Dispositional behavioral activation: Relationships with cardiovascular activity during anger and sadness

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ABSTRACT

Anger arises when goals are thwarted, but remain potentially attainable, and can motivate persistent engagement in goal pursuit. Sadness occurs when goals appear beyond reach, often leading to disengagement. Variations in engagement, or motivational intensity, are reflected in cardiovascular reactivity. Motivational intensity in goal-relevant situations is subject to regulation by individual differences in dispositions involving behavioral activation (BAS) and inhibition systems (BIS). This study examined whether cardiovascular responses indicative of motivational intensity are influenced by attributes measured by Carver and White’s (1994) BAS Drive, BAS Reward Responsiveness, and BIS scales. Anger and sadness were induced on a within-subjects basis using an autobiographical recall task. Cardiovascular measures were systolic and diastolic blood pressure (SBP and DBP) and heart rate (HR). Post-task affect ratings indicated that the anger–sadness manipulation was successful. As expected, BAS Drive was positively associated with SBP and DBP (but not HR) elevations while participants related an anger-inducing event. Also as expected, this pattern was reversed for sadness, where Drive scores were inversely related to SBP and DBP elevations. Neither Reward Responsiveness nor BIS were associated with cardiovascular reactivity. These findings contribute to understanding of dispositions that influence physiologic activity reflective of motivational intensity in emotional situations.

1. Introduction

The Carver and White (1994) Behavioral Inhibition/Behavioral Activation System (BIS/BAS) scales measure dispositional constructs grounded in the work of Gray (1990) (for a review see Smillie, Pickering, & Jackson, 2006). Behavioral activation concerns energization and engagement in goal pursuit, or motivational intensity, with facets including persistence (Drive), positive responses to goal attainment (Reward Responsiveness), and desire for new rewards (Fun-Seeking). However, whereas Drive and Reward Responsiveness appear to be components of BAS, Fun-Seeking seems primarily to involve impulsivity (Smillie, Dalgleish, & Jackson, 2007). Behavioral inhibition is an avoidant disposition, reflecting sensitivity to aversive events. This article examined Drive, Reward Responsiveness, and BIS in relation to motivational intensity reflected in cardiovascular reactivity (CVR) accompanying anger and sadness.

As dispositions linked to goal-related activity, the BIS/BAS constructs have relevance for states of anger and sadness. Emotion theorists argue that negative emotions arise when situations are cognitively appraised as unfavorable for goal attainment (Moors, Ellsworth, Scherer, & Frijda, 2013). A similar conclusion has been reached in theory that construes emotions as states elicited by rewards and punishers (Rolls, 2005). In these frameworks, both anger and sadness are approach-related emotions because they are responses to impediments to efforts to secure goals or incentives (Carver & Scheier, 2013). However, because of differences in the extent and context of goal blockage, anger and sadness are accompanied by different action tendencies and therefore differ in motivational intensity. Anger arises when goal thwarting is judged unfair, a responsible agent has been identified and, most relevant from a motivational standpoint, corrective action appears possible and urgent. Energy is mobilized to support efforts at removing the obstacle, reflecting increased motivational intensity. In sadness, by contrast, goals appear out of reach. Therefore, corrective action is not possible, and urgency, motivational intensity, and energy mobilization are diminished.

Action tendencies and physiologic activity are major components of emotions (others include subjective feelings and behavior) (Moors et al., 2013). Variations in preparation for behavioral engagement, which distinguish anger and sadness but also may vary within each of
these states, are marked by corresponding neuroendocrine changes that regulate CVR (e.g., Cannon, 1929; Lang & Bradley, 2010; Obrist, 1981; Wright & Barreto, 2012). Therefore, by influencing motivational intensity in states of anger and sadness, dispositional BAS should be related to alterations in CVR that accompany these emotions.

It has been suggested that individual differences in BAS are associated with variations in motivational intensity that occur in response to impediments to goal pursuit (Carver & Scheier, 2013). When goals are thwarted, but not entirely lost, it might be expected that resulting anger is accompanied by greater motivation to reassert goal pursuit in higher BAS individuals than in lower BAS individuals. We hypothesized that under these conditions BAS will be positively associated with CVR. By the same reasoning, when goals are blocked and attainment is infeasible, sadness in higher BAS individuals should be accompanied by greater reductions in motivational intensity and greater disengagement than in those lower in BAS (Carver & Scheier, 2013). This should be accompanied by dampened CVR, bringing about an inverse relationship between BAS and CVR.

