreatic lipase was not different between the test groups or among time points in each group.

CRP levels were not different between the control and the treatment group or among time points in each group. Glucose levels increased at 1.5 and 3 h and decreased to the initial level at 5 h in both groups but there were no differences between groups ($P > 0.05$) (Table 4).

Discussion

The significance of the findings reported herein derives from the implication that tea may affect the postprandial oxidative stress and the rise of lipids and glucose concentration which in turn are linked to atherosclerotic procedures [4], particularly in patients with CAD [1].

The first finding was that in CAD patients, plasma TAC did not change postprandially when the tea and the water groups were compared. However, plasma TAC increased 1.5 and 3 h after breakfast with tea but not after breakfast with water. The discrepancy in the finding when comparing between or within groups may signify a weak effect of tea on antioxidant capacity of plasma which nonetheless

Table 2 Breakfast constitution in tea group (330 ml tea, 80 g bread and 20 g butter) and control group (330 ml water, 80 g bread and 20 g butter)

	Calories (kcal)	Water (g)	Carbohydrates (g)	Fat (g)	Proteins (g)	Na (g)
<i>Tea group</i>						
Tea	4	324	0.6	0	0.4	0.02
Butter	174.7	3.12		16.34	0.1	
Bread	202.4	38.41	38.97	1.93	7.45	1.1
Total	380.7	365.53	39.57	18.27	8.95	1.12
<i>Water group</i>						
Tea		330				
Butter	174.7	3.12		16.34	0.1	
Bread	202.4	38.41	38.97	1.93	7.45	1.1
Total	376.7	371.53	38.97	18.27	7.55	1.1

Table 3 Plasma total antioxidant capacity, measured with ferric reducing antioxidant power of plasma (FRAP) and oxygen radical absorbance capacity (ORAC) assays, and serum uric acid levels for the two groups in each time point

	Baseline	1.5 h	3 h	5 h
<i>FRAP**</i>				
Tea group	494.45 ± 129.27a*	562.17 ± 123.05b	569.51 ± 115.64b	512.9 ± 160.35a
Control group	539.57 ± 100.42a	552.07 ± 126.74a	554.52 ± 66.16a	536.45 ± 140.27a
<i>ORAC</i>				
Tea group	9539.7 ± 929.63a	10,212.82 ± 888.51b	10,172.7 ± 884.62b	10055.99 ± 1,066.92a
Control group	9,957.15 ± 826.17a	9,776.38 ± 884.24a	9,811.78 ± 936.03a	9,990.1 ± 994.05a
<i>Uric acid</i>				
Tea group	406.05 ± 112.9a	383.652 ± 104.22b	389.31 ± 96.92a	387.78 ± 100.38a
Control group	397.66 ± 120.05a	377.51 ± 95.45a	371.19 ± 153.79a	381.86 ± 117.55a

* Values are Mean ± SD. Different letters in rows represent statistical significant differences among time points at $P < 0.05$, ($n = 23$ for tea group, $n = 20$ for control group)

** Plasma FRAP values measured as $\mu\text{mol FeSO}_4/\text{L}$, ORAC as $\mu\text{mol Trolox/l}$ and uric acid as mmol/l

Table 4 Serum lipids (triglycerides, total-, high-density lipoprotein (HDL)-, low-density lipoprotein (LDL)-cholesterol), pancreatic lipase, glucose and C-reactive protein (CRP) levels for the two groups in each time point

