Original Article

Acute Effect of Coconut Oil on Peak Forearm Blood Flow in Healthy Men: A Randomized Crossover Trial

Kamin Naothaworn MSc[1], Onanong Kulaputana MD, PhD[1,2,4], Wilai Anomasiri PhD[1,3]

[1] Medical Sciences Program, Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand

[2] Department of Physiology, Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand

[3] Department of Biochemistry, Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand

[4] King Chulalongkorn Memorial Hospital, Bangkok, Thailand

Background: A high-fat meal can induce vascular dysfunction. Despite containing a high amount of saturated fats, coconut oil is claimed to have cardiovascular health benefits. However, the information regarding the acute effect of coconut oil on vascular function in humans is unknown.

Objective: To determine the effects of coconut oil ingestion experiment (Coco) on peak forearm blood flow (FBF_{peak}) and plasma biomarkers in healthy subjects.

Materials and Methods: Seventeen healthy young men completed two separate experimental visits, Coco and control experiment (Con) in random order. The outcomes were FBF_{peak} measured by venous occlusion plethysmography and biomarkers as plasma triglycerides, free fatty acids, and malondialdehyde. The outcomes were collected at baseline (12 hour fasting), 2-hour and 4-hour after Coco (45 mL) in the Coco visit and at the same timeline in the control visit. Statistical analyses were performed to compare the data between the two experimental groups and within the group.

Results: FBF_{peak} at 4-hour was significantly increased from the baseline (24.2±4.7 versus 21.7±3.8 mL/100 mL tissue.minute, p=0.009). Plasma triglycerides at 2-hour (75±25 mg/dL, p=0.03) and 4-hour (72±22 mg/dL, p=0.039) were significantly increased from the baseline (65±20 mg/dL). Coco significantly increased plasma free fatty acids at 2-hour (125.1±60.3 μEq/L, p=0.042) and at 4-hour (166.9±35.3 μEq/L, p<0.001) compared to the baseline (87.2±34.0 μEq/L). There were no significant changes in vascular resistance and plasma malondialdehyde.

Conclusion: Coconut oil augmented vascular function in healthy young men by increasing FBF_{peak} despite the accompanying postprandial elevations of plasma triglycerides and free fatty acids.

Keywords: Virgin coconut oil, Peak forearm blood flow, Vascular function, Saturated fatty acid, Medium chain triglyceride

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Vascular homeostasis is maintained by a balance between vasodilation and vasoconstriction[1]. Disturbance of vascular tone regulation, particularly impaired vasodilation, facilitates the processes of atherosclerosis[1]. Reactive hyperemia is an elevation of blood flow following a transient arterial occlusion and an indicator of vasodilating capacity of blood vessels[2]. Tissue ischemia after transient arterial occlusion induces vasoactive substances production, resulting in an increased blood flow during post-occlusion vasodilation[2]. Venous occlusion plethysmography is a well-accepted non-invasive technique for blood flow measurement. The blood flow during post-occlusion vasodilation is used to assess the vasodilating capacity. Reactive hyperemia of the forearm vascular system has been related to the mortality of renal failure[3] and is able to predict endothelial dysfunction associated with heart failure[4].

Coconut oil has long been used in traditional cuisines in many tropical nations, including India, Malaysia, Indonesia, Philippines, and Thailand. Recently, coconut oil has gained popularity worldwide from being heavily promoted for health benefits. There are many anecdotes and claims in the commercial literature and websites that coconut oil is a very good dietary supplement with health benefits, including aiding weight loss, protection against Alzheimer's disease, diabetes, and cardiovascular disease[5,6]. The quality of studies on the effects of coconut oil

Correspondence to:

Kulaputana O.

Department of Physiology, Faculty of Medicine, Chulalongkorn University and King Chulalongkorn Memorial Hospital, Bangkok 10330, Thailand.

Phone: +66-2-2564267, +66-89-6410331

Email: onanong.k@chula.ac.th

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in humans is limited by confounding factors such as eating out, duration of oil consumption, and physical exercise as well as the fact that this oil is well blended into local diets.

