Published in final edited form as:

J Res Pers. 2011 October ; 45(5): 430–440. doi:10.1016/j.jrp.2011.05.002.

Reactive and self-regulatory dimensions of temperament: Interactive relations with symptoms of general distress and anhedonia

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Abstract

Converging evidence indicates that shared temperamental diatheses partly underlie the covariance between anxiety and depression. Although developmental psychopathology research suggests that self-regulatory temperament (e.g., effortful control or EC) mitigates reactive risks associated with negative affectivity (NA) and positive affectivity (PA), and their respective counterparts, behavioral inhibition- and activation sensitivity (BIS and BAS), no studies have established EC's protective effects in adulthood. This study examined concurrent relations between temperament and distress symptoms shared by anxiety and depression, and anhedonic symptoms unique to depression, in young adults. Anticipated two- and three-way interactions emerged supporting EC's moderating effect between reactive temperament (i.e., high BIS and low BAS) and both symptom dimensions. However, no interactive relations emerged between symptoms and NA, PA, and EC.

Keywords

Effortful control; Emotional reactivity; Self-regulation; Anxiety; Depression

1. Introduction

Given the high prevalence of disorders involving anxiety and depression (Kessler et al., 1994) and their high costs and associated functional impairments (Satcher, 2000), prevention efforts are clearly important (Barrera, Torres, & Muñoz, 2007; Flannery-Schroeder, 2006). The success of such efforts hinges on understanding the risk factors associated with such disorders. Although these factors are undoubtedly diverse, mounting evidence and theory strongly suggest that reactive aspects of temperament play important roles as vulnerabilities to such disorders (Brown, 2007; Clark, Watson, & Mineka, 1994; Kotov, Watson, Robles, & Schmidt, 2007; Muris & Ollendick, 2005; Nigg, 2006), although they also have clear potential to influence their course through pathoplastic and complication effects (see Tackett, 2006). These temperamental factors reflect variations in affective-motivational reactivity that are both inherited and shaped by experience (Rothbart & Posner, 2006).

Two aspects of temperamental reactivity of clear importance are the traits of negative affectivity (NA) and positive affectivity (PA) (Anthony, Lonigan, Hooe, & Phillips, 2002; Clark et al., 1994; Watson, Clark, & Harkness, 1994). NA reflects variability in sensitivity to negative stimuli where elevated levels are characterized by heightened reactivity to

negative stimuli associated with behavioral inhibition and displeasurable environmental engagement (Gray, 1994). NA is closely related to both neuroticism and Gray's behavioral inhibition system (BIS; Watson et al., 1994). Heightened BIS activity is associated with nonspecific arousal, inhibition of behavior, and increased attention to conditioned threat stimuli (Gray, 1994), and cognitive processes like rumination and worry (Muris, Roelofs, Rassin, Franken, & Mayer, 2005). Thus, heightened NA/BIS is viewed as a vulnerability to the general distress symptoms that are common to anxiety and depressive disorders (e.g., Brown, Chorpita, & Barlow, 1998; Mineka, Watson, & Clark, 1998).

PA reflects individual differences in reactivity to positive stimuli, where high levels of PA are characterized by pleasurable environmental engagement and attendant feelings of assertiveness, confidence, vigor, and venturesomeness (Tellegen, 1985). PA is linked with extraversion as well as Gray's behavioral activation system (BAS; Watson et al., 1994). Low levels of PA/BAS are typified by cognitive and motor slowing, disinterest, lethargy, low sensitivity to reward, behavioral withdrawal and anhedonia (Gray, 1994) and are thought to confer vulnerability to symptoms that are specific to depression (i.e., anhedonia) and social anxiety (Brown et al., 1998; Mineka et al., 1998).

Rather than being mere variants of the same underlying construct, the bulk of the empirical evidence appears to support the distinction between NA/BIS, PA/BAS and psychopathological symptoms (e.g., Lemery, Essex, & Smider, 2002; Lengua & Kovacs, 2005; Muris & Ollendick, 2005; Rapee & Coplan, 2010). The links between NA/BIS, PA/BAS and symptoms of anxiety and depression have been well-established both cross-sectionally (e.g., Anthony et al., 2002; Lonigan, Hooe, David, & Kistner, 1999; Muris, 2006; Muris, De Jong, & Engelen, 2004; Muris, Meesters, & Blijlevens, 2007; Muris, Meesters, & Spinder, 2003) and longitudinally (e.g., Caspi, Henry, McGee, Moffitt, & Silva, 1995; Eisenberg et al., 2005; Hirshfeld et al., 1992; Lonigan, Phillips, & Hooe, 2003). For example, composite measures of NA and PA at age 18 are predictive of later symptoms, such that low PA predicts risk for depression and high NA predicts both anxiety and depression (Krueger, Caspi, Moffitt, Silva, & McGee, 1996). Similarly, high NA and low PA are predictive not only of concurrent symptomatology in youth, but they also predict later depression (e.g., Joiner & Lonigan, 2000). Finally, high NA/BIS and low PA/BAS predict more chronic course of anxiety and depression and poorer overall outcome with or without treatment (Andrews, Neilson, Hunt, Stewart, & Kiloh, 1990; Brown, 2007; Duggan, Lee, & Murray, 1990; Kasch, Rottenberg, Arnow, & Gotlib, 2002; Lonigan et al., 2003; McFarland, Shankman, Tenke, Bruder, & Klein, 2006).

Despite evidence linking high NA/BIS and low PA/BAS to risk for anxiety and depressive disorders, these links are not yet fully understood. For example, although high NA/BIS is associated with heightened risk for such disorders, many individuals with this reactive vulnerability do not develop such problems, at least not at clinical levels (Degnan & Fox, 2007; Rapee, 2002). Undoubtedly, there are important factors that serve to mitigate the risks associated with reactive temperamental vulnerability. Recent theories of vulnerability to childhood emotional problems (e.g., Lonigan & Phillips, 2001; Muris & Ollendick, 2005; Nigg, 2006) have emphasized the protective effects of self-regulatory aspects of temperament that enable individuals who are reactively at risk to modulate their emotional, cognitive, and behavioral responses. This self-regulatory dimension of temperament, labeled "effortful control" (EC) by Rothbart (1989), includes the capacity to override or compensate for reactive vulnerability through voluntary inhibitory control, activation control, and attentional control (Posner & Rothbart, 2000; Rothbart & Bates, 1998). Thus, an individual who is high in both NA/BIS and EC can override his/her negative reactive tendencies (e.g., to avoid aversive stimuli or to worry or ruminate) and substitute more adaptive responses (e.g., actively re-engage in the task at hand). Similarly, an individual who is low in PA/BAS

but high in EC can override his/her lethargy and approach potentially pleasurable or rewarding situations despite lacking immediate motivation to do so, or having strong avoidance motivation.

