A longitudinal analysis of coping style and cardiovascular reactivity to laboratory stressors

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ABSTRACT

The association between active and passive coping and cardiovascular reactivity has been of interest because of its theoretical implications. However, most past studies utilized laboratory manipulations and cross-sectional data. A complementary approach would be to examine individual differences in active and passive coping and their links to lab-based reactivity over time. The present longitudinal study used the COPE Inventory to assess active and passive coping styles, which were used to predict cardiovascular reactivity of 107 individuals to a laboratory stressor at a 10-month follow-up. Consistent with hypotheses, results showed that Time 1 active coping scores predicted significantly greater heart rate reactivity at Time 2, β = 0.16, p = .02. In contrast, passive coping did not predict any indices of cardiovascular reactivity over time. These findings are discussed in light of their theoretical implications.

1. Introduction

Active and passive coping are two different approaches an individual may use when responding to stress. While active coping is typically associated with efforts to directly affect event outcomes (e.g., planning, Obrist, 1981), passive coping is generally defined by the lack of an instrumental response to do so (e.g., avoidance, Sherwood, Dolan, & Light, 1990). These coping options are typically associated with distinct cardiovascular response patterns, with active coping eliciting primarily a myocardial β-adrenergic response, and passive coping an α-adrenergic response (Lovallo, Pincomb, & Wilson, 1986; Obrist, 1981; Sherwood et al., 1990). These relatively distinct cardiovascular responses are thought to represent differences in preparatory responses for action or defense (Obrist, 1981; Tuer, 1994).

There are at least two approaches that can inform theory and research on active/passive coping. The first approach examines active and passive coping as a situational factor that emerges in a particular coping context. This is the most common approach in this literature and examines tasks designed to have active/passive coping features in a laboratory setting and measure their influence on cardiovascular reactivity (Bongard, Hodapp, Frisch, & Lennartz, 1994; Ginter, Hollandsworth, & Intriere, 1986; Sherwood, Allen, Obrist, & Langer, 1986). Early work by Obrist et al. (1978) showed that active coping tasks typically increased heart rate and cardiac output and decreased vascular resistance which is consistent with a myocardial β-adrenergic response. Consistent with an α-adrenergic response, passive coping tasks are related to smaller increases in heart rate and cardiac output and more observable increases in vascular resistance. Although discrepant findings exist, by and large these patterns have been supported (Bolli, Ammann, Hulthen, Kiowski, & Buhler, 1981; Light & Obrist, 1980; Light & Obrist, 1983; Obrist, 1981; Sherwood et al., 1990).

A second approach is to examine active and passive coping as individual differences factors that influence cardiovascular responses across situations. However, very few studies have taken this approach. In contrast to lab-based manipulations, individual differences in active and passive coping are important to consider because they model an individual's typical response to stress across situations (Carver, Scheier, & Weintraub, 1989). Thus, such individual differences should be related to laboratory stress reactivity because lab assessments are thought to index how individuals respond to stress in their daily life (Gerin et al., 1998; Kamarck et al., 1992). Such an assumption is not without controversy, however, although increased reliability and generalizability can be gained via aggregation across multiple tasks (Kamarck et al., 1992).

Consistent with an individual difference approach, participants have been shown to differ in their tendency toward an active or passive coping style (independent of task type), with distinct cardiovascular response patterns (Sherwood et al., 1990). For example, one of the few studies in this area found that men relatively high in active coping tendencies showed relatively greater increases in cardiac output during...
a handgrip test, whereas men relatively high in passive coping showed greater increases in total peripheral resistance (Malan et al., 2006). However, this study was cross-sectional and hence not able to examine change over time. Prospective studies can provide stronger theoretical evidence for a possible link between individual differences in coping styles across situations, including lab-based assessments.

The present study used longitudinal data to evaluate whether individual differences in active and passive coping predicted future cardiovascular reactivity. Given their distinct biological mechanisms (Obrist, 1976; Sherwood et al., 1990), it was predicted active coping would be associated with relatively greater increases in heart rate, whereas passive coping would be associated with relatively greater increases in blood pressure (Garwood, Engel, & Capriotti, 1982; Light & Obrist, 1980). Although some work indicates that active coping processes are also associated with systolic blood pressure (SBP, Jennings et al., 1997), the magnitude of this response appears greater for passive coping tasks (Gregg, James, Matyas, & Thorsteinsson, 1999). In addition, the interpretation of SBP as reflecting active coping in prior work is complicated by several factors. First, blood pressure is influenced by heart rate which is one reason SBP may be elevated during active coping tasks (Obrist, 1981). Second, the predicted β-adrenergic mechanisms associated with active coping may result in vasodilation (hence decreasing blood pressure) via β2 receptors in the vasculature (Smith & Kampine, 1990). This would make it a relatively less sensitive measure of active coping processes compared to heart rate, albeit heart rate is also influenced by the parasympathetic nervous system.

