Resting Plasma Lipids and Cardiovascular Reactivity to Acute Psychological Stress

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Contents

Articles

Different Patterns of Heart Rate Variability During Acute Withdrawal in Alcohol Dependent Patients With and Without Comorbid Anxiety and/or Depression
Tien-Yu Chen, Chuan-Chia Chang, Nian-Sheng Tzeng, Terry B. J. Kuo, San-Yuan Huang, Ru-Band Lu, and Hsin-An Chang 87

Resting Plasma Lipids and Cardiovascular Reactivity to Acute Psychological Stress
Anthony W. Austin, Michael R. Kushnick, Michael J. Knutson, Mark L. McGlynn, and Stephen M. Patterson 99

Individual Differences in Personality Traits: Perfectionism and the Brain Structure
A. Karimizadeh, Amin Mahnam, M. R. Yazdehi, and M. A. Besharat 107

Psychological Response to Sound Stimuli Evaluated by EEG: Joint Consideration of AAE Model and Comfort Vector Model
Xi Chen, Isao Takahashi, Yoshimitsu Okita, Hisashi Hirata, and Toshifumi Sugiura .112

Oral Immune Activation by Disgust and Disease-Related Pictures
Richard J. Stevenson, Deborah Hodgson, Megan J. Oaten, Luba Sominsky, Mehmet Mahmut, and Trevor I. Case 119
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Resting Plasma Lipids and Cardiovascular Reactivity to Acute Psychological Stress

Anthony W. Austin,1 Michael R. Kushnick,2 Michael J. Knutson,2 Mark L. McGlynn,2 and Stephen M. Patterson3

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Abstract. Prior research suggests that hyperlipidemia is associated with elevated blood pressure responses to acute stress but whether lipid levels influence underlying cardiac and vascular determinants of blood pressure during stress is not known. Thus, we examined whether lipids were associated with stress-induced blood pressure responses and responses of stroke volume (SV), cardiac output (CO), and total peripheral resistance (TPR). In 19 healthy university students (15 men), blood was drawn to measure lipid levels (triglycerides, low-density lipoprotein cholesterol [LDL-c], high-density lipoprotein cholesterol [HDL-c], total cholesterol) after a 10-min rest period. Participants then completed a 6-min mental arithmetic stressor and a 3-min cold pressor (separated by a 10-min recovery). This procedure was repeated twice, approximately 6 weeks apart. Lipids and hemodynamic values were averaged across the two sessions. Multiple linear regression analyses revealed that a model including LDL-c, HDL-c, and triglycerides significantly predicted diastolic blood pressure (DBP), \( R^2_{\text{adj}} = .45, \ p = .007 \), and systolic blood pressure (SBP) cold pressor reactivity, \( R^2_{\text{adj}} = .35, \ p = .023 \). Individually, only LDL-c significantly predicted DBP, \( b = .64, \ p = .003 \), and SBP cold pressor reactivity, \( b = .64, \ p = .005 \). The same model marginally predicted CO, \( R^2_{\text{adj}} = .24, \ p = .069 \), and TPR, \( R^2_{\text{adj}} = .21, \ p = .091 \), reactivity to mental arithmetic, but only triglycerides were independently associated with CO, \( b = .63, \ p = .012 \), and TPR, \( b = .54, \ p = .029 \) reactivity. Lipids were not associated with heart rate (HR) or SV reactivity. LDL-c was positively associated with the blood pressure response to the cold pressor, whereas triglycerides were positively and negatively associated with the TPR and CO responses, respectively, to mental arithmetic. Endothelial dysfunction and greater release of vasoconstrictors in those with high lipids may explain these relationships.

