Aerobic Capacity and Postprandial Flow Mediated Dilation

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ABSTRACT

Int J Exerc Sci 1(4) : 163-176, 2008. The consumption of a high-fat meal induces transient vascular dysfunction. Aerobic exercise enhances vascular function in healthy individuals. Our purpose was to determine if different levels of aerobic capacity impact vascular function, as measured by flow mediated dilation, following a high-fat meal. Flow mediated dilation of the brachial artery was determined before, two- and four-hours postprandial a high-fat meal in young males classified as highly trained (n = 10; VO2max = 74.6 ± 5.2 ml·kg·min^{-1}) or moderately active (n = 10; VO2max = 47.3 ± 7.1 ml·kg·min^{-1}). Flow mediated dilation was reduced at two- ($p < 0.001$) and four-hours ($p < 0.001$) compared to baseline for both groups but was not different between groups at any time point ($p = 0.108$). Triglycerides and insulin increased at two- ($p < 0.001$) and four-hours ($p < 0.05$) in both groups. LDL-C was reduced at four-hours ($p = 0.05$) in highly trained subjects, and two- and four-hours ($p \leq 0.01$) in moderately active subjects. HDL-C decreased at two- ($p = 0.024$) and four-hours ($p = 0.014$) in both groups. Glucose increased at two-hours postprandial for both groups ($p = 0.003$). Our results indicate that a high-fat meal results in reduced endothelium-dependent vasodilation in highly trained and moderately active individuals with no difference between groups. Thus, high aerobic capacity does not protect against transient reductions in vascular function after the ingestion of a single high-fat meal compared to individuals who are moderately active.

KEY WORDS: Endothelial function, ultrasound, reactive hyperemia, aerobic exercise, athletes, high-fat meal, insulin, lipids

INTRODUCTION

Vascular endothelial dysfunction is present in healthy subjects with risk factors for atherosclerosis years before the appearance of atheromatous plaques and can be assessed non-invasively through the endothelium-dependent method of flow mediated dilation (FMD) (6). A FMD test results in arterial dilation due to increased nitric oxide secretion by the endothelium in response to increased shear stress (21). In addition, other factors have been suggested to contribute to FMD, including a balance between vasodilators (i.e., bradykinin, adenosine, vascular endothelial growth factor, and prostacyclin) and vasoconstrictors (i.e., endothelin, prostanoids, and angiotensin II) (11). The consumption of a high fat meal (HFM) has been shown to induce transient vascular dysfunction in healthy, young men (4, 29,

46) and is thought to occur due to the oxidation of postprandial triglyceride-rich lipoproteins (38, 46), hyperglycemia (47), or hyperinsulinemia (2).

Conversely, endurance training improves vascular function in diseased populations (20, 28) as well as in healthy, young men (7, 18, 22) by increasing nitric oxide availability (16). A cross-sectional study demonstrated that young, endurance-trained men had higher FMD responses of the brachial artery than sedentary young men (22).

The acute effects of a single HFM on vascular function in individuals who perform different amounts of physical activity is unknown. The purpose of this study was to determine if different levels of aerobic capacity evoke a differential effect on vascular function as measured by FMD prior to and following a single HFM in apparently, healthy young men. The primary aim was to determine if individuals who were highly trained had a protective effect against the negative impact of a HFM on vascular function. It was hypothesized that FMD would be similar between groups at baseline, as demonstrated previously (15), and would be less impaired postprandially in highly trained individuals compared to moderately active individuals. A secondary aim was to determine if blood lipids, insulin, or glucose were correlated to FMD at baseline or postprandial a HFM.

