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The current study examined the implications of Gray's Reinforcement Sensitivity Theory for internalizing psychopathology. Previous research indicates that low activation of the Behavioral Activation System (BAS) predicts depression and high activation of the Behavioral Inhibition System (BIS) predicts anxiety and in some studies depression. However, few studies have examined BIS/BAS levels in relation to both depressive and anxious symptomatology. A sample of 285 undergraduates was administered questionnaires that tap BIS and BAS activation and internalizing symptoms. General distress and anxious arousal scores were collapsed into one symptom category because results indicated that the measure of psychopathology did not discriminate the two. Higher BIS activation predicted all types of internalizing psychopathology, lower BAS predicted anhedonic depression, and negative life events predicted anxious arousal and general distress. BIS, BAS and life events interacted to predict both symptom groups, and these results are discussed in terms of the Joint Subsystems Hypothesis.

THE IMPLICATIONS OF REINFORCEMENT SENSITIVITY THEORY FOR DEPRESSION
AND ANXIETY

by

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TABLE OF CONTENTS

	Page
LIST OF TABLES	v
CHAPTER	
I. INTRODUCTION.....	1
II. REVIEW OF THE LITERATURE.....	5
III. STATEMENT OF PURPOSE.....	8
IV. METHOD.....	9
Participants.....	9
Measures.....	9
Procedure.....	12
V. RESULTS	13
VI. DISCUSSION	21
REFERENCES.....	26

LIST OF TABLES

	Page
1. Cronbach's Alpha and Pearson Correlations of MASQ Subscales with BIS/BAS and SPSRQ Subscales	14
2. Structure Matrix of PCA with BIS/BAS and SPSRQ Subscales.....	15
3. Stepwise Regression Predicting Anxious and Distressed Symptoms.....	18
4. Stepwise Regression Predicting Anhedonic Depression Symptoms.....	19

CHAPTER I

INTRODUCTION

In the current *Diagnostic and Statistical Manual of Mental Disorders* (DSM IV-TR; APA, 2000), anxiety and depression are considered distinct disorders, categorized respectively within anxiety disorders and mood disorders. However, there are many indications that anxiety and depression are related. First, they are highly comorbid, with rates around 60% in most studies (Dobson, 1985). Furthermore, anxiety symptoms precede depression in the majority of cases (Belzer & Schneider, 2004; Hettema, Prescott, & Kendler, 2003). Thus, having an anxiety disorder may be a risk factor for the development of depression. Genetic data indicate that nonspecific negative affect may be a common diathesis to both disorders. Family studies have found that relatives of individuals with depressive and anxiety symptoms have higher rates of internalizing disorders than relatives of individuals with depressive symptoms only (Leckman, Merikangas, Pauls, Prusoff, & Weissman, 1983; Merikangas, 1990), which may suggest that these individuals have greater genetic risk, possibly in the form of a personality style such as neuroticism.

Because depression and anxiety are so highly correlated, this adds complexity to the differential diagnoses of these two disorders. Many individuals present with a mixed anxious and depressive symptom picture that does not lend itself to categorization as either type of disorder or that meets criteria for both. Under the current diagnostic criteria, there is substantial symptom overlap that may in part represent a measurement problem causing artificially increased

comorbidity. However, it may also suggest that these disorders are etiologically related, perhaps due to a similar personality diathesis or similar sensitivity to stressful environmental circumstances.

To address this mixed symptom picture, the International Classification of Diseases 10th edition (ICD-10, World Health Organization, 2003) includes the diagnosis of Mixed Anxiety-Depressive Disorder (MAD), which requires the presence of four symptoms of anxiety and depression for at least one month and can only be diagnosed if the individual does not meet criteria for any other anxiety or mood disorder. The DSM-IV-TR currently includes this as a diagnosis warranting further study. Results from the DSM-IV field trial for MAD indicate that the most common symptom pattern for individuals with subclinical internalizing psychopathology was a mixed anxiety- depression presentation, and that these individuals experienced clinically significant distress and impairment in functioning (Zinbarg, Barlow, Liebowitz, Street, Broadhead, Katon, et al., 1994). However, it is likely that depression and anxiety represent a continuum from normal to psychopathology, suggesting that a mixed symptom pattern may be common in both clinical and nonclinical samples. Thus, it is important to gain a clearer understanding of the etiology and relationship between these disorders.