Dispositional BAS involves sensitivity to aversive stimuli (Carver & Scheier, 2013; Cooper, Gomez, & Aucocte, 2007). It is therefore associated with anxiety and fear or, with successful avoidance, relief/calmness. However, anxiety or fear may co-occur with anger or sadness if aversive consequences are expected, such as harm (anger) or loneliness (sadness). Possible implications suggested in the literature include reduced motivation and CVR (Tomaka & Palacios-Esquivel, 1997), approach-avoidance conflict that increases CVR (Stanley & Conrada, 2012), vigilance leading to decreased CVR (Obrist, 1981), and active avoidance and CVR augmentation (Wright & Barreto, 2012).

The foregoing line of thought was tested using an autobiographical memory task. Participants recalled and described aloud one recent event that provoked anger and one that induced sadness. Use of personally-relevant events was intended to induce homogeneous levels of these states (Labouvie-Vief, Lumley, Jain, & Heinze, 2003). Cardiovascular measures were obtained throughout.

Systolic and diastolic blood pressure (SBP, DBP) and heart rate (HR), were the focus as they reflect neuroendocrine processes activated by motivationally significant situations (Berntson, Quigley, & Lozano, 2007; Obrist, 1981). Elevations in SBP reflect increased myocardial contractility and, to a lesser extent, increased vascular resistance, both resulting from sympathetic nervous system (SNS) activity and circulating epinephrine produced by the adrenal medullae (Berntson et al., 2007; Obrist, 1981). Sympathetic activation mobilizes energy in preparation for vigorous efforts to respond to situational demands. It was predicted that, in the anger condition, dispositional BAS would be positively associated with CVR, especially SBP, reflecting greater motivational intensity. With sadness, the opposite was expected: dispositional BAS was expected to be inversely associated with CVR, reflecting less motivational intensity.

Drive was the focus of predictions because it involves pursuit of goals, and anger and sadness are responses to obstacles that prevent goal attainment. By contrast, Reward Responsiveness is more relevant when goals are attained. No directional predictions were made regarding BIS.

2. Methods

2.1. Participants

The sample, 117 undergraduates (60.7% female), ranged in age from 18 to 28 (M = 18.9, SD = 1.5). All were enrolled in General Psychology and received course credit.

2.1.1. Measures

2.1.1.1. BIS/BAS scales. Participants completed Carver and White’s (1994) Drive, Reward Responsiveness, and BIS scales, which possess desirable psychometric properties and show evidence of construct validity (Carver, 2004; Cooper et al., 2007; Harmon-Jones, Price, Peterson, Gable, & Harmon-Jones, 2013).

2.1.1.2. Other self-report measures. A brief questionnaire assessed demographic factors. Participants periodically rated their “anger,” “sadness,” and “distress” on a 5-point scale.

2.1.1.3. Cardiovascular measures. Measures of SBP, DBP, and HR were obtained with a DINAMAP Pro 100 (GE) monitor. Blood pressure was ascertained oscillometrically in millimeters of mercury (mm Hg), and HR was based on pressure pulses in beats per minute (bpm). Readings were recorded at 60-s intervals during an initial 8-min rest period from which baseline measures for SBP, DBP, and HR were derived as the mean of the last 2 readings. They also were recorded at 60-s intervals during the two speaking tasks.

2.1.1.4. Procedure. Participants were instructed to refrain from caffeine, nicotine, alcohol, other drugs, and exercise for at least 2 h prior to the session. After informed consent was obtained questionnaires were administered by PC. An inflatable cuff then was placed on the non-dominant arm, the experimenter left the room, and baseline readings recorded. The participant then completed the affect scales.

The experimenter returned to initiate the task. Like other autobiographical techniques (Berntsen & Rubin, 2002; Labouvie-Vief et al., 2003), memories of a recent emotional event were activated. In the anger condition, participants recalled a life event that occurred within the past 6 months and caused anger. Instructions for the sadness condition were identical, except participants recalled and related an event that caused them to feel depressed.  

During a 2-min task preparation phase, subjects were asked to concentrate on thoughts, feelings, and sensations experienced at the time of the event, to recreate the incident mentally, and to describe it aloud when prompted. The focus on sensory and motor cues was based on a previous research on emotional imagery (Lang & Bradley, 2010). In the ensuing, 3-min task phase, speaking was used to take advantage of its ability to amplify affect and its physiological concomitants (Kirschbaum, Pirke, & Hellhammer, 1993). Order of administration of anger and sadness task versions was counterbalanced. The experimenter left the room before the task commenced.