Keywords: cholesterol, mental stress, stress reactivity, hemodynamics, impedance cardiography

Cardiovascular disease is the leading cause of death in the United States. Excess blood lipid levels, including triglycerides, nonesterified fatty acids, and low-density lipoproteins (LDL-c), have been implicated in the initiation and progression of atherosclerosis (Steinberg, 2004), which may result in a decrease in vessel diameter and elasticity and therefore, have the potential to negatively impact blood pressure, leading to hypertension. A primary candidate mechanism linking high blood lipids to hypertension is endothelial dysfunction. Vascular tone and the endothelium are disrupted by hyperlipidemia through impairments in endothelium-dependent vasodilation (Hamasaki et al., 2000; Kim, Montagnani, Chandrasekran, & Quon, 2012). In turn, endothelial dysfunction is a hallmark feature of hypertension and provides prognostic value for future cardiovascular events (Schulz, Gori, & Munzel, 2011; Tang & Vanhoutte, 2010).

The cardiovascular reactivity hypothesis suggests that exaggerated cardiovascular responses to acute psychological stress increase risk for the development of hypertension and cardiovascular disease (Carroll, 2011; Phillips & Hughes, 2011; Treiber et al., 2003). Supporting this hypothesis, individuals with high blood pressure (Deter, Blecher, & Weber, 2007; Flaa, Mundal, Eide, Kjeldsen, & Rostrup, 2006) or family history of cardiovascular disease risk (Pierce, Grim, & King, 2005; Wright, O’Donnell, Brydon, Wardle, & Steptoe, 2007) demonstrate relatively greater blood pressure responses to and poorer recovery from laboratory stressors. Moreover, heightened cardiovascular reactivity and blunted recovery are associated with future incidence of hypertension and carotid intima-media thickness (Chida & Steptoe, 2010).

Given that both elevated lipids and cardiovascular reactivity may impose harm on the cardiovascular system, it is possible that they have interactive effects. Though little research has been conducted on whether diet influences cardiovascular reactivity, some studies suggest that diets or meals high in saturated fat are associated with increased cardiovascular reactivity. For example, in a 2-week crossover study in healthy normotensive participants, consuming...
a diet with a low polyunsaturated-to-saturated fat ratio was associated with a greater systolic blood pressure (SBP) response to the cold pressor compared to consuming a diet with a high polyunsaturated-to-saturated fat ratio (Straznicky, Louis, McGrade, & Howes, 1993). Moreover, compared to a low-fat meal, consumption of a high-fat meal prior to mental stress is associated with increased blood pressure response tasks (Faulk & Bartholomew, 2012; Jakulj et al., 2007). If dietary fat is associated with cardiovascular reactivity, the relationship may be mediated by plasma lipid levels. In individuals with normal (Minami et al., 1999) and high normal blood pressure (Borghi et al., 2004), those with hyperlipidemia had greater blood pressure responses to mental stress than individuals with normal lipid levels. Finally, cholesterol-lowering statin therapy mitigates the blood pressure response to acute psychological stress in patients with hyperlipidemia (Minami et al., 2003), providing converging evidence that cholesterol levels impact blood pressure responses to mental stress. However, previous studies examined those with high versus low lipids, and it is not known whether lipids across the normal range have a linear relationship with blood pressure responses.

Only one known study has examined the influence of serum cholesterol on measures of heart contractility during acute stress (Clark, Moore, & Adams, 1998). Total cholesterol was negatively correlated with stroke volume (SV) and positively correlated with ejection velocity index in African Americans after watching a racially noxious video. However, whether change in heart contractility from baseline was associated with total cholesterol was not reported. Moreover, only one known study has examined whether lipid levels are associated with total peripheral resistance (TPR) or cardiac output (CO) changes during stress (van Doornen, Snieder, & Boomsma, 1998). Specifically, in women, but not in men, CO response to mental stress was positively associated with high-density lipoprotein cholesterol (HDL-c) in women and TPR response was associated with total cholesterol.

The primary objective of this study was to examine whether cholesterol was associated with stress-induced blood pressure responses and responses of the underlying cardiac and vascular components of blood pressure, (i.e., SV, CO, and TPR) in young, healthy individuals. We hypothesized that triglycerides and LDL-c levels would be associated with blood pressure, SV, CO, and TPR reactivity and that lipids would at least partially statistically be associated with blood pressure, SV, CO, and TPR reactivity and that lipids would at least partially statistically be associated with blood pressure, SV, CO, and TPR reactivity and that lipids would at least partially statistically be associated with blood pressure, SV, CO, and TPR responses.