METHOD

Participants

Apparently healthy, highly trained (n = 10) and moderately active (n = 10) young men between the ages of 19 and 26 years were recruited to participate in the study. No subject had a history of disease, smoked or was presently taking any medications or dietary supplements that may have an impact on vascular function. Subjects with a body mass index (BMI) ≥ 28 $kg \cdot m^2$ were excluded to eliminate obesity having an impact on endothelium-dependent vasodilation (44). Highly-trained subjects were members of a Division I university cross country team and had participated in high-intensity endurance training for at least two years. The moderately active subjects reported occasional endurance/ recreational exercise (i.e., jogging, basketball, cycling, etc.) but were not consistently exercising (> 3 $days \cdot week^{-1}$, 20 $min \cdot day^{-1}$) over the past six months. Self-reported physical activity levels were obtained from each subject prior to participation in the study to determine training status. All subjects signed a written informed consent approved by the review board of the University of Louisville.

Protocol

Aerobic capacity, defined by the maximal oxygen consumption (VO_2max), of each subject was measured using a treadmill ramp protocol and indirect open circuit spirometry (ParvoMedics, Sandy, UT). Subjects reported to the laboratory for vascular testing within two-weeks of the VO_2max test. They were instructed to abstain from exercise for 24-hours to avoid any confounding influences of exercise on lipid metabolism (35), to fast for 12-hours (38), and to avoid caffeine and alcohol consumption for 12-hours prior to vascular testing.

Body composition was determined using bioelectrical impedance (RJL Systems,

Clinton Township, MI). Total body resistance and reactance to an alternating electrical current were used to calculate fat mass (FM). Lean body mass (LBM) was calculated using a validated multiple regression equation: *LBM = -8.98751 + 0.36273 [Height^2 (cm)/Resistance) + 0.21411 (Height) + 0.13290 (Weight (kg)]* (41).

Endothelium-dependent FMD of the brachial artery was determined non-invasively using high-resolution ultrasound with an upper arm cuff occlusion to induce reactive hyperemia as previously described (17, 33, 42, 45). Briefly, subjects were placed supine, in a quiet, dark, temperature controlled room for 10-min. Vascular function was measured in the right brachial artery using quantitative Doppler ultrasound (Philips HDI 5000, Seattle, WA) by a single investigator. The brachial artery was imaged longitudinally, 2 cm above the antecubital fossa by B-mode ultrasound, using a 12-5 mHz linear array transducer. Reactive hyperemia was induced by inflation of a pneumatic cuff placed on the upper arm (3-4 cm above the transducer) proximal to the transducer using a rapid cuff inflator (Hokanson E20, Bellevue, WA) at 60-80 mmHg above the systolic pressure for five-minutes. A five-minute period was measured both before and after cuff occlusion to determine baseline brachial artery diameter and FMD after cuff occlusion, respectively. Peak FMD for each individual was determined as the greatest diameter following cuff occlusion release and expressed as a percent change from baseline. A video file was collected throughout the entire test (ULead Video Studio 7, Taipei, Taiwan) to allow data analysis after the test. A custom-made software program using LabView version 7.1 (National Instruments, Austin, TX) measured the changes in the brachial diameter beat by beat during diastole throughout the testing period and thereby allowed second by second data collection. All video files were analyzed by the same investigator who performed the vascular measurements. Blood pressure was measured throughout the entire test to verify that changes in blood flow were not dependent on changes in blood pressure.

Ten ml of blood was collected from an antecubital vein to obtain fasting triglycerides (TG), total cholesterol (TC), high density lipoproteins (HDL-C), low density lipoproteins (LDL-C), insulin, and glucose. Samples were centrifuged and the serum was stored at -80^o C for less than six months until analyzed. TG, TC, and HDL-C were measured enzymatically by reflectance spectrophotometry and LDL-C was calculated by the Friedewald Formula (40). Insulin was measured by an electrochemiluminescent double monoclonal immunometric assay (Roche Diagnostics, Indianapolis, IN). Glucose was measured via an enzymatic method (Ortho Clinical Diagnostics, Raritan, NJ) using glucose oxidase coupled to peroxidase.

Following the baseline blood draw, subjects consumed a meal consisting of an Enormous Omelet Sandwich® [740 kcals, 46 g fat, 16 g saturated fat, 330 mg cholesterol (56% fat, 24% carbohydrate, 20% protein)] and a medium order of hash browns [(310 kcals, 20 g fat, 5.5 g saturated fat, 0 mg cholesterol (58% fat, 40% carbohydrate, 2% protein)] (Burger King Corporation, Miami, FL) with water. The meal fat content used in this study was consistent with other literature which has

Table 1. Characteristics of highly trained and moderately active subjects.