The role of EC as a moderator of reactive vulnerability has been considered in a model of risk for childhood anxiety offered initially by Lonigan and Phillips (2001; also see Lonigan, Vasey, Phillips, & Hazen, 2004), which itself is informed by the work of Rothbart and colleagues (e.g., Rothbart, 1989; Rothbart & Bates, 1998). In this model, high NA/BIS is viewed as a necessary but not sufficient condition for the development of the general distress seen in anxiety and depressive disorders. Such reactivity is likely to lead to clinical levels of distress only when paired with low capacity for self-regulation (i.e., low EC). In other words, while a person high in NA/BIS is predisposed toward the experience of intrusive negative thoughts, behavioral inhibition, and cognitive biases favoring negative stimuli, these tendencies can be successfully controlled provided he/she has good capacity for EC. There is growing evidence supporting this view. For example, Lonigan and Vasey (2009) showed that children high in NA and low in EC exhibited a significant attentional bias toward threat cues on a probe detection task whereas those high in NA and high in EC did not differ from low NA controls, who showed no bias toward threat regardless of their level of EC. Derryberry and Reed (2002) reported a similar pattern in adults. Thus, the risk linking high levels of NA/BIS to general distress should be moderated by EC.

Although Lonigan et al. (2004) focused primarily on EC's role as a moderator of the relation between NA and anxiety they made it clear that the $NA \times EC$ interaction should also hold for depressive symptoms. Further, they noted that EC should also modulate the link between PA and depression (see also Anthony et al., 2002). Specifically, low PA individuals who are high in EC should be able to engage in adaptive, active approach of pleasurable stimuli despite their lack of reactive motivation to do so and, thus, attenuate their risk for depression or reduce its persistence. Thus, the risk linking low levels of PA/BAS to the anhedonic features of depression should also be moderated by EC.

The notion that reactive temperamental vulnerabilities to psychopathology can be moderated by a regulatory trait dimension is consistent with other "dual process" models of cognition and behavior that conceptualize the processing of experience in terms of separate though interacting systems within the human nervous system (e.g., see Carver, Johnson, & Joorman, 2008; Depue & Lenzenweger, 2005). Such models emphasize interactions between primitive, lower-order processing systems on the one hand (mapping onto NA/BIS and PA/BAS in the present context) and a more recently evolved higher-order processing system (which maps onto EC).

In addition to the modulating effects of EC on the risks associated with high NA/BIS and low PA/BAS, there is clear reason to expect an interaction between the two reactive dimensions themselves, in relation to symptoms of anxiety and depression (Fredrickson & Joiner, 2002). A growing number of studies of youth samples have shown that the link between high levels of NA and depressive symptoms is moderated by PA. Specifically, heightened NA predicts elevated depressive symptoms much more strongly at low versus high levels of PA (e.g., Joiner & Lonigan, 2000; Loney, Lima, & Butler, 2006; Wetter & Hankin, 2009). Similar results have been reported for BIS and BAS (e.g., Hundt, Nelson-Gray, Kimbrel, Mitchell, & Kwapi, 2007; Knyazev, Slobodskaya, & Wilson, 2004).

Given that reactive risk appears to be an interactive function of NA/BIS and PA/BAS, we suggest that the focus on 2-way interactions in past studies of the moderating role of EC is insufficient. Instead, the protective role of EC should be considered in the context of the $NA/BIS \times PA/BAS$ interaction, wherein risk for symptoms is markedly stronger at the

combination of high NA/BIS and low PA/BAS than would be expected if their effects were merely additive. It is in the context of that joint synergistic risk that low levels of EC should be most problematic, thus implying the need to test the NA/BIS $\times$ PA/BAS $\times$ EC interaction (for a thorough discussion of this 3-way interaction, see Harbaugh et al., 2011). By way of illustration, individuals who are highly reactive to stress (i.e., high NA/BIS), but who also have low approach motivation (i.e., low PA/BAS), are at particular risk for depressive symptoms, because they are not only prone to experience distress but also have little motivation to engage in pleasurable activities that could counteract their distress. This dual, synergistic reactive vulnerability would be particularly strong among those with low EC. However, it should be attenuated among individuals with high EC because they are able to effectively stop, through attention control, the worrisome or ruminative thought processes associated with heightened NA/BIS and to initiate approach behaviors, through activation control, despite their low motivation to do so. Thus, it seems reasonable to hypothesize a three-way interaction between NA/BIS, PA/BAS, and EC. However, to our knowledge, that interaction has not been tested to date.

Although the foregoing discussion underscored the role of EC as a moderator of reactive temperamental vulnerability to anxiety and depression, it should be noted that a main effect of EC is also expected. Studies of youth samples have repeatedly shown that, independent of reactive temperament, deficits in EC are associated with internalizing symptoms, including depression (De Boo & Kolk, 2007; De Boo & Spiering, 2010; Loukas & Robinson, 2004; Muris, van der Pennen, Sigmond, & Mayer, 2008; Muris et al., 2007; Oldehinkel, Hartman, Ferdinand, Verhulst, & Ormel, 2007; Verstraeten, Vasey, Raes, & Bijttebier, 2009). In light of the relevance of effortful self-regulation to everyday domains of behavior that have the potential to generate stress, including nonconformity to social norms, failure to meet work deadlines, and the mismanagement of one's finances (e.g., Faber & Vohs, 2004; Tangney, Baumeister, & Boone, 2004), it seems reasonable to expect that low levels of EC may serve as a vulnerability for anxiety and depression that is independent of reactive temperament in adults.