Coconut oil consists of mostly saturated fatty acids and more than 50% of its fat content is medium chain saturated fat[7]. As a result, coconut oil is usually classified as a source of saturated fat similar to palm oil, animal fats, and butter. Recommendation of saturated fats intake is less than 10% of total calories per day[8,9]. Unlike other long-chain saturated fats, coconut oil has different physiological properties on human health[10]. Many attempts have been made to link coconut oil to medium-chain triglycerides. Research on commercially manufactured medium-chain triglycerides may not be applicable to coconut oil because the differences of structure, absorption, and metabolism of triglycerides components between the manufactured oils and coconut oil[10].

High fat meals could induce vascular dysfunction[11], resulting in impaired vasodilation determined by flow mediated dilation[12,13]. The vasodilation impairment associated with a high fat composition diet has been reported in various populations, including coronary artery patients[12] and healthy individuals[12,13]. A decrease in vasodilating function simultaneously occurs with postprandial elevation of triglycerides in the blood circulation[12,13]. Evidence regarding the effects of coconut oil is unclear, particularly on the acute changes of vasodilation function. In addition, it is unknown whether coconut oil ingestion (Coco) could produce detrimental vascular effects similar to high fat meals in previous reports. The objective of the present study was to determine the postprandial effects of a single Coco on peak forearm blood flow (FBF_{peak}) and plasma biomarkers in healthy young people. The authors hypothesized that coconut oil would impair postprandial FBF_{peak} and increase plasma triglycerides and fatty acids in healthy young men.

Materials and Methods

Subjects

The sample size was calculated from data of the authors' pilot study by using the G-power program. The FBF_{peak} was used in the calculation as an outcome variable with the effect size of 0.725, alpha error of 0.05 and 80% power. The appropriate number of subjects required for the present study was at least 17.

Participants in the present study were young men aged between 19 to 25 years old. All subjects were screened according to the inclusion and exclusion criteria. Inclusion criteria were being healthy and physically inactive. Being healthy in the present study was defined as non-smoking, normotensive with a blood pressure below 120 over 80 mmHg, normal resting electrocardiogram (EKG), normal body mass index (BMI) below 23 kg/m^2, normolipidemic with a fasting triglycerides of less than 150 mg/dL, cholesterol of less than 200 mg/dL, and a low-density lipoprotein (LDL) cholesterol of less than 130 mg/dL, and non-diabetic with a fasting blood glucose of less than 100 mg/dL. Physically inactive was defined as engaging in sports or regular physical exercise at moderate intensity such as jogging, cycling, or recreational sports of no longer than 30 minutes per session, less than three sessions per week for the past three months[14]. Exclusion criteria were open wound on upper extremities, musculoskeletal or nervous system abnormalities, unable to ingest coconut oil, and taking antioxidants or vitamins supplements within one week prior to the experiments. Subjects in the present study were volunteers recruited from the Chulalongkorn University and surrounding community. All participants were part of a larger study designed to investigate the effects of coconut oil and exercise on the vascular function in individuals who were at risk of hypertension. A written informed consent was obtained from every participant.

Study design and experimental protocol

The study was carried out in accordance with the Declaration of Helsinki (2000) guidelines. Written informed consent was obtained from all participants. All procedures were approved by the Ethics Committee of the Faculty of Medicine, Chulalongkorn University (COA No. 040/2017).

On a separate occasion prior to the experimental visits, all participants were screened and tested for baseline characteristics, blood sugar concentration, and lipid profile. The experimental visits included Coco visit and control (Con) visit (Figure 1). Each participant completed two separate visits with the washout period between the visits of five to seven days. The order of the visits was randomly assigned to each participant, for example the Coco as the first visit and the Con as the second visit or vice versa. All participants were instructed to refrain from high fat composition diets with no more than 1 g fat per 1 kg body weight, within 24 hours, any moderate to vigorous physical exercise within 24 hours, alcohol consumption within 24 hours, and caffeine consumption within 8 to 12 hours prior to each