An 8-min recovery period was interposed between experimental conditions. Immediately following each speaking phase, participants provided affect ratings. Following task completion the experimenter returned, disconnected the cuff, and debriefed participants.

3. Results

3.1. BIS/BAS scores

The BIS/BAS scales showed adequate internal consistency (Cronbach’s alpha = .85 for Drive, .84 for Reward Responsiveness, and .76 for BIS). The correlation between Drive and Reward Responsiveness was .27 (p < .01). Relationships of BIS to Drive and Reward Responsiveness were weaker, rs = .03 (ns) and .20 (p < .05).

3.2. Affect ratings

There were no significant associations between BIS/BAS scores and baseline affect (ps > .50). Paired-samples t-tests showed that the manipulations had the intended effects: anger recall induced a significant increase in anger ratings comparing baseline (M = 1.82, SD = .90) to post-task values (M = 2.29, SD = 1.3), p < .001. Similarly, sadness recall

1 Although task instructions asked participants to recall and describe a recent event that caused them to feel “depressed,” we refer to this as the sadness condition to be explicit about the fact that it was intended to cause sad feelings, or depressed affect, but not a state or condition of depression in any clinical sense.
induced a significant increase in sadness (M = 1.99, SD = 0.89 versus M = 2.47, SD = 1.24). Both effect sizes fell between small and medium at d = .36 (Cohen, 1988). The manipulations were distinct in their impact: anger recall produced a significantly larger increase in anger (M = +0.5, SD = 1.3) than in sadness (M = 0.0, SD = 1.1), and sadness recall produced a significantly larger increase in sadness (M = +0.5, SD = 1.3) than in anger (M = −0.1, SD = 1.1), ps < .001.

Distress ratings (SD in parentheses) for baseline and following the anger and sadness tasks were 2.26 (0.92), 2.21 (1.02), and 2.22 (1.03), showing little variation, p > .50.

Mixed-model regression analyses evaluated main effects of emotion and BIS/BAS scores, and emotion × scale interactions, on change-scores reflecting responses to the tasks, adjusting for baseline values. The BIS/BAS scales and baselines were continuous between-subject predictors and emotion (anger versus sadness) a 2-level, within-subject factor. Terms involving gender were dropped after they were found to be non-significant (ps > .38).

A main effect indicated that the non-induction produced significantly higher elevations in anger than did the sadness induction, F(1/115) = 23.16, p < .001 (Fig. 1). At f² = .2285, this was a medium-to-large effect (Cohen, 1988). The only other significant term reflected an inverse association between baseline anger and anger change-scores (p < .001).

Results for sadness also revealed an emotion main effect (Fig. 1). The sadness induction produced significantly higher elevations in sadness than did the anger induction, F(1/115) = 30.72, p < .001. At f² = .3198, this fell slightly short of a large effect (Cohen, 1988). There was also a significant inverse association between baseline sadness and sadness change-scores (p < .001).

In addition, there was a Drive effect (p < .05) and an emotion × Drive interaction, F(1/112) = 4.04, p < .05, the latter a small effect at f² = .0345. The interaction was explicated by computing predicted sadness change-scores for subjects with higher and lower Drive scores (M ± 1 SD)(Cohen, Cohen, West, & Aiken, 2003). For sadness, higher Drive subjects had a predicted sadness elevation of 0.77 whereas the predicted sadness elevation for lower BAS subjects was 0.19. Corresponding sadness change-scores in the anger condition for subjects with higher and lower Drive scores were smaller and more similar at −0.08 and −0.21, respectively. No other effects were significant.

Results for distress indicated no significant effects, F < 1.0, ps > .80, except for an inverse association between baseline distress and distress change-scores (p < .001).

3.3. Cardiovascular reactivity

Descriptive data for CVR are presented in Table 1. There were no significant associations between BIS/BAS scores and cardiovascular baselines (ps > .50).

For SBP change-scores, there was a significant emotion × Drive interaction, F(1/112) = 8.48, p < .005, f² = .0724. None of the other main effect or interaction terms were significant.