In summary, a growing body of cross-sectional and longitudinal research supports this interactive model in youth samples (e.g. Caspi et al., 1995; Joiner & Lonigan, 2000; Lengua, 2002; Lonigan et al., 2004; Muris, 2006; Muris et al., 2007; Oldehinkel et al., 2007; Wetter & Hankin, 2009). However, no studies to date have tested this interactive model in adults. The purpose of this study was to provide the first cross-sectional test of this model in adults. Before listing our specific hypotheses it is important to note that in contrast to most past studies, which have focused on symptoms of anxiety and depression broadly construed, we have chosen to distinguish between those symptoms common to anxiety and depression (i.e., general distress) and those symptoms unique to depression (i.e., anhedonia).¹ According to the tripartite model (Clark & Watson, 1991), NA/BIS should be most related to distress and PA/BAS should be most associated with anhedonia. Thus, it is possible that these temperament dimensions will relate in different ways to these two symptom dimensions. In the context of these dependent variables, we hypothesized that: (1) EC will be associated with symptoms of generalized distress and symptoms of anhedonia; (2) EC will moderate the association between NA/BIS and symptoms of distress and between PA/BAS and symptoms of anhedonia; (3) NA/BIS and PA/BAS will interact to predict both types of symptoms; and (4) NA/BIS $\times$ PA/BAS $\times$ EC will interact to predict symptoms of general

¹The tripartite model identifies a third dimension of physiological arousal as being a specific aspect of anxiety. We chose not to examine such symptoms because they are not clearly related to the two reactive dimensions of temperament considered in this study. Such symptoms are linked to the dimension of physiological hyperarousal (PH) and it is unclear to what extent that dimension is temperamental (Clark et al., 1994). Furthermore, that dimension is primarily related to panic disorder (Brown et al., 1998). Consequently, in the absence of clear theory-based predictions, we chose not to inflate our Type I error rate by including such symptoms in the analyses.

distress and anhedonia. Finally, we chose to test for moderation of these effects by sex because sex differences in the pattern of links between temperament and depression may help to explain the greater prevalence of depression in females versus males (Hyde, Mezulis, & Abramson, 2008). Consistent with this view, Verstraeten et al. (2009) reported that sex moderated the NA $\times$ EC and PA $\times$ EC interactions in relation to depressive symptoms in their youth sample. Therefore, we included interactions involving sex in all regression models.

2. Material and methods

2.1. Participants and procedure

Participants were 477 undergraduate students (56% female) at The Ohio State University who received partial course credit for enrolling in the study. They had a mean age of 19.2 years ($SD = 2.1$) and were predominantly Caucasian (80.4%). Small groups of 20–40 respondents completed questionnaires, which were administered in a randomly ordered packet.

2.2. Measures

Attentional Control Scale (ACS; Derryberry & Reed, 2002). The ACS is a 20-item measure that assesses the general capacity for attentional control. Items on the ACS require participants to rate their degree of voluntary attentional control on a 4-point Likert response scale. The scale has good internal consistency ($\alpha = .88$ in the present sample), and high test–retest reliability across a 6 week interval ($r = .83$; Vasey, 2008).

Balanced Inventory of Desirable Responding (BIDR; Paulhus, 1984). The BIDR is a 40-item measure of two constructs: self-deception positivity (the tendency to provide honest yet positively biased self-reports) and impression management (IM; deliberate positive self-presentation). The BIDR subscales have good internal consistency and adequate test–retest reliability (Paulhus, 1984).

Behavioral Inhibition System and Behavioral Activation System Scales (BIS/BAS Scales; Carver & White, 1994). The BIS/BAS is a 20-item self-report measure designed to assess the primary constructs of Gray's theory (Gray, 1994). The BIS/BAS asks respondents to indicate on a 4-point Likert response scale the extent to which they agree with statements concerning their emotional reactions to potentially harmful or rewarding events. The BIS/BAS subscales have adequate internal consistency and test–retest reliability (Carver & White, 1994).

Effortful Control Scale – Persistence/Low Distractibility Subscale (ECS-P/LD; Lonigan, 1998). The *persistence and low distractibility* subscale of the ECS is a 12-item measure designed to tap the attention and activation control facets of Rothbart's EC dimension. Although originally designed for use with children, the items are appropriate for college students and factor analyses support the ECS-P/LD scale in undergraduate samples (Vasey, 2008). The scale has good internal consistency and excellent test–retest reliability across a 7-week interval ($r = .80$; Vasey, 2008).

Mood and Anxiety Symptom Questionnaire (MASQ; Watson & Clark, 1991; Watson et al., 1995; Watson et al., 1995). The MASQ is a 90-item self-report measure designed explicitly to assess Clark and Watson's (1991) tripartite model of anxiety and depression. MASQ items were developed from the symptom criteria for the anxiety and mood disorders. For this study, we used the General Distress (MASQ-GD) and Anhedonia (MASQ-A) subscales identified by Watson, Weber, et al. (1995). These scales have adequate reliability and validity.

Positive and Negative Affect Schedule – Trait Form (T-PANAS; Watson, Clark, & Tellegen, 1988). The T-PANAS is a self-report questionnaire composed of 20 mood adjectives designed to measure the trait dimensions of PA and NA. The NA and PA scales have good internal consistency, test–retest reliability, and validity (Watson et al., 1988).

3. Results and discussion

3.1. Preliminary analyses

All analyses reflect data for 467 (56% female) participants having complete data. Table 1 presents descriptive statistics and correlations for all measures. In general, all variables were correlated in a manner consistent with expectations. However, although NA and PA were positively correlated with BIS and BAS respectively, these correlations were modest and therefore we analyzed these scales separately rather than aggregating them as originally planned. EC was represented by a composite formed by averaging the standardized item scores from the ACS and the EC P/LD scale ($\alpha = .90$; $r = .45$, $p < .001$).² Additionally, we chose to sum BAS items across the three BAS subscales on the BIS/BAS to create a single BAS scale ($\alpha = .89$). Finally, preliminary analyses demonstrated that all variables adequately met the assumptions underlying multiple linear regression (MLR) analysis (see Cohen, Cohen, West, & Aiken, 2003).

