



A High-Fat Meal Increases Cardiovascular Reactivity to Psychological Stress in Healthy Young Adults

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Abstract

The consumption of high levels of saturated fat over the course of several weeks may lead to exaggerated cardiovascular reactivity. The consumption of a single high-fat meal has been associated with a transient impairment of vascular function. In a randomized, repeated measures, crossover study we tested whether the consumption of a single high-fat meal by healthy, normotensive participants would affect cardiovascular reactivity when compared with an isocaloric, low-fat meal. Thirty healthy participants ate a high-fat (42 g) and a low-fat (1 g) meal on 2 separate occasions, and their cardiovascular response to 2 standard laboratory stressors was measured. Systolic blood pressure, diastolic blood pressure, and total peripheral resistance were greater in participants following the consumption of the high-fat meal relative to the low-fat meal. The findings of the present study are consistent with the hypothesis that even a single high-fat meal may be associated with heightened cardiovascular reactivity to stress and offer insight into the pathways through which a high-fat diet may affect cardiovascular function. *J. Nutr.* 137: 935–939, 2007.

Introduction

Evidence implicates dietary fat as an important contributor to sustained high blood pressure (1). Specifically, high levels of saturated fat and cholesterol may contribute to increases in blood pressure through the development of plaques on vessel walls, which result in the reduction of both their diameter and elasticity (2). Thus, a diet high in fat may contribute to the development of atherosclerosis, instigating elevations in blood pressure, which in turn can lead to a further risk of other cardiovascular events (3,4). In addition, high-fat meals have been shown to have acute effects on vascular tone, mediated in part by reductions in endothelium-dependent vasodilation (5). This suggests another mechanism for the effects of saturated fat on hypertension.

Cardiovascular reactivity is the acute change in cardiovascular function that occurs in response to stress (6,7). Exaggerated cardiovascular reactivity has been postulated as a possible mechanism in the development of hypertension and cardiovascular disease (CVD)⁴ (8,9). Specifically, the reactivity hypothesis states that exaggerated responses to stress cause a cascade of pathophysiologic events that can eventually lead to sustained increases in blood pressure (1,3,10–12). For example, increases

in vascular reactivity have been linked to enhanced peripheral vascular resistance, which is a hemodynamic alteration associated with hypertension (10,13). Several prospective studies have established exaggerated cardiovascular reactivity as an independent predictor of future hypertension (8,14–18), in some cases, as much as 36 y later (3). Increased and prolonged cardiovascular responses to stress can pose serious strain on the arteries and the myocardium (6,19). Furthermore, recent prospective studies have shown that high reactors to acute stressors have more extensive arterial plaques and more aggressive plaque growth (3,20).

A diet high in saturated fat and cholesterol has been associated with exaggerated cardiovascular reactivity in healthy populations over relatively short periods of time. For example, Straznicky et al. (21) reported increased blood pressure and heart rate reactivity in healthy, normotensive participants after consuming a high-fat diet compared with an isocaloric, low-fat diet in a 2-wk crossover study. In the same study, Straznicky et al. also observed simultaneous increases in cholesterol and triglyceride concentrations in the blood. These findings have important implications insofar as they show that even short-term exposure of healthy individuals to high levels of dietary fat can have considerable effects on cardiovascular function. In a seminal study, Vogel et al. (5) reported that even a single meal high in saturated fat may be associated with a transient impairment of vascular function. In that study, flow-mediated vasoactivity of the brachial artery was impaired by 50% 4 h after the consumption of a high-fat meal (5). Furthermore, a

⁴ Abbreviations used: CO, cardiac output; CV, cardiovascular; CVD, cardiovascular disease; DBP, diastolic blood pressure; HR, heart rate; SBP, systolic blood pressure; SV, stroke volume; TPR, total peripheral resistance.

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significant correlation was reported between elevated levels of triglycerides in the blood and endothelial dysfunction (5). Impairment of vasoactivity may have important implications on cardiovascular function: endothelial dysfunction can instigate and enhance the development of hypertension (4,6,7). The results of this study further imply that a high-fat diet can affect the cardiovascular system through a direct, endothelium-dependent pathway as well as an indirect, cholesterol-dependent pathway. In addition, diets that specifically reduce fat intake have been shown to improve blood pressure and vascular function (22). However, no previous studies have examined the effects of fat content from a single meal on cardiovascular reactivity to stress in the postprandial period.