The emotion × Drive interaction was explicated by examining simple effects of Drive on SBP change-scores separately for the anger and sadness conditions (Cohen et al., 2003). Baseline SBP was omitted given the absence of a baseline effect in the initial analysis. Because hypotheses focus on prediction of CVR specific to either anger or sadness, a second model was evaluated: to adjust for the correlation between SBP change-scores for anger and for sadness (r = .46, p < .001). change-scores for the sadness condition were added to the simple effect model for anger, and change-scores for the anger condition were added to the simple effect model for sadness. This allowed an estimation of unique, emotion-specific effects of Drive on SBP change.

Results for anger (Table 2, columns 1–4) indicated that, in the first model, none of the BIS/BAS scales were significantly related to SBP reactivity, and the overall model was nonsignificant. However, when SBP change-scores for sadness were added, they showed a significant relationship with SBP change-scores for anger, p < .01, and there was significant improvement in the overall model, p < .01. More importantly, this led to emergence of a significant, positive relationship between Drive and the unique variance in SBP change-scores for anger, p < .05, as well as a significant final model, p < .01.

For sadness (Table 2, columns 5–8) the initial model contained a significant, inverse relationship between Drive and SBP change-scores, p < .05, though the overall model was not significant. When SBP change-scores for anger were entered, they showed a significant relationship with SBP change-scores for anger, p < .01, and there was significant improvement in the overall model significantly improved (p < .01), and the final model was significant, p < .01.

Fig. 2 depicts effects of Drive on SBP for the anger and sadness conditions based on the final models focusing on emotion-specific effects. Consistent with hypotheses, Drive was significantly related to change-scores in both anger and sadness conditions, but in opposite directions. The relationship between Drive and SBP change-scores in the anger

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Descriptive statistics for cardiovascular measures.</th>
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</thead>
<tbody>
<tr>
<td>Measure</td>
<td>Experimental condition</td>
</tr>
<tr>
<td></td>
<td>Baseline</td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>107.6 (10.2)</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>63.8 (5.9)</td>
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<tr>
<td>HR (mm Hg)</td>
<td>72.9 (10.9)</td>
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</tbody>
</table>

Note. N = 117. Values are means with SDs given in parentheses. SBP = systolic blood pressure, DBP = diastolic blood pressure, HR = Heart Rate.

The emotion × Drive interaction was explicated by examining simple effects of Drive on SBP change-scores separately for the anger and sadness conditions (Cohen et al., 2003). Baseline SBP was omitted given the absence of a baseline effect in the initial analysis. Because hypotheses focus on prediction of CVR specific to either anger or sadness, a second model was evaluated: to adjust for the correlation between SBP change-scores for anger and for sadness (r = .46, p < .001). change-scores for the sadness condition were added to the simple effect model for anger, and change-scores for the anger condition were added to the simple effect model for sadness. This allowed an estimation of unique, emotion-specific effects of Drive on SBP change.

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<table>
<thead>
<tr>
<th>Table 2</th>
<th>SBP reactivity: simple and unique effects of BAS Drive for anger and sadness conditions.</th>
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</thead>
<tbody>
<tr>
<td>Variable</td>
<td>Anger condition</td>
</tr>
<tr>
<td></td>
<td>Simple effectsa</td>
</tr>
<tr>
<td></td>
<td>B</td>
</tr>
<tr>
<td>BAS Drive</td>
<td>0.351</td>
</tr>
<tr>
<td>BAS RR</td>
<td>0.263</td>
</tr>
<tr>
<td>BIS</td>
<td>0.087</td>
</tr>
<tr>
<td>SBP change</td>
<td></td>
</tr>
<tr>
<td>Anger</td>
<td>−−</td>
</tr>
<tr>
<td>Sadness</td>
<td>−−</td>
</tr>
<tr>
<td>R² change</td>
<td>0.232</td>
</tr>
<tr>
<td>R²</td>
<td>0.012</td>
</tr>
</tbody>
</table>


a Simple effects test each predictor separately for the anger and sadness conditions.

b Unique effects adjust for the positive relationship between anger- and sadness-related reactivity.

p < .05.

** p < .01.
condition was positive, whereas there was an inverse relationship between Drive and SBP change-scores in the sadness condition. The squared semi-partial correlation coefficients ($\hat{r}^2$) for these two associations were .0306 and .0488, respectively. At about 3% and 5% unique variance explained, these fell between small and medium (Cohen, 1988). None of the other simple effects for SBP were statistically significant for either anger or sadness.

Analysis of DBP also indicated a significant emotion × Drive interaction, $F(1/112) = 4.80, p < .04 f^2 = .0409$. There also was a significant inverse association between baseline and change-scores, $p < .03$. Remaining main effects and interactions did not approach significance.