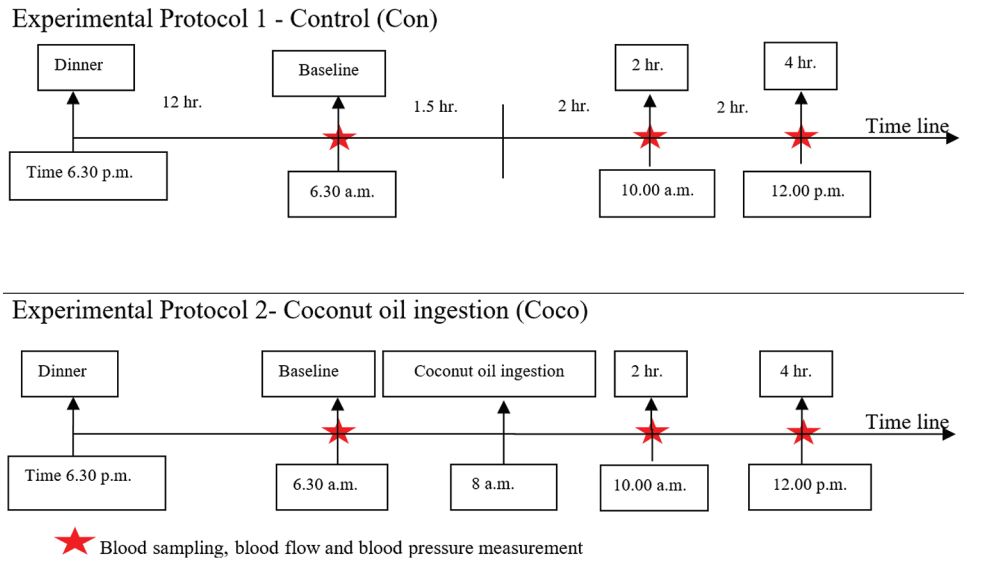

Figure 1. Experimental protocols.

experimental visit. The participants were required to fast for 12 hours with water ad libitum.

On each visit, the measurements of resting forearm blood flow (FBF_{rest}), FBF_{peak}, blood pressure, and blood sample for biomarkers were obtained at baseline, at 2-hour, and at 4-hour after Coco. On the Coco visit, the participants were asked to drink 45 mL, which is three tablespoons of coconut oil after the baseline data were obtained while the participants on the control visit continued fasting throughout the experiment. To aid the drinking, the coconut oil was mixed with 150 mL water immediately prior to ingestion. All participants finished drinking the mixture within 15 minutes. The total energy from coconut oil was approximately 360 kcal.

Product used in the study

Coconut oil: Virgin coconut oil (VCO) extracted by GMP certified cold pressed method was purchased from a local supermarket. The nutrition facts, Thai Food and Drug Administration (FDA) number, lot number, expiration date and quantity of product were specified on the package. The fat composition of the VCO was analyzed by an accredited laboratory complying with ISO/IEC 17025 (Table 1). The 45 mL amount of coconut oil was indicated as the recommended dose on the coconut oil product label, which was approved by the Thai FDA. The 45 mL coconut oil had been tested in the authors' pilot subjects and shown to be tolerable without adverse effects.

Table 1. Major fatty acid composition of coconut oil

Composition	Results (%)
Saturated fatty acids	92.73
Caproic acid (6:0)	0.68
Caprylic acid (8:0)	8.32
Capric acid (10:0)	6.51
Lauric acid (12:0)	47.51
Myristic acid (14:0)	18.13
Palmitic acid (16:0)	8.60
Stearic acid (18:0)	2.77
Lignoceric acid (24:0)	0.03
Monounsaturated fatty acids	5.90
Oleic acid (18:1)	5.69
Others	1.37

Measurements

Forearm blood flow (FBF): The participants rested quietly in a supine position for 10 to 15 minutes in a controlled temperature (25°C) room. The forearm blood flow was measured on the non-dominant arm using automatic venous occlusion strain gauge plethysmography (EC6, D.E. Hokanson Inc., USA). The blood flow data were collected via NIVP3 software (D.E. Hokanson Inc., USA). The measurement was performed according to the protocol described in a previous study[15]. Briefly, a mercury-in-silastic strain gauge was placed around the widest part at the proximal area of the forearm. To measure FBF, the venous occlusion cuff was inflated above systemic

venous pressure (approximately 50 mmHg) using an automatic rapid cuff inflator for 10 seconds followed by a release of cuff pressure for five seconds. FBF_{rest} was measured during quiet rest just prior to transient ischemia induction for each measurement of reactive hyperemic blood flow. A transient ischemia was induced by inflating an arterial occlusion cuff placed on the upper arm to 200 mmHg for five minutes. During all blood flow measurements, an arterial cuff was placed on the wrist and inflated to a suprasystolic level (200 mmHg) to exclude hand circulation. FBF_{peak} during reactive hyperemia was defined as the highest blood flow occurring within the first couple cardiac cycles immediately after releasing the ischemic cuff. All values of FBF were reported in milliliters per minute per 100 milliliters of forearm volume (mL/100 mL tissue.minute).