3.2. Data-analytic strategy

For all MLR models, following Aiken and West (1991), continuous variables were centered. They were additionally standardized to enhance interpretability. Each analysis involved six hierarchical steps. In Step 1, we included Sex and both the BIDR Impression Management (BIDR-IM) and Self Deception (BIDR-SD) to control for socially desirable responding.³ Step 1 also included the EC Composite and either NA and PA or BIS and BAS (the remaining steps are illustrated using NA and PA). Step 2 added the two-way interactions between the temperament dimensions (e.g., $NA \times EC$, $PA \times EC$, $NA \times PA$) and Step 3 added the three-way interaction (e.g., $NA \times PA \times EC$). Steps 4–6 tested for moderating effects of Sex. Specifically, Step 4 added the two-way interactions involving sex (e.g., $NA \times Sex$, $PA \times Sex$, and $EC \times Sex$). Step 5 added the three-way interactions (e.g., $NA \times EC \times Sex$, $PA \times EC \times Sex$, and $NA \times PA \times Sex$). Finally, Step 6 tested the four-way interaction (e.g., $NA \times PA \times EC \times Sex$).

3.3. NA, PA, and EC results

3.3.1. MASQ general distress (MASQ-GD) results—As summarized in Table 2, the variables entered on Step 1 accounted for 57% of the variance in MASQ-GD. Steps 2–6 failed to yield any significant increments in R^2 . Considering Step 1, Sex was positively associated with MASQ-GD, with females reporting more than males. With regard to

²Rothbart and colleagues (e.g., Evans & Rothbart, 2007) have proposed that effortful control (EC) involves three facets: Attentional Control, Activation Control, and Inhibitory Control. However, the present study predates our realization that there may be important facet effects. Nevertheless, our composite EC measure principally consists of items tapping Activation Control (EC-P/LD) and Attentional Control (ACS). Although Inhibitory Control is not included in our composite EC measure, it is worth noting that such items in Rothbart's measures involve the control of impulsive approach/appetitive motor responses, which are not central to internalizing psychopathology. Moreover, in a separate sample of 143 young adults (Vasey, 2011), our EC Composite evidenced the following correlations with the EC-related subscales of the Adult Temperament Questionnaire (ATQ-EC): Attentional Control ($r = .77$, $p < .001$), Activation Control ($r = .70$, $p < .001$), Inhibitory Control ($r = .43$, $p < .001$), and EC Broadband Score ($r = .79$, $p < .001$). It should be noted that, in that sample, the correlation between the ACS and EC P/LD scales was similar in magnitude to that found in the current study, $r = .49$.

³Controlling for such response tendencies has the potential to remove relevant variance from the constructs of interest. However, in this case we thought it important to include the Impression Management and Self Deception scales to make it clear that the effects of EC were not merely explainable in terms of those constructs. However, the pattern of results was the same when these variables were not included.

temperament, MASQ-GD was positively associated with NA and negatively associated with PA and EC.

3.3.2. MASQ anhedonia (MASQ-A) results—As summarized in Table 2, the variables entered on Step 1 accounted for 50% of the variance in MASQ-A. Beyond Step 1, only Step 4 yielded a significant increment in R^2 . Although the three temperament variables were all significant in Step 1, only PA remained significant in Step 4. Examination of the simple slopes for males and females stemming from the NA $\times$ Sex interaction revealed that NA was significantly associated with MASQ-A only among females (slope_{females} = .16, p = .004; slope_{males} = -.06, p = .62). Finally, there was a significant effect of BIDR-SD, with higher self-deception being associated with lower MASQ-A.

3.4. BIS, BAS, and EC results

3.4.1. MASQ-GD results—As summarized in Table 3, the variables entered on Step 1 accounted for 33% of the variance in MASQ-GD. Steps 2 and 3 each produced a significant increment in R^2 , whereas Steps 4–6 did not. The significant BIS $\times$ BAS $\times$ EC interaction found in Step 3 was interpreted following Preacher, Curran, and Bauer (2006) and using their internet-based utility (available at <http://www.quantpsy.org>). Specifically, we examined simple slopes at high (+1 SD) and low (–1 SD) values for z-BIS, z-BAS, and z-EC. Fig. 1 illustrates this interaction with lines representing z-EC's association with MASQ-GD at the four combinations of reactivity (i.e., low (–1 SD) and high (+1 SD) values for z-BIS and z-BAS). Simple slopes for these four lines were: low BAS + high BIS = –.45, p < .001; low BAS + low BIS = –.32, p < .001; high BAS + high BIS = –.60, p < .001; and high BAS + low BIS = –.19, p < .05. Examination of the region of significance revealed that EC was significantly associated with MASQ-GD for all levels of BIS at z-BAS = –1 SD . At z-BAS = +1 SD , EC was significantly associated with MASQ-GD except when BIS was low (i.e., z-BIS < –1.15 SD). Examination of Fig. 1 shows that predicted values for MASQ-GD were highest when high BIS co-occurred with low EC, regardless of level of BAS.

Supplemental analyses revealed that the predicted level of general distress associated with low EC + high BIS was .82 when BAS was low and .76 when BAS was high. Both values are significantly higher than the average general distress score (i.e., higher than zero, p < .001). Thus, when EC was low, high levels of BIS predicted distress significantly above average at both low and high levels of BAS. In contrast, general distress at high EC + high BIS was –.08 (p = .60) when BAS was low and –.44 (p = .001) when BAS was high. Thus, when EC was high, high levels of BIS predicted a level of distress significantly below average when BAS was high and no different from average when BAS was low. When BIS was low and EC was high, predicted levels of distress were significantly below average for both low BAS (–.44, p < .001) and high BAS (–.74, p < .001). When BIS was low and EC was low, predicted levels of distress were no different than average when BAS was low (.12, p = .31) but below average when BAS was high (–.37, p = .005).

3.4.2. MASQ-A results—As summarized in Table 3, the variables entered on Step 1 accounted for 33% of the variance in MASQ-A. Steps 2 and 6 each produced a significant increment in R^2 , whereas Steps 3–5 did not. The significant BIS $\times$ BAS $\times$ EC $\times$ Sex interaction was interpreted by fitting separate regression models for males and females. These models are summarized in Table 4. Table 4 shows that although there was a significant BIS $\times$ BAS $\times$ EC interaction for males, this 3-way interaction was not significant for females.