	Highly Trained (n=10)	**Moderately Active** (n=10)	**p-value**
Age (yrs)	20.8 ± 1.8	20.9 ± 2.2	0.914
Height (cm)	180.7 ± 7.5	181.6 ± 10.0	0.826
Weight (kg)	67.3 ± 7.3	77.5 ± 14.0	0.057
Blood Pressure (mmHg)	120/66	124/68	0.890
BMI ($kg \cdot m^2$) *	20.5 ± 1.1	23.4 ± 3.1	0.014
LBM (kg)	60.5 ± 5.0	63.9 ± 6.5	0.207
FM (kg) *	6.9 ± 3.3	13.7 ± 8.5	0.029
VO_2max ($ml \cdot kg \cdot min^{-1}$) †	74.6 ± 5.2	47.3 ± 7.1	< 0.001

Values are mean ± SD. * $p < 0.05$, difference between groups. † $p < 0.001$, difference between groups. BMI, body mass index; LBM, lean body mass; FM, fat mass; VO_2max, maximal oxygen consumption.

shown adverse effects of an acute meal on endothelial function (8, 29). Between testing sessions subjects rested quietly in the laboratory while reading and were not allowed to consume any other food or drink other than water. Vascular function and blood markers were reassessed at two- and four-hours postprandial.

Statistical Analysis

An independent samples t-test was used to determine any mean differences between group characteristics. A two-way repeated measures ANOVA was performed to assess differences within subjects for vascular and blood variables prior to and following the HFM. Correlational analyses were used to determine any relationship between FMD and blood variables at baseline, two-, and four-hour postprandial. Differences were considered to be statistically significant at a P value < .05.

RESULTS

The highly trained and moderately active groups were not different in age, height, weight or LBM (Table 1). As expected, differences were found between groups for VO_2max ($p < 0.001$). Body mass index ($p = 0.014$) and FM ($p = 0.029$) were found to be

Table 2. Serum Blood Lipid Responses.

Time Period	TG (mg/dL)	TC (mg/dL)	LDL-C (mg/dL)	HDL-C (mg/dL)
Highly Trained				
Baseline	66.6 ± 18.0	139.7 ± 29.4	77.0 ± 21.1	49.4 ± 11.7
Two-hour	95.5 ± 28.3 ‡	140.4 ± 23.3	72.8 ± 21.6	48.6 ± 10.6 §
Four-hour	102.3 ± 38.0 ‡	142.8 ± 22.9	72.4 ± 22.8 ‡	49.9 ± 10.9
Moderately Active				
Baseline	75.2 ± 32.3	154.0 ± 28.9	90.5 ± 25.9	48.4 ± 7.5
Two-hour	134.6 ± 57.6 ‡	145.9 ± 20.9	73.0 ± 18.3 ‡	46.0 ± 8.9 §
Four-hour	144.4 ± 66.3 ‡	156.8 ± 32.9	80.6 ± 23.8 ‡	47.3 ± 8.3

Values are mean ± SD. ‡ $p \leq 0.05$, difference compared to baseline. § $p < 0.05$, difference compared to baseline and four-hour. TG, triglycerides; TC, total cholesterol; LDL-C, low density lipoprotein-cholesterol; HDL-C, high density lipoprotein-cholesterol.

higher in the moderately active compared to the highly trained group.

