Vigilance, Active Coping, and Cardiovascular Reactivity During Social Interaction in Young Men

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This study of 72 undergraduate men examined the effects of two determinants of cardiovascular response—active coping and vigilance—on blood pressure and heart rate responses to social stressors. Observation of a future debate partner (i.e., vigilance) evoked larger increases in blood pressure than did observation of a less relevant person, apparently through the combination of increases in cardiac output and vascular resistance. Preparation and enactment of efforts to exert social influence (i.e., active coping) evoked heightened blood pressure and heart rate responses through increased cardiac contractility and output. Thus, both vigilance and active coping in social contexts increased cardiovascular reactivity, but apparently through different psychophysiological processes.

Key words: cardiovascular reactivity, vigilance, active coping, psychosocial risk

Social processes influence the development of cardiovascular disease. Social isolation increases risk of coronary heart disease (CHD; Hazuda, 1994), as does the personality trait of hostility (T. Q. Miller, Smith, Turner, Guifarro, & Hallet, 1996). Further, hostile people have strained relationships (Smith, Pope, Sanders, Allred, & O'Keefe, 1988). In monkeys, social conflict promotes atherosclerosis, and individual differences in social dominance moderate this effect (Manuck, Marsland, Kaplan, & Williams, 1995). In humans, dominance confers risk of CHD and premature death (Houston, Babyak, Chesney, Black, & Ragland, 1997; Whitman, Deary, Lee, & Fowkes, 1997). Thus, aspects of the social environment and individual differences in social behavior contribute to the multifactorial etiology of cardiovascular disease. Cardiovascular reactivity may be an important mechanism in these effects (Manuck, 1994). According to related models, risk factors such as hostility promote larger, more frequent, and prolonged heart rate (HR) and blood pressure responses to stressors. Reactivity, in turn, could promote development of essential hypertension and atherosclerosis (Manuck, 1994; Obrist, 1981), and might precipitate manifestations of CHD (Kop, 1999).

Because psychosocial risk factors involve interpersonal processes (Adler & Matthews, 1994), tests of potential mechanisms must examine the social determinants of reactivity (Smith & Gerin, 1998). In developing a social psychophysiology of cardiovascular response, researchers have used two approaches. In the first, the psychosocial risk process is modeled in the laboratory. For example, the provision of social support can attenuate responses to stressful tasks (Kamarck, Peterman, & Raynor, 1998; Lepore, 1998; Uchino, Cacioppo, & Keicolt-Glaser, 1996), and trait hostility is associated with heightened cardiovascular responses to interpersonal stressors (Houston, 1994; Suls & Wan, 1993). A second strategy extends the literature on nonsocial stressors by evaluating their effects in social contexts. Psychosocial risk factors may evoke more frequent, pronounced, or prolonged activation of reactivity patterns observed in nonsocial paradigms. Active coping is a central example (Obrist, 1981), defined as effort to influence or control the outcome of an event. Studies of this concept have compared signaled reaction time with contingent outcomes (e.g., monetary rewards or shock) to watching an unpleasant film (e.g., Obrist, 1981). However, such tasks differ in ways beyond the critical distinction of opportunity and effort to influence outcomes (Sherwood, Dolan, & Light, 1990).

Active coping has been evaluated more directly by manipulations of outcome contingency within otherwise equivalent tasks. Compared with conditions in which no influence is available, performing reaction time tasks or solving cognitive problems to obtain rewards or avoid punishment produces increased HR, blood pressure, and cardiac output, primarily through sympathetically mediated increases in the rate and force of myocardial contraction (Light & Obrist, 1983; Lovato et al., 1985). This effect also occurs during anticipation of active coping (Contrada, Wright, & Glass, 1984) and is moderated by incentive magnitude (Tranel, Fisher, & Fowles, 1982) and task difficulty (Wright, Dill, Green, & Anderson, 1998). Active coping has been likened to the concepts of sensory rejection (Williams, 1986), defense (Schneiderman & McCabe, 1989), and challenge (Tomaka, Blascovich, Kelsey, & Leiten, 1993), although these concepts are defined in terms of the cognitive demands of the task or appraisals of coping efficacy rather than opportunity and incentive to influence outcomes.

A social analogue of active coping produces a similar pattern of responses. Compared with otherwise equivalent tasks, contingent incentives to influence an interaction partner produce increased HR and systolic blood pressure (Brown & Smith, 1992; Smith,
Allred, Morrison, & Carlson, 1989; Smith, Nease, Kircher, & Limon, 1997). These effects also occur during anticipation of exerting influence (Smith, Baldwin, & Christensen, 1990; Smith et al., 1989, 1997) and are moderated by the magnitude of incentive (Smith et al., 1989) and task difficulty (Brown, Smith, & Benjamin, 1998; Smith et al., 1990). Such responses could contribute to the effect of psychosocial risk factors on disease. For example, social dominance may affect health (Houston et al., 1997; Manuck et al., 1995) because efforts to control others evoke heightened reactivity.

