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POSTPRANDIAL INVESTIGATION ON PLANT AND ANIMAL DERIVED PALMITIC ACID VERSUS OLEIC ACID-RICH HIGH FAT DIETS ON ENDOTHELIAL DYSFUNCTION BIOMARKERS IN HEALTHY MEN: A PILOT STUDY

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ABSTRACT

Prolonged postprandial lipemia has been associated with endothelial dysfunction and an increased risk of cardiovascular disease. While the effect of dietary fatty acids on postprandial lipemia is well recognised, less is known about their effects on endothelial dysfunction biomarkers. We investigated the effects of palmitic acid rich high-fat diets from either plant (palm olein) or animal (lard) compared with an oleic acid-rich high-fat diet (virgin olive oil) on endothelial dysfunction biomarkers in healthy young men. This pilot study employed a randomised crossover design involving 10 healthy men. Each subject consumed 50 g of test fat incorporated in a diet. The diets were consumed on three different days, with blood samples collected at baseline and hourly over 4 hr post-meal. Plasma concentrations of monocyte chemoattractant protein-1 (MCP-1), E-selectin, soluble vascular cell adhesion molecule-1 (sVCAM-1), soluble CD40 ligand (sCD40L) and Plasminogen activator inhibitor-1 (PAI-1) were analysed. All types of fat consumption were linked with a significant decrease in plasma PAI-1 and sCD40L throughout 4 hr, but did not influence other pro-inflammatory markers measured during the postprandial state. In conclusion, palmitic acid-rich high-fat diets from different sources had limited effects on endothelial dysfunction biomarkers in the postprandial state, underscoring the need for further investigation in larger cohorts.

Keywords: endothelial function, oleic acid, palmitic acid, postprandial lipemia, saturated fat.

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INTRODUCTION

Prolonged postprandial lipemia has been associated with endothelial dysfunction and increased cardiovascular risk (Zhao *et al.*, 2021). While numerous studies have explored how saturated fats (SFA) influence the magnitude of postprandial lipemia, less is known about their effects on endothelial markers in the postprandial state (Berry *et al.*, 2007; Masson & Mensink, 2011; Sanders *et al.*, 2011; Teng *et al.*, 2011; 2015; 2017; Tholstrup *et al.*, 2011).

Animal fats and vegetable oils, such as lard and palm oil, are rich sources of palmitic acid (16:0), a dietary SFA known for its cholesterol-raising effects. However, the cholesterol response to palm oil containing 44% palmitic acid does not align with predictions made by Mensink's equations (Mensink *et al.*, 2003). This may be due to the differences in the positional distribution of palmitic acid within triacylglycerol (TAG) molecules. In palm oil, palmitic acid is mainly positioned at the outer *sn*-1 and *sn*-3 positions, whereas in lard, it is predominantly at the *sn*-2 position. Research suggests that fats containing *sn*-2 fatty acids are efficiently absorbed as monoglycerides and transported to the liver, where they may influence cholesterol homeostasis differently compared to when occupying the *sn*-1,3 positions (Hunter *et al.*, 2001; Kritchevsky, 2000; Kritchevsky *et al.*, 2000).

The quantity and quality of dietary fats have a substantial influence on the postprandial lipemia (Kdekian *et al.*, 2020). Monounsaturated fatty acids

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(MUFA) and polyunsaturated fatty acids (PUFA) have been shown to reduce postprandial lipemia compared to SFA in both healthy individuals and those with metabolic syndrome (Dror *et al*., 2017; Margioris, 2009; Masson & Mensink, 2011; Telle-Hansen *et al*., 2017). However, some findings are contradictory. Sciarrillo *et al*. (2019) reported higher postprandial lipemia with MUFA and PUFA than with SFA. Notably, a study found lower TAG responses after consuming SFA-rich lard compared to palm olein (Teng *et al*., 2011).