Blood pressure and vascular resistance: Blood pressure was measured on the contralateral arm during quiet rest before transient ischemia and at the end of ischemia using an automatic blood pressure measurement cuff (BSM 6000 series, Nikon Kohden). Mean arterial pressure (MAP) was calculated using the values of systolic and diastolic blood pressure. Vascular resistance (R) of the forearm was calculated as MAP divided by the corresponding FBF and reported as arbitrary units (u).

Blood collection and biochemistry analyses: A 5 mL blood sample was collected according to the protocol for the biomarkers, which were plasma triglycerides, free fatty acids (FFA), and malondialdehyde. Plasma triglyceride level was determined by colorimetry technique using the Architect C system (Abbott Diagnosis) in the accredited clinical laboratory of King Chulalongkorn Memorial Hospital. FFA concentration was determined by NEFA C quantification kit (Wako, Osaka, Japan) using colorimetric absorbance at 550 nm. Concentration of serum malondialdehyde was measured by the thiobarbituric acid-reactive-substances reaction and the colorimetric absorbance was performed at 532 nm.

Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics software, version 22.0 (IBM Corp., Armonk, NY, USA). Data were presented as means ± standard deviation unless stated otherwise. Repeated measures analysis of variance (ANOVA) was used to assess the effects of coconut oil on the outcome parameters. Pairwise comparisons using least significant difference (LSD) method was employed to determine the differences between the two experiments and between the different times within each experiment. A p-value of less than 0.05 was regarded as statistically significant.

Table 2. Characteristics of the participants (n=17)

Characteristics	Mean±SD
Age (year)	21.5±1.7
Weight (kg)	61 ±8
Height (cm)	173.2±6
BMI (kg/m^2)	20±2
Resting SBP (mmHg)	111±9
Resting DBP (mmHg)	63±10
MAP (mmHg)	95±9
FBG (mg/dL)	86±4
Triglyceride (mg/dL)	60±16
HDL-C (mg/dL)	48±12
LDL-C (mg/dL)	98±20
Cholesterol (mg/dL)	156±24

BMI=body mass index; SBP=systolic blood pressure; DBP=diastolic blood pressure; MAP=mean arterial pressure; FBG=fasting blood glucose; HDL-C=high-density lipoprotein cholesterol; LDL-C=low-density lipoprotein cholesterol; SD=standard deviation

Results

Table 2 describes the characteristics of the 17 participants. Their baseline characteristics satisfied the eligibility criteria.

Forearm blood flow

Reactive hyperemic blood flow or FBF_{peak} of the Coco visit at 4-hour was significantly increased from the baseline (24.2±4.7 versus 21.7±3.8 mL/100 mL tissue.minute, p=0.009) and higher than the FBF_{peak} at 2-hour (24.2±4.7 versus 21.9±3.7 mL/100 mL tissue.minute, p=0.007). The FBF_{peak} at 2-hour did not change from the baseline (21.9±3.7 versus 21.7±3.8 mL/100 mL tissue.minute, p=0.78). The FBF_{peak} of the control experiment (Con) did not change at the 4-hour course. The FBF_{peak} values were not different between the two experiments at the baseline (p=0.13), 2-hour (p=0.48), and 4-hour (p=0.14). However, the percentage change from the baseline of the FBF_{peak} at 4-hour in the Coco was greater than the Con (112.2% versus 97.4%, p=0.023) (Figure 2A).

There was no difference in the FBF_{rest} at the baseline between the two experimental visits (Con 2.4±0.6 versus Coco 2.5±0.5 mL/100 mL tissue. minute, p=0.59). In both experimental visits, the FBF_{rest} measured at 2-hour and at 4-hour did not change from the baseline.

