



Palmolein and olive oil consumed within a high protein test meal have similar effects on postprandial endothelial function in overweight and obese men: A randomized controlled trial



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ABSTRACT

Objective: This study assessed the postprandial effects of high fat, high protein meals containing either palmolein or olive oil on endothelial function in overweight/obese men.

Design: 28 men (32–65 yr; 25–35 kg/m²) consumed, in random order 1 wk apart, isocaloric high protein, high fat meals (2791 kJ, 40 g protein (~3 g L-arginine), 44 g fat, 21 g carbohydrate) prepared with either 40 g palmolein or 40 g olive oil after an overnight fast. The SFA:MUFA:PUFA ratio of the oils were: palmolein, 42:47:12; olive oil, 17:76:7. Brachial artery flow-mediated dilatation (FMD), circulating endothelial function markers, nitrotyrosine (oxidative stress marker), triglycerides, glucose and insulin were assessed pre-meal and hourly for 5 h. Mixed model procedures were used to analyze the data.

Results: Meal consumption increased serum triglycerides (time effect, $P < 0.001$); with no meal differences (meal $\times$ time interaction, $P = 0.93$). Serum insulin peaked 1 h post-consumption and returned to pre-meal concentrations by 5 h with both meals (time effect, $P < 0.001$; meal $\times$ time effect, $P = 0.68$). FMD, serum intercellular adhesion molecule-1 (ICAM-1) and E-selectin did not change (meal $\times$ time effect, $P > 0.4$). Olive oil transiently increased plasma nitrotyrosine after 1 h compared to palmolein (meal $\times$ time interaction, $P = 0.002$) whereas both meals increased serum vascular cell adhesion molecule-1 (VCAM-1) after 1 h (time effect, $P < 0.001$; meal $\times$ time interaction, $P = 0.98$). Both nitrotyrosine and VCAM-1 returned to pre-meal concentrations after 2 h.

Conclusion: In the context of a high protein meal, palmolein similarly to olive oil did not affect postprandial endothelial function in overweight/obese men.

Trial registration: Australian New Zealand Clinical Trials Registry (ANZCTR) (<http://www.anzctr.org.au/default.aspx>). Trial ID: ACTRN12613000136707.

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1. Introduction

Reducing saturated fatty acid (SFA) intake has been a key dietary strategy for lowering coronary heart disease (CHD) risk based primarily on its cholesterol raising effects [1,2]. However, recent evidence suggests this relationship may not be as straightforward [1,3]

and may be affected by co-consumption of other dietary components. Meals or foods high in SFA may contain other constituents that counteract its CHD potential [1]. Furthermore, dietary effects on CHD risk are mediated through multiple pathways; it is therefore insufficient to base dietary recommendations solely on its lipid modulating effects and evaluating the effects of SFA on multiple biomarkers will assist to characterize its CHD potential [1].

Endothelial dysfunction, involving increased endothelial permeability to lipoproteins and other plasma constituents, reduced vasodilatation and activation of thrombotic and inflammatory pathways has been proposed as the earliest identifiable event in the atherosclerosis process and therefore represents an important clinical target for cardiovascular disease (CVD) prevention through dietary interventions [4,5]. To date, the effects of SFA on vascular function have not been well-researched [2,6].

Abbreviations: %E, percentage of total energy; CHD, coronary heart disease; CVD, cardiovascular disease; FMD, flow-mediated dilation; ICAM-1, intercellular adhesion molecule-1; MUFA, monounsaturated fatty acids; PAI-1, plasminogen activator inhibitor 1; PUFA, polyunsaturated fatty acids; SFA, saturated fatty acids; TG, triglycerides; tPA, tissue plasminogen activator; VCAM-1, vascular cell adhesion molecule-1.

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Considering most humans spend the majority of their day in the postprandial state, it is important to investigate the acute effects of fat-containing meals on endothelial function. High-fat meals have previously been shown to impair postprandial vascular function as indicated by reduced brachial artery flow-mediated dilatation (FMD) [6], a recognized marker of endothelial dysfunction [7], and increased markers of vascular function such as cell adhesion molecules including vascular cell adhesion molecule-1 (VCAM-1), intercellular adhesion molecule-1 (ICAM-1) and E-selectin [8]. This effect may be ascribed to increased inflammation and oxidative stress mediated by elevated non-esterified fatty acids and postprandial triglyceride rich lipoproteins [6]. However, evidence for the differential postprandial effects of SFA vs. unsaturated fatty acids on vascular function is inconclusive [6] and may be dependent on other concurrent meal components such as protein. Westphal et al. [9] showed that consumption of SFA from dairy (whipped cream) had a neutral effect on FMD postprandially when consumed with a 50 g protein load (soy protein, caseinate). This response may be due to the presence of the amino acid L-arginine in the meals [10]. This suggests consumption of SFA in combination with a high protein and/or L-arginine load provided by dietary sources such as fish, meat (beef, poultry, pork), seeds, nuts and soy [11] may not impair FMD and endothelial function.