Vigilance (Schneiderman & McCabe, 1989), or sensory intake (Williams, 1986), is a second pattern of response. It is not equivalent to passive coping (Obrist, 1981), defined as exposure to aversive stimuli without opportunity to alter outcomes. Rather, tasks evoking vigilance require alert, watchful observation of the external environment. In an early study, mental arithmetic (i.e., active coping or sensory rejection) evoked increased blood pressure and HR and decreased forearm vascular resistance. In contrast, a word-identification task involving blurred words presented upside down and backward evoked smaller increases in HR and blood pressure and increased vascular resistance (Williams, Bitker, Buchsbaum, & Wynne, 1975). Comparisons of different nonsocial tasks (e.g., mental arithmetic vs. mirror tracing) support the conclusion that sensory intake or vigilance is associated with minimal cardiac response but increased blood pressure and vascular resistance (Williams, Bitker, Buchsbaum, & Wynne, 1975; Williams, 1986). If this pattern occurred in social contexts, then personality characteristics or social environments that promote vigilant observation of others could contribute to disease in this way. Some evidence from studies of behavior during interviews suggests that vigilance in social contexts produces responses similar to those seen in nonsocial tasks (Bitker, Buchsbaum, Williams, & Wynne, 1975; Williams, Kimball, & Willard, 1972). However, a lack of precise manipulation of vigilance in these studies precludes firm conclusions.

To evaluate this second major pattern of psychophysiological response—vigilance or sensory intake—in an interpersonal context, we asked participants anticipating a current-events discussion task to watch a video depicting one of two individuals: an individual who would present an opposing view on the topic or another individual discussing an irrelevant topic. We predicted that the high-relevance video would evoke larger increases in blood pressure than would the low-relevance video. Impedance cardiography assessments (Sherwood, 1993) permitted a test of the prediction that these effects on blood pressure would reflect increases in vascular resistance rather than cardiac output. While they watched the video and prepared for and then engaged in the task, half of the participants expected to receive a monetary reward if they were rated as more effective than their discussion partner; the others expected to receive it regardless of their relative performance. This manipulation of active coping, combined with impedance cardiography techniques and assessment of respiratory sinus arrhythmia (RSA), permitted an evaluation of the presumed determinants of active coping responses. We predicted that active coping would produce increased blood pressure accompanied by increases in cardiac output (CO) and decreases in vascular resistance, and increases in HR accompanied by sympathetically mediated cardiac activity (i.e., greater prejection period shortening) rather than a decrease in parasympathetic activity (i.e., RSA). Recent findings suggest that prior vigilance can potentiate later cardiovascular reactivity (Gump & Matthews, 1998). Therefore, we also examined the effects of vigilance during the subsequent task.

Method

Participants

A total of 72 men were recruited from undergraduate psychology courses. The mean age of participants was 23.8 years (SD = 4.7), and the mean years of education was 14.2 (SD = 1.5). Of the participants, 93% identified themselves as Caucasian with the remainder self-identifying as Asian American (3%), Native American (3%), and Hispanic (1%). In addition to the money they received as part of the incentive manipulation, all participants received extra credit in their psychology course. Participants were randomly assigned to one of four experimental conditions.

Observation of and anticipated interaction with same- versus opposite-gender partners could affect cardiovascular responses (Krantz & Ratliff-Cruin, 1989). Therefore, use of same-gender partners was advisable in an initial study of vigilance. We chose to include men only for two reasons. First, given inherent difficulty in standardizing stimulus properties across genders and the resulting confounding of gender of participant with a variety of aspects of the social stimuli when same-gender pairs are studied, we elected to study only one gender. We chose to study men because the particular stressor we used to manipulate vigilance could be construed as primarily activating agency-related motives (e.g., dominance or achievement). Hence, vigilance might be more readily evoked in men than in women in this setting (Helgeson, 1994). As discussed later, this creates an important limitation on the interpretation of our results.

Procedure

Baseline period. Sessions were conducted in a single-occupant chamber with adjoining space for monitoring equipment. Except for brief interactions, participants were alone. On arrival, they listened to an audiorecording introducing the study as examining "cardiovascular responses to listening and speaking" and "everyday social interaction." They were told that their responses would be measured during a baseline period and while they watched a video of an individual describing his opinions about a topic in the news. They were told that their responses would then be measured while they discussed a topic, taking turns listening and speaking. To limit anxious anticipation during baseline, we avoided any specific description of the task or mention that the video might depict the discussion partner or that performance might be evaluated. Following initial informed consent, mylar bands were placed on the participant and connected to an impedance cardiograph. A blood pressure cuff was attached to the upper portion of the nondominant arm. The 10-min baseline involved a minimally demanding task (i.e., "vanilla baseline"); Jennings, Kamarck, Eddy, & Johnson, 1992), in which participants were asked to examine and rate pairs of pictures at 1-min intervals.

Task instructions and manipulations. After the baseline, we told participants that they would watch a video of someone discussing a topic in the news and then prepare for a discussion of a current topic (i.e., taking the pro vs. con position on student uniforms in public schools or raising social security retirement age). They were informed that they would present a point of view opposed to that presented by the partner, taking turns listening and responding to a prerecorded video of the partner describing his views. They were also told that it was important for the discussion to be realistic, so their comments would be recorded and evaluated by the experimenter immediately after the task to "judge whether or not your remarks were as clear, organized, and effective as those of the tape-recorded remarks of the other student in the discussion."