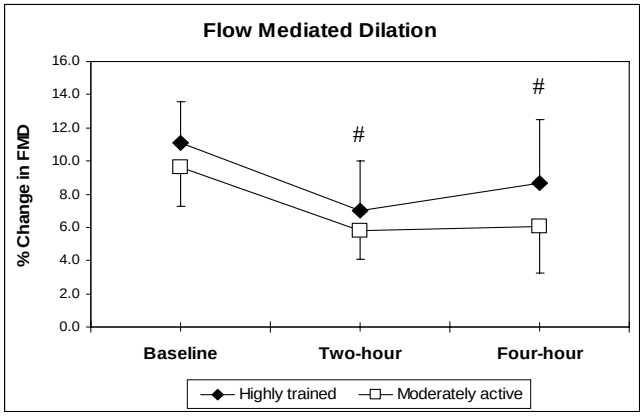

Figure 1. Flow mediated dilation (FMD) of the brachial artery in highly trained and moderately active subjects as determined by Doppler ultrasound (means ± SD). # $p < 0.001$, difference from baseline for both groups.

Resting brachial artery diameter was not different between groups (0.48 ± 0.04 vs. 0.46 ± 0.05 cm, highly trained vs. moderately active, respectively) ($p = 0.398$) at any time point, suggesting that any differences found in FMD across time were not due to changes in baseline diameter. Flow mediated dilation was not different between groups ($p = 0.108$) across time points (Figure 1). However, a time effect was found after the HFM ($p < 0.001$) as FMD was reduced (≈ 37-40%) from baseline to two-hour postprandial ($p < 0.001$) in both groups. FMD from baseline to four-hour postprandial was decreased by 20% in the highly trained group while the moderately active group remained at a 37% reduction ($p < 0.001$). When FMD was normalized to

Table 3. Serum Glucose and Insulin Responses.

Time Period	Glucose (mmol/L)	Insulin [a] (μU/ml)
Highly Trained		
Baseline	5.2 ± 0.3	4.4 ± 2.3
Two-hour	5.5 ± 0.4 ‡	11.2 ± 3.3 ‡
Four-hour	5.1 ± 0.5 ‡	5.4 ± 2.0 ‡ ¶
Moderately Active		
Baseline	5.1 ± 0.3	4.7 ± 1.8
Two-hour	5.4 ± 0.4 ‡	16.4 ± 7.2 ‡
Four-hour	5.3 ± 0.2 ‡	8.3 ± 2.1 ‡ ¶

Values are mean ± SD. ‡ $p \leq 0.05$, difference compared to baseline. ¶ $p \leq 0.05$, difference compared to two-hour. [a] $p = 0.056$, difference between groups.

baseline diameter to control for diameter differences between subjects, the results were the same, with two- and four-hour FMD reduced from baseline in both groups ($p < 0.001$) and no difference between groups ($p = 0.257$).

The average time to peak dilation was not different between groups ($p = 0.518$) nor did it differ over time (88.2 ± 15.0 and 97.5 ± 18.3 s, highly trained vs. moderately active, respectively) ($p = 0.167$). Blood pressure was not different between groups ($p = 0.890$), did not change throughout the test ($p = 0.689$), and did not differ across time point ($p = 0.783$) suggesting that blood pressure did not affect the blood velocity (i.e. shear stress stimulus) on the vessel.

No between group differences were found in TG levels ($p = 0.093$) across all time points (Table 2). However, a time effect for both groups was found ($p < 0.001$). Compared to baseline, TG increased by approximately 43% and 52% ($p < 0.001$) at two- and four-hour postprandial, respectively, with no difference between two- and four-hour postprandial ($p = 0.323$). Total cholesterol was not different between groups ($p = 0.310$) or across time ($p = 0.138$).

An interaction effect was found between time and group ($p = 0.049$) for LDL-C. Levels of LDL-C were found to be different across time ($p = 0.002$). The highly trained group had a reduction in postprandial LDL-C from baseline by 6.0% ($p = 0.05$) at

Table 4. Correlational Analyses.