2. Method

2.1. Participants

Participants were recruited as part of a larger longitudinal study. In the original study (see Uchino, Uno, Holt-Lunstad, & Flinders, 1999) 64 men and 69 women 30–70 years of age were recruited through advertisements placed in local newspapers. The following self-reported inclusion criteria were utilized to ensure healthy participants were selected for the study: (a) no existing hypertension, (b) no cardiovascular prescription medication use, (c) no past history of chronic disease with a cardiovascular component (e.g., diabetes), (d) no recent history of psychological disorder (e.g., major depressive disorder), (e) no tobacco use, and (f) no consumption of > 10 alcoholic beverages a week (see Cacioppo et al., 1995).

Of the 108 individuals who returned for the follow-up (out of 133 participants), all but one gave sufficient data for inclusion in the present analysis (N = 107). Participants were reevaluated against the inclusion criteria and follow-up (Time 2) data were collected an average of 10 months (SD = 1.6) after initial study participation. At each time point participants were financially compensated $35 for approximately 2.5 h of time. Table 1 presents the basic demographics from the follow-up sample, as well as information on the major study variables.

2.2. Procedure

At Time 1, participants were contacted via telephone and screened according to the inclusion criteria previously detailed. Self-report data obtained from participants were also checked for consistency with the inclusion criteria. Qualifying participants were then scheduled for a laboratory appointment. Upon arrival participants completed an informed consent document, background information, and the COPE questionnaire. Participants’ height and weight were measured using a standard medical scale, and their body mass index (BMI) was calculated (i.e., kg/m²).

Next, participants were escorted to a separate, sound attenuated room where sensors and an occluding blood pressure cuff of appropriate size were attached. Individuals were seated in a comfortable chair and instructed to relax for the next 12 min while resting measures of cardiovascular function were obtained. During the final 5 min of the resting assessment, cardiovascular assessments of heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP) were obtained once every 90 s. Following the resting assessments, participants took part in a stress protocol developed by Cacioppo et al. (1995) which included 6 mins of a speech stressor and 6 mins of a math stressor (counterbalanced across years; see Uchino, Holt-Lunstad, Bloor, & Campo, 2005 for full details). The same procedures were completed at Time 2 but changes in task content were implemented to minimize habituation (e.g., different speech topic and different set of serial subtractions; see Uchino et al., 2005).

2.3. Measures

2.3.1. Cardiovascular measures

A Minnesota Impedance Cardiograph Model 304B was used to measure HR, whereas a Dinamap Model 8100 monitor (Critikon Corporation, Tampa, Florida) was used to measure blood pressure. The Dinamap used the oscillometric method to estimate blood pressure (see Gorback, Quill, & Lavine, 1991 for validation). Mean SBP, DBP, and HR for each epoch was averaged across minutes (e.g., values for speech and mental arithmetic were averaged) to increase the reliability of these assessments (Kamarck et al., 1992). Reactivity was computed as a change score (i.e., stress task value – resting baseline value).

2.3.2. Active/passive coping assessment

At Time 1, the COPE Inventory was used to evaluate individual differences in participants’ active and passive coping (Carver et al., 1989). Internal consistency in the present study was adequate for active coping (active coping subscale, Cronbach’s α = 0.78) and passive coping (behavioral disengagement subscale, α = 0.65).

2.3.3. Demographics

Participants completed a basic demographic form which assessed age, gender, highest level of education, and annual family income.

2.4. Analytic approach

Analyses were conducted using the SAS System. Participants with missing data were removed prior to analysis, and the corresponding degrees of freedom adjusted accordingly. Primary analyses employed simultaneous regression via Proc Reg. Several relevant covariates were identified a priori based on prior work in the literature including age,

### Table 1: Sample characteristics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Sample</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time 1 age frequency distribution (%)</td>
<td></td>
</tr>
<tr>
<td>29–40</td>
<td>30</td>
</tr>
<tr>
<td>41–49</td>
<td>34</td>
</tr>
<tr>
<td>50–59</td>
<td>28</td>
</tr>
<tr>
<td>60–70</td>
<td>15</td>
</tr>
<tr>
<td>Mdn education level (T1)</td>
<td>College graduate</td>
</tr>
<tr>
<td>yearly income (T1)</td>
<td>$20,000 to $29,000</td>
</tr>
<tr>
<td>M (SD) age</td>
<td></td>
</tr>
<tr>
<td>Active coping scale</td>
<td>3.12 (0.61)</td>
</tr>
<tr>
<td>Passive coping scale</td>
<td>1.60 (0.50)</td>
</tr>
<tr>
<td>SBP reactivity (T1)</td>
<td>15.36 (7.70)</td>
</tr>
<tr>
<td>DBP reactivity (T1)</td>
<td>8.48 (5.35)</td>
</tr>
<tr>
<td>Heart rate reactivity (T1)</td>
<td>9.90 (6.36)</td>
</tr>
<tr>
<td>SBP reactivity (T2)</td>
<td>13.29 (8.58)</td>
</tr>
<tr>
<td>DBP reactivity (T2)</td>
<td>7.13 (5.02)</td>
</tr>
<tr>
<td>Heart rate reactivity (T2)</td>
<td>8.05 (5.05)</td>
</tr>
</tbody>
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