In the simple effect analysis for anger (Table 3, columns 1–4), the first model contained no significant BIS/BAS effects, and the overall model was nonsignificant. Given their correlation with anger change-scores, $r = .45, p < .01$, DBP change-scores for sadness were added as a predictor and showed a significant relationship with DBP change-scores for anger, $p < .01$, and the overall model significantly improved, $p < .01$. However, unlike SBP, this did not lead to emergence of a significant effect of Drive on unique variance in DBP change-scores for anger, despite a significant final model, $p < .01$.

Results for sadness (Table 4, columns 5–8) showed a significant, inverse relationship between Drive and DBP change-scores in the initial model, $p < .05$, though the overall model was not significant. When DBP change-scores for anger were added to the model, they showed a significant relationship with DBP change-scores for sadness, $p < .01$. The inverse effect for Drive became stronger, $p < .01$, the overall model significantly improved $p < .01$, and the final model was significant, $p < .01$.

Fig. 3 depicts the unique effects of Drive on DBP change-scores for anger and sadness. Consistent with hypotheses, and similar to SBP, Drive was related to DBP change-scores in both the anger and sadness conditions, but in opposite directions, though only significantly so for sadness. The relationship between Drive and DBP change-scores in the anger condition was positive, albeit nonsignificant, whereas there was a significant inverse relationship between Drive and DBP responses to the sadness induction. The $\hat{r}^2$ coefficients were .0128 and .0420, respectively. At 4% unique variance explained, the relationship for the sadness condition fell between small and medium. None of the other simple effects for DBP were statistically significant for either anger or sadness.

For HR, the only significant effect indicated that HR baselines were inversely related to change-scores, $p < .01$.

### 4. Discussion

This study supports hypotheses concerning relationships linking the BAS Drive disposition measured by the Carver and White’s (1994) scale to CVR accompanying anger and sadness. During anger, Drive scores were positively associated with CVR, whereas in the sadness condition, Drive scores were inversely associated with CVR. This pattern was as predicted in that Drive enhances motivational intensity during anger and diminishes it during sadness, and was seen for SBP and DBP. These novel findings increase understanding of motivational dispositions, emotion, and CVR.

#### 4.1. Anger and sadness ratings

There was minimal evidence that BAS or BIS were related to affect, contrasting with previous studies (e.g., Carver, 2004, Study 3; Kasch, Rottenberg, Arnow, & Gotlib, 2002). This may be due to task differences. Our participants recalled and re-experienced self-selected situations involving anger and sadness. To follow instructions was to experience these states however provoked. By contrast, prior work examined stimulus conditions selected for relevance to BAS. Therefore, previous literature focused on differential determinants of affects, whereas we demonstrated differential CVR given a state of anger or sadness. These questions are complementary; ours moves the focus from elicitation of affect to its physiological concomitants once elicited.

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**Table 3**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Anger condition</th>
<th>Sadness condition</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Simple effects$^a$</td>
<td>Unique effects$^a$</td>
</tr>
<tr>
<td></td>
<td>$B$</td>
<td>$SE$</td>
</tr>
<tr>
<td>DBP baseline</td>
<td>−0.249</td>
<td>0.108</td>
</tr>
<tr>
<td>BAS Drive</td>
<td>0.084</td>
<td>0.268</td>
</tr>
<tr>
<td>BAS RR</td>
<td>−0.114</td>
<td>0.305</td>
</tr>
<tr>
<td>BIS</td>
<td>−0.132</td>
<td>0.177</td>
</tr>
<tr>
<td>DBP change</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anger</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sadness</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$R^2$ change</td>
<td>0.056</td>
<td>0.245$^c$</td>
</tr>
</tbody>
</table>


$^a$ Simple effects test each predictor separately for the anger and sadness conditions.

$^b$ Unique effects adjust for the positive relationship between anger- and sadness-related reactivity.

$^c$ $p < .05$.

$^{**} p < .01$. 