Participants in the high-incentive condition were told they would receive $5 if the experimenter judged their remarks to be as clear, organized, and
effective as those of the tape-recorded discussant and that about half of the previous participants had received that rating. In the low-incentive condition, participants were told that if they simply completed the study they would receive $5. Vigilance was manipulated by varying the relevance of a 3-min video. In the high-relevance condition, participants were told the video they would watch depicted the other discussant presenting his views on the discussion topic. Those in the low-relevance condition were told the video depicted a different individual discussing an unrelated topic.

Participants then watched a 3-min video of one of two confederates (described as other undergraduates) presenting one of the topics. Confederates and topics were counterbalanced across participants. After the observation period, participants were given notes on arguments for their assigned position. They reviewed the notes for 3 min. During the task, an audiotape presented the confederate's remarks with prompts by another speaker to guide the participant through three alternating 1-min segments of listening and 1-min responses.

Measures

Following the instructions, participants rated the extent to which they could influence their receipt of the $5 on a scale of 1 (not at all) to 5 (completely) and the likelihood of receiving payment from 1 (about zero) to 5 (about 100%). Following the baseline, observation, preparation, and discussion periods, participants completed a questionnaire consisting of twelve 5-point Likert items reflecting alertness (e.g., attentive and concentrating), anxiety (e.g., jittery and nervous), and anger (e.g., irritable and angry) taken from the Positive and Negative Affect Scales (Watson & Clark, 1992). They also completed an adjective checklist following the vigilance period assessing their appraisals of the target and again after the discussion task to assess their discussion partner. The checklist is a shortened version of the Interpersonal Adjective Scales (IAS-R; Wiggins, Trapnell, & Phillips, 1988), a measure of the interpersonal circumplex. We administered four items per octant and calculated Dominance and Friendliness factor scores (Wiggins & Broughton, 1991). For Dominance, values greater than 0 reflect dominant behavior; ratings less than 0 reflect submissiveness. Friendliness values greater than 0 reflect friendly or warm behavior; ratings less than 0 reflect hostility.

Physiological Data

A Minnesota Impedance Cardiograph Model 304B (Surcom, Inc., Minneapolis, MN) assessed electrocardiogram (ECG), basal thoracic impedance (Zth), and the first derivative of the impedance signal (dZ/dt). Four mylar bands were placed in the tetrapolar configuration (Sherwood, Allen, et al., 1990). A 4 mA AC current at 100 kHz was passed through the two outer bands, and Zth and dZ/dt were recorded from the two inner bands. The ECG, Zth, and dZ/dt signals were digitized at 500 Hz. We ensemble averaged these data within 1-min epochs and verified or edited waveforms prior to analyses. Stroke volume (SV) was estimated using the Kubicek equation (Sherwood, Allen, et al., 1990), and CO in l/min was calculated with the equation HR × (SV/1000). Total peripheral resistance (TPR) was measured in resistance units (dynes-s/cm"5) on the basis of mean arterial pressure (MAP) and CO (i.e., TPR = MAP/CO X 80). Pre-ejection period (PEP) was calculated as the time interval in milliseconds between the Q-point of the ECG and the B-point of the dZ/dt signal. PEP reflects sympathetic control of the heart (see Cacioppo et al., 1994).

RSA provides an index of parasympathetic control of the heart. It was calculated on the basis of the digitized interbeat intervals (IBI) and checked and edited for artifacts using the detection algorithm of Bernston, Quigley, Jang, and Boysen (1990). A heart period time series was created from the IBI series using a "weighted" beat algorithm (Bernston, Cacioppo, & Quigley, 1995). We detected sharp transitions in the heart period time series (e.g., because of arrhythmia) using the Bernston et al. (1990) algorithm and removed them by smoothing. A linear (first order) polynomial was fit to, and subtracted from, the heart period time series (Litvack, Oberlander, Carney, & Saul, 1995). Subtraction of this first-order polynomial (linear detrending) acted as a high pass filter, removing very large ultralow frequency trends (including the DC component) from the input signal. After linear detrending, the heart period time series was band-pass filtered from 0.12 to 0.40 Hz using an interpolated finite impulse response filter (Neuvo, Cheng-Yu, & Mitra, 1984). The power spectrum of the heart period time series was calculated using a fast Fourier transform and scaled to msc2/Hz. RSA was calculated as a natural log of the area under the heart period power spectrum within the corner frequencies of the band-pass filter (see Litvack et al., 1995). Average RSA was calculated for each minute. A Dinamap Model 8100 monitor (Critikon Corporation, Tampa, FL) was used to measure systolic blood pressure (SBP), diastolic blood pressure (DBP), and MAP. It uses the oscillometric method to estimate blood pressure. Blood pressure was measured once per minute.

