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RELATIONSHIP OF CARDIOVASCULAR REACTIVITY AND ANGER EXPRESSION TO SERUM LIPID CONCENTRATIONS IN HEALTHY YOUNG MEN

SHARI R. WALDSTEIN,* JOANNA M. POLEFRONE,* ELIZABETH A. BACHEN,* MATTHEW F. MULDOON,† JAY R. KAPLAN,‡ and STEPHEN B. MANUCK*

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Abstract—The relationship between behaviorally evoked cardiovascular reactivity, preferred mode of anger expression, and serum lipid concentrations was examined in 63 healthy, young adult males. Subjects derived from three studies, each evaluating cardiovascular response to laboratory stressors. All participants completed the Spielberger Anger Expression Scale and provided fasting blood samples for lipid determinations. A significant negative correlation, calculated by meta-analytic procedures, was noted between a baseline-free measure of heart rate reactivity and high density lipoprotein-cholesterol (HDL-C) concentrations ($r = -0.26$, $p = 0.05$). However, the previously reported relationship between cardiovascular reactivity and elevated total serum cholesterol (TSC) was not found. Additionally, men scoring high on a self-report measure of the tendency to express anger outwardly had significantly higher HDL-C concentrations than men scoring low on this measure ($r = 0.30$, $p = 0.02$); when subjects were stratified by level of cardiovascular reactivity, this relationship was apparent only among those showing the greatest magnitude of heart rate and blood pressure responses to acute mental stress.

INTRODUCTION

VARIABILITY of serum lipid concentrations has been associated with both psychosocial factors and the psychophysiologic response characteristics of individuals. For example, relative elevations in total serum cholesterol (TSC) have been found in persons exposed to a variety of psychological stressors (e.g., perceived life events) [1] and among individuals scoring high on self-report or observational measures of Type A behavior and hostility [2, 3]. Among hypertensive patients, persons with a history of coronary heart disease, and healthy middle-aged adults, individual differences in cardiovascular reactivity to mental stress have also been found to correlate positively with fasting TSC concentrations, low-density lipoprotein-cholesterol (LDL-C), and triglycerides, and inversely with high-density lipoprotein cholesterol (HDL-C) [4–8]. That these associations may be mediated by the well-established metabolic effects of circulating catecholamines is suggested by the observation that infusion of catecholamines in physiologic doses raises TSC in dogs and nonhuman primates, [9–11] and induces lipolysis in humans [12].

In this paper, we further examine the relationship between cardiovascular arousal, preferred mode of anger expression, and serum lipid concentrations in healthy, young adult men. In addition, since self-reported hostility has been shown to

*Behavioral Physiology Laboratory, Department of Psychology, University of Pittsburgh.
†Department of Medicine, University of Pittsburgh.
‡Department of Comparative Medicine, Bowman Gray School of Medicine.
Address correspondence to: Shari R. Waldstein, Behavioral Physiology Laboratory, University of Pittsburgh, 506 Old Engineering Hall, 4015 O'Hara Street, Pittsburgh, PA 15260, U.S.A.

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modulate relationships between cardiovascular reactivity and lipid levels [13], it is conceivable that the effects of anger expression on cholesterol are also modulated by psychophysiologic reactivity. That is, anger–lipid relationships may be strongest among persons who show the greatest magnitude of sympathetic response to behavioral challenge. In this regard, associations between anger expression and serum lipid concentrations are examined separately among subjects comprising clearly differentiated groups of high and low heart rate and blood pressure 'reactors,' as defined by their responses to laboratory stressors.

METHOD

Subjects

Subjects derived from three studies of healthy, white men [Sample A ($N = 22$); Sample B ($N = 20$); and Sample C ($N = 21$)], each evaluating cardiovascular responsivity to laboratory stressors, conducted for other purposes. Subjects were selected for the current analyses on the basis of complete cardiovascular, lipid, and affect data. All participants were students (aged 18–30) recruited from the university community through bulletin board advertisements and published announcements. Individuals were ineligible for participation if they had a history of hypertension, cardiovascular or pulmonary disease, psychiatric disorder, or obesity (greater than 25% overweight according to Metropolitan Life Insurance tables). Experimental procedures were explained to all subjects, and informed consent was obtained in accordance with the University of Pittsburgh Biomedical Institutional Review Board guidelines. Subjects were paid for study participation.