Postprandial lipemia has been associated with low-grade-systemic inflammation and endothelial dysfunction (Zhao *et al*., 2021). Endothelial dysfunction is characterised by increased permeability, allowing low-density lipoprotein (LDL) infiltration and oxidation. Oxidised LDL promotes pro-inflammatory responses by stimulating the release of cytokines like TNF-α and IL-6. These cytokines increased endothelial adhesion molecule expression (*e.g*., ICAM-1 and sVCAM-1) and the secretion of chemokines such as MCP-1, fostering immune cell adhesion and migration, which accelerates the development of endothelial dysfunction and atherosclerosis (Badimon & Vilahur, 2014; Fritsche, 2015; Meessen *et al*., 2020; Wang *et al*., 2017; Zhao *et al*., 2021). Epidemiological evidence linked SFAs with increased markers of inflammation (Fernández-Real *et al*., 2003; Klein-Platat *et al*., 2005). This was also supported by human trials (Baer *et al*., 2004; Dumas *et al*., 2016; Lopez-Moreno *et al*., 2017). However, the findings were inconsistent, with some studies reporting no significant effects (Dehghan *et al*., 2017; Siri-Tarino *et al*., 2010). Conversely, PUFA- and MUFA-rich meals have been shown to lower postprandial inflammatory markers at 4-6 hr compared to SFA-rich meals (Cruz-Teno *et al*., 2012; Masson & Mensink, 2011; Rathnayake *et al*., 2018).

Despite having similar fatty acid content, the distinct physical properties of fats from different sources, such as palm olein (plant-based) and lard (animal-based), may result in varying effects on postprandial endothelial function. To address the limited understanding of these divergent impacts, this study aimed to investigate the postprandial responses of endothelial dysfunction biomarkers after consuming SFA-rich high-fat meals derived from different sources, compared to meals rich in MUFA (oleic acid), in healthy individuals.

MATERIALS AND METHODS

Subjects

Ten healthy males with a body mass index (BMI) between 18.5-30.0 kg/m^2 and a mean age of 21.9 ± 0.7 yr were recruited. A history of cardiovascular disease, diabetes, hypertension, plasma cholesterol levels more than 5.2 mmol/L, TAG levels more than 1.7 mmol/L, intake of medication and smoking were among the exclusion criteria. Prior to the enrolment, all potential participants attended a screening session for medical evaluation and blood collection for further determination of their health and fitness. In the end, 10 eligible participants met the inclusion criteria. All participants gave informed written consent. This study protocol was approved by the University Malaya Medical Centre Research Ethics Committee, Reference number 732.22 and was registered at ClinicalTrials.gov under the identifier NCT01124487. *Table 1* presents the baseline characteristics of study participants.

TABLE 1. BASELINE CHARACTERISTICS OF THE STUDY PARTICIPANTS

Variables	Mean ± SD
Male, n	10
Age, yr	21.9 ± 0.7
BMI, kg/m^2	21.0 ± 1.6
Systolic BP, mmHG	126.8 ± 8.6
Diastolic BP, mmHG	71.3 ± 4.9
Waist, cm	79.5 ± 6.2
Total cholesterol, mmol/L	4.8 ± 0.7
TAG, mmol/L	0.9 ± 0.7
LDL cholesterol, mmol/L	2.6 ± 0.8
HDL cholesterol, mmol/L	1.6 ± 0.4

Note: BP - blood pressure.

Experimental Procedures

Study design. This study employed a single-blind, randomised crossover design, with all subjects receiving three experimental meals in a random order. A three days wash out period was given after each meal. Subjects were instructed to avoid consuming high-fat meals, caffeinated drinks and alcohol. Additionally, they were asked to refrain from strenuous physical activities a day before the study visit. A regular low-fat dinner (containing less than 10 g of fat) was also given to the subjects the night before their postprandial testing. Subjects reported to the study centre at 7.30 am after 10 hr overnight fast and baseline blood samples were collected. Subjects finished their meals within 15 min and blood samples were obtained hourly for 4 hr. Drinking water *ad libitum* was permitted.

Test meals. The meals were made at the study centre. The test meals comprise 60 g of mashed potato incorporated with 50 g test fat (virgin olive

oil, palm oil, or lard); baked beans (180 g), skimmed milk (50 mL) and orange juice (200 mL). The meals provided a total energy of 754 kcal. *Table 2* presents the nutrient compositions of the test meals. Unilever Best Foods Italia s.r.l., Italy, supplied the olive oil. Wilmar International Limited, Singapore, provided the palm olein. Sainsbury's in the United Kingdom provided the lard.