FMD	**Baseline**	**Two-hour**	**Four-hour**
VO_2max			
r	0.146	0.220	0.330
Sig.	0.270	0.198	0.084
TG			
r	-0.422	-0.053	-0.203
Sig.	0.032 *	0.420	0.202
TC			
r	0.145	0.180	0.193
Sig.	0.271	0.245	0.214
LDL-C			
r	0.088	0.154	0.150
Sig.	0.357	0.277	0.270
HDL-C			
r	0.385	0.130	0.449
Sig.	0.047 *	0.309	0.027 *
Insulin			
r	-0.140	-0.208	-0.104
Sig.	0.279	0.211	0.337
Glucose			
r	0.115	-0.015	0.325
Sig.	0.315	0.477	0.087

Correlational analyses for respective time points. * $p < 0.05$. FMD, flow mediated dilation; VO_2max, maximal oxygen consumption; TG, triglycerides; TC, total cholesterol; LDL-C, low density lipoprotein-cholesterol; HDL-C, high density lipoprotein-cholesterol.

four-hour postprandial but not at two-hour ($p = 0.110$). While in the moderately active group LDL-C was reduced from baseline by 19% ($p = 0.010$) and 11% ($p < 0.001$) at two- and four-hour postprandial, respectively. However, no between group differences in LDL-C were found at any time period ($p = 0.457$).

No differences in HDL-C were found between groups at any time point ($p = 0.634$). The HDL-C two-hour postprandial period for both groups was reduced compared to baseline ($p = 0.024$) and four-hour postprandial ($p = 0.014$). No differences were determined between baseline and four-hour postprandial ($p = 0.572$).

Glucose concentrations were found to be greater at two-hour postprandial compared to baseline in both groups ($p = 0.003$) (Table 3). At four-hour postprandial, glucose levels decreased and were lower than the two-hour measurement ($p = 0.05$) but were not different from baseline. No between group differences were found for serum glucose concentrations at any time point ($p = 0.895$). Serum insulin concentration was greater at two- ($p < 0.001$) and four-hour ($p = 0.003$) postprandial a HFM compared to baseline for both groups (Table 3). Furthermore, insulin was lower at four-hour compared to two-hour ($p < 0.001$). In addition, the moderately active individuals demonstrated greater insulin concentrations compared to the highly trained group at all time periods ($p = 0.056$).

Analyses were conducted to determine if blood markers or VO_2max were correlated to FMD. Baseline FMD was correlated to baseline TG and HDL-C levels (Table 4). No significant correlations between FMD and any blood markers were found at two-hour postprandial. At four-hours, post-prandial FMD was correlated to HDL-C.

DISCUSSION

The primary finding of this study was that flow mediated dilation of the brachial artery was impaired following the consumption of a single high-fat meal, independent of aerobic capacity. No differences in FMD were detected between groups at any time point suggesting that a high aerobic capacity does not augment or protect vascular function prior to or following a HFM. These findings demonstrate that vascular function is not improved, and can be impaired by a single HFM, to the same extent whether the subject is highly trained or moderately active. In addition, our finding that resting brachial artery diameter was unaffected by the test meal is in agreement with previous studies (29, 36, 45) and is important as an increased diameter prior to occlusion results in a decreased FMD response after reactive hyperemia (17). When we normalized FMD to resting diameter in order to control for diameter differences between subjects, we found similar changes to those of FMD alone.

Our findings of reduced endothelial function after a HFM confirms the results of several studies using healthy, young men (4, 8, 29, 46). On the other hand, several others have found conflicting results (13, 39). We found an approximate 40% reduction in FMD at two- and four-hour postprandial which is consistent with the results of Tsai et al. (46). Discrepancies in studies which have found no changes may be explained by age of the subject, the dietary lipid composition of the meals (10), the timing of the vascular measurements, and/or the gender of the subjects studied (32).

A potential mechanism for the reduction in FMD postprandially may be increased serum insulin concentrations. Our results indicated that insulin levels at two- and four-hour postprandial compared to baseline were elevated. Acute hyperinsulinemia has been shown to have a negative impact on the FMD in healthy subjects (2). While other work has found that the degree of hyperinsulinemia independently predicted decreases in FMD in healthy volunteers (3). The mechanism for the reduction in FMD with