Jeffrey Gray's Reinforcement Sensitivity Theory (RST, Gray & McNaughton, 2000) may provide a useful framework for understanding depressive and anxious symptomatology. RST is a personality model based upon an approach motivational system related to impulsivity called the Behavioral Approach System (BAS) and an avoidance motivational system related to anxiety called the Behavioral Inhibition System (BIS). Developed as a modification of Eysenck's personality theory of neuroticism and extraversion, Gray's model represents a 30° rotation such

that high BAS is a combination of high extraversion and high neuroticism and high BIS is related to low extraversion and high neuroticism. Implicit in Gray's theory is the idea that an individual is predisposed to certain levels of BIS and BAS activation by genetic and biological factors and that environmental contingencies and learning modify these predispositions throughout development. According to RST, BIS and BAS system activations underlie normal mood, orienting, and appetitive functioning. However, extreme under- or over- activation leads to increased risk for developing psychopathology, especially when an individual experiences negative life events or stressful circumstances.

The BIS or anxiety system, on the one hand, causes inhibition, increased attention, orienting, arousal, and passive avoidance in the face of cues to punishment, novel stimuli, and non-reward. The BIS involves the septo-hippocampal system and its connections to the frontal cortex, the locus coeruleus, and the raphe nucleus (Gray & McNaughton, 2000), areas identified by neuropsychological research as important in anxiety.

The BAS, on the other hand, is sensitive to cues of reward and relief from punishment and activates reward-seeking behavior, feelings of elation, and desire for reward despite risk or threat to the individual (Pickering & Gray, 1999). Activation of the BAS, however, can also increase anger (Carver, 2004) which motivates aggressive behaviors that produce rewards. The system responds to unconditioned stimuli such as food, social contact, or sex, as well as to conditioned stimuli of reward. When activated by potential reward, the BAS activates the dopamine system in various brain circuits (Reuter et al., 2004) which stimulates an emotion similar to hope. The ventral tegmental area has been implicated in the BAS (Depue & Collins, 1999) as well as the basal ganglia, ventral striatum, and the dopaminergic fibers connecting the

mesencephalon and mesolimbic system to the basal ganglia and thalamic nuclei (Harmon-Jones, Abramson, Sigelman, Bohlig, Hogan, & Harmon-Jones, 2002).

CHAPTER II

REVIEW OF THE LITERATURE

Hypothesized behavioral effects of low BAS activation include decreased responsiveness to reward and decreased reward-seeking behavior. Low BAS has been found to predict depression (Depue, Krauss, & Spont, 1987; Meyer, Johnson, & Carver, 1999) and to be related to EEG abnormalities often found in depressed participants (Harmon-Jones & Allen, 1997). Furthermore, low BAS activation is associated with left frontal hypoactivation, which has been linked to depression (Harmon-Jones, Abramson, Sigelman, Bohlig, Hogan, & Harmon-Jones, 2002). Low BAS was also significantly associated with persistence of depression over an 8-month interval (Campbell-Sills, Liverant, & Brown, 2004), whereas BIS was not. Higher BAS scores on the Reward Responsiveness and Drive subscales of Carver & White's (1994) BIS/BAS scales in depressed patients predict recovery from the disorder, whereas there was no relation between BIS scores and recovery. Beevers and Meyer (2002) found that the relationship between low BAS and depression is mediated by lack of positive expectancies; that is, that individuals with low activation of the BAS tend not to expect positive events to happen to them and thus are not motivated to seek out pleasant experiences. In fact, depressed and dysphoric persons perform reduced amounts of behaviors that elicit reinforcement, such as recreational activities (Heiby, Dubanoski, Kameoka, & Saito, 2002; Nelson & Craighead, 1981). Individuals with lower BAS activation experienced fewer positive events than those with high BAS activation, presumably due to differences in tendencies to seek out such events (Gable, Reis & Elliot, 2000). However,

some studies have found no relationship between BAS and depression (Johnson, Turner, & Iwata, 2003).