Procedures

Each cohort participated in a laboratory session consisting of baseline and task (stress) periods, during which automated measures of systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR) were collected using a Critikon Dinamap Vital Signs Monitor. In Sample C, HR measurements were also obtained continuously by photoplethysmography. Individual laboratory sessions are described in greater detail below.

All subjects provided blood samples for cholesterol determinations following a 12 hr fast. Blood samples were stored at −80°C for later analysis at a CDC standardized lipid analytic laboratory. TSC concentrations were determined enzymatically [14]. Determinations of HDL-C were made with the heparin-manganese chloride precipitation procedure [15].

All participants completed the Spielberger Anger Expression Scale [16]. Two subscales were scored: anger-out (denoting the outward expression of anger); and anger-in (the experience and suppression of anger).

Sample A. Subjects initially rested supine, in a reclining chair for 30 min, the last 4 min of which served as a baseline period. A 20-min stress period ensued in which participants performed two computerized tasks: a modified Stroop color word interference test [17] and mental arithmetic (addition and subtraction of one to three digit numbers), presented in alternating 5-min blocks.

Cardiovascular measurements (SBP, DBP, HR) were obtained twice during the last 4 min of the baseline period, and at 2 min intervals throughout the task period. Mean baseline levels of SBP, DBP and HR were calculated by averaging the two readings obtained during the baseline period. Task levels of all cardiovascular measurements were calculated as an average of the 10 readings obtained during the task period. Change in cardiovascular measures (task value−baseline value) averaged 9.49 (SD = 6.65) mmHg for SBP, 5.49 (SD = 6.18) mmHg for DBP, and 12.42 (SD = 8.23) beats per minute for HR.

Sample B. For Sample B, subjects rested in a seated position and performed only the Stroop test [17] during the 20-min task period, but were otherwise treated the same as Sample A. Cardiovascular measurements (SBP, DBP, HR) were obtained at 90 sec intervals during the baseline and task periods. Mean baseline levels of SBP, DBP and HR were calculated by averaging the last two readings obtained during the baseline period. Task levels of all cardiovascular measurements were calculated as an average of the 14 readings obtained. Average baseline-to-task changes were 15.10 (SD = 6.15) mmHg for SBP, 10.67 (SD = 7.61) mmHg for DBP, and 12.73 (SD = 5.67) beats per minute for HR.

Sample C. Subjects initially rested in a seated position for 9 min, the last 3 min of which served as a baseline period; the remainder of the laboratory session consisted of serial presentation of five experimental stressors with 8 min (5 min rest, 3 min baseline) intertask periods. The order of task presentation was: mental arithmetic, mirror tracing, video game, speech anticipation, and speech delivery.

During mental arithmetic, subjects subtracted serially by 17, aloud, beginning with a new four-digit

number at the beginning of each 1-min interval, for a total of 3 min. Next, using an automated mirror tracing apparatus (Lafayette Instruments), subjects used a stylus to trace the outline of a star, guided only by its inverted mirror image, for a period of 3 min. Subjects then played a commercially available video game, *Defender*, for 6 min. Finally, subjects prepared and delivered a brief impromptu speech described as a 'simulated job interview.' Two 3-min task periods followed: (a) mental preparation for the speech (anticipation); and (b) delivery of the speech.

Blood pressure measurements were taken at 1-min intervals during the last 3 min (baseline) of each rest period, and throughout each task period. Heart rate was recorded continuously during baseline and task periods. Mean baseline levels of SBP, DBP, and HR were calculated as an average of all measurements taken during the first rest period. Task levels reflect an average of all cardiovascular measures taken during each task period. Baseline-to-task changes (averaged across tasks) were 13.24 (SD = 6.43) and 7.38 (SD = 4.38) mmHg for SBP and DBP, respectively; and 5.64 (SD = 5.45) bpm for HR.