The significant BIS $\times$ BAS $\times$ EC interaction found for males was interpreted following Preacher et al. (2006). Specifically, we again examined simple slopes for z-EC at the four

combinations of levels of BIS and. Fig. 2 illustrates this interaction with lines representing EC's association with MASQ-A among males at the four combinations of low (-1 *SD*) and high ($+1$ *SD*) values for *z*-BAS and *z*-BIS. Examination of the simple slopes for these lines revealed that, whereas the slope for low BAS + high BIS was significant (slope = $-.60$, $p < .001$), the simple slopes for the other three combination were not: low BAS + low BIS = $-.07$, $p = .54$; high BAS + high BIS = $-.22$, $p = .21$; and high BAS + low BIS = $-.12$, $p = .29$. Examination of the region of significance revealed that EC was not significantly associated with MASQ Anhedonia for any value of BIS at *z*-BAS = $+1$ *SD*. However, at *z*-BAS = -1 *SD*, EC was significantly associated with MASQ-A except when BIS was low (*z*-BIS $< -.51$).

Supplemental analyses revealed that the predicted level of anhedonia associated with low EC + low BAS was $.20$ ($p = .18$) when BIS was low and $.72$ ($p = .0001$) when BIS was high. When EC was low and BAS was high, anhedonia was $-.27$ ($p = .16$) when BIS was low and $.27$ ($p = .17$) when BIS was high. When EC was high and BAS was low, predicted levels of anhedonia were $.06$ ($p = .68$) when BIS was low and $-.47$ ($p = .04$) when BIS was high. When EC was high and BAS was high, predicted levels of anhedonia were significantly below average for low BIS ($-.52$, $p = .0002$) but no different than average for high BIS ($-.16$, $p = .52$).

As shown in Table 4, the 3-way interaction was not significant for females. Instead, Step 2 revealed significant BIS $\times$ EC and BAS $\times$ EC interactions. These interactions were interpreted following Preacher et al. (2006). As shown in Fig. 3, at *z*-EC = -1.0 , BAS was significantly negatively correlated with anhedonia (simple slope = $-.37$, $p < .001$). In contrast, at *z*-EC = $+1$, BAS was unrelated to anhedonia (simple slope = $.04$, $p = .68$). Examination of the region of significance revealed that BAS was significantly associated with anhedonia only when *z*-EC was less than $+0.15$. Supplemental analyses revealed that the predicted level of anhedonia when both BAS and EC were low was $.79$ ($p < .001$) whereas when BAS was low but EC was high, anhedonia was significantly below average ($-.56$, $p < .001$). In contrast, high BAS combined with low EC predicted anhedonia near the average ($.06$, $p = .67$) whereas high BAS plus high EC predicted anhedonia significantly below average ($-.51$, $p < .001$).

As shown in Fig. 4, for low levels of *z*-EC (-1 *SD*), BIS was significantly positively associated with anhedonic symptoms (simple slope = $.25$, $p = .0011$). However, for high levels of *z*-EC ($+1$ *SD*), BIS was unrelated to anhedonia (simple slope = $.01$, $p = .90$). Examination of the region of significance showed that BIS was significantly associated with anhedonia only when *z*-EC was less than $+0.76$. Supplemental analyses revealed that the predicted level of anhedonia for high BIS plus low EC was $.67$ ($p < .001$) whereas when high BIS was combined with high EC, anhedonia was significantly below average ($-.51$, $p < .001$). In contrast, low BIS combined with low EC predicted anhedonia near the average ($.17$, $p = .20$) whereas low BIS plus high EC predicted anhedonia significantly below average ($-.53$, $p < .001$).

3.5. Exploration of failure to find effects involving NA and PA

These findings leave unclear why EC interacted as expected with BIS and BAS but did not do so with NA and PA. One possibility reflects the fact that the associations of NA and PA with symptoms were substantially stronger than the comparable correlations involving BIS or BAS. As shown in Table 1, whereas MASQ-GD correlated $r = .70$ with NA, it correlated only $r = .40$ with BIS. Similarly, MASQ-A correlated $r = -.63$ with PA, but only $r = -.27$ with BAS. This overlap of NA and PA with the symptom measures led us to hypothesize that NA and PA may carry variance associated with EC and with the BIS $\times$ EC and BAS $\times$ EC interactions. With few exceptions, the items on the BIS/BAS scales ask about one's

reactions to various situations rather than the extent to which those reactions persist (e.g., “When I get something I want, I feel excited and energized.”). In contrast, the trait version of the PANAS asks participants to rate the extent to which they feel various positive and negative affective states (e.g., “distressed” or “nervous”) “most of the time.” In our view, people who indicate that they usually feel very distressed and nervous, are saying something not only about their emotional reactions but also about their ability to regulate them. Consistent with this view, as shown in Table 1, EC was more strongly correlated with NA ($r = .39$) than with BIS ($r = -.28$) and with PA ($r = .49$) than with BAS ($r = .17$), although this difference was significant only in the latter case. Furthermore, as shown in Table 5, supplemental regression analyses revealed that EC interacted with both BIS and BAS in predicting NA and with BIS in predicting PA. Specifically, examination of the regions of significance revealed that BIS was significantly positively correlated with NA but only when EC was low (i.e., $z\text{-EC} < 1.12$). BAS was significantly negatively correlated with NA but only when EC was low (i.e., $z\text{-EC} < -.18$), and BIS was significantly negatively correlated with PA but only when EC was low (i.e., $z\text{-EC} < -.41$).

4. Conclusions

The results of this study provide initial support for extending to adults an interactive temperamental model of risk for symptoms of anxiety and depression (specifically general distress and anhedonia), heretofore tested only in youth samples. Although the main effect links between such symptoms and the two reactive aspects of temperament (i.e., NA/BIS and PA/BAS) are well established among adults, the present results strongly suggest that future research on these links should consider also main effects of EC (i.e., self-regulatory aspects of temperament) as well as its moderating effect on the association between these dimensions of reactivity and symptoms. Finally, the present study extends previous findings in youth samples by testing for the first time a three-way (NA/BIS $\times$ PA/BAS $\times$ EC) interaction, which was based on a model of dual, synergistic reactive risk (i.e., high NA/BIS plus low PA/BAS) interacting with EC. Results supported this model and suggest that future studies should be designed to optimally test this interaction.