Results

Affective Responses, Manipulation Checks, and Baseline Equivalence of Groups

Self-reports of changes in affect were examined to determine the general impact of tasks periods, and changes in alertness during the observation period were examined as a check on the relevance manipulation. As presented in Table 1, self-reported anxiety and anger were elevated over baseline levels during all three periods, all Fs(1, 136) > 4.00, p < .05. Although the magnitude of the increases in anxiety and anger varied across task periods, both Fs(2, 136) > 5.9, p < .01, there were no significant effects involving relevance or incentive on these responses. Self-reported alertness was also elevated over baseline during all three task periods, all Fs(1, 136) > 6.0, p < .01. Alertness increased more during the preparation and discussion periods than during the observation period, F(2, 136) = 6.31, p < .005. This effect was qualified by a significant Task Periods X Relevance interaction, F(2, 136) = 4.56, p < .02. As expected, participants in the high-relevance condition reported a larger increase in alertness during the observation period than did those in the low-relevance condition (0.46 vs. 0.18), t(71) = 1.82, p < .04. No other effects on alertness were significant. Thus, participants reported overall increases in anxiety, anger, and alertness, and the relevance manipulation increased alertness during the observation period. Participants in the high-incentive condition reported that their payment depended on their task performance to a greater degree than did participants in the low-incentive condition (4.0 vs. 1.4), t(71) = 16.0, p < .001, and they indicated less certainty of

<table>
<thead>
<tr>
<th>Measure</th>
<th>Observation</th>
<th>Preparation</th>
<th>Speaking</th>
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<tbody>
<tr>
<td>Alertness</td>
<td>0.32, 0.08</td>
<td>0.53, 0.08</td>
<td>0.56, 0.08</td>
</tr>
<tr>
<td>Anxiety</td>
<td>0.14, 0.06</td>
<td>0.46, 0.08</td>
<td>0.32, 0.09</td>
</tr>
<tr>
<td>Anger</td>
<td>0.30, 0.07</td>
<td>0.21, 0.07</td>
<td>0.40, 0.08</td>
</tr>
</tbody>
</table>

Note. Means with different subscripts within rows are different at p < .05.
receiving it (3.0 vs. 4.9), t(71) = 30.9, p < .001. Thus, participants appeared to understand the incentive manipulation instructions.

A mixed analysis of variance (ANOVA; High vs. Low Incentive × High vs. Low Relevance × Observation vs. Speech Period) on participants' IAS-R ratings of target friendliness revealed a main effect for periods, F(1, 68) = 8.29, p < .01. Participants rated the target during the debate task as more hostile than they receiving it (3.0 vs. 4.9), t(71) = 30.9, p < .001. Thus, participants main effect for periods, F(1, 68) = 8.29, p < .01. Participants on participants' IAS-R ratings of target friendliness revealed a

tative X High vs. Low Relevance X Observation vs. Speech Period) high-relevance group tended to rate the "old" target as slightly

group rated the "new" target as somewhat dominant, whereas the

of the target's dominance during the observation period (—2.4 vs. —3.1), t(68) = 2.46, p < .01. However, among participants observing their partner in the upcoming discussion, those in the high-incentive condition made

indicated that the high- versus low-incentive condition did not influence the extremity of ratings made by participants evaluating the low-relevance target, t(68) = 1.33. However, among participants observing their partner in the upcoming discussion, those in the high-incentive condition made more extreme attributions than did low-incentive participants, t(68) = 2.46, p < .01. Thus, motivation for success in the task altered the cognitive process of attributions only when participants observed a relevant target.

Overview of Primary Analyses

Consistent with prior recommendations (Llabre, Spitzer, Saab, Ironson, & Schneiderman, 1991), change scores (i.e., task minus average baseline) were calculated for each minute of the observation, preparation, and speaking periods. Because baseline levels can affect subsequent change (Benjamin, 1967), baseline values were included as a covariate in mixed analyses of covariance (ANCOVAs). During the observation period, we computed three-way mixed ANCOVAs (i.e., High vs. Low Relevance × High vs. Low Incentive × Three Trials) for each cardiovascular measure and derived two a priori directional contrasts (Keppel, 1982) from this overall analysis as tests of the predicted effects of relevance (i.e., vigilance) and incentive (i.e., active coping). SBP, DBP, and HR were considered to be the primary cardiovascular reactivity indexes. The effects of CO and TPR were examined as determinants of blood pressure responses, whereas PEP and RSA were examined as determinants of HR responses. As discussed above, the effects of relevance (i.e., vigilance) during the observation period were expected for SBP and DBP reactivity, accompanied by increases in TPR. The effects of incentive during the observation period were expected for HR (Tranel et al., 1982), accompanied by decreases in PEP. For blood pressure, incentive effects were expected on SBP, accompanied by increases in CO and decreases in TPR.