Palmolein, derived from dry fractionation of palm oil is a rich source of both SFA (42%) and unsaturated fat (47% monounsaturated fatty acids (MUFA), 12% polyunsaturated fatty acids (PUFA)) (Table 1). It's inherent stability and resistance to oxidation makes it a popular choice for food manufacturers and as replacement for *trans* fats [12,13]. Hence, assessing its health effects is important.

The aim of this study was to assess postprandial effects of typical meals high in protein containing either palmolein or olive oil on endothelial function in overweight or obese men, a target group with high prevalence globally [14] with increased risk of endothelial dysfunction [15]. We hypothesized that palmolein relative to olive oil, in the context of a high protein meal, will have similar effects on endothelial function as assessed by FMD as primary outcome. Secondary outcomes were circulating markers of endothelial function including serum adhesion molecules (VCAM-1, ICAM-1 and E-Selectin), endothelium derived fibrinolytic factors (plasminogen activator inhibitor-1 [PAI-1], tissue plasminogen activator [tPA]), a marker of oxidative stress (nitrotyrosine formation), insulin, glucose and triglycerides.

2. Methods

The dietary intervention (<http://www.anzctr.org.au>; ACTRN12613000136707) was conducted at CSIRO's Nutrition and

Health Research Clinic, Adelaide, South Australia between September and December 2013 according to the guidelines of the National Health and Medical Research Council National Statement on Ethical Conduct in Human Research. Ethical approval for the trial was obtained from the CSIRO Human Ethics Committee reference 12/13 and written informed consent was obtained from all participants.

2.1. Participants

Twenty eight [28] overweight and obese men (aged 18–65 yr, BMI between 25 and 35 kg/m²) who were weight stable over the previous 3 months were recruited by local advertisement and a pre-existing participant database. Overweight and obesity is associated with endothelial dysfunction [15] and it was therefore considered important to establish the effects of the interventions being tested in this population. Women were excluded to remove the potential confounding effects of the menstrual cycle on FMD [7]. Other exclusion criteria were known medical conditions or diseases including type 1 or 2 diabetes, kidney, respiratory, gastrointestinal, cardiovascular or peripheral vascular disease, smoking, history of heavy alcohol consumption (>5 standard drinks/d), use of nitrate medication, non-steroidal anti-inflammatory medication or medication and supplements that may affect the study outcomes and gastrointestinal function (such as antibiotics, laxatives, fish oil, etc) during the 3 months prior to the study.

2.2. Study design

This acute (5 h) nutrition intervention was conducted using a randomized double-blind cross-over study design. Participants attended the CSIRO clinic on two occasions separated by 1 wk during which they consumed two high protein, high fat test meals prepared with either palmolein or olive oil. Participants were randomly assigned by computer generation (<http://www.randomisation.com>) to treatment orders matched on age and BMI. Medication usage remained constant throughout the study and during test days. Prior to each test day participants were requested to avoid alcohol and strenuous exercise for 24 h and 48 h respectively, and to consume the same evening meal. Participants recorded the content of their evening meal and research staff verified whether the same meal was consumed. Two participants deviated from this protocol, but their outcome responses did not differ from other participants. Participants arrived at the clinic between 8.00 and 8.30 am after an overnight fast; height (at first visit), weight and blood pressure were recorded before an intra-venous cannula was inserted in the left arm for venous blood collection. A baseline blood sample and FMD measurement (of the right arm) were then obtained after which participants consumed the test meals within 15 min. After 1 h from commencing consumption of the test meal and hourly thereafter for 5 h, blood samples and FMD measurements were obtained.

2.3. Test meals

The test meals were prepared in the CSIRO clinic kitchen using safe food handling practices. The test meals were identical in appearance and energy and macronutrient content and only differed in the oil type used to prepare the meals. No obvious difference in flavor between the meals was apparent. The meals consisted of 200 g (raw weight) of lean chicken strips fried in 40 g test oil, either palmolein (RBD Palmolein) or olive oil (Moro Pure Olive Oil 100% pure, produced and packed in Spain for Conga Foods Pty. Ltd). The cooked chicken was served with lightly fried white bread (fried in the remaining oil after cooking the chicken to absorb

Table 1
Fatty acid composition of the test oils (% of total fatty acids).

Fatty acids	Palmolein	Olive oil
Lauric acid (C12:0)	0.20	0.00
Myristic acid (C14:0)	0.98	0.00
Palmitic acid (C16:0)	36.2	11.7
Palmitoleic acid (C16:1n-9)	0.22	0.69
Stearic acid (C18:0)	4.14	4.20
Elaidic acid (C18:1n-9t)	0.25	1.04
Oleic acid (C18:1n-9)	46.1	74.1
Linoleic acid (C18:2n-6)	11.3	6.80
Alpha-linolenic acid (C18:3n-3)	0.19	0.45
Arachidic acid (C20:0)	0.37	0.51
Gondoic acid (C20:1n-9)	0.21	0.33
Behenic acid (C22:0)	0.00	0.16
Total SFA	41.9	16.6
Total MUFA	46.8	76.2
Total PUFA	11.5	7.25

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