Unsurprisingly, the zero-order correlations between the reactive dimensions of temperament and symptoms of general distress and anhedonia were consistent with previous findings. However, when these reactive dimensions were considered in the context of a model emphasizing not only reactivity but also EC, a more complex picture emerged. First, consistent with theory-based predictions and with findings from studies of youth samples, as predicted given the many paths by which poor self-regulatory capacity may lead to anxiety and depression, EC was negatively correlated with symptoms of general distress and anhedonia above and beyond their associations with NA/BIS and PA/BAS. These associations were observed not only at the zero-order level, but also in the regression analyses, showing that they were not merely a function of EC's associations with other variables in the regression models. For example, although EC was positively associated with PA, and PA may buffer against the effects of NA (e.g., Fredrickson & Joiner, 2002), that association did not account for EC's negative correlation with symptoms. Similarly, although EC was positively correlated with self-deceptive response bias and, less strongly, to impression management as measured by the BIDR, EC remained negatively correlated with symptoms when both BIDR scores were partialled out.

It was in interaction with the two reactive dimensions that the importance of considering EC became most apparent. Consistent with expectations, EC proved to interact with both BIS and BAS in predicting symptoms, although it did not do so in the case of NA and PA. Possible reasons for the latter finding are discussed below, but it is first worth considering EC's interactive relations with BIS and BAS, in regards to both symptoms of general distress

and anhedonia. In both cases, the pattern of results pointed not only to the importance of the two-way interactions, as emphasized in past studies of youth, but to the three-way interaction between these temperament dimensions.

Regarding general distress, examination of the $BIS \times BAS \times EC$ interaction revealed that EC was negatively associated with general distress except at levels of BIS and BAS that signal little reactive vulnerability (i.e., low BIS scores and high BAS scores). In other words, EC proved irrelevant only when there was little reactivity to regulate. In considering the predicted levels of general distress at the various combinations of levels of BIS, BAS, and EC depicted in Fig. 1, it is noteworthy that the highest levels were predicted when EC was low and BIS was high, regardless of the level of BAS. It may initially seem surprising that at low EC, high BAS was not associated with a reduction in the magnitude of the association between BIS and general distress. However, this is consistent with findings in at least one other study. Hundt et al. (2007) reported the combination of high BIS and high BAS predicted elevated mixed anxiety – depression symptoms (measured with the MASQ-General Distress scale). Hundt et al. (2007) suggest that this may reflect the fact that high BIS, combined with high BAS, may engender more frequent or intense approach – avoidance conflicts which contribute to elevation in general distress. Indeed, other findings suggest that high BAS is related to low frustration tolerance and impulsivity, which may engender conflict and stress (e.g., Hundt, Kimbrel, Mitchell, & Nelson-Gray, 2008), something that would seem particularly likely when EC is low. It will be important for future research to further examine the link between high BIS combined with high BAS and symptoms of mixed anxiety and depression. Furthermore, we suggest that such a combination may also have potential relevance to understanding risk for (hypo)manic symptoms (Alloy et al., 2009).

A $BIS \times BAS \times EC$ interaction also emerged in the case of anhedonic symptoms. Although that effect was further moderated by sex, it should be noted that the three-way interaction was significant on average (see Step 3 of the final model in Table 3). Separate regression models for males and females revealed that this three-way interaction was significant only among males. Although the pattern of the interaction was similar for females, it did not achieve significance. Instead, several two-way interactions emerged. Among women, both low BAS and high BIS were predictive of elevated anhedonia, except when EC was high (see Figs. 3 and 4). By contrast, among men, although the $BIS \times EC$ interaction was significant at average levels of BAS, this interaction was further moderated by BAS (see Table 4). As depicted in Fig. 2, this $BIS \times BAS \times EC$ interaction revealed that the highest levels of anhedonia were seen at low EC when both reactive risks were present (i.e., high BIS and low BAS). However, remarkably when EC was high, the predicted level of anhedonia among those with this dual reactive vulnerability was very similar to the level predicted when reactive risk was low (i.e., low BIS and high BAS). Thus, while these sex differences might initially appear complex, they ultimately converge in a manner consistent with the prediction that high self-regulatory capacity (i.e., high EC) attenuates reactive vulnerability to symptoms of anhedonia. In the case of males, that reactive vulnerability reflected the synergistic combination of high BIS and low BAS whereas among females, these reactive dimensions did not interact. Although Verstraeten et al. (2009) also reported sex differences in interactions between reactive dimensions and EC the pattern was not entirely consistent with that observed in the present study. It remains to be seen in future studies if these sex differences prove replicable.

One puzzling question raised by our findings is why moderating effects of EC were observed in the case of BIS and BAS but not for NA and PA. Results of our exploratory analyses stemming from this question suggest that the trait form of the PANAS taps NA and PA in a manner that leads them to share substantial variance with measures of EC. As noted

in the results section, people who report that they generally experience high levels of NA they are saying not only that they are prone to experience strong negative emotional reactions but, because they chronically experience such emotion, they are also have low capacity to regulate such reactions. Consistent with this view, our regression analysis showed that NA was as strongly correlated with EC as with BIS. Moreover, it was predicted by both the $BIS \times EC$ and $BAS \times EC$ interactions. Results for PA were similar. PA was as strongly correlated with EC as with BAS. Although it was not predicted by the $BAS \times EC$ interaction, the $BIS \times EC$ interaction was significant. These results suggest that the trait form of the PANAS is not a good candidate for future studies of this sort in that it taps NA and PA in such a manner that they already carry the main and moderating effects of EC. That said, it should be noted that past youth studies have found the predicted interactions using such measures. Thus, the present results may be, at least in part, sample specific. Still, it would appear to be unwise in future research to rely purely on such measures of reactive risk.

4.1. Limitations and future directions

Although the results of this study are consistent with the interactive temperament model of vulnerability to anxiety and depression, a number of important limitations must be noted. First, the correlational nature of these results also leave open the possibility that other variables associated with the reactive and self-regulatory temperamental variables and symptoms of anxiety and depression may account for the observed relations. Related to this, the fact that all variables in the study were measured at the same point in time means that the direction of the associations among the variables of interest is unclear. In the present context our primary emphasis is on their potential to interact synergistically as vulnerabilities and protective factors. However, as discussed by Tackett (2006) there is also clear potential for other models to operate. For example, temperamental factors may have pathoplastic effects, in which they serve to moderate the expression of a disorder without necessarily having a direct causal role in its onset. Complication effects are also possible, in which aspects of temperament are altered by virtue of developing a disorder. In our view both are likely in the present context. For example, given the link between depression and impaired cognitive function it seems likely that the development of depression may result in further reductions in EC. The potential of this and similar complicating effects (e.g., reduced PA) to set up a self-perpetuating process by which depression is maintained is readily apparent. Finally, it should be noted that these alternative models are not incompatible with the vulnerability model. In other words, temperamental factors may serve not only as vulnerabilities to the onset of anxiety and depression, but may also modulate and/or complicate their course. Such possibilities will require prospective research designs to sort out.