TABLE 2. NUTRIENT COMPOSITION OF TEST MEALS

Nutrient composition	Olive oil	Palm olein	Lard
Total calorie, kcal	754	754	754
Energy (%)			
Carbohydrates	33.0	33.0	33.0
Protein	7.0	7.0	7.0
Fat	60.0	60.0	60.0
SFAs	9.2	39.7	44.1
MUFAs	44.6	27.7	27.8
PUFAs	6.2	8.6	5.9
Fatty acid composition (mol %)			
Palmitic acid (C16:0)	12.4	33.7	25.4
Stearic acid (C18:0)	2.7	3.7	17.0
Oleic acid (C18:1)	72.1	46.1	42.2
Linoleic acid (C18:2)	10.1	13.8	9.1
Linolenic acid (C18:3)	0.6	0.3	0.6
Others	2.1	2.4	5.8

Note: Values are means of double determinations.

Laboratory Procedures

Blood sampling. Blood samples were drawn into K2 EDTA vacutainers (Becton Dickinson, USA) and centrifuged at 1,300xg for 15 min at 4°C. Plasma samples were stored at -80°C until they were analysed.

Endothelial biomarkers. Plasma endothelial biomarkers (sVCAM-1, MCP-1, PAI-1, E-selectin and sCD40L) were analysed with the (Panomics Inc. USA) Procarta® Cytokine Assay Kit (Panomic Inc. USA). Multiple protein targets were detected and quantified simultaneously using xMAP® technology and multi-analyte profiling beads. Intra-assay values were 4.4% for sVCAM (n = 20), 9.3% for MCP-1 (n = 20), 7.8% for E-selectin (n = 16), 8.3% for PAI-1 (n = 20) and 6.5% for sCD40L (n = 20).

Statistical Analysis

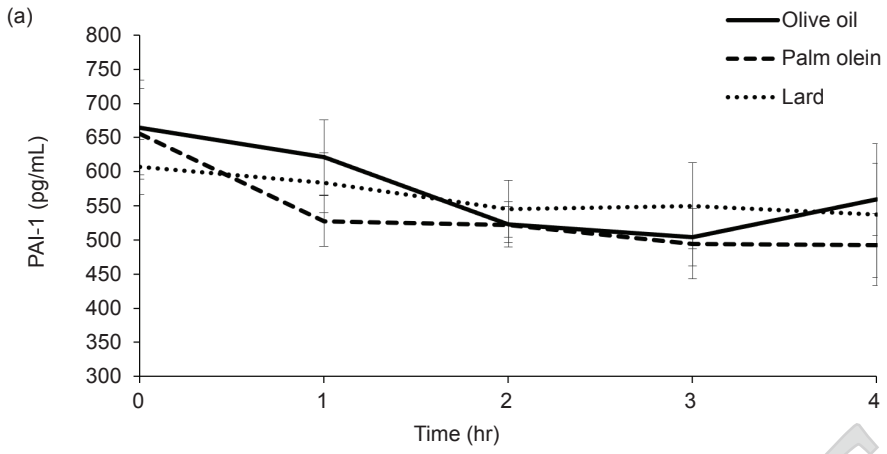
All data were corrected with baseline values and a general linear model for repeated measures was used for statistical analysis. GraphPad Prism 5.03 for Windows and PASW Statistics 18.0 from Chicago, USA were used for all analyses (Graph Pad Software, USA).