For the preparation and speech phases, we computed four-way mixed ANCOVAs (i.e., High vs. Low Relevance × High vs. Low Incentive × Preparation vs. Speaking × Three Trials) and derived a priori directional contrasts of the expected effects of incentive and relevance. Because speaking can influence cardiovascular response (Friedman, Thomas, Kulick-Cuifffo, Lynch, & Sugino-hara, 1982), significant overall contrasts were followed by contrasts within the preparation and speaking periods. The incentive (i.e., active coping) effects during preparation and speech were

Table 2

Means and Standard Deviations for Baseline Physiological Measures

<table>
<thead>
<tr>
<th>Measure</th>
<th>High relevance</th>
<th>Low relevance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>High incentive</td>
<td>Low incentive</td>
</tr>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>SBP (mm/Hg)</td>
<td>113.28</td>
<td>10.86</td>
</tr>
<tr>
<td>DBP (mm/Hg)</td>
<td>63.69</td>
<td>6.49</td>
</tr>
<tr>
<td>CO (l/min)</td>
<td>5.89</td>
<td>1.50</td>
</tr>
<tr>
<td>TPR (dyne<em>s</em>cm^-2)</td>
<td>1238.14</td>
<td>596.46</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>68.74</td>
<td>7.75</td>
</tr>
<tr>
<td>RSA (natural log)</td>
<td>6.23</td>
<td>0.86</td>
</tr>
<tr>
<td>PEP (msec)</td>
<td>128.96</td>
<td>15.30</td>
</tr>
</tbody>
</table>

Note. SBP = systolic blood pressure; DBP = diastolic blood pressure; CO = cardiac output; TPR = total peripheral resistance; HR = heart rate; bpm = beats per minute; RSA = respiratory sinus arrhythmia; PEP = preejection period.

For the preparation and speech phases, we computed four-way mixed ANCOVAs (i.e., High vs. Low Relevance × High vs. Low Incentive × Preparation vs. Speaking × Three Trials) and derived a priori directional contrasts of the expected effects of incentive and relevance. Because speaking can influence cardiovascular response (Friedman, Thomas, Kulick-Cuifffo, Lynch, & Sugino-hara, 1982), significant overall contrasts were followed by contrasts within the preparation and speaking periods. The incentive (i.e., active coping) effects during preparation and speech were

1 Analysis of the strength or extremity of participants' responses to the IAS-R items provides additional support for the effectiveness of the vigilance and incentive manipulations. Increased motivation for accurate social judgments leads people to process available information more extensively and make stronger attributions or evaluations (Pittman, 1998), as when people anticipate future interactions with the target and those interactions have important outcomes (e.g., D. T. Miller, Norman, & Wright, 1978; Monson, Keel, Stephens, & Genung, 1982). A Relevance × Incentive interaction on the extremity (i.e., average absolute value of the difference from scale midpoint) of ratings on the IAS-R, F(1, 68) = 7.15, p < .01, indicated that the high- versus low-incentive condition did not influence the extremity of ratings made by participants evaluating the low-relevance target, t(68) = 1.33. However, among participants observing their partner in the upcoming discussion, those in the high-incentive condition made more extreme attributions than did low-incentive participants, t(68) = 2.46, p < .01. Thus, motivation for success in the task altered the cognitive process of attributions only when participants observed a relevant target.
expected for HR and SBP, accompanied by decreases in PEP, increases in CO, and decreases in TPR. Other impedance cardiography parameters (e.g., SV) are considered exploratory. If expected effects were qualified by interactions with trials within a period, we adjusted interaction effects (Greenhouse & Geisser, 1959) and examined them with comparisons using the appropriate error term (Bernhardson, 1975).

Effects of Vigilance During the Observation Period

As expected, compared with participants watching the tape of an irrelevant target, those watching their discussion partner displayed larger increases in SBP (7.8 mmHg vs. 3.3 mmHg), t(67) = 2.43, p < .01, and DBP (4.6 mmHg vs. 2.1 mmHg), t(67) = 2.32, p < .02, as depicted in Figure 1 (Panels A and B). The effect of relevance on HR responses during the observation period was not significant, F(1, 67) = 0.59. Unexpectedly, participants observing the high-relevance target displayed a larger increase in CO than did those observing the low-relevance target (0.57 L/min vs. 0.22 L/min), F(1, 67) = 4.54, p < .04. As depicted in Figure 1 (Panel C), this effect was qualified by a significant Relevance x Trials interaction, F(2, 114) = 4.14, p < .02. Participants in the high-relevance condition displayed larger increases in CO than did their counterparts in the low-relevance condition during the first 2 min of the observation period, both t(57) > 2.35, p < .05, but not during the third minute, t(57) < 1.0. Further, among high-relevance participants, CO decreased from the 1st to 3rd minute of observation, t(114) = 1.98, p < .05, whereas low-relevance participants displayed a nonsignificant increase, t(114) = 1.25. Analyses of TPR did not reveal the expected effect of relevance, but a similar Relevance x Trials interaction approached significance, F(2, 114) = 3.30, p < .06. As depicted in Figure 1 (Panel D), high-relevance participants displayed a trend toward increasing TPR over the observation period, t(114) = 1.92, p < .06, whereas those in the low-relevance condition displayed a nonsignificant decrease, t(114) = 1.50. No other effects involving target relevance approached significance during the observation period.²