Materials and Methods

Participants

Twenty college students (15 men; 5 women) between the ages of 18 and 21 were recruited. Prior to the study, participants abstained from food and drink for 4 hr, exercise for 12 hr, and alcohol for 24 hr. One woman’s cardiovascular data was unusable due to equipment malfunction, leaving 19 (15 men; 4 women) for analyses. Exclusion criteria were: (a) personal history of angina, heart disease, diabetes, or other chronic physical diseases, (b) use of prescription medications that would affect blood pressure (e.g., beta blockers, diuretics), (c) diet prescribed by a physician, (e) current smoker, (f) obesity, and (g) women who were pregnant, nursing, or taking oral contraceptives. Written, informed consent was given by the participants. Institutional Review Board approval was obtained from Ohio University.

Procedure

Initially, participants completed a health history questionnaire to verify eligibility. Then, weight (kg) was measured on a standard hospital balance beam scale and height (cm) was measured with a stadiometer, and BMI was calculated. Next, tetrapolar band electrodes and bipolar silver-silver chloride electrodes were placed on the participant for electrocardiogram and cardiac impedance assessments, respectively. Participants were then taken to a testing room where they sat on a comfortable chair. A blood pressure cuff was placed on the left arm and participants were connected to impedance cardiograph equipment. Participants rested quietly for a 10-min baseline period. At the end of baseline, a blood sample was collected for lipid analysis. A 6-min mental arithmetic stressor was presented next, followed by a 10-min recovery period and a 2.5-min cold pressor test. Cardiovascular measures were recorded during the final 3 min of baseline and throughout mental arithmetic, recovery, and the cold pressor. Systolic (SBP) and diastolic (DBP) blood pressure and heart rate (HR) were measured each min, whereas CO, SV, and TPR were measured continuously. Participants underwent the same protocol twice, approximately 6 weeks apart at the same time of day. We aggregated cardiovascular and lipid data across the two sessions in order to obtain more robust individual differences in cardiovascular responses (Kamarck, Debski, & Manuck, 2000).

Materials

Blood Pressure and Heart Rate

SBP and DBP were monitored using a Dinamap™ Compact T automated sphygmomanometer applied over the upper part of the left arm. Electrocardiograms using three bipolar silver-silver chloride electrodes were utilized to measure HR.

Impedance Cardiography

CO and SV were measured using the Minnesota Impedance Cardiograph Model 304B with a tetrapolar band electrode configuration on the neck and thorax. The method is
noninvasive and relates aortic flow to changes in thoracic resistance. The Kubicek formula was used to calculate SV. CO (l/min) was calculated as the product of mean SV (mL) and HR during each minute. Analysis of impedance cardiography used the Cardiac Output Program (COP) (Bioimpedance Technology, Inc., Lafayette, Co, USA) run on a microcomputer. Blood pressure readings were manually entered into the COP after all subject data was collected. The program calculates mean arterial pressure (MAP) using the formula: MAP = ((SBP − DBP)/3) + DBP. Total peripheral resistance (TPR) is then calculated as follows: TPR (dyne-seconds/cm$^5$) = (MAP/CO) × 80.

**Lipid Analyses**

Blood was drawn into a 7 mL vacutainer tube (Beckson Dickson) and centrifuged immediately at 3,000g for 10 min to obtain plasma. Plasma was aliquoted into 1.5 mL micro-centrifuge tubes (Eppendorf) and frozen at −80 °C until assayed in one batch per analyte. Total cholesterol and triglycerides were assayed using commercially available kits (Infinity™, Thermo DMA, Louisville, CO). HDL-c was measured according to manufacturer’s specifications using standard spectrophotometry (Evolution 300, Thermo Scientific). LDL-c was determined with the Friedewald formula. Coefficients of variation were less than 5% for all lipid measures.

**Task Descriptions**

Two stress tasks were used, an active coping (mental arithmetic) and a passive coping (cold pressor).

**Mental Arithmetic**

Participants performed a 6-min mental arithmetic task with verbal harassment from the experimenter. Each participant was instructed to subtract aloud by sevens from a prerecorded four-digit number as quickly and as accurately as possible. The experimenter instructed the participant to be more accurate and to work more quickly throughout the task, regardless of the participant’s performance.