hyperinsulinemia is unknown but it may be due to oxidative stress (2). Another potential mechanism to explain our findings may be elevated blood lipid concentrations as research has indicated that impairments in FMD after a HFM are due to increased oxidation of elevated TG levels (4, 38, 46). Elevations in TG results in a rise in superoxide anion production (4) which impairs vascular function through direct inactivation of nitric oxide and increases in lipid oxidation (27); thereby, limiting nitric oxide availability necessary for arterial dilation. Despite our observation of differing levels of LDL-C during the postprandial period, we do not believe that this mechanism contributed to our results as brachial artery FMD has not correlated with several traditional cardiovascular risk factors, including LDL-C in healthy men (48). Our findings support this as no significant correlations were found between FMD and LDL-C in this study. Interestingly, our correlational analyses did not suggest that any one variable was strongly related to postprandial FMD. Thus, other factors such as increased oxidative stress, free radicals, apolipoprotein B, reduced endogenous antioxidants, inflammatory cytokines, and/or neutrophils may be responsible for the decrease in FMD at two- and four-hour postprandial. Future studies should examine these factors to determine if they exert detrimental effects on vascular function following a HFM.

Our results indicated that possessing a high aerobic capacity did not protect individuals from the acute negative effects of a HFM on vascular function to a greater extent than those who are moderately active. We did not find a difference in FMD at baseline between the highly trained and moderately active men (11.1 vs. 9.6%, respectively). Results are mixed in regards to the effect of exercise training improving (7, 22) or having no effect (12, 15) on vascular function in young, healthy individuals. Goto et al. (18) found an augmentation in endothelium-dependent dilation in young (25 ± 2.5 years), healthy men following 12-weeks of moderate-intensity aerobic exercise, but not following the same period of mild or high-intensity exercise. It was postulated by these authors that the elevated oxidative stress levels seen in individuals performing high-intensity exercise may have impaired endothelium-dependent dilation through a reduction in nitric oxide bioavailability. Increasing exercise intensity has been shown to progressively increase the production of nitric oxide (30). It is possible that the high-intensity exercise performed by our highly trained group increased nitric oxide production while at the same time resulted in increased oxidative stress thus counteracting the beneficial effects of increased nitric oxide production. We did not measure nitric oxide production, however, so this can only be postulated based on the current research. Future research warrants looking at this mechanism as well as other factors modulating endothelial function (i.e. bradykinin, prostacyclin, endothelin) (11).

The young age of our subjects may be another reason why we did not find a difference in FMD between our subjects. It has been shown that age is an important contributor to vascular function and has an interactive effect with disease and lifestyle on cardiovascular health (25). Indeed, the FMD response is preserved in older athletes

compared to their age-matched, sedentary counterparts and is similar to the responses seen in both younger athletic and sedentary individuals (15). Thus, we may not have seen a difference in vascular function either prior to or after the HFM because our subjects were young enough that they did not exhibit the arterial stiffening that occurs due to aging.

In addition, the health status of our subjects may explain our findings as all of our subjects were considered healthy (i.e. no history of disease or smoking) via self-reported medical history. Research has demonstrated that exercise training improves endothelium-dependent dilation in diseased individuals (20, 28) as well as in aged individuals (15), with inconsistent results observed in young, healthy individuals (7, 12, 24, 32). Vascular dysfunction induced by a high-fat meal may occur due to enhanced oxidative stress (4, 46). In contrast to our hypothesis, our study demonstrated that high aerobic capacity did not offer increased protection against the acute, negative effects of a high-fat meal on vascular function. Several mechanisms have been suggested by which exercise may protect endothelial function following a high-fat meal (i.e. increased shear stress, diminished oxidative stress and inflammation (46), increased antioxidant enzyme activity (31), and the release of anti-inflammatory cytokines (43)). The scarcity of studies that have investigated the effects of exercise training on postprandial endothelial function further demonstrates the need for future research to expand our understanding of the role of diet, exercise, age, and health status on cardiovascular health.