² Observation of the high-relevance target tended to evoke greater changes in SV than did observation of the low-relevance target (1.85 ml vs. -1.11 ml), F(1, 60) = 3.28, p < .08. This may account for the effect of relevance on CO despite the lack of a parallel effect on HR.
Effects of Active Coping During the Observation Period

As expected and depicted in Figure 2 (Panel A), participants anticipating a reward contingent on their performance displayed larger increases in HR than did those anticipating a noncontingent reward (7.5 beats per minute [bpm] vs. 3.0 bpm), \( t(67) = 2.65, p < .01 \). They also displayed greater PEP shortening than did those in the low-incentive condition (−9.45 msec vs. −5.14 msec), \( t(67) = 2.05, p < .025 \) (see Figure 2, Panel B). There were no effects involving the incentive manipulation on RSA during the observation period. Incentive did not alter blood pressure responses during the observation period, perhaps because of the combined, offsetting effects for CO, \( t(57) = 2.27, p < .02 \), and TPR, \( t(57) = 2.73, p < .005 \). As expected (see Figure 2, Panel C), compared with low-incentive participants, those in the high-incentive condition displayed larger increases in CO (0.58 L/min vs. 0.21 L/min). They displayed a decrease in TPR during observation, whereas low-incentive participants displayed an increase (−66.6 dynes-s \( \times \) cm vs. 26.2 dynes-s \( \times \) cm; see Figure 2, Panel D).

Effects of Active Coping During Preparation and Speaking

As depicted in Figure 3 (Panel A), high-incentive participants displayed larger increases in SBP than did low-incentive participants (17.6 mmHg vs. 12.1 mmHg), \( t(67) = 2.19, p < .02 \). This effect was significant during both the preparation and speaking periods, both \( t(67) > 1.98, p < .03 \). This contrast was also significant for DBP (10.6 mmHg vs. 8.0 mmHg), \( F(1, 67) = 4.2, p < .04 \), but only during the speaking phase (14.5 mmHg vs. 11.7 mmHg), \( t(67) = 2.05, p < .05 \). Participants in the high-incentive condition displayed a larger increase in CO during the task than did their counterparts in the low-incentive condition (1.01 L/min vs. 0.47 L/min), \( t(57) = 2.55, p < .01 \) (see Figure 3, Panel C) while participants both prepared for and engaged in the discussion, both \( t(57) > 2.09, p < .025 \). High-incentive participants also displayed an overall decrease in TPR during the task, whereas participants in the low-incentive condition displayed an increase (−24.8 dynes-s \( \times \) cm vs. 48.8 dynes-s \( \times \) cm), \( t(57) = 1.79, p < .04 \). However,
analyses within the preparation and speaking periods separately were not significant, both $t(57) < 1.6$.

As expected, high-incentive participants displayed larger increases in HR during the task than did low-incentive participants (13.5 bpm vs. 9.6 bpm), $t(67) = 2.14, p < .02$, but this effect was significant only during the speaking phase, $t(67) = 2.57, p < .01$, (see Figure 3, Panel B). The parallel contrast of task changes in PEP indicated that high-incentive participants tended to display greater shortening in PEP than did low-incentive participants ($-13.67$ ms vs. $-9.37$ ms), $t(60) = 1.57, p < .07$. Overall task decreases in RSA were greater among high- than among low-incentive participants ($-0.44$ vs. $-0.14$, respectively), $F(1, 61) = 4.45, p < .04$, but this effect was significant only during the speaking phase of the task, $t(61) = 2.78, p < .01$ (see Figure 3, Panel D).

**Effects of Prior Vigilance on Responses During Speaking**

To test the hypothesis that prior vigilance can potentiate responses to subsequent stressors (Gump & Matthews, 1998), we examined the effects of high versus low relevance on cardiovascular responses during the speaking phase. There were no main effects on SBP, DBP, CO, TPR, HR, PEP, or RSA responses during speaking. However, participants who had previously watched the video of their discussion partner displayed larger increase in SV while responding during the discussion than did participants who had observed an irrelevant target (0.84 ml vs. $-3.84$ ml), $F(1, 60) = 7.37, p < .01$. No Incentive X Relevance interactions emerged in the analyses of speaking period changes in SBP, DBP, CO, TPR, HR, PEP, or RSA, but the general pattern of results suggested a potential influence of prior vigilance. None of the simple main effects of incentive were significant among low-relevance participants. However, among participants who had previously observed the discussion partner, each simple main effect of incentive was significant, all $p$s $< .05$, except for TPR, as depicted in Table 3.