**Cold Pressor Test**

Participants performed a cold pressor test in which they submerged their right hand into a bucket of ice water maintained at 4 °C (±1 °C) for 2.5 min. The cold pressor was administered at the end of the testing session to minimize carryover effects.

**Analyses**

Cardiovascular data (SBP, DBP, HR, SV, CO, TPR) was averaged during each period (i.e., baseline, math, recovery, cold pressor) during each session. In the first session, one participant withdrew participation prior to the cold pressor. Therefore, the mean difference between cold pressor and recovery was computed for the remaining participants for each cardiovascular measure. The mean difference was then added to the recovery values of each cardiovascular measure for the participant with missing cold pressor data. Values for cardiovascular and lipid data were averaged across the two sessions. A series of repeated measures analyses were conducted to verify whether the stressors elicited the expected cardiovascular changes. Then, to assess whether cholesterol or triglycerides accounted for any of the variability in cardiovascular reactivity, repeated measures analyses were repeated with covariate adjustment for total cholesterol, LDL-c, HDL-c, and triglycerides, after mean centering (Delaney & Maxwell, 1981). As determined by the Kolmogorov-Smirnov test all lipid and cardiovascular measures were normally distributed. The Huynh-Feldt correction was applied to account for any violations of the sphericity assumption and adjusted degrees of freedom are reported when appropriate.

We also calculated Pearson’s correlations between each lipid measure and change scores of each cardiovascular measure using the aggregated data. Change scores were calculated as math minus baseline and cold pressor minus baseline. Then, a series of multiple linear regressions were conducted in which reactivity change scores (math minus baseline and cold pressor minus baseline) were regressed on lipid values. No multivariate outliers were detected with the Mahalanobis distance procedure. Data were analyzed using SPSS (version 21.0, Chicago, IL). All tests were two-tailed with level of significance set at $p < .05$. However, given the small sample size, we also report marginally significant results ($p < .10$) in an effort to guide hypothesis testing for future studies.

**Results**

Participant characteristics by sex are presented in Table 1. Mean BMI was 23.46 ($SD = 2.60$) and all participants had a BMI less than 30. Overall, LDL-c levels were borderline high (132.02 mg/dL ± 24.93), HDL-c levels were in the healthy range (51.19 mg/dL ± 15.82), total cholesterol was in the high range of normal (198.35 mg/dL ± 30.67), and triglycerides were in the healthy range (7,546 mg/dL ± 17.21). No sex differences were observed for age, BMI, or any lipid measure.

**Preliminary Analyses**

With the exception of SV, the repeated measures analyses violated the sphericity assumption. Therefore, the Huynh-Feldt adjusted degrees of freedom are reported. Without adjustment for lipid levels, repeated measures analyses indicated significant main effects of period for HR, F(2.48, 44.57) = 41.82, $p < .001$, DBP, F(2.42, 43.58) = 30.46, $p < .001$, SBP, F(2.34, 42.19) = 19.54, $p < .001$,
Table 1. Participant characteristics

<table>
<thead>
<tr>
<th></th>
<th>Females (N = 4)</th>
<th>Males (N = 15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age in years</td>
<td>18.75 (0.96) [18, 20]</td>
<td>19.13 (1.13) [18, 21]</td>
</tr>
<tr>
<td>BMI</td>
<td>23.82 (1.79) [22.01, 25.54]</td>
<td>23.37 (2.82) [18.83, 28.55]</td>
</tr>
<tr>
<td>Baseline SBP</td>
<td>113.13 (5.86) [105.83, 119.67]</td>
<td>122.75 (6.83) [112.83, 138.50]</td>
</tr>
<tr>
<td>Baseline DBP</td>
<td>61.08 (4.25) [55.00, 64.17]</td>
<td>62.97 (7.74) [47.00, 74.00]</td>
</tr>
<tr>
<td>Baseline HR</td>
<td>64.21 (13.29) [48.00, 80.17]</td>
<td>60.06 (7.62) [49.17, 77.83]</td>
</tr>
<tr>
<td>HDL-c (mg/dL)</td>
<td>58.00 (23.45) [36.28, 86.03]</td>
<td>49.37 (13.68) [29.31, 74.15]</td>
</tr>
<tr>
<td>LDL-c (mg/dL)</td>
<td>137.69 (15.35) [117.25, 154.45]</td>
<td>130.58 (27.15) [77.20, 160.60]</td>
</tr>
<tr>
<td>Total cholesterol (mg/dL)</td>
<td>210.16 (13.96) [192.04, 222.62]</td>
<td>195.21 (33.41) [133.97, 246.08]</td>
</tr>
<tr>
<td>Triglycerides (mg/dL)</td>
<td>72.44 (13.86) [54.30, 85.10]</td>
<td>76.27 (18.34) [45.02, 106.30]</td>
</tr>
</tbody>
</table>