Data analysis

The data analytic strategy employed is as follows. First, age, body mass index (BMI), and baseline levels of physiologic variables are compared among groups using analysis of variance. Next, Pearson coefficients are reported to describe relationships between basal cardiovascular measurements and cholesterol, and associations between task-associated cardiovascular reactivity and cholesterol concentrations. In these sections, a meta-analytic approach to the data analysis is employed [18]. Use of meta-analysis here is based on two considerations: (a) neither baseline nor task values of HR and blood pressure could be collapsed across groups due to the procedural differences among studies; and (b) on the basis of effect sizes noted in prior investigations, the small individual samples described here would likely provide insufficient statistical power to detect significant associations.

To obtain a measure of cardiovascular response free of potential baseline influence (e.g., law of initial values), [19] residualized change scores were calculated for each cardiovascular variable, for each task and in each study, by regressing baseline measurements taken from the rest periods immediately preceding each task onto corresponding task values for SBP, DBP, and HR. The regression residuals (deviations of actual from predicted task values) were then standardized (converted to z scores) and taken as indices of baseline-adjusted HR and blood pressure reactivity. For Sample C, these residuals were averaged across all five tasks to provide a single value for SBP, DBP, and HR. Resting cardiovascular measurements were also standardized in order to equate the distributions of scores across samples.

The baseline and reactivity measurements for SBP, DBP, and HR were first correlated with cholesterol levels (TSC, HDL-C) for each sample separately (Pearson coefficients).* Chi-square tests for heterogeneity of results among samples [18] did not achieve significance for any variable. The individual correlation coefficients were therefore combined across samples using meta-analytic procedures to yield a single correlation coefficient for each variable [18]. The following formula was used to combine effect sizes: $(z_{r1} + z_{r2} + z_{r3})/K$ where z_r = Pearson Product–Moment correlation coefficient transformed to Fisher's z score, and K = number of groups. The aggregate correlation coefficients were then tested for statistical significance.

Next, Pearson coefficients were calculated between scores on the anger expression subscales (i.e., anger-in, anger-out) and concentrations of TSC and HDL-C across all subjects ($N = 63$). Finally, since hostility has been shown to modulate relationships between cardiovascular reactivity and lipid levels, [13] the possibility that the effects of anger expression on cholesterol levels are also modulated by psychophysiologic reactivity was explored. Subjects were first divided into high and low cardiovascular reactors based on median division of the distribution of reactivity scores for each sample; correlations were then calculated between anger expression scores and cholesterol concentrations separately for high and low HR, SBP, and DBP reactors.

RESULTS

Basal cardiovascular measurements and cholesterol

Mean age, BMI, and baseline SBP, DBP, HR, TSC and HDL-C are displayed in Table I for each sample. Total serum cholesterol concentrations ranged from 104 to 227 mg/dl (2.69–5.87 mmol/l); HDL-C concentrations ranged from 30.2 to 65 mg/dl (0.78–1.68 mmol/l). Analysis of variance indicated that groups did not differ

*The relationship between cardiovascular reactivity and TSC/HDL-C ratios was also examined, but did not yield significant findings.

significantly in fasting cholesterol concentrations, resting blood pressure, or BMI. However, groups differed significantly in age ($F = 17.05$, $p < 0.00001$). *Post-hoc* comparisons (Tukey's HSD) indicated that Sample A was significantly older than Samples B or C ($p < 0.05$); Sample B and Sample C did not differ. Additionally, resting HR differed significantly among groups ($F = 9.30$, $p < 0.0003$). *Post-hoc* comparisons revealed that Sample A had significantly lower resting HR's than did Sample B and Sample C ($p < 0.05$); the latter two groups did not differ.

TABLE I.—DEMOGRAPHIC MEASURES AND BASELINE LEVELS OF PHYSIOLOGIC VARIABLES

Measure	Sample A Mean (SD)	Sample B Mean (SD)	Sample C Mean (SD)
Age*	23.77 (2.98)	20.05 (1.54)	21.05 (1.53)
BMI	23.41 (2.71)	24.19 (1.82)	22.39 (2.57)
SBP (mmHg)	115.82 (5.36)	118.03 (7.74)	112.99 (8.21)
DBP (mmHg)	61.64 (8.89)	59.50 (7.74)	64.49 (7.45)
HR (bpm)**	55.76 (6.91)	63.98 (6.56)	65.09 (9.43)
TSC (mg/dl)	156.77 (25.86)	163.15 (27.26)	163.19 (26.15)
TSC (mmol/l)	4.05 (0.67)	4.22 (0.70)	4.22 (0.68)
HDL-C (mg/dl)	42.69 (8.66)	46.37 (6.97)	44.43 (10.15)
HDL-C (mmol/l)	1.10 (0.22)	1.20 (0.18)	1.15 (0.26)

*$p < 0.00001$; **$p < 0.0003$.