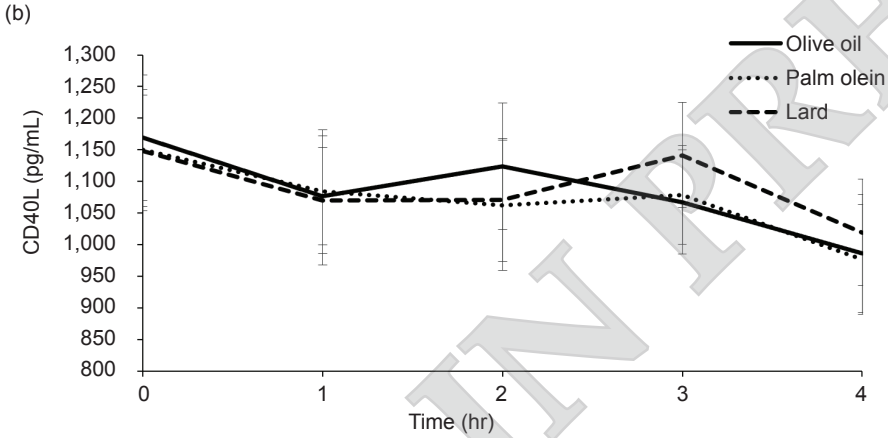
RESULTS AND DISCUSSION

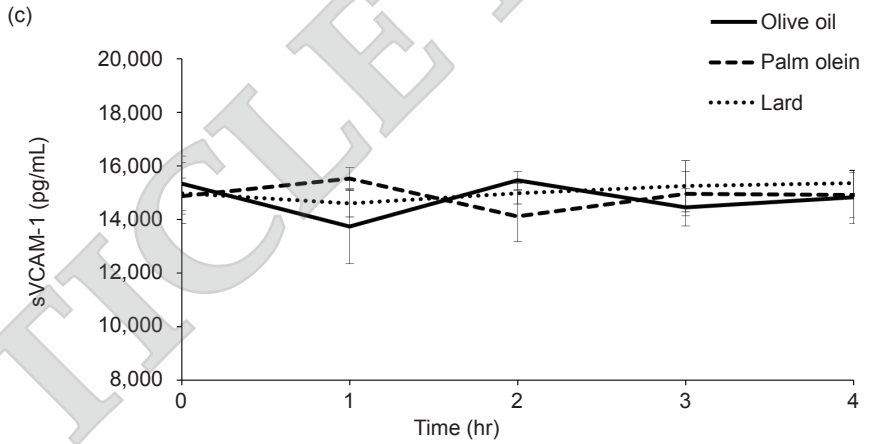
The primary findings of the present study reveal that ingesting a high-fat meal, irrespective of the fat type, is linked to a significant decrease in plasma PAI-1 and sCD40L concentrations (time effect, $P_{sCD40L} = 0.007$; $P_{PAI-1} = 0.041$) over time up to 4 hr as shown in *Figure 1a-1b*. This observation was consistent with other studies that have also reported a decreased response in PAI-1 level in a postprandial setting due to diurnal variation (Teng *et al.*, 2015; Tholstrup *et al.*, 2003). SFA has a tendency to decrease PAI-1 antigens 8 hr after ingestion when compared to the unsaturated fatty acids (Tholstrup *et al.*, 2003). Elevated plasma free fatty acids (FFAs) levels have been shown to further elevate endothelial injury via endothelial activation with an increase in plasma TNF-a, IL-6, sICAM-1 and sVCAM-1 after an intake of a high-fat meal (Nappo *et al.*, 2002). We previously reported that FFA decreased after a post-meal period and displayed a transient increase after 1 hr. Notably, lard resulted in a smaller increase in plasma FFAs at 2 and 3 hr compared to olive oil and palm olein (Teng *et al.*, 2011). However, this observation was not consistent with the plasma PAI-1 levels. Lack of response of PAI-1 levels indicated that healthy and lean subjects were included in the trial. Intake of stearic, oleic and linoleic acids for 5 weeks had similar effects on thrombotic factors in healthy subjects (Thijssen *et al.*, 2005). Similarly, Delgado-Lista *et al.* (2008) also found that dietary SFAs, MUFAs, and carbohydrate intake did not show significant differences in PAI-1 levels after 4 weeks in healthy subjects.