Finally, this study relied exclusively on self-report data and thus it is possible that some part of the observed relations may reflect shared method variance. Future studies would benefit from inclusion of other reporters as well as measurement modalities other than questionnaires (e.g., performance-based measures of self-regulatory capacity).

4.2. Summary

In summary, the results of this study support the core hypothesis that self-regulatory aspects of temperament must be considered in understanding the association between temperamental reactivity and symptoms of anxiety and depression. In particular, the present results provide the first evidence in support of a synergistic triple vulnerability model in which symptoms of general distress and anhedonia are especially likely to occur and/or become problematic in individuals high in negative reactivity, low in positive reactivity, and low in EC.

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Fig. 1. BIS $\times$ BAS $\times$ EC interaction at high (+1 SD) and low (-1 SD) values of BIS, BAS, and EC, with tests of significance of predicted levels of MASQ-General Distress compared to zero (i.e., average levels).

Fig. 2. BIS $\times$ BAS $\times$ EC interaction among males at high (+1 SD) and low (-1 SD) values of BIS, BAS, and EC, with tests of significance of predicted levels of MASQ-Anhedonia compared to zero (i.e., average levels).

Fig. 3. BAS $\times$ EC interaction among females at high (+1 SD) and low (-1 SD) values of BAS, and EC, with tests of significance of predicted levels of MASQ-Anhedonia compared to zero (i.e., average levels).

Fig. 4. BIS $\times$ EC interaction among females at high (+1 SD) and low (-1 SD) values of BIS, and EC, with tests of significance of predicted levels of MASQ-Anhedonia compared to zero (i.e., average levels).

Table 1

Correlations and descriptive statistics for primary measures.

	1	2	3	4	5	6	7	8	9	10	M	SD
1. Sex	-											
2. PANAS NA	.07 [‡]	(.82)									21.2	6.1
3. PANAS PA	.00 [‡]	-.25	(.84)								34.4	6.3
4. BIS	.31	.31	-.21	(.78)							19.5	3.9
5. BAS	.12	-.12	.26	.12	(.89)						41.8	6.5
6. EC composite	-.05 [‡]	-.39	.49	-.28	.17	(.90)					0.0	0.85
7. MASQ-general distress	.12	.70	-.39	.40	-.14	-.50	(.97)				77.6	28.6
8. MASQ-anhedonia	.04 [‡]	.43	-.63	.24	-.27	-.50	.60	(.93)			39.2	12.1
9. BIDR-IM	.12	-.18	-.18	.01 [‡]	.01 [‡]	.26	-.14	-.18	(.76)		4.7	3.4
10. BIDR-SD	-.08 [‡]	-.29	-.29	-.34	.18	.51	-.34	-.44	.43	(.76)	5.4	3.7

Note: $n = 467$.

[‡] All r s > are significant at $p < 0.05$, two-tailed except those noted. Cronbach's alphas are shown on the diagonal. Sex = 0 (male) or 1 (female). PANAS NA = Negative Affectivity Subscale of Positive and Negative Affect Schedule – Trait Form; PANAS PA = Positive Affectivity Subscale of Positive and Negative Affect Schedule – Trait Form; BIS = Behavioral Inhibition System Subscale of Behavioral Inhibition System and Behavioral Activation System Scales; BAS = Behavioral Activation System Subscale of Behavioral Inhibition System and Behavioral Activation System Scales; EC Composite = Averaged standardized item scores from Attentional Control Scale and Effortful Control Scale – Persistence/Low Distractibility Subscale (ECS – P/LD); MASQ-General Distress = General Distress Subscale of Mood and Anxiety Symptom Questionnaire; MASQ-Anhedonia = Anhedonia Subscale of Mood and Anxiety Symptom Questionnaire; BIDR-IM = Impression Management Subscale of Balanced Inventory of Desirable Responding; BIDR-SD = Self-Deception Subscale of Balanced Inventory of Desirable Responding.

Table 2
NA, PA, and EC regression models predicting MASQ general distress and MASQ Anhedonia.

Step/variables added	MASQ general distress			MASQ anhedonia				
	R ²	Δ R ²	B (SE) at final significant step	sr at Final significant step	R ²	Δ R ²	B (SE) at final significant step	sr at Final significant step
Step 1:	.57***				.50***			
Constant			−.07 (.05)				−.01 (.05)	
Sex			.12** (.06)	.06**			−.01 (.07)	−.00
z-BIDR-IM			.04 (.03)	.04			.04 (.04)	.04
z-BIDR-SD			−.01 (.04)	−.01			−.12*** (.05)	−.09***
z-EC			−.23*** (.05)	−.15***			−.11 (.16)	−.02
z-NA			.58*** (.03)	.53***			−.06 (.12)	−.02
z-PA			−.15*** (.04)	−.13***			−.50*** (.12)	−.13***
Step 2:	.57***	.00			.51***	.00		
NA × EC							−.02 (.04)	−.01
PA × EC							.03 (.04)	.02
NA × PA							.02 (.04)	.02
Step 3:	.57***	.004*			.51***	.00		
NA × PA × EC							−.05 (.03)	−.05
Step 4:	.58***	.006*			.52***	.01**		
NA × Sex							.22*** (.07)	.10***
PA × Sex							.02 (.08)	.01
EC × Sex							−.03 (.10)	−.01
Step 5:	.58***	.00			.52***	.00		
NA × EC × Sex,								
PA × EC × Sex,								
NA × PA × Sex								
Step 6:	.58***	.00			.53***	.01		

Step/variables added	MASQ general distress				MASQ anhedonia			
	R^2	ΔR^2	B (SE) at final significant step	sr at Final significant step	R^2	ΔR^2	B (SE) at final significant step	sr at Final significant step
NA $\times$ PA $\times$ EC $\times$ Sex								
Note: sr = semi-partial correlation; Sex = 0 (male) or 1 (female); BIDR-IM = Balanced Inventory of Desirable Responding – Impression Management; BIDR-SD = Balanced Inventory of Desirable Responding – Self Deception; EC = EC Composite. B (SE) and sr values excluded for steps where additional entries do not result in a significant change in R^2 (ΔR^2).								
* $p < .10$.								
** $p < 0.05$.								
*** $p < 0.01$.								