Notes. BMI = body mass index; HDL-c = high-density lipoprotein cholesterol; LDL-c = low-density lipoprotein cholesterol.

Table 2. Correlations between cardiovascular reactivity and lipid markers

<table>
<thead>
<tr>
<th></th>
<th>Total-c</th>
<th>LDL-c</th>
<th>HDL-c</th>
<th>Triglycerides</th>
</tr>
</thead>
<tbody>
<tr>
<td>Math reactivity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR</td>
<td>-.16</td>
<td>-.30</td>
<td>.23</td>
<td>-.33</td>
</tr>
<tr>
<td>DBP</td>
<td>-.13</td>
<td>-.26</td>
<td>.17</td>
<td>-.06</td>
</tr>
<tr>
<td>SBP</td>
<td>-.10</td>
<td>-.22</td>
<td>.19</td>
<td>-.17</td>
</tr>
<tr>
<td>CO</td>
<td>-.15</td>
<td>-.15</td>
<td>.06</td>
<td>-.60**</td>
</tr>
<tr>
<td>SV</td>
<td>-.08</td>
<td>.14</td>
<td>-.35</td>
<td>-.09</td>
</tr>
<tr>
<td>TPR</td>
<td>-.09</td>
<td>-.25</td>
<td>.14</td>
<td>.37</td>
</tr>
<tr>
<td>Cold pressor reactivity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR</td>
<td>.24</td>
<td>.22</td>
<td>.01</td>
<td>.45*</td>
</tr>
<tr>
<td>DBP</td>
<td>.64**</td>
<td>.59**</td>
<td>.36</td>
<td>-.25</td>
</tr>
<tr>
<td>SBP</td>
<td>.62**</td>
<td>.61**</td>
<td>.26</td>
<td>-.12</td>
</tr>
<tr>
<td>CO</td>
<td>-.12</td>
<td>-.13</td>
<td>.01</td>
<td>-.20</td>
</tr>
<tr>
<td>SV</td>
<td>-.34</td>
<td>-.27</td>
<td>-.16</td>
<td>-.29</td>
</tr>
<tr>
<td>TPR</td>
<td>0.01</td>
<td>-.01</td>
<td>.06</td>
<td>-.11</td>
</tr>
</tbody>
</table>

Notes. *p = .052, **p < .01. HDL-c = high-density lipoprotein cholesterol; LDL-c = low-density lipoprotein cholesterol; HR = heart rate; DBP = diastolic blood pressure; SBP = systolic blood pressure; CO = cardiac output; SV = stroke volume; TPR = total peripheral resistance.

Multiple Linear Regression Analyses

Total cholesterol was highly correlated with LDL-c, r = .87. Therefore, total cholesterol was excluded from the multiple linear regression analyses to avoid multicollinearity. The overall model for DBP cold pressor reactivity was significant, F(3, 18) = 5.91, p = .007, R² = .54, R² adjusted = .45. That is, 45% of the variance in DBP cold pressor reactivity was explained by the model. However, LDL-c made the only significant contribution to the prediction of DBP cold pressor reactivity, β = .64, t = 3.56, p = .003. The squared semipartial correlation between LDL-c and DBP cold pressor reactivity was .386, indicating that 38.6% of the variance in DBP cold pressor reactivity was uniquely explained by LDL-c. The scatterplot in Figure 1 demonstrates the linear relationship between LDL-c and DBP cold pressor reactivity.