	Baseline	1.5 h	3 h	5 h
<i>Triglycerides**</i>				
Tea group	2.14 ± 1.55a*	2.19 ± 1.5a	2.33 ± 1.7a	2.18 ± 1.66a
Control group	1.48 ± 0.95a	1.75 ± 0.97a	1.9 ± 1.03b	1.75 ± 1.06a
<i>Total cholesterol</i>				
Tea group	4.45 ± 1.32a	4.24 ± 0.96a	4.31 ± 1.31a	4.3 ± 1.30a
Control group	3.97 ± 1.22a	3.89 ± 1.29a	3.89 ± 1.14a	3.86 ± 1.18a
<i>HDL-C</i>				
Tea group	0.9 ± 0.28a	0.89 ± 0.27a	0.89 ± 0.27a	0.91 ± 0.28a
Control group	0.88 ± 0.2a	0.87 ± 0.21a	0.86 ± 0.2a	0.87 ± 0.19a
<i>LDL-C</i>				
Tea group	3.89 ± 1.59a	4.41 ± 3.54a	4.38 ± 3.75a	4.37 ± 3.68a
Control group	2.75 ± 1.11a	2.66 ± 1.2a	2.65 ± 1.07a	2.61 ± 1.07a
<i>Pancreatic lipase</i>				
Tea group	36.97 ± 22.38a	35.8 ± 15.53a	35.82 ± 14a	35.67 ± 12.63a
Control group	31.25 ± 8.23a	33.14 ± 9.78a	33.37 ± 9.54a	35.43 ± 12.21a
<i>Glucose</i>				
Tea group	7.04 ± 2.51a	10.46 ± 4.43b	8.56 ± 4.38b	7.09 ± 3.4a
Control group	6.49 ± 1.98a	9.3 ± 3.01b	7.86 ± 2.87b	5.98 ± 2.09a
<i>CRP</i>				
Tea group	3.56 ± 4.41a	3.78 ± 4.89a	3.99 ± 5.2a	3.98 ± 5.1a
Control group	8.86 ± 23.18a	10.22 ± 29.41a	10.7 ± 30.56a	12.59 ± 37.98a

* Values are Mean ± SD. Different letters in rows represent statistical significant differences among time points at $P < 0.05$, ($n = 23$ for tea group, $n = 20$ for control group)

** Serum lipid and glucose levels measured as mmol/l , while pancreatic lipase as U/l and CRP as mg/l

deserves further exploitation. The increase in plasma TAC following tea ingestion may be attributed to increased plasma concentration of polyphenols [18, 23]. Although many studies have shown acute increase in plasma TAC 1–2 h after black or green tea consumption [9], few studies have investigated plasma oxidative status after tea consumption with a meal [15]. Plasma TAC reflects a plethora of endogenous and exogenous, mainly dietary, antioxidants, such as thiol groups of proteins, uric acid, endogenous enzymes as glutathione peroxidase, catalase and superoxide dismutase, endogenous and dietary tocopherols,

ascorbic acid and carotenoids, reduced glutathione, bilirubin and albumin. Therefore, plasma TAC is a useful biomarker of oxidative stress suggested as crucial in CAD pathophysiology [24–29]. It must be noted, however, that it is not established yet that changes in TAC may be claimed in direct link with human health.

In the present study, we used both FRAP and ORAC assays; measurement of TAC by a single assay cannot reflect on the multiple reactions and mechanisms involved in oxidative stress because it represents only the chemical reactivity under the specific conditions of this assay [25]. A

relatively large standard deviation was observed in the TAC readings. Previous research indicates that individuals may respond in different ways to phenolic-rich foods/beverage. The genetic differences among patients affect the extent of polyphenols absorption in lumen and thus polyphenol bioavailability and final antioxidant biomarkers [24–29]. This was considered when power analysis was carried out in order to decide the number of participants in each group.

The acute rise of plasma TAC may be associated with the presence of phenolic compounds metabolites; however, there are studies which support that change in plasma TAC following food ingestion may be attributed not only to dietary antioxidants but also to uric acid [30]. Uric acid is an endogenous antioxidant which increases after food consumption [25] and may contribute up to 58 % of plasma TAC. Lottito and Frei [30] support that an increase in plasma TAC after tea consumption is mainly the result of acute rise of uric acid via methylxanthines and caffeine metabolism while tea polyphenols are converted to inactive metabolites. In the present study, uric acid concentration decreased after tea consumption, while did not change after water consumption. Others have observed stable uric acid levels in parallel with increased plasma antioxidant activity after tea consumption [9]. This observation suggests that the increase in plasma TAC may be attributed to tea components, most probably polyphenols, and not to an effect on uric acid levels.