The goal of the present study was to examine the effect of a single high-fat meal on cardiovascular reactivity. Reactivity was measured in terms of changes scores in systolic blood pressure (SBP) and diastolic blood pressure (DBP), total peripheral resistance (TPR), heart rate (HR), stroke volume (SV), and cardiac output (CO). We hypothesized that healthy, normotensive individuals would exhibit higher cardiovascular reactivity to a set of standard laboratory stress tasks following a single high-fat meal vs. an isocaloric, low-fat meal.

Materials and Methods

Participants

Thirty healthy undergraduate students (18 women) between the ages of 18 and 25 y of age participated in the study. All participants completed a health history questionnaire. Exclusion criteria included history of hypertension or suspected hypertension uncovered during screening; current use of medications for pain, blood pressure control, cardiovascular disease, sleep disorders, anxiety, or depression; previous diagnosis of diabetes mellitus, atrial fibrillation, myocardial infarction, alcohol or drug abuse (within the last 12 mo), and renal or hepatic dysfunction; inability to comply with the assessment procedures or inability to provide informed consent; and pregnancy. Written and informed consent was obtained, and approval for the study was granted by the University of Calgary Conjoint Faculties Research Ethics Board.

Measures and apparatus

Measurements of SBP and DBP (mm Hg) were obtained at 1-min intervals using an automatic blood pressure monitor (Accutor Plus, Data Scope) and a blood pressure cuff on the upper part of the dominant arm. Measures of HR (bpm), and SV (mL) were recorded noninvasively using a HIC-2000 Bio-electric Impedance Cardiograph, the Cardiac Output Program, developed by Bio-Impedance Technology, and an IBM PC. From these measures both TPR (dyne-s/cm⁵) and CO (L/min) were calculated using standard equations (23). A tetrapolar band-electrode configuration was used. The 2 inner recording electrode bands were positioned circumferentially at the base of the neck and at the thorax. The 2 outer current electrode bands were placed around the neck and the thorax 3 cm apart from the inner bands. Three adhesive silver-silver chloride electrodes were utilized in conjunction with an electrode gel to independently measure the electrocardiogram signal. Two electrodes were placed bilaterally on the upper rib cage, with a ground electrode placed on the right hipbone. For each minute, means of TPR, HR, SV, and CO were obtained from these recordings by the Cardiac Output Program system.

Procedure

Design. The present study utilized a randomized, repeated measures, crossover design. Participants fasted and abstained from caffeine, tobacco, and alcohol for 10 h before testing. Each testing session began at 0830. Participants ate either a high- or low-fat meal, and 2 h later cardiovascular parameters were measured at rest and during 4 behavioral stress tasks. An identical procedure was followed on the 2nd testing

day 1 wk later, and the order of treatment presentation was randomized and counterbalanced.

Test meals. The high-fat meal consisted of a McDonald's breakfast: 2 hash brown patties, a Sausage McMuffin and an Egg McMuffin [820 kcal (3433 kJ), 42 g of fat, 17 g of saturated fat, and 270 mg of cholesterol]. The isocaloric low-fat meal consisted of Kellogg's Frosted Flakes, skim milk, Source fat-free yogurt, a Kellogg's Fruit Loops fruit bar, and Sunny Delight orange juice [830 kcal (3475 kJ), 1g of fat and 15 mg of cholesterol]. The low-fat meal included a 1000 mg sodium supplement to balance sodium intake between the 2 meals (see Table 1). Participants were instructed to resume fasting for a 2-h postprandial period after which testing was conducted. The 2-h time point was selected because of previous studies indicating that postprandial lipemia reaches a peak at 2 h and is sustained at 4 h (24).

Each testing session was ~90 min long. At test initiation, participants' height and weight were measured. All stress-task procedures were conducted in a temperature-controlled (21°C) room. Following instrumentation, participants were instructed to sit quietly for a 5-min baseline period. Following the baseline period, participants engaged in 4 laboratory stress tasks. Impedance and electrocardiograph measurements were recorded continuously during baseline, during each task, and during the subsequent recovery period. Blood pressure was measured at 1-min intervals throughout the test session.

Stress tasks. Tasks were selected on the basis that they are established procedures for inducing sympathetic cardiovascular responses, although through different mechanisms (25–28). Each task was 5-min long and was followed by a 5-min recovery period. The tasks were presented in the following order:

Mental arithmetic. Participants were presented with a series of mathematical subtraction equations with the answers included on a computer screen. They were asked to determine whether the answer to each equation was correct or incorrect. A researcher informed the participant that he or she was being judged on time and accuracy. Each correct answer was followed by a beep emitted from a speaker located below the participant's chair. Each incorrect answer was followed by a noxious blare emanating from the same speaker. The task was designed to change in difficulty according to the participant's ability to maintain a 60% correct rate.