For SBP cold pressor reactivity, the overall model was significant, F(3, 18) = 4.26, p = .023, R² = .46, R² adjusted = .35. That is, 35% of the variance in SBP cold pressor reactivity was explained by the model. Similar to SV, F(3, 54) = 21.96, p < .001, CO, F(2.42, 43.52) = 8.58, p < .001, and TPR, F(1.25, 22.45) = 11.85, p = .001. Specifically, SBP, DBP, and HR increased and SV decreased significantly during both math and the cold pressor, whereas CO increased only during math and TPR increased only during the cold pressor. Adjusting for lipid levels did not influence these relationships.

Table 2 shows the correlations between change scores and lipid values. HR cold pressor reactivity was marginally correlated with triglycerides, r = .45, p = .052. DBP cold pressor reactivity was correlated with total cholesterol, r = .64, p = .003, and LDL-c, r = .59, p = .008. Similar correlations were observed for SBP cold pressor reactivity and total cholesterol, r = .62, p = .005, and LDL-c, r = .61, p = .006. CO math reactivity was negatively correlated with triglycerides, r = -.60, p = .007. No significant correlations were observed between lipid values and HR, SV, and TPR reactivity.

Figure 1. Scatterplot of LDL cholesterol and DBP reactivity to the cold pressor.
ally significant, \( F(3, 18) = 2.60, \ p = .091 \), \( R^2 = .34 \), \( R_{\text{adjusted}}^2 = .21 \), explaining 21% of the variance in TPR math reactivity. Triglycerides made a significant predictive contribution, \( \beta = .54, t = 2.41, p = .029 \), whereas LDL-c made a marginally significant predictive contribution, \( \beta = -.39, t = -1.78, p = .096 \). The squared semipartial correlation coefficients for triglycerides and LDL-c were .255 and .138, respectively. That is, triglycerides uniquely explained 25.5% of the variance in TPR math reactivity and LDL-c uniquely explained 13.8% of the variance.

Finally, the models for HR, DBP, SBP, and SV math reactivity and HR, CO, SV, and TPR cold pressor reactivity were not significant. All regression models are summarized in Table 3.

Table 3. Multiple linear regression models predicting cardiovascular reactivity to mental arithmetic and the cold pressor

<table>
<thead>
<tr>
<th></th>
<th>Overall model</th>
<th>LDL-c</th>
<th>HDL-c</th>
<th>Triglycerides</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>( R^2 )</td>
<td>( R_{\text{adj}}^2 )</td>
<td>( F(3, 18) )</td>
<td>( p )</td>
</tr>
<tr>
<td><strong>Math</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR</td>
<td>.20</td>
<td>.03</td>
<td>1.21</td>
<td>.34</td>
</tr>
<tr>
<td>DBP</td>
<td>.11</td>
<td>-.07</td>
<td>.59</td>
<td>.63</td>
</tr>
<tr>
<td>SBP</td>
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<td>-.09</td>
<td>.52</td>
<td>.68</td>
</tr>
<tr>
<td>CO</td>
<td>.37</td>
<td>.24</td>
<td>2.91</td>
<td>.07</td>
</tr>
<tr>
<td>SV</td>
<td>.21</td>
<td>.06</td>
<td>1.36</td>
<td>.29</td>
</tr>
<tr>
<td>TPR</td>
<td>.34</td>
<td>.21</td>
<td>2.60</td>
<td>.09</td>
</tr>
</tbody>
</table>