Table 3
BIS, BAS, and EC regression models predicting MASQ general distress and MASQ Anhedonia.

Step/variables added	MASQ general distress				MASQ anhedonia			
	R^2	ΔR^2	B (SE) at final significant step	sr at Final significant step	R^2	ΔR^2	B (SE) at final significant step	sr at Final significant step
Step 1:	.33***				.33***			
Constant			-.09 (.06)				-.04 (.06)	
Sex			.07 (.08)	.03			-.02 (.08)	-.01
z-BIDR-IM			-.07 (.04)	-.06			-.02 (.04)	-.02
z-BIDR-SD			-.04 (.05)	-.03			-.23*** (.05)	-.17
z-EC			-.42*** (.05)	-.30***			-.05 (.05)	-.01
z-BIS			.35*** (.05)	.29***			.13 (.05)	.03
z-BAS			-.11*** (.04)	-.10***			-.17 (.04)	-.04
Step 2:	.35***	.02***			.35***	.02***		
BIS $\times$ EC			-.11** (.04)	-.10***			-.20 (.04)	-.04
BAS $\times$ EC			.03 (.04)	.02			.01 (.04)	.00
BIS $\times$ BAS			.00 (.04)	.00			.30*** (.04)	.09**
Step 3:	.37***	.02***			.35***	.00		
BIS $\times$ BAS $\times$ EC			-.10*** (.03)	-.12***			.27*** (.03)	-.09**
Step 4:	.37***	.00			.36***	.01		
BIS $\times$ Sex							.01 (.09)	.00
BAS $\times$ Sex							.00 (.09)	.00
EC $\times$ Sex							-.20* (.11)	-.07*
Step 5:	.37***	.00			.37***	.01*		
BIS $\times$ EC $\times$ Sex							.04 (.09)	.02
BAS $\times$ EC $\times$ Sex							.09 (.10)	.03
BIS $\times$ BAS $\times$ Sex							-.18** (.08)	-.09**

Step/variables added	MASQ general distress				MASQ anhedonia			
	R ²	Δ R ²	B (SE) at final significant step	sr at Final significant step	R ²	Δ R ²	B (SE) at final significant step	sr at Final significant step
Step 6:								
BIS × BAS × EC × Sex	.37***	.00			.38***	.01**	-.16** (.07)	-.09**

Note: sr = semi-partial correlation; Sex = 0 (male) or 1 (female); BIDR-IM = Balanced Inventory of Desirable Responding – Impression Management; BIDR-SD = Balanced Inventory of Desirable Responding – Self Deception; EC = EC Composite. B (SE) and sr values excluded for steps where additional entries do not result in a significant change in R^2 (ΔR^2).

* $p < .10$.
** $p < 0.05$.
*** $p < 0.01$.

Table 4
BIS, BAS, and EC regression models predicting MASQ anhedonia for males and females.

Step/variables added	Males			Females		
	R^2	ΔR^2	B (SE) at final significant step	sr at Final significant step	R^2	B (SE) at final significant step
Step 1:		.34***			.35***	
Constant			-.02 (.06)			-.05 (.06)
z-BIDR-IM			.08 (.06)	.07		-.08 (.06)
z-BIDR-SD			-.28*** (.07)	-.22***		-.20*** (.07)
z-EC			-.25*** (.09)	-.16***		-.47*** (.07)
z-BIS			.11 (.07)	.09		.13*** (.06)
z-BAS			-.15*** (.06)	-.14**		-.16*** (.06)
Step 2:	.36***	.02			.38***	
BIS $\times$ EC			-.15*** (.07)	-.13**	.03**	-.12*** (.06)
BAS $\times$ EC			.08 (.07)	.07		.21*** (.07)
BIS $\times$ BAS			.12*** (.05)	.12**		-.02 (.05)
Step 3:	.37***	.01**			.39***	
BIS $\times$ BAS $\times$ EC			-.11*** (.05)	-.12**		

Note: sr = semi-partial correlation; BIDR-IM = Balanced Inventory of Desirable Responding – Impression Management; BIDR-SD = Balanced Inventory of Desirable Responding – Self Deception; EC = EC Composite. B (SE) and sr values excluded for steps where additional entries do not result in a significant change in R^2 (ΔR^2).

* $p < .10$.

** $p < 0.05$.

*** $p < 0.01$.

Table 5

BIS, BAS, and EC regression models predicting NA and PA.

Step/variables added	NA			PA		
	R^2	ΔR^2	B (SE) at final significant step	sr at Final significant step	R^2	B (SE) at final significant step
Step 1:	.21***				.33***	
Constant			-.06 (.07)			.01 (.06)
Sex			.02 (.07)	.01		.02 (.08)
z-BIDR-IM			-.11** (.05)	-.10**		.02 (.04)
z-BIDR-SD			-.03 (.06)	-.02		.28*** (.06)
z-EC			-.31*** (.06)	-.22***		.39*** (.05)
z-BIS			.26*** (.05)	.22***		-.05 (.04)
z-BAS			-.07 (.05)	-.07***		.20*** (.04)
Step 2:	.23***	.02**			.35***	.02**
BIS $\times$ EC			-.11** (.05)	-.10**		.09** (.04)
BAS $\times$ EC			.10** (.05)	.09**		-.01 (.04)
BIS $\times$ BAS			.06 (.04)	-.06		.06* (.03)
Step 3:	.23***	.00			.35***	.00
BIS $\times$ BAS $\times$ EC						

Note: sr = semi-partial correlation; Sex = 0 (male) or 1 (female); BIDR-IM = Balanced Inventory of Desirable Responding – Impression Management; BIDR-SD = Balanced Inventory of Desirable Responding – Self Deception; EC = EC Composite. B (SE) and sr values excluded for steps where additional entries do not result in a significant change in R^2 (ΔR^2).

* $p < .10$.

** $p < 0.05$.

*** $p < 0.01$.