On the other hand, it is well established that high BIS is related to anxiety in adults (Johnson, et al., 2003; Turner, Beidel, & Wolff, 1996), and behavioral inhibition is a risk factor for anxiety disorders in children (Biederman, Rosenbaum, Hirshfeld, & Faraone, 1990). Some studies have also found that high BIS leads to risk for depression (Kasch, Rottenberg, Arnow, & Gotlib, 2002; Meyer, Johnson, & Carver, 1999). However, 57-58% of depressed patients have a comorbid anxiety disorder (Mineka, Watson, & Clark, 1998), and others may have elevated levels of subclinical anxiety, which may account for findings of high BIS in depressed patients. Higher BIS sensitivity has been found to magnify reactions to negative events (Gable, Reis, & Elliot, 2000), which indicates that it may play a role in both depression and anxiety.

To date, only one study (Johnson, et al., 2003) has attempted to account for the comorbidity of anxiety and depression using Gray's theory. This epidemiological study of adolescents classified participants as meeting criteria for major depression, anxiety disorder or both based upon DSM-IV diagnostic criteria. They predicted that low BAS would be associated with depression, high BIS would be associated with anxiety disorders, and low BAS and high BIS would be associated with comorbid anxiety and depression. High BIS did predict both depression and anxiety alone, but BAS was not associated with either. There was no significant association of BIS or BAS with the group of adolescents with comorbid anxiety and depression, although only 31 participants composed this group and power may have been too low to detect differences. In addition, this study did not measure subclinical levels of psychopathology, which leaves open the possibility that the association of high behavioral inhibition with the depression-

only group was due to subclinical anxiety symptoms in this population. Thus, the relation of BIS and BAS to anxious and depressive symptoms remains unclear.

Whereas BIS and BAS are generally assumed to act as personality diatheses, negative life events may be important in triggering the underlying vulnerability to psychopathology. Negative life events predict depression, anxiety, and overall psychopathology in both cross-sectional and longitudinal studies (Paykel, 2003; Tennant, 2002; Williamson, Birmaher, Dahl, and Ryan, 2005). Most studies find a moderate relationship, $r = .3$ (Miller & Rahe, 1997), although few have examined the interaction of personality and life stress in predicting internalizing symptoms.

CHAPTER III

STATEMENT OF PURPOSE

The current study attempted to determine whether BIS and BAS levels predict symptoms of depression and anxiety. It was hypothesized that 1) higher BIS activation predicts greater anxious arousal; 2) lower BAS activation predicts greater anhedonic depression; 3) lower activation of the BAS and higher activation of the BIS predicts higher rates of negative affect or general distress; and 4) stressful life events interact with BIS and BAS to predict all symptoms.

CHAPTER IV

METHOD

Participants

Two hundred and eighty five introductory psychology students at the University of North Carolina at Greensboro were recruited to participate in this study. The sample (mean age = 20.3) was predominantly Caucasian (68%) and female (66%), which was consistent with the university demographics. Data from 32 participants were excluded for missing responses or high scores on the Infrequency Scale, indicating random responding. Participants were not excluded on the basis of any demographic variable.

Measures

Mood and Anxiety Symptom Questionnaire – Short Form

The MASQ short form (Watson & Clark, 1991) is a 62 item self-report measure based upon Watson and Clark's tripartite model of anxiety and depression, which postulates that these disorders share a general distress feature but each have distinct components. The Anxious Arousal scale attempts to measure components unique to anxiety, such as somatic tension and physiological hyperarousal. The Anhedonic Depression subscale measures loss of interest or pleasure in activities, low energy, and withdrawal. Two General Distress scales, a General Distress Depression scale and a General Distress Anxiety scale, measure negative emotions

common to both anxiety and depression. As these scales were highly correlated, $r = .8$, $p < .05$, they were collapsed to form one General Distress scale (see Results). In the current sample, the

overall General Distress scale had an internal consistency of .94, indicating that these items tap a homogenous construct. For each scale, participants rated on a five point Likert scale the extent to which they had experienced a particular symptom in the past week. The MASQ has good internal consistency and validity (Keogh & Reidy, 2000; Reidy & Keogh, 1997).