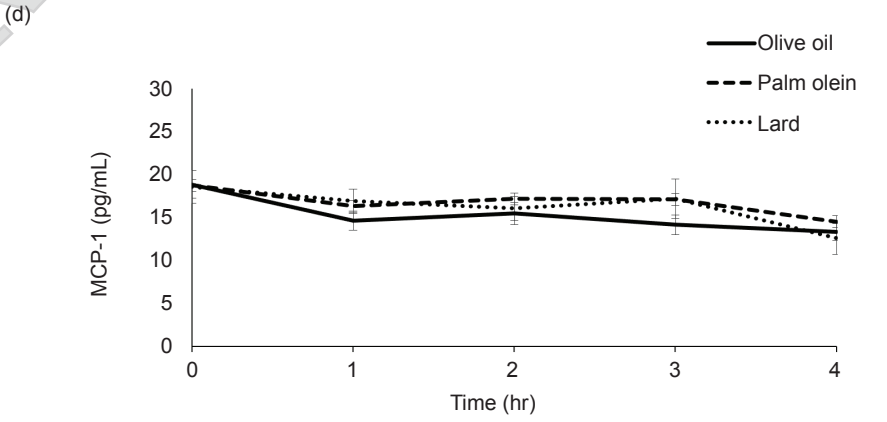
Elevated sCD40L levels were reported in obese people (Unek *et al.*, 2010) but decreased after weight loss. Higher waist circumferences, which indicate higher visceral fat, are associated with elevated sCD40L concentrations. Our finding suggests that individuals with a healthier body composition, as indicated by normal BMI and waist circumference, may exhibit different sCD40L responses compared to obese individuals with larger waist circumferences. Understanding the role of sCD40L and its relationship with body composition may provide valuable insights into metabolic and cardiovascular health.

No apparent changes were noted in plasma MCP-1, sVCAM-1 and E-selectin (*Figure 1c-1e*) measured to identify inflammatory responses related to atherosclerosis. Corroborating with our data, De Souza *et al.* (2022) and Rubin *et al.* (2008) have observed no alterations in these









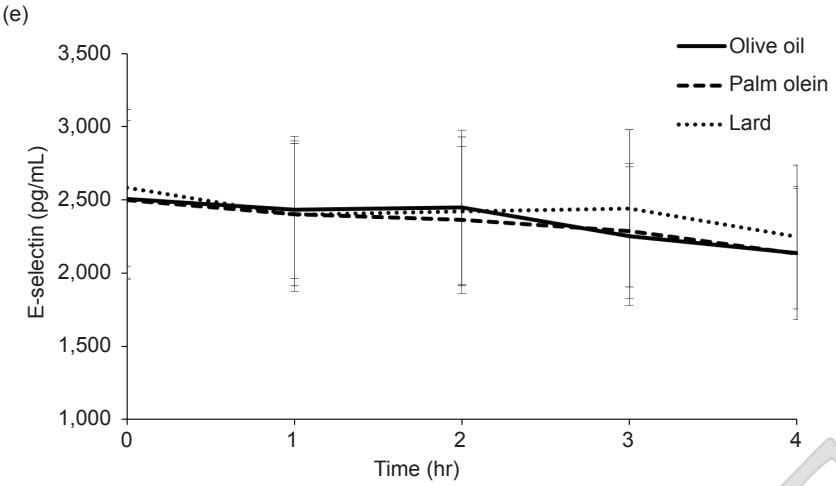


Figure 1. Mean changes for endothelial markers (a) PAI-1, (b) sCD40L, (c) sVCAM-1, (d) MCP-1 and (e) E-selectin after high- fat meals containing palm olein or lard or olive oil. Data are mean ± SEM (n = 10). Changes from baseline values were assessed using repeated-measures ANOVA, with Bonferroni's multiple comparison tests used to compare the three test meals across the time points (0-4 hr). No significant differences were observed for type test meals, but a significant time effect was observed for sCD40L, (P = 0.007) PAI-1, (P = 0.041) over time up to 4 hr.

biomarkers after high-fat meals in healthy and obese individuals. Another study has also reported that dietary fats in meals had varying effects on acute inflammation and endothelial activation in overweight and obese individuals. PUFA has activated the inflammatory factor, nuclear factor-Kappa B (NF-κB) with no response in sICAM-1. Whereas SFA induced sICAM-1 and sVCAM-1 are slightly reduced following all meals in these individuals (Peairs *et al.*, 2011). In a different study with lean and obese men there was a decrease of sICAM-1 and sVCAM-1 after consuming milkshakes rich in SFA, MUFA and PUFA, although postprandial TAG responses were increased in MUFA compared to SFA and PUFA (Esser *et al.*, 2013). Similarly, other studies have reported a lack of response of sVCAM-1 and E-selectin after high-fat meals (Cortés *et al.*, 2006; Tsai *et al.*, 2004). In contrast with ours and others, a postprandial study has reported significant elevation in adhesion molecules for 4 hr after a high-fat meal compared to a high-carbohydrate meal in both healthy and type 2 diabetes mellitus individuals (Nappo *et al.*, 2002). Fuentes *et al.* (2008) have reported that chronic ingestion of SFA – rich diet increased the plasma levels of sVCAM-1 in the postprandial state versus MUFA – rich meals. The effects were also more pronounced in patients with type 2 diabetes mellitus when a high-fat meal and glucose were combined and prolonged.