It is possible that we may not have seen differences in FMD between our groups due to methodology (i.e. upper arm versus forearm occlusion) or the population (i.e. diseased (20, 28) or aged (12, 15, 34) that was used. In the present study we induced FMD by utilizing the proximal cuff position method. In young, healthy subjects brachial artery FMD has been found to be significantly higher after upper arm occlusion compared to forearm occlusion and thus proposed as a better means of evaluating vascular function in this population (1, 5). Occlusion of the forearm has been suggested to provide a more accurate assessment of endothelial dysfunction induced by smoking (19). The partial attenuation of FMD with the NO synthase inhibitor N^G-monomethyl-L-arginine (L-NMMA) following upper arm occlusion demonstrates that dilation induced by the proximal occlusion method is not entirely mediated by NO (14). The regional ischemia produced by occlusion of the upper arm is associated with the release of vasodilators (i.e. potassium, adenosine, ATP) and changes in local pH that could explain the greater dilation observed with proximal occlusion (14). However, we chose to use the upper arm occlusion method due to our population studied and because previous studies examining the effect of a high fat meal on FMD have also utilized this method (4, 29, 36, 37). Exercise training improves endothelium-dependent dilation in asymptomatic older individuals (15), as well as patients with chronic heart failure (20) and type 2 diabetes (28). However, the beneficial effects of exercise training on vascular function in young individuals exhibiting normal arterial function have not been consistently observed (7, 12, 24, 32). Future studies

warrant investigation into the effects of both cuff position and aging on postprandial vascular function.

Another possibility is that the difference in aerobic capacity between subjects may not have been large enough to elicit difference in FMD at baseline. However, this does not seem likely as a difference in FMD with a population that had similar differences in VO_2max has been found previously (22). Alternatively, differences in postprandial FMD between the highly trained and moderately active group may have become apparent if we had extended the study (i.e. six-hour postprandial HFM) as Tsai et al. (46) found a reduction in FMD at six-hour postprandial in healthy, young men. Future studies are warranted that may address some of these factors.

The increased calories or the type of fat used in our meal could have contributed to the impairment of postprandial vascular function seen in this study rather than the fat intake. However, we do not think this is likely as an isocaloric low-fat meal has no effect on FMD when compared to a HFM (23, 38). Furthermore, a meal high in saturated fat, which was comparable to our study, resulted in reduced FMD while meals high in carbohydrates, monounsaturated fat, or polyunsaturated fat had no effect on FMD (23).

A limitation to the study is the lack of chronic dietary information for our subjects as chronic high fat (26) or low fat (9) diets may alter FMD. Although the chronic dietary patterns between our subjects may have been different, we compared the FMD response for each individual to baseline; thus, the changes in FMD are indicative of the acute response to a HFM. Another limitation to this study is the lack of a true sedentary group as a control. Despite being classified as moderately-active, this group still displayed a VO_2max of 47.3 ± 7.1 ml·kg·min^{-1} and thus can be considered as possessing a high level of fitness. The inclusion of a sedentary control group with a lower aerobic capacity may have allowed us to detect differences between groups not seen in the present study design and to further elucidate the role of fitness level on postprandial FMD. Future studies should include individuals with lower aerobic capacities and different age populations to allow for further investigation into the relationship between fitness, aging and postprandial endothelial function.

This study demonstrated that FMD was similar with differing aerobic capacities and was reduced in both highly trained and moderately active young men for up to four-hours after consuming a HFM. Our results suggested that a high aerobic capacity did not augment FMD and did not offer increased protection against the acute, negative effects of a HFM on vascular function compared to individuals possessing a moderate aerobic capacity. Moderate-intensity exercisers demonstrated similar FMD responses compared to elite runners, suggesting that exercise performed at a moderate-intensity is sufficient to elicit positive responses in overall cardiovascular risk and vascular health. Future studies are warranted with a larger sample size to determine the mechanism for the decrease in FMD after a HFM and to determine if differences may exist between groups if followed for a longer period of time (i.e. six- or eight-hours postprandial). These findings are significant as it demonstrates that moderately active individuals have

similar acute responses to a HFM compared to highly trained individuals, suggesting that increased aerobic capacity and/or high intensity aerobic exercise is not necessary to maintain endothelial function.

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