**Discussion**

These results replicate and extend our prior findings regarding the cardiovascular effect of active coping in social contexts and provide novel evidence that vigilant observation of the social environment evokes increases in blood pressure. As in prior studies (e.g., Brown & Smith, 1992; Smith et al., 1989, 1997), an incentive for successful performance of an interpersonal task produced larger blood pressure and HR responses, both while partic-
VIGILANCE AND CARDIOVASCULAR REACTIVITY

Table 3

Effects of Incentive Manipulation (Including Means and Standard Errors) on Cardiovascular Reactivity During Speaking, Within High- and Low-Relevance Conditions

<table>
<thead>
<tr>
<th>Measure</th>
<th>High relevance</th>
<th>Low relevance</th>
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<tbody>
<tr>
<td></td>
<td>High incentive</td>
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<td>High incentive</td>
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<td>(M)</td>
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<td>SE</td>
<td>(M)</td>
<td>SE</td>
<td>(t)</td>
<td>(M)</td>
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<tr>
<td>SBP</td>
<td>25.90</td>
<td>3.09</td>
<td>18.70</td>
<td>3.11</td>
<td>1.88*</td>
<td>20.40</td>
<td>3.09</td>
<td>16.30</td>
<td>3.13</td>
<td>1.07</td>
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<td>DBP</td>
<td>15.30</td>
<td>1.53</td>
<td>11.60</td>
<td>1.53</td>
<td>1.92*</td>
<td>13.70</td>
<td>1.54</td>
<td>11.90</td>
<td>1.53</td>
<td>0.93</td>
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<tr>
<td>CO</td>
<td>2.05</td>
<td>0.34</td>
<td>1.06</td>
<td>0.30</td>
<td>3.09**</td>
<td>0.93</td>
<td>0.30</td>
<td>0.78</td>
<td>0.32</td>
<td>0.47</td>
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<tr>
<td>TPR</td>
<td>116.00</td>
<td>54.36</td>
<td>22.30</td>
<td>48.03</td>
<td>1.33</td>
<td>6.13</td>
<td>47.51</td>
<td>52.50</td>
<td>50.81</td>
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<tr>
<td>HR</td>
<td>22.60</td>
<td>2.35</td>
<td>13.40</td>
<td>2.36</td>
<td>3.22**</td>
<td>17.70</td>
<td>2.37</td>
<td>16.02</td>
<td>2.35</td>
<td>0.59</td>
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<tr>
<td>RSA</td>
<td>-0.96</td>
<td>0.21</td>
<td>-0.23</td>
<td>0.20</td>
<td>3.17**</td>
<td>-0.37</td>
<td>0.20</td>
<td>0.14</td>
<td>0.20</td>
<td>1.00</td>
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<tr>
<td>PEP</td>
<td>-20.83</td>
<td>3.97</td>
<td>-13.80</td>
<td>3.50</td>
<td>1.72*</td>
<td>-13.80</td>
<td>3.65</td>
<td>-12.50</td>
<td>3.78</td>
<td>0.32</td>
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</table>

Note. SBP = systolic blood pressure; DBP = diastolic blood pressure; CO = cardiac output; TPR = total peripheral resistance; HR = heart rate; RSA = respiratory sinus arrhythmia; PEP = pre-ejection period.

* \(p < .05\), one-tailed. ** \(p < .01\), one-tailed.

Participants silently prepared for the task (i.e., SBP and HR). None of our previous studies included impedance cardiographic assessments of determinants of these responses. Hence, it is important to note that blood pressure and HR responses here were accompanied by increases in CO and decreases in PEP, consistent with the presumed cardiac origin of active coping responses (Schniederman & McCabe, 1989; Williams, 1986). During the task, these changes were also accompanied by some dilation of the peripheral vasculature (i.e., decrease in TPR) and parasympathetic withdrawal (i.e., decreased RSA). During the initial observation period, the active coping manipulation produced a substantial increase in HR reactivity, accompanied by increased sympathetic activity (i.e., PEP shortening) without a decrease in parasympathetic activity (i.e., RSA). Also during this period, the incentive manipulation had the expected effect of increasing CO but did not produce a corresponding increase in SBP, perhaps because of the large, parallel reduction in peripheral resistance. These results are consistent with the view that active coping in social contexts produces effects on cardiovascular reactivity through the same physiological mechanism—sympathetic stimulation of the heart—as is seen in nonsocial stressors.

As expected, vigilant observation of the social environment produced increased blood pressure but not HR, consistent with the literature on vigilance in nonsocial contexts (Schneiderman & McCabe, 1989; Williams, 1986). However, rather than being accompanied by an increase in peripheral resistance, this increase in blood pressure was initially accompanied by greater CO. Over the 3-min observation period, the effect of vigilance on blood pressure was constant, but the effect on CO waned. At the same time, vigilance produced increasing resistance (i.e., TPR). Thus, although the limitation of vigilance effects to blood pressure reactivity is consistent with prior research, the effects on CO and TPR might indicate that vigilance in social contexts produces effects on blood pressure through different mechanisms than those operating in nonsocial tasks. Alternatively, the changing effects on CO and TPR may indicate a somewhat different, dynamic basis for the effect on blood pressure. Participants in the high-relevance condition might have considered counterarguments or engaged in related active coping early in the observation period, shifting to simple vigilance over time. However, the relevance manipulation was not simply a second manipulation of active coping, as it produced blood pressure reactivity without a strong impact on cardiac response. Hence, in this specific situation, vigilant observation of the social environment may have raised blood pressure through a mixture of cardiac and vascular mechanisms that changed over time.