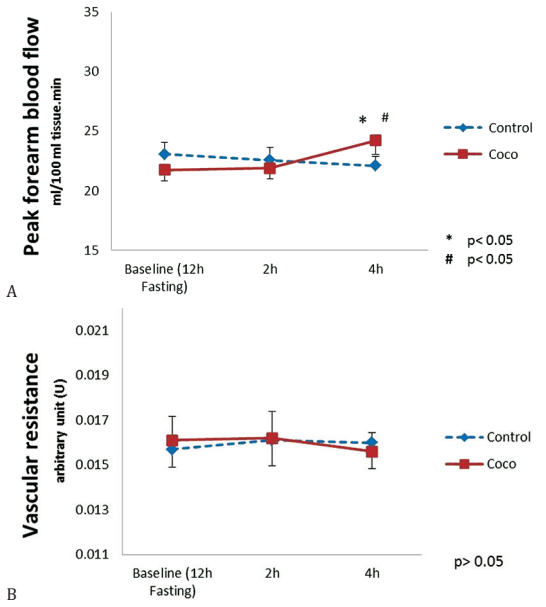

Figure 2. Effect of coconut oil ingestion on A) peak forearm blood flow (n=17), values presented as mean and SEM (* p<0.05 versus baseline in the same experiment, # p<0.05 versus 2 hours); and B) vascular resistance (n=17), values presented as mean and SEM.

Coco=coconut oil ingestion experiment; Control=no coconut oil ingestion experiment

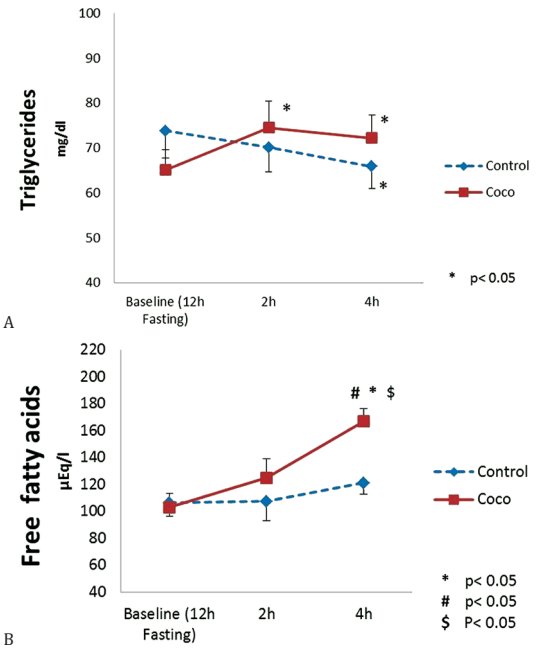

Figure 3. Effect of coconut oil on A) triglyceride levels (n=17), values presented as mean and SEM (* p<0.05 vs. baseline in the same experiment) and B) free fatty acids (n=17), values presented as mean and SEM (* p<0.05 versus baseline in the same experiment, # p<0.05 versus 2 hours, $ p<0.05 versus control at 4 hours).

Coco=coconut oil ingestion experiment; Control=no coconut oil ingestion experiment

Blood pressure and forearm vascular resistance

Resting blood pressure measured at the baseline was similar between the two experimental visits. There were no differences in blood pressure between the baseline and after Coco, at 2-hour and at 4-hour. Forearm vascular resistance remained constant throughout the Con. The forearm vascular resistance tended to decrease at 4-hours after Coco, although not statistically significant (p=0.43). There were no differences in the forearm vascular resistance between the two experiments, at the baseline (p=0.58), 2-hour (p=0.98), and 4-hour (p=0.74). There were no differences in percentage change of forearm vascular resistance between the two experiments at 2-hour (p=0.85) and 4-hour (p=0.65) (Figure 2B).