**Cold pressor**

|                |               |       |       |               |
| HR             | .24           | .09   | 1.56  | .24           | .11        | .47    | .01  | .65         | .14          | .59    | .02   | .57       | .47         | 1.93    | .19   | .07       |               |
| DBP            | **.54**       | **.45** | **5.91** | **.007**  | **.64**  | **3.56** | **.39** | **.003**  | **.23**       | **1.27** | .05   | **.23**  | -.32        | **-1.69** | .09   | **.11**   |               |
| SBP            | **.46**       | **.35** | **4.25** | **.023**  | **.64**  | **3.28** | **.39** | **.005**  | **.16**       | **1.81** | .02   | **.43**  | -.21        | **-1.03** | .04   | **.32**   |               |
| CO             | .05           | -.14  | .25   | .86           | -.09       | -.33   | .01  | .74         | -.04         | -.15   | .001  | .88       | -.19        | -.70    | .03   | .50       |               |
| SV             | .18           | .02   | 1.12  | .37           | -.19       | -.78   | .03  | .34         | -.24         | -.98   | .05   | .34       | -.32        | -.12    | .09   | .22       |               |
| TPR            | .01           | -.18  | .07   | .98           | .01        | .04    | .000 | .97        | .01          | .12    | .01   | .91       | -.10        | -.37    | .009  | .72       |               |

Notes. Bold values are at \( p < .01 \). Italicized values are at \( p < .10 \). LDL-c = low-density lipoprotein cholesterol; HDL-c = high-density lipoprotein cholesterol; HR = heart rate; DBP = diastolic blood pressure; SBP = systolic blood pressure; CO = cardiac output; SV = stroke volume; TPR = total peripheral resistance.

Figure 2. Scatterplot of LDL cholesterol and SBP reactivity to the cold pressor.

Discussion

Unsurprisingly, mental arithmetic and the cold pressor task evoked robust cardiovascular changes in our study and these results were unchanged when adjusting for resting lipids in our ANCOVAs, suggesting that cardiovascular responses during acute psychological stress are independent of blood lipids. Nevertheless, as shown with our multiple linear regression analyses, LDL-c was associated with both the DBP and SBP responses to the cold pressor. To a lesser extent, triglycerides were associated with the CO and TPR responses to mental arithmetic.
Our results support older work showing that blood pressure responses were greater in those with high cholesterol ( McKinney et al., 1987 ). However, this previous work also reported that those with high triglycerides or lower HDL levels had a greater blood pressure response. Conversely, we did not observe an association between blood pressure response to the cold pressor and triglycerides or HDL-c. Similarly, blood pressure reactivity during the mental arithmetic task was unrelated to any lipid measure. However, previous research reporting that high lipids were associated with the blood pressure response to mental arithmetic split participants into two different groups based on total cholesterol levels, either using median split ( Fredrikson, Lundberg, & Tuomisto, 1991 ) or standard cutoffs ( Borghi et al., 2004; Minami et al., 1999 ). In our sample, there was high multicollinearity between total cholesterol and LDL-c. We removed total cholesterol and kept LDL-c in the model in order to examine independent associations of LDL-c and HDL-c. Furthermore, our sample was not large enough to split participants into two groups like in previous studies. Therefore, it is possible that if we had a larger sample and were able to split it into high and low total or LDL-c cholesterol groups, we would have observed significant differences in mental arithmetic reactivity. Additionally, it is possible that cholesterol would have accounted for cardiovascular reactivity in our ANCOVAs with a larger sample. Elevated blood pressure response to the cold pressor in those with high LDL-c could be due, at least in part, to the potentially negative impact of LDL-c on peripheral vascular tone. High LDL-c seems to reduce vasodilatory capabilities, possibly through upregulated gene expression of angiotensin II Type 1 ( AT1 ) receptors, which may modulate blood pressure reactivity ( Borghi et al., 2004 ). Angiotensin II administration results in greater blood pressure ( Campbell, 2013; Nickenig et al., 1999 ), whereas statins downregulate AT1 receptor density and eliminate the elevated blood pressure associated with angiotensin II administration ( Nickenig et al., 1999 ).

Although no associations between lipids and blood pressure responses to mental arithmetic were observed, triglycerides were predictors of CO and TPR responses. We acknowledge that the overall models for CO and TPR responses were only marginally significant, but this was likely due to low power from the small sample size. However, the amount of variance explained by the models was substantial (24% of the variance in CO math reactivity and 21% of the variance in TPR math reactivity). Therefore, the models would likely have emerged as significant with a larger sample.