Public speech task. Participants were allowed 2 min to prepare a 5-min speech that would be given to 2 of the researchers on 1 of 3 topics. The choice of topics included: 1) the rights of smokers vs. nonsmokers, 2) cheating on a significant other, and 3) decriminalization of marijuana. A researcher informed each participant that his or her performance would be evaluated on content and quality. Participants were encouraged to continue talking for the full 5 min and were prompted to do so throughout the task. They were also instructed to limit the use of words "umm," "like," and "ahh," and to refrain from using hand gestures.

Arm ischemia. Participants were instructed to repeatedly squeeze a hand dynamometer to their maximum ability at a rate of once per second for 2 min using their dominant hand. Participants then lowered their arm, and a blood pressure cuff was placed with the lower rim positioned

TABLE 1 Nutrient composition of the high-fat and low-fat test meals

Meal type	High-fat	Low-fat
Nutrients		
Energy, kcal (kJ)	820 (3433)	830 (3475)
Total fat, g	42.0	1.3
Saturated fat, g	16.5	0.8
Cholesterol, mg	270.0	15.0
Sodium, mg	2010.0	1904.0
Carbohydrates, g	73.0	172.0
Fiber, g	6.0	1.30
Protein, g	31.0	15.0

~2 cm above the elbow. The cuff was inflated to 100 mm Hg above the participant's baseline systolic blood pressure, thus effectively obstructing blood flow and causing a dull, aching sensation.

Cold pressor. Participants were instructed to immerse their right hand, up to the wrist fold, into 4° ice water for two 2-min periods separated by a 1-min rest interval. They were told to keep their hand motionless and fingers spread for the entire immersion period.

Data analysis

The present study utilized a within-subjects, repeated measures design. Data were analyzed using SPSS, version 14.0, and results were reported as means ± SE. Measures of SBP, DBP, TPR, HR, SV, and CO were averaged for each participant to obtain mean absolute values during baseline and during each of the 4 stress tasks. Differences on any of the baseline CV measures, recorded 2 h following the low-fat vs. the high-fat meal, were determined using Student's *t* tests. A series of task (5) × meal (2) × treatment order (2) repeated measures analyses of variance (RM-ANOVA) were performed on each of the 6 cardiovascular variables (SBP, DBP, TPR, HR, SV, and CO). Meal (high-fat or low-fat) and task (baseline, mental arithmetic, public speech, arm ischemia, and cold pressor) were the within-subjects variables and treatment order was the between-subjects variable. Gender and BMI were included in preliminary models. However, these effects were uniformly nonsignificant and were therefore removed from subsequent models. Tukey's post-hoc analyses were performed on significant interaction effects. To correct for possible violations of the assumption of the homogeneity of covariance, Greenhouse-Geisser corrected probability levels were used for analyses involving >2 levels of a repeated measure. Although mean values were used in the analyses and tables, changes scores (task – baseline) were used in the figures. This was done to allow for clearer representation of the main effects of meal and the meal-task interaction. The alpha level was set at 0.05.

Results

Participant characteristics. All participants had a body mass index (BMI) of < 35 (22.8 ± 3.9) and were normotensive with none displaying a resting blood pressure >140/90 mm Hg. There were no significant differences in any of the baseline CV measures recorded 2 h after consumption of the 2 meals (Table 2).

Diet effects. Results of the RM-ANOVA revealed significant interaction effects between meal and task for SBP (*P* < 0.05) and DBP (*P* < 0.01), reflecting increased reactivity to the stress tasks after intake of the high-fat rather than the low-fat meal but no effects of the meals on baseline values (Fig. 1A), (Fig. 1B). In addition, RM-ANOVA conducted on the hemodynamic variables underlying changes in BP revealed a main effect of meal on TPR (*P* = 0.05), such that overall TPR was greater after subjects consumed the high-fat compared with the low-fat diet (Fig. 1C). There were no significant main effects of meal on HR, SV, or

CO. There were also no significant meal × task interaction effects for TPR, HR, SV, or CO. Unsurprisingly, results of the RM-ANOVA revealed significant main effects of task for SBP, DBP, TPR, HR, SV, and CO variables, reflecting well-known differences in patterns of cardiovascular reactivity associated with the various stressors (29,30).