Triglycerides, free fatty acids, and malondialdehyde

Figure 3A showed that the plasma triglycerides at 4-hour of the control visit decreased from the baseline (p=0.024). The plasma triglycerides levels of the Coco visit at 2-hour (75±25 mg/dL, p=0.03) and 4-hour (72±22 mg/dL, p=0.039) were significantly increased from the baseline (65±20 mg/dL). When compared between the two experiments, the values of plasma triglycerides were not different, at the baseline (p=0.17), 2-hour (p=0.48), and 4-hour (p=0.27). However, the percentage change from the baseline of plasma triglycerides in the Coco visit was greater than control at 2-hour (116.4% versus 95.7%, p=0.003) and 4-hour (113.9% versus 90.9%, p=0.001).

FFA concentration did not change during the Con (Figure 3B). The FFA levels of the Coco visit at 2-hour (125.1±60.3 µEq/L, p=0.042) and 4-hour (166.9±35.3 µEq/L, p<0.001) were significantly elevated from the baseline (87.2±34.0 µEq/L). The value of FFA at 4-hour of the Coco was greater than control (166.93±35.3 versus 121.16±38 µEq/L, p=0.003). The percentage change from the baseline of FFA in Coco was greater than control at 4-hour (93.91% versus 30.83%, p=0.05). Plasma malondialdehyde did not change in either experiment (Figure 4).

Discussion

In the present study, the acute effect of Coco on peak vasodilation of the forearm was investigated in healthy young men. Only male participants were recruited in the present study to avoid the confounding effects of the female hormones on vascular function[16].

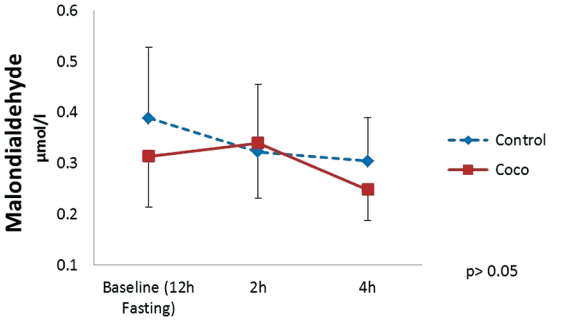

Figure 4. Effect of coconut oil on malondialdehyde (n=17), values presented as mean and SEM.

Coco=coconut oil ingestion experiment; Control=no coconut oil ingestion experiment

The results revealed that FBF_{peak} during reactive hyperemia was increased after a single dose of Coco and concurrent with the rising of blood triglycerides and FFA levels. The increased peak blood flow did not support the hypothesis that coconut oil would impair forearm vasodilation. This is the first study that demonstrates the effects of coconut oil on human vascular reactivity. The results suggest that coconut oil may have a distinctive influence on human vascular control despite its high saturated fat content.

Many stimulations such as vasoactive substances, exercise, and transient ischemia have been used to study vasodilatory function of various vascular beds[17,18]. Reactive hyperemia, which is an increased blood flow in the area of prior transient ischemia, was used as an indicator of dilating capacity of vasculature in the present study. In the control visit, both FBF_{rest} and peak reactive hyperemic blood flow did not change over the four hours course of study. In addition, both resting blood flow and peak blood flow measured at the baseline were similar between the two experimental visits. These findings indicate that the alteration of peak blood flow in response to transient ischemia observed in the Coco visit was due to the coconut oil intervention. Furthermore, coconut oil did not have an impact on the FBF_{rest}.

An increased blood flow after transient ischemia is associated with a myogenic mechanism and the accumulation of endothelial derived vasodilator substances such as nitric oxide, adenosine, prostaglandins, and endothelial hyperpolarizing factors[19,20]. How coconut oil alters FBF_{peak} was not resolved by the current study. Evidence in healthy individuals[12,21] and patients with coronary artery diseases[12] showed that a significant decrease in flow-mediated dilation, which reflects the function of endothelium, occurred two to four hours after a high fat meal and the phenomenon was linked to the elevated plasma levels of triglycerides[12,21]. The results of the present study, however, revealed an enhanced FBF_{peak} at 4-hour after Coco despite an increase in plasma triglycerides. Different methods of blood flow measurement that reflect different vascular properties may account for this discrepancy. Other possible explanations for this discrepancy may be due to the differences in types of diet and the amount of calorie intake. A previous study[12] used a mixed meal with 980 kcal while the present study used VCO with only 360 kcal. A high caloric meal could cause more oxidative stress than a low caloric meal[22]. Oxidative stress can attenuate the endothelial derived vasodilator substances[17]. Impaired endothelial dependent vasodilation as a result of oxidative stress associated with postprandial lipemia has been reported[23,24]. Therefore, the much lower calories used in the present study was likely insufficient to cause significant oxidative stress to induce an impairment of vasodilation.