Behavioral Inhibition System/Behavioral Activation System Scales

The BIS/BAS scales (Carver & White, 1994) include 20 items that measure a person's emotional responding in situations that may evoke anxiety or impulsivity. Although there is a single BIS scale, Carver and White (1994) created three related scales to assess impulsivity and the BAS dimension: Drive, Reward Responsiveness, and Fun Seeking. The Drive scale measures goal-motivated behavior, Fun Seeking relates to desire for rewards and willingness to attempt to secure new rewards, and Reward Responsiveness focuses on emotional responsiveness to reward. Confirmatory factor analyses show that these scales load on a general BAS factor, but several studies have found that the Reward Responsiveness scale also problematically correlates positively with BIS ($r = 0.2$ to $r = 0.3$, Leone, Perugini, Bagozzi, Pierro, & Mannetti, 2001; Campbell-Sills, Liverant, & Brown, 2004). The BIS/BAS Scales has moderate internal consistency and good convergent and discriminant validity (Campbell-Sills, Liverant, & Brown, 2004).

Sensitivity to Punishment/Sensitivity to Reinforcement Questionnaire

The SPSRQ (Torrubia, Avila, Molto, & Caseras, 2001) contains a 24 item scale to measure sensitivity to reinforcement (SR) or behavioral activation and a 24 item scale to measure sensitivity to punishment (SP) or behavioral inhibition. The subscales showed good internal

consistency and validity (O'Connor, Colder & Hawk, 2004). SR and SP scores have been shown to predict performance on anxiety and impulsivity related tasks in the laboratory (Avila & Parcet, 2000; Avila & Parcet, 2001) and correlate with other measures of anxiety and impulsivity (Torruba, Avila, Molto, & Caseras, 2001).

Recent Life Change Questionnaire

The RLCQ (Miller & Rahe, 1997) is a 74 item self-report inventory that assesses stressful life events and changes in five domains: work, health, home/family, financial, and personal/social. Respondents indicate if the event occurred in the past two years and if so, when. Events were weighted by severity based upon the 1995 rescaling in which a normative sample was asked to indicate the stressfulness of each event compared to a baseline event, marriage. Overall Life Change scores were standardized. The RLCQ has good reliability and predictive validity (Miller & Rahe, 1997).

Infrequency Scale for Personality Measures

The Infrequency Scale (Chapman & Chapman, 1986) is a 13-item scale designed to assess careless responding. It contains items that are very unlikely to be true and indicate a random response style. Participants who endorsed three or more items on the Infrequency Scale were excluded from the analysis.

Procedure

Upon arrival, participants signed a paper consent form. They completed study measures online in a university computer lab and were monitored by a research assistant. After completion, participants were debriefed and received course credit for participation.

CHAPTER V

RESULTS

All subscales had acceptable to good internal consistency reliabilities ($\alpha = .73$ to $.94$; see Table 1). Anxious Arousal, General Distress and the total negative life events score had a skewed distribution and were log transformed. As expected, measures of BAS were intercorrelated, $r = .33$ to $.50$, $p < .05$, and BIS subscales intercorrelated, $r = .43$, $p < .05$, indicating that the SPSRQ and BIS/BAS scales tap similar constructs. The Anxious Arousal scale correlated moderately with the Anhedonic Depression scale, $r = .33$, $p < .05$, indicating that each scale measures at least some individual variance and thus allows examination of anxious and depressive symptoms independently. The General Distress scale correlated with Anxious Arousal, $r = .75$, $p < .05$, and with Anhedonic Depression, $r = .54$, $p < .05$, suggesting this measure taps negative affect co-occurring with both types of internalizing symptoms.

Pearson correlations (see Table 1) indicated a significant positive relationship between Anxious Arousal and both BIS, $r = .15$, $p < .05$, and SP, $r = .26$, $p < .01$, such that higher sensitivity of the Behavioral Inhibition System is linked with higher anxiety symptoms. Similarly, Anhedonic Depression correlated with both BIS, $r = .21$, $p < .01$, and SP, $r = .43$, $p < .01$. Anhedonic Depression correlated negatively with the BAS Reward Responsiveness subscales from the BIS/BAS Scales, $r = -.31$, $p < .01$, the BAS Drive subscale, $r = -.15$, $p < .05$, and the BAS Fun Seeking subscale, $r = -.17$, $p < .01$. There was no significant relationship of

Anhedonic Depression with the SR scale from the SPSRQ, although the correlation was in the predicted direction. General Distress correlated positively with both BIS and SP and not with any

measures of BAS.

Table 1

Cronbach's Alpha and Pearson Correlations of MASQ Subscales with BIS/BAS and SPSRQ

Subscales

	Anxious Arousal	Anhedonic Depression	General