Results of the meta-analysis indicated a significant positive correlation between basal SBP and TSC concentration ($r = 0.33$, $p = 0.01$). Neither resting DBP nor resting HR correlated significantly with TSC ($r = 0.12$ and 0.10, respectively). HDL-C concentration correlated negatively with basal HR ($r = -0.35$, $p = 0.009$). However, correlations of resting SBP and DBP with HDL-C concentration ($r = -0.07$ and -0.22, respectively) failed to achieve statistical significance.

Cardiovascular reactivity and cholesterol

Correlations of TSC with SBP reactivity ($r = 0.15$), DBP reactivity ($r = 0.01$) and HR reactivity ($r = 0.19$) were all nonsignificant. However, there was a significant negative correlation between HR reactivity and HDL-C concentration ($r = -0.26$, $p = 0.05$). Neither SBP reactivity nor DBP reactivity correlated significantly with HDL-C ($r = -0.13$ and -0.11 respectively).

Anger expression and cholesterol

A significant positive correlation was found between anger-out and HDL-C concentration ($r = 0.30$; $p = 0.02$). Correlations between individual items of the anger-out scale and HDL-C concentrations are displayed in Table II. Anger-out was not significantly related to TSC ($r = -0.04$). In addition, anger-in failed to correlate significantly with either TSC ($r = -0.04$) or HDL-C ($r = -0.05$).

Anger expression, cardiovascular reactivity, and cholesterol concentrations

As noted above, correlations were also computed between measures of anger expression and cholesterol concentrations within groups of high and low HR, SBP, and DBP reactors. Results, shown in Table III, indicate a significant positive

correlation between anger-out and HDL-C concentration among the high reactors, for each of the three cardiovascular measures (SBP, DBP, HR). Anger-out did not correlate significantly with TSC in any group, nor was anger-in associated significantly with cholesterol concentrations in either high or low reactors.

TABLE II.—CORRELATIONS BETWEEN ANGER-OUT ITEMS AND HDL-C CONCENTRATIONS

Item	*r*	*p*
I argue with others	0.25	0.05
I make sarcastic remarks at others	0.25	0.05
If someone annoys me, I'm apt to tell him or her how I feel	0.23	0.07
I express my anger	0.19	NS
I say nasty things	0.18	NS
I strike out at whatever infuriates me	0.12	NS
I lose my temper	0.07	NS
I do things like slam doors	0.06	NS

TABLE III.—PEARSON PRODUCT–MOMENT CORRELATIONS BETWEEN ANGER-OUT AND CHOLESTEROL CONCENTRATIONS IN HIGH AND LOW CARDIOVASCULAR REACTORS

	Anger-out					
	SBP		DBP		HR	
	High	Low	High	Low	High	Low
HDL-C	0.55*	0.09	0.51**	0.14	0.40***	−0.18
TSC	0.07	−0.17	0.14	−0.25	−0.18	0.19

*$p < 0.001$; **$p < 0.003$; ***$p < 0.03$.

DISCUSSION

Consistent with findings of large, epidemiologic investigations [20], we observed a positive relationship between resting SBP and TSC concentrations in healthy young men. In addition, higher resting HRs were associated with lower concentrations of HDL-C. We also found a significant, negative association between HDL-C concentrations and a baseline-free measure of behaviorally-elicited HR reactivity. Interestingly, HR reactivity has previously been found to be correlated with an elevated TSC/HDL-C ratio in both male and female cynomolgus monkeys [21, 22]. Male coronary patients with high TSC/HDL-C ratios have also shown a more pronounced catecholamine response to mental stress than patients with relatively lower TSC/HDL-C ratios [5]. In a fourth study, HDL-C correlated negatively with the DBP and total peripheral resistance response to laboratory stress in a group of patients having cardiovascular and/or gastrointestinal disorders [6]. Interpretation of the latter two studies is complicated, though, by the fact that many patients were receiving antihypertensive medications which may have independently influenced both cardiovascular reactivity and lipid levels. Finally, and in contrast to these findings, two studies of healthy middle-aged adults have failed to find a relationship between cardiovascular reactivity and HDL-C concentrations [7, 8].