The VCO was used in the present study because of its distinctive properties. It is richer in bioactive compounds, such as vitamin E, sterols, and polyphenols, which have antioxidant properties[7,10,25-27]. In vitro studies demonstrated that the polyphenol fraction of VCO can prevent LDL oxidation and reduced carbonyl formation[7,26]. Hence, the effects of coconut oil on enhancing vasodilation capacity may be due to the reduction of oxidative stress, which leads to a decrease in the degradation of vasoactive substances, such as nitric oxide[7,26]. Malondialdehyde, a product of lipid peroxidation was assessed to evaluate whether the oxidative stress status was altered by Coco. There was no significant change in malondialdehyde with Coco. It could be interpreted that coconut oil did not cause an alteration in oxidative stress or that malondialdehyde was not a proper blood biomarker for oxidative stress in the research setting. Previous studies[28] reported that malondialdehyde has insufficient sensitivity and specificity due to its reaction with highly reactive carbonyl groups of the blood contain compounds such as sugar, amino acid, and albumin[29]. Furthermore, both visible and invisible hemolysis could contribute to bias in the interpretation of malondialdehyde analysis[28]. In addition, the present study did not measure antioxidant status. Therefore, the effect on oxidant-antioxidant balance with a single coconut oil consumption could not be evaluated.

Elevations of FFA can impair endothelial

dependent vasodilation[30]. In contrast, the results of the present study demonstrated an enhanced FBF_{peak} despite the increased plasma FFA following Coco. This finding suggests that the increased plasma FFA may not always cause impaired vasodilation. The types of circulating fatty acids may be responsible for this discrepancy. In agreement with this assumption, Steer et al reported that long chain fatty acids and medium chain fatty acids had different impact on forearm blood flow in healthy humans[31]. In addition, saturation of fatty acids also determined the biological effects. Another study in cultured endothelial cells demonstrated that oleic acid, a long chain unsaturated fatty acid, reduced the activity of nitric oxide synthase[32]. This enzyme is responsible for nitric oxide production. However, steric acid, another saturated fatty acid with equal chain length did not have the same effect[32]. Moreover, an in vitro experiment showed that oleic acid impaired vasorelaxation of the rabbit femoral artery[32]. Although the types of FFA were not determined in the present study, coconut oil contains 92% of saturated fatty acids of which greater than 50% consists of medium chain fatty acids (C8-C14)[7]. Since medium chain fatty acids have a faster metabolic conversion along with other biological properties[10,31], it may be that the vascular effects on human blood vessels of coconut oil with high medium chain fats are different than other oils such as animal oil or palm oil that contain high amounts of long chain fatty acids. Further investigation on the effects of coconut oil on vascular reactivity in humans may be helpful, either the study on whole coconut oil or its major fatty acid components, particularly lauric acid.

The present study had some limitations. First, since antioxidant markers were not measured, the relationships between the role of oxidant and antioxidant balance and the vascular function influenced by Coco could not be determined. Second, the authors determined the effects of coconut oil in an acute phase, hence, the longer-term effects were not evaluated. Finally, the subjects in the present study were healthy young men, therefore, the results may be limited when they are applied to other groups.

Conclusion

Coconut oil appeared to have unique effects on vascular function. Postprandial FBF_{peak} after Coco was increased in healthy young men. However, the mechanism mediating such vascular enhancement has yet to be determined. Longer term investigations to establish the role of VCO in improving vascular control are needed. In the meantime, coconut oil should be regarded as other saturated fats that should be consumed in a limited amount, not more than 10% of total calories intake.

What is already known on this topic?

High fat meals and postprandial lipemia can cause impaired vascular function.

What this study adds?

Coconut oil enhances peak vasodilatory response despite a postprandial increase in triglycerides and free fatty acids in healthy men.

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Conflicts of interest

The present study was initiated by the investigators. Data collection, analyses, and interpretation were performed by the investigators without external interference. The authors declare no conflict of interest.

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