Triglycerides were negatively associated with CO math reactivity and positively associated with TPR reactivity. These opposite associations would be expected given that CO and TPR are inversely related. As with LDL-c and blood pressure response to the cold pressor, elevated triglycerides may be associated with increased TPR response to mental arithmetic through endothelial dysfunction. Hypertriglyceridemia was inversely associated with the dilation response of small arteries in individuals at intermediate to high cardiovascular risk ( Ferre et al., 2011 ). The increase in resistance, in turn, makes the heart work harder in order to maintain elevated blood pressure during stress, resulting in elevated cardiac output.

It is established that blood lipids increase during stress ( Dimsdale & Herd, 1982; Patterson, Matthews, Allen, & Owens, 1995; Stoney, Bausserman, Ni aura, Marcus, & Flynn, 1999 ), although this effect is largely due to increases in hemoconcentration ( Patterson et al., 1995 ). Given that elevated lipids are associated with blood pressure reactivity, reactivity may provide a mechanism linking high lipid levels to coronary heart disease. Unfortunately for proponents of the cardiovascular reactivity hypothesis, little evidence exists supporting the generalizability of reactivity to laboratory tasks to the “real world” and causal explanations for a link between reactivity in the laboratory and disease states remain elusive ( Schwartz et al., 2003 ). That is, the ecological validity of laboratory stressors appears dubious. On the other hand, tasks that involve social competence ( e.g., anger recall, Trier Social Stress Test ) may be more characteristic of daily life stressors than traditional laboratory stressors ( Linden, Rutledge, & Con, 1998 ), which is supported by the finding that greater occupational stress is associated with elevated LDL-c and triglycerides and lowered HDL-c ( Dijndjic et al., 2013 ). Future studies should examine whether lipids are differentially associated with reactivity to traditional laboratory stressors versus social competence stressors.

Our study had notable strengths. To our knowledge, this is the first study to examine whether lipids across the normal range are associated with cardiovascular reactivity, as well as the first to examine whether lipids are associated with CO and TPR reactivity. Despite its strengths, some limitations must be addressed. First, due to the small sample size and the number of tests performed, it is possible that our results were due to random effects, and our results should be interpreted with caution. However, the mental arithmetic task and the cold pressor test likely produced effects independent of each other, thereby reducing the number of relative comparisons. Additionally, it has convincingly been argued that adjusting for multiple comparisons is only necessary when one believes that the universal null hypothesis is indeed correct and results are completely due to chance ( Rothman, 1990 ). Given previous research in this area, it is unlikely that our findings were completely due to chance. Second, though both testing sessions occurred at the same time of day for each participant, we could not ensure that testing occurred at the same time of day across participants, due to participant schedules. Therefore, it is possible that differences in circadian rhythms in blood pressure or lipids could have modulated our findings. Finally, several unmeasured variables could have influenced the results observed in our study. Cardiovascular reactivity to stress is probably the result of several, interacting factors. For instance, hostility ( Suls, 2013 ), physical fitness ( Rimmele et al., 2009 ), chronic life stress ( Low, Salomon, & Matthews, 2009 ), depression ( Salomon, Clift, Karlsdottir, & Rottenberg, 2009 ), and social support ( Schwerdtfeger & Schlager, 2011 ) are all associated with reactivity. Future research with larger sample sizes should take into account these potential moderating variables.
In conclusion, LDL-c was positively associated with the blood pressure response to the cold pressor, whereas triglycerides were positively associated with the TPR response and negatively associated with the CO response to mental arithmetic. Over time, poorer cardiovascular responding to psychological stress in those with poor lipid profiles could lead to worse cardiovascular outcomes, potentially through endothelial dysfunction or greater levels of vasoconstrictors such as angiotensin II. More research is needed to untangle the interlinking mechanisms of stress, lipids, and cardiovascular responses.

Ethics and Disclosure Statements

Informed consent was obtained from all participants. The study conformed to Ohio Universities Policy on human subjects research and was approved by the Ethics Committee at Ohio University. The authors disclose no actual or potential conflicts of interest including any financial, personal, or other relationships with other people or organizations that could inappropriately influence (bias) their work.

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