The second finding reported herein refers to the postprandial lipidemic profile in CAD patients. Total, HDL- and LDL-cholesterol levels did not change, as was expected for such an acute intervention; however, triglyceride levels increased 3 h after breakfast with water but not after breakfast with tea. Other researchers have observed a decrease in triglyceride levels after postprandial polyphenol consumption. For example, Unno [6] showed a reduced postprandial plasma triglyceride response. They observed that triglyceride levels decreased 1, 2, 3, 4 and 6 h after consumption of a fatty meal enriched with tea catechins in comparison with the same meal without tea catechins. Esposito [2] observed a decrease in serum triglyceride levels 3 and 4 h after ingestion of a meal enriched with vegetable polyphenols, respectively. It is plausible that interaction between meal and tea components results in inhibition of lipid absorption [4]. Serum pancreatic lipase was low in all groups; this index may be elevated in subjects suffering from pancreatitis which was not the case in this study; herein, serum pancreatic lipase was measured as a reflection of luminal pancreatic lipase levels but no difference was detected at any time point. The inactivation of pancreatic lipase by polyphenols has been proposed as a potential mechanism [31], but in the present study, serum pancreatic lipase levels did not change in any group or time

point. Alternatively, the observed results may be explained by processes that link tea components to inhibition of lipid absorption; it is proposed that tea polyphenols may change the physicochemical attributes of the lipid emulsion such as micelle composition or fat particle size; also, they may affect cholesterol transfer into the enterocyte [6, 31, 32].

Although several studies support possible hypolipidemic activity of tea [33], few studies have observed a positive effect of tea on postprandial serum lipids [6, 32], while some showed no effect [1]. In the present study, the observed prevention of acute triglyceride rise after tea consumption is of value [1–4]. In patients with CAD, a postprandial increase in plasma lipids and glucose is a crucial parameter for the disease development [7].

Another finding was that glucose curves were similar between the two groups while there was no effect on inflammation parameters. Few studies have examined the effect of tea consumption on serum glucose levels [34]. Postprandial glycemia and inflammation parameters have important role on atherosclerotic mechanisms, while diets rich in antioxidant compounds may prevent the consequences of acute postprandial glucose rise or act as anti-inflammatory agents, especially after chronic consumption [7]. In the present study, these observations were not confirmed. Baseline glucose levels were higher than normal, indicative of insulin resistance which may well be expected in a CAD population. This would not make a difference to the potential effect of polyphenols in modifying the glycemic response because in both groups, glucose levels were significantly increased at 1.5 h compared to baseline. CRP levels did not differ among the groups or among the time points of each group, as was expected in an acute study. The variation in CRP measurement was extensive, which indicate that some people within the cohort had an acute inflammatory response.

The results of the study may be interpreted under the limitations of the experiment. One limitation was that this study was single blinded, and the control was water. This derives from the fact that tea is a well-recognized beverage; moreover, it is difficult to prepare a control beverage with the same taste and characteristics with tea. Another limitation derives from the diversity of the characteristics of the patients recruited. The patients in the tea and control group had some differences, yet not significant, in baseline lipid profiles and CRP, previous CAD treatments (bypass, stent fitted) and smoking habits. Another limitation was that all patients were taking their medication at the time of recruitment and were not instructed to stop the medication for entering the study, according to ethical procedures in medical research; however, they did not take their medication during the experimental day. Furthermore, the analyses performed without adjusting for potential confounder's factors (age, sex, previous dietary habits).

Finally, it is noticeable the large variation of some indices which may affected the lack of significance in the results.