The Diet-Body-Brain (DietBB) postprandial study was carried out with older adults at risk for cardiometabolic diseases revealing that consuming an energy-rich Mediterranean diet compared to two types of Western diets [approximately 4,300 kilojoules (kJ)] resulted in significant increases in glycemia, lipemia and inflammatory responses. Notably, the Mediterranean diet exhibited more favourable effects on these postprandial responses compared to the Western diets (Diekmann *et al.*, 2019; Schönknecht *et al.*, 2020). In the secondary data analysis of the DietBB study, which examined the changes in inflammatory response to meal intake over a 5 hr periods, the magnitude of the inflammatory response was similar between subjects with pro-inflammatory markers and those without for all meals. In other words, individuals who exhibited proinflammatory markers prior to the meal showed a comparable inflammatory response to the meal as those without such markers. This suggests that the presence of pre-existing proinflammatory markers did not significantly influence the acute inflammatory response to the meal in this group of older individuals with metabolic syndrome traits (Schönknecht *et al.*, 2020; 2022).

Limitations of This Study

Some limitations of our study should be noted. Firstly, the ideal postprandial period should have been extended from 4-6 hr, as recommended in previous literature (De Souza *et al.*, 2022). However, due to the discomfort it would inflict on the participants, performing sequential blood sampling for more than 4 hr would be challenging. In our study, the high-fat meal used might not have been high enough to induce significant endothelial damage and inflammatory response. Obtaining different results might have been possible by including higher proportions of SFA and extending the testing period to at least 4 hr or observing the effects after the meals throughout the day. It is worth noting that while SFA levels were comparable between the two palmitic acid-rich fats, other types of fatty acids such as MUFA

and PUFA were present in the test fats as well. These fatty acids could still contribute to the different metabolic responses despite the similar SFA content between the two tested palmitic acid-rich diets.

It is crucial to note that this study was conducted on healthy male subjects, which means that the interpretation of the data may not be directly applicable to other populations, including women. Gender differences can significantly influence postprandial lipemia and inflammation (Kolovou *et al.*, 2006; Manning *et al.*, 2008; Orem *et al.*, 2018; Schillaci *et al.*, 2001). Men and women have distinct physiological and biological characteristics that can affect metabolic outcomes, including the regulation of lipid metabolism, which can vary due to hormonal factors (Lee *et al.*, 2016; Lindegaard *et al.*, 2018). In women, protective hormones such as estrogen play a crucial role in maintaining vascular health and metabolic stability (Schillaci *et al.*, 2001). These hormones help regulate lipid profiles, reduce inflammation and improve overall cardiovascular function. However, following menopause, the protective effects of female sex hormones diminish, leading to a narrowing of the gender disparity in risk for cardio-metabolic diseases (Lee *et al.*, 2016). This shift suggests that estrogen and possibly other sex hormones are significant contributors to the differential risk profiles between men and women for conditions such as cardiovascular disease and metabolic syndrome (Henstridge *et al.*, 2019; Lindegaard *et al.*, 2018).

It should also be noted that the small sample size in this pilot study may limit the ability to detect significant changes acutely following a high-fat meal. While this study provides valuable preliminary insights, a future study involving a larger cohort, including patients with metabolic syndrome, would be necessary to observe meaningful changes in plasma endothelial biomarkers and better understand the broader implications for gender-specific metabolic responses.

CONCLUSION

In summary, this pilot work indicates that the acute consumption of high-fat meals containing lard and palm olein, both rich in palmitic acid, does not have a significant effect on endothelial dysfunction biomarkers. The specific mechanisms underlying this outcome remain unclear based on these findings. Moreover, in-depth investigations are required to assess the long-term health implications of consuming different quantities and types of dietary fats in larger cohorts.

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