Discussion

The main finding of the present study was that participants displayed enhanced SBP, DBP, and TPR reactivity to stress tasks following a single high-fat meal in comparison with a low-fat meal. These findings are consistent with results of previous studies, which suggest that a short-term, 2-wk dietary intake of high levels of fat is associated with enhanced blood pressure reactivity (21,31) and that even a single high-fat meal may lead to transient impairments of endothelium-dependent vascular function (5). The results of the present study support and extend these findings by suggesting that a single high-fat meal may lead to heightened cardiovascular reactivity in healthy, normotensive individuals. Specifically, the enhanced reactivity observed by Straznicky et al. (21) may have been a result of changes in cardiovascular risk factors following 2 wk of dietary intervention and/or short-term changes in reactivity due to the acute postprandial lipemia following a high-fat meal. The results of the current study suggest that the effects observed may be attributed, at least in part, to the short-term effects of a high-fat meal.

The reactivity hypothesis suggests that exaggerated cardiovascular responses to stress may be an important predictor of future development of hypertension (3,4,32). Hypertension is one of the most important risk factors in the development of atherosclerosis and coronary heart disease through mechanisms such as endothelial dysfunction and hemodynamic stress (13). The finding that reactivity may be augmented by a single high-fat meal could have important implications in the context of the reactivity hypothesis and could lead to a more thorough understanding of the mechanisms and progression of CVD. The results of the current study emphasize the importance of reducing dietary levels of saturated fat as a vital component in preventing hypertension, atherosclerosis, and CVD.

In the present study, cardiac output reactivity to stress was not significantly changed with the treatments, whereas total peripheral vasoconstriction was significantly increased following the high-fat meal. Vogel et al. (5) proposed that a single high-fat meal is sufficient to induce a transient impairment in coronary artery vasoactivity thus potentially impairing the adaptability of the vascular system to physiological demands during stress. Reduced elasticity of arteries can instigate and enhance blood pressure increases (6). In accordance with this

TABLE 2 SBP, DBP, and other hemodynamic variables in study participants at baseline and during stress tasks after consuming the low-fat and the high-fat test meals¹

Meal	SBP, mm Hg		DBP, mm Hg		HR, bpm		TPR, dyne-s/cm ⁵		SV, mL		CO, L/min	
	LF	HF	LF	HF	LF	HF	LF	HF	LF	H	LF	HF
Baseline	114.0 ± 10.2	115.0 ± 10.9	70.4 ± 5.5	69.7 ± 6.1	70.8 ± 8.5	70.2 ± 8.7	704.7 ± 89.7	699.94 ± 90.3	143.0 ± 37.2	147.2 ± 31.4	10.0 ± 2.3	10.2 ± 1.8
Mathtask	118.8 ± 11.7	123.6 ± 11.9	73.6 ± 5.4	77.9 ± 6.8	74.5 ± 8.1	73.2 ± 9.0	664.5 ± 36.5	693.7 ± 98.6	149.2 ± 39.2	150.7 ± 31.0	10.9 ± 2.3	10.8 ± 1.7
Public speaking	130.3 ± 13.8	135.9 ± 14.7	83.8 ± 9.3	86.9 ± 9.6	81.7 ± 10.5	82.0 ± 10.8	697.8 ± 107.7	754.3 ± 90.4	141.3 ± 32.6	141.1 ± 28.1	11.4 ± 2.6	11.3 ± 1.7
Arm ischemia	118.4 ± 13.8	124.5 ± 12.3	75.3 ± 7.5	80.4 ± 7.2	68.2 ± 7.7	68.5 ± 7.0	696.9 ± 108.6	740.6 ± 92.6	122.5 ± 29.8	157.2 ± 32.6	10.3 ± 2.0	10.6 ± 1.6
Cold pressor	126.6 ± 11.9	131.3 ± 10.7	81.1 ± 7.2	85.9 ± 7.1	73.7 ± 8.8	75.2 ± 8.7	750.5 ± 86.2	795.4 ± 107.0	141.0 ± 34.9	140.2 ± 31.0	10.2 ± 2.1	10.3 ± 1.7

¹ Values are means ± SD, *n* = 30.