The main result of the study was that postprandial tea consumption did not lead to significant difference in any biomarker studied at any time point (1.5, 3, 5 h), in comparison with water consumption ($P > 0.05$). After analysis for determination of differences in the tea and water groups separately, plasma total antioxidant capacity significantly increased 1.5 and 3 h after tea consumption and serum triglycerides significantly decreased 3 h after tea consumption, in comparison with control. These biomarkers are relevant to postprandial lipemia and oxidative stress and are linked to cardiovascular disease. However, further research is needed for understanding the possible effect of tea components on postprandial state.

Conflict of interest On behalf of all authors, the corresponding author states that there is no conflict of interest.

References

- Hodgson J, Puddey I, Burke V, Beilin L, Mori T, Chan S (2001) Acute effects of ingestion of black tea on postprandial platelet aggregation in human subjects. *Br J Nutr* 87:141–145
- Esposito K, Nappo F, Giugliano F, Marfella R, Giugliano D (2003) Effect of dietary antioxidants on postprandial endothelial dysfunction induced by a high-fat meal in healthy subjects. *Am J Clin Nutr* 77:139–143
- Hodgson J, Burke V, Puddey I (2005) Acute effects of tea on fasting and postprandial vascular function and blood pressure in humans. *J Hypertens* 23:47–54
- Sies H, Stahl W, Sevanian A (2005) Nutritional, dietary and postprandial oxidative stress. *J Nutr* 135:969–972
- Shankar V, Kaur H, Dahiya K, Gupta MS (2008) Comparison of fasting and postprandial lipid profile in patients of coronary heart disease. *BHJ* 50(3):445–449
- Unno T, Tago M, Suzuki Y, Nozawa A, Sagesaka M, Kakuda T et al (2005) Effect of tea catechins on postprandial plasma lipid responses in human subjects. *Brit J Nutr* 93:543–547
- O’Keefe HJ, Gheewala M, O’Keefe J (2008) Dietary strategies for improving post-prandial glucose, lipids, inflammation, and cardiovascular health. *J Am Coll Cardiol* 51:249–255
- Cabrera A, Artacho R, Jimenez R (2006) Beneficial effects of green tea—a review. *J Am Coll Nutr* 6:79–99
- Rietveld A, Wiseman S (2003) Antioxidant effects of tea: evidence from human clinical trials. *J Nutr* 133:3285S–3292S
- Mukamal KJ, McDermott MA, Vinson JA, Oyaman N, Manning WJ, Mittleman MA (2007) A 6-month randomized pilot study of black tea and cardiovascular risk factors. *Am Heart J* 154:1–6
- Hirano-Ohmori R, Takahashi R, Momiyama Y, Taniguchi H, Yomemura A, Tamai S et al (2005) Green tea consumption and serum malondialdehyde-modified LDL concentrations in healthy subjects. *J Am Coll Nutr* 24(5):342–346
- Hodgson JM, Puddey IB, Croft KD, Burke V, Burke V, Mori TA, Caccetta RA et al (2000) Acute effects of ingestion of black and green tea on lipoprotein oxidation. *Am J Clin Nutr* 71:1103–1107
- Steptoe A, Gibson EL, Vuononvirta R, Hamer M, Wardle J, Rycroft JA et al (2007) The effects of chronic tea intake on platelet activation and inflammation: a double-blind placebo controlled trial. *Atherosclerosis* 193:277–282
- Widlansky ME, Hamburg NM, Anter E, Holbrook M, Kahn DF, Elliot M et al (2007) Acute EGCG supplementation reverses endothelial dysfunction in patients with coronary artery disease. *J Am Coll Nutr* 6(2):95–102
- Young JF, Dragsted LO, Haraldsdottir J, Daneshvar B, Kall MA, Loft S et al (2007) Green tea extract only affects markers of oxidative status postprandially: lasting antioxidant effect of flavonoid-free diet. *Brit J Nutr* 87:343–355