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**Fig. 3.** SBP reactivity to each experimental condition. Interactive effects of dispositional BAS Drive and experimental condition on DBP reactivity. Predicted values of change in DBP for the anger and sadness conditions are plotted using coefficients from the regression analysis for BAS Drive scores at 1 standard deviation above (13.2) and below (8.3) the mean.
4.2. Cardiovascular reactivity

The emotion × Drive interactions demonstrated significant differences in the relationship of Drive with SBP and DBP reactivity for anger compared with sadness. They suggest that higher levels of Drive increased motivational intensity during anger and decreased it during sadness. Simple effect analysis then estimated the Drive-CVR relationship separately for each emotion. Results showed that the associations of Drive with both SBP and DBP reactivity were statistically significant (and inverse) for the sadness condition, but the simple (positive) associations seen for anger were not. However, because emotion was manipulated within subjects, CVR during anger and sadness was correlated. Simple effect analysis does not adjust for this association. Accordingly, we estimated the relationship between Drive and SBP reactivity for both emotions, and led to a significant effect for anger in addition to that for sadness. It also strengthened the Drive-DBP effects for both anger and sadness, but statistical results were unchanged, significant for sadness but not anger.

There were no main effects of emotion on CVR. This may be because the task did not yield sufficiently qualitatively distinct and intense states of anger and sadness. However, it is also consistent with literature concerning autonomic concomitants of discrete emotions, including evidence suggesting that other factors, such as motivational disposi-
tions, may need to be considered when characterizing physiologic variations between and within different emotional states (Quigley & Barrett, 2014).

Theoretically, the findings for blood pressure reflect a positive relationship between BAS Drive scores and an active behavioral stance during a state of anger, and between Drive and behavioral disengagement in the case of sadness. There are ample theoretical and empirical bases for the inference that cardiovascular elevations in general, and blood pressure reactivity in particular, can reflect varying degrees of physiologic mobilization for action, or motivational intensity (e.g., Obrist, 1981; Wright & Barreto, 2012). Nonetheless, behavioral engagement was not measured directly in this study. Hypothesized action tendencies can only be inferred from task requirements, the emotion manipulation, and CVR.

We can find no precedent for these findings. The few studies that examined BAS and CVR involved hypertensives (Sadeghi et al., 2013) and/or tasks that did not target specific emotions (Hepomieni, Keltikangas-Järvinen, Kettunen, Puttonen, & Ravaja, 2004; Sadeghi et al., 2013). These and other methodological differences preclude direct comparisons.

Although the present findings extend previous work, SBP, DBP, and HR provide a picture of cardiovascular activity but not its underlying neural, endocrine, and hemodynamic influences. Emotion research leaves many issues unresolved, including the status of emotions as natural categories, whether they show distinct physiologic patterns, and which physiologic variables are most relevant (Quigley & Barrett, 2014). Our approach is silent on these issues; SBP, DBP, and HR were intended to capture only one dimension of emotion, namely, motivational intensity. However, it is worth noting that these measures reflect processes that may explain heightened risk of cardiovascular disease associated with anger, depression, and other emotions (Stanley & Contrada, 2012).

It is not possible to draw firm conclusions regarding the more pronounced pattern seen for SBP compared to DBP. However, SBP is typically more sensitive than DBP and HR to effects of psychological manipulations and personality attributes. This may be because, whereas both myocardial and vascular effects of sympathetic activity influence SBP, DBP is mainly affected by vascular effects, and these can be mixed since vasodilation in large muscles can offset vasoconstriction elsewhere (Miron & Brehm, 2012). Regarding HR, sympathetic effects can be overridden by parasympathetically-mediated cardiac deceleration.

4.3. Dispositional BAS and BIS constructs

Relationships of BAS Drive to SBP and DBP reactivity to anger and sadness are consistent with the view that BAS is relevant to negative affective states in addition to positive ones (e.g. Carver, 2004; Carver & Scheier, 2013). Depending on circumstances, BAS may contribute to elaboration and joy, but also to anger and sadness. By contrast with Drive, Reward Responsiveness may be more relevant when rewards are experienced, rather than thwarted. The notion of a general BAS disposition is likely useful for some purposes, but it may be important to examine subscales separately. Although BIS was unrelated to CVR, there are findings linking it to anger (e.g., Cooper, Gomez, & Buck, 2008), to responses to stimuli designed to produce anxiety or fear (Carver & White, 1994; Sadeghi et al., 2013), and to physiologic measures other than SBP, DBP, and HR (Tomaka & Palacios-Esquível, 1997). Future research should determine more precisely the conditions under which BAS and BIS are related to physiologic concomitants of affective states. States of mixed emotion may be of particular interest.

5. Conclusions

This study encourages further examination of dispositional BAS/BIS constructs in relation to emotion, motivational intensity, and CVR. Such work should involve a larger sampling of emotional states, alternative emotion induction techniques, and the use of direct measures of behavioral engagement with more comprehensive measures of CVR.

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References