In the present study, there was no relationship between cardiovascular reactivity

and TSC. This result differs from those of previous investigations which found HR or blood pressure responsivity to correlate with TSC in healthy, middle-aged individuals [7, 8] and in patients with mild hypertension [4, 6]. This inconsistency may be explained, in part, by sampling differences among studies. In contrast to the studies described above, the current investigation employed healthy young men.

The mechanism accounting for any association between cardiovascular reactivity and serum lipid concentrations is unknown, but may involve stress-associated increases in plasma catecholamines since many facets of lipid metabolism are affected by adrenergic stimuli. However, because alpha and beta receptors on hepatocytes and adipocytes have opposite actions, the net effect of adrenergic activation on serum HDL concentrations is difficult to predict [23]. Other potential mechanisms which may account for the observed relationship between HR reactivity and HDL-C concentration include lifestyle variables such as customary diet and physical exercise, which were not measured in the present investigation.

Results of this study also indicate that young men scoring high on a self-report measure of the tendency to express anger (i.e., anger-out) had higher concentrations of HDL-C than men who scored relatively lower on this measure. This cannot be compared directly with results of previous studies because the Anger Expression Scale has never been examined in conjunction with lipid levels. However, two recent investigations found no relationship between HDL-C and hostility, but did find higher cholesterol (TSC, LDL-C) concentrations among individuals scoring high on measures of Type A behavior and hostility [2, 3]. This apparent inconsistency may be explained by several methodologic differences among studies.

First, and most importantly, the anger-out scale employed in the present study measures a somewhat different construct than the hostility measures employed in previous investigations (i.e., paranoid ideation subscale of the SCL-90, Videotaped Structured Interview ratings). Spielberger's anger-out subscale measures the tendency to express angry emotions, either verbally or physically, [16] and contains items relating to both assertive and aggressive behaviors. It is possible that the assertiveness dimension of the anger-out scale may relate positively to HDL-C. Indeed, an examination of correlations between individual subscale items and HDL-C (see Table II) indicates that several items which reflect verbal discussion of angry feelings (assertive behaviors) correlated most highly with cholesterol concentrations. The self-report measure of hostility used in previous research (SCL-90 paranoid ideation subscale) measures a cognitive or attitudinal set characterized by suspicion, cynicism and mistrust [3]. In addition, the observational rating of hostility used previously (Videotaped Structured Interview) subsumes a compilation of ratings including experience of anger, hostile attitude, and antagonistic behavior [2]. Finally, it is also notable that measures of hostility and anger-out frequently do not correlate significantly with one another [24].

Another consideration is that, in previous investigations, hostility correlated significantly with cholesterol concentrations only in men who were also rated as Type As. Hostility alone did not relate significantly to cholesterol in men. Additionally, those studies which found hostility to correlate with TSC employed middle-aged subjects; the present sample was a relatively younger, healthy sample. It is possible that the influence of anger-related variables on physiologic measures (such as fasting cholesterol levels) differs across the life span [25].

The current data also indicate that the relationship between expressed anger and HDL-C may be modulated by psychophysiologic arousal. Specifically, the outward expression of anger may provide a protective influence on HDL-C only in individuals who display more pronounced cardiovascular reactivity (whether defined by SBP, DBP or HR). Prior research has shown greater HR and catecholamine response to mental stress in middle-aged men having both elevated TSC and high scores on the Cook–Medley Hostility Scale, as compared to low hostile men with lower TSC [13]. Again, these contrasting findings might conceivably be explained by sampling differences and the use of different measures of anger and hostility. And once again, the possibility remains that the relationships among anger expression, reactivity, and lipids may be accounted for by differences in health habits such as dietary factors, smoking, alcohol consumption or exercise, all of which affect cholesterol concentrations [26–28].

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