- Jochman N, Bauman G, Stangl V (2008) Green tea and cardiovascular disease: from molecular targets towards human health. *Curr Opin Clin Nutr Metab Care* 11:758–765
- Frayn KN (1998) Dietary factors and postprandial lipaemia. *Brit J Nutr* 80:409–410
- Koutelidakis AE, Argyri K, Serafini M, Proestos C, Komaitis M, Pecorari M et al (2009) Green tea, white tea and Pelargonium purpureum increase the antioxidant capacity of plasma and some organs of mice. *Nutrition* 25:453–458
- Huang D, Ou B, Hampsch-Woodill M, Flanagan JA, Prior RL (2002) High-throughput assay of oxygen radical absorbance capacity (ORAC) using a multichannel liquid handling system coupled with a microplate fluorescence reader in 96-well format. *J Agric Food Chem* 50:4437–4444
- Spanos GA, Wrolstad RE (1990) Influence of variety, maturity, processing and storage on the phenol composition of pear juice. *J Agri Food Chem* 38:817–824
- Mantala I, Polaki A, Yanniotis S (2009) Influence of frozen storage on bread enriched with different ingredients. *J Food Eng* 92(2):137–145
- Trichopoulou A, Vasilopoulou A (2004) Food composition databases in Greece, 3rd edn. Parisianos edition, Greece
- Scalbert A, Morand C, Manach C, Remesy C (2002) Absorption and metabolism of polyphenols in the gut and impact on health. *Biomed Pharmacother* 56:276–282
- Ghiselli A, Serafini M, Natella F, Scaccini C (2000) Total antioxidant capacity as a tool to assess redox status: critical view and experimental data. *Free Radic Biol Med* 29(11):1106–1114
- Niki E (2010) Assessment of antioxidant capacity in vitro and in vivo. *Free Radic Biol Med* 49(4):503–515
- Sies H (2007) Total antioxidant capacity: appraisal of a concept. *J Nutr* 137(6):1493–1495
- Prior RL (2000) Plasma antioxidant measurements. *J Nutr* 134:3184–3185
- Sialvera TE, Koutelidakis AE, Richter DJ, Yfanti G, Kapsoképhalou M, Pounis GD, Goumas G, Diamandopoulos E, Zampelas A (2012) Phytosterol supplementation does not affect plasma antioxidant capacity in patients with metabolic syndrome. *Int J Food Sci Nutr* 64(1):21–27
- Karabela D, Koutelidakis AE, Proestos C, Komaitis M, Kapsoképhalou M (2011) Ingesting iron together with white tea (*Camellia Sinensis*) may decrease its antioxidant capacity and phenolic content in human plasma. *Trace Elem Electrolytes* 29(1):16–21
- Lottito S, Frei B (2006) Consumption of flavonoid-rich foods and increased plasma antioxidant capacity in humans: cause, consequence, or epiphenomenon? *Free Radic Biol Med* 41:1727–1746
- Koo S, Noh S (2007) Green tea as inhibitor of the intestinal absorption of lipids. Potential mechanism for its lipid-lower effects. *J Nutr Biochem* 18:179–183
- Fujita H, Yamagani T (2008) Extract of black tea (Pu-Ehr) inhibits postprandial rise in serum cholesterol in mice, and with long term use reduces serum cholesterol and low density lipoprotein levels and renal fat weight in rats. *Phytother Res* 22:1275–1281

33. Shirai N, Suzuki H (2008) Effects of simultaneous intakes of fish oil and green tea extracts on plasma, glucose, insulin, C-peptide and adiponectin and on liver lipid concentrations in mice fed low- and high-fat diets. *Ann Nutr Metab* 52:241–249
34. Bryans JA, Judd PA, Ellis PR (2007) The effect of consumption instant black tea on postprandial plasma glucose and insulin concentrations in healthy humans. *J Am Coll Nutr* 26(5):471–477