There was some, albeit limited, evidence that prior vigilance altered later reactivity. Prior vigilance did have an effect on SV during the task, but this result should be considered exploratory. Although no Incentive X Relevance interactions occurred during speaking, the simple effect of the incentive manipulation was significant for most cardiovascular responses among participants who had previously observed the discussion partner, but was not significant on any measure among low-relevance participants. The lack of interactions despite this consistent pattern may reflect the relatively low power to detect ordinal interactions in this design. Further, main effects of prior vigilance on task responses might have been obscured by the pervasive effects of active coping during this period. Also, the previously observed partner (i.e., high-relevance condition) was appraised as less dominant than the novel partner (i.e., low relevance), perhaps masking otherwise potentiating effects of prior vigilance. Dominant interaction partners can evoke greater reactivity (e.g., Newton, Bane, Hores, & Greenfield, 1999), and in the present study perceptions of the partner as dominant were associated with larger increases in DBP during the task, \(r(72) = .23, p < .05\). Thus, although vigilant observation of the social environment may have influenced subsequent responses, the concurrent effects were much more apparent.

Qualifications and Limitations

It is important to note that the procedures used in this study to operationalize vigilance may limit the generalizability of the results. Vigilance prior to an instrumental activity may produce different effects than vigilance without such anticipation. As noted
above, observation of a target in anticipation of competitive interaction may evoke a mixture of sensory intake and active coping, affecting both cardiac and vascular influences on blood pressure responses. Observation of the social environment when no future instrumental response is anticipated may produce a simpler, largely vascular response. Therefore, conceptual replication of the effects of vigilant observation of others is warranted. Of course, vigilance in anticipation of future competitive or conflictual interaction may be an important aspect of several psychosocial influences on health. Hence, this confounding of task components may be ecologically valid. Nonetheless, given the inconsistent and unexpected effects of vigilance on TPR and CO during observation, a direct comparison of social and nonsocial contexts is needed.

Our findings are obviously limited to young men. It will be important to replicate the findings with other groups. Given our choice of a task that emphasizes agency (Bakan, 1966; Wiggins, 1991), it is possible that the response of women to the same procedures could be quite different (Davis & Matthews, 1996; Helgeson, 1994; Smith, Gallo, Goble, Ngü, & Stark, 1998; Smith, Limon, Gallo, & Ngü, 1996). Vigilant observation of others may have differing effects on men and women, depending on whether it is in the service of agency or communion. Also, several studies have suggested gender differences in the cardiac versus vascular mechanisms underlying blood pressure responses (e.g., Allen, Stoney, Owens, & Matthews, 1993; Girdler, Turner, Sherwood, & Light, 1990).

Finally, although some studies indicate that individual differences in cardiovascular reactivity predict rising in blood pressure (Matthews, Woodall, & Allen, 1993), atherosclerosis (Barnett, Spence, Manuck, & Jennings, 1997), and manifestations of CHD (Blumenthal, Jiang, Waugh, 1995), support for this hypothesis is tentative (Manuck, 1994). Further, the fact that individual differences in cardiovascular reactivity predict later disease provides only indirect evidence of the role of this construct as a mediator of the effects of psychosocial risk factors. The individual difference and mediating mechanism views of cardiovascular reactivity are conceptually distinct (Christenfeld, Glynn, Kulik, & Gerin, 1998; Smith et al., 1997), and support for one does not imply support for the other. Animal research provides more direct support: Kaplan, Manuck, Adams, Weingand, & Clarkson (1987) demonstrated that pharmacologic blockade of sympathetic input to the heart eliminated the otherwise atherogenic effects of social stress on dominant male monkeys. Yet, there is only limited direct evidence that cardiovascular reactivity links psychosocial risk factors and disease. In fact, these effects of vigilance and active coping during exposure to acute stressors could even reflect adaptive responses (Dienstbier, 1989).

**Conclusions and Future Directions**

These results support the view that efforts to influence or control the social environment produce sympathetically mediated increases in HR and blood pressure. In a novel extension of the prior literature on the cardiovascular effects of vigilance, our findings also indicate that motivated observation of others can increase blood pressure. If such cardiovascular responses are involved in the development of cardiovascular disease, then vigilance in social contexts may have psychosomatic consequences.

In addition to research addressing the limitations of this study, this latter speculation suggests more avenues for research. It may be worthwhile to examine the specific association of psychosocial risk factors with cardiovascular responses to interpersonal equivalents of active coping and vigilance. For example, as noted above, dominance may confer increased risk through the mechanism of more frequent and vigorous efforts to control others and the associated cardiovascular effects of active coping in social contexts. Several personality traits such as hostility, suspiciousness, and mistrust (Barefoot et al., 1998; Smith, 1992) are likely to promote vigilant observation of others. Further, prior to a potentially conflictual social interaction, hostile men and women display physiological responses consistent with vigilance (Davis, Matthews, & McGrath, 2000). Aspects of social relationships, demographic characteristics, and features of the social environment may also be associated with more frequent vigilance. The present findings suggest that this motivated cognitive activity could account for at least part of the influence of such factors on health.

**References**


VIGILANCE AND CARDIOVASCULAR REACTIVITY