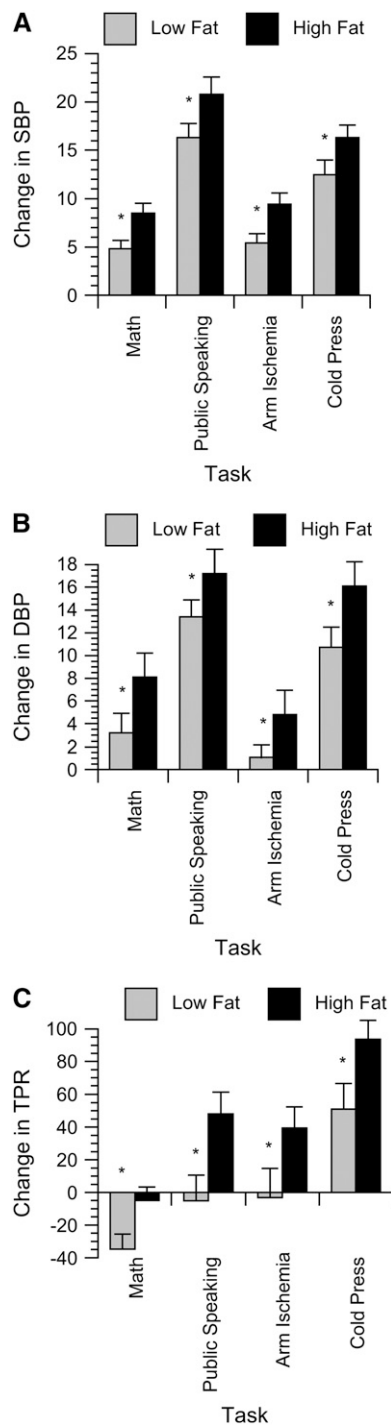


Figure 1 Change in SBP (A), DBP (B) and total peripheral resistance (C) in healthy young adults during the 4 stress tasks after consuming a high-fat (42 g) and a low-fat (1 g) meal on 2 separate occasions. Values are means \pm SEM, $n = 30$. *Different from after the high-fat meal, $P < 0.05$.

theory, the observed pattern of underlying hemodynamics in the current study suggests that a high-fat meal may result in increased vascular constriction as indicated by increased TPR reactivity. Enhanced TPR reactivity could represent a possible mechanism through which blood pressure changes were affected. Regardless of its origin, an increase in blood pressure is mediated by an increase in cardiac output and/or TPR (4).

Participants exhibited varied response patterns to the 4 different stress tasks. Variations in the stress response to

different tasks have been described in previous research (29,30). Therefore, the observed main effect of tasks in the present study was not surprising. Although the tasks were designed to elicit different patterns of reactivity (e.g., the mental arithmetic task often elicits a greater change in heart rate, whereas blood pressure reactivity is often greater during the cold pressor task), a consistent hemodynamic response to the meals emerged. Regardless of task, enhanced blood pressure reactivity following the high-fat meal was accompanied by increases in TPR. Taking into account both impedance and blood pressure parameters, as well as reactivity across multiple stress tasks, allowed for a more generalized reactivity profile.

Study limitations. Based on the observations by Vogel et al. (5), it seems reasonable to assume that the effects of a single high-fat meal would be transient in nature. However, the longevity of enhanced reactivity was not examined in the present study. This issue may need to be addressed in future research.

The present study utilized a large fat load (42 g). Although high-fat meals are commonly consumed, especially in fast-food restaurants across North America (33), this study did not attempt to determine whether meals with smaller fat loads would yield the same effects on cardiovascular reactivity. The 2 meals tested in this experiment were reasonably well matched for sodium and energy, but they differed on fat and cholesterol content. Although we have attributed exaggerated cardiovascular reactivity to the high-fat content of the fast-food meal, it is also possible that dietary cholesterol contributed to this effect, and future research should attempt to clarify the mechanisms involved. Furthermore, although there are some recent animal findings that examine the impact of saturated fat vs. unsaturated fat on cardiac hypertrophy (34–36), this study did not examine whether a high-fat meal containing less saturated, more unsaturated, and polyunsaturated fats may have the same effect on the observed cardiovascular reactivity parameters.

Although enhanced postprandial blood pressure and TPR responses correlated with the consumption of the high-fat meal, the specific triglyceride or lipoprotein levels responsible for the observed effect were not identified or measured. These data would also be valuable to confirm the fasting state of subjects prior to consuming the test meals.

In conclusion, the results of this study suggest that even a single high-fat meal may be associated with heightened cardiovascular reactivity in healthy, normotensive individuals. The observed association contributes further insight into the pathways through which a high-fat diet may affect cardiovascular function, and provides new directions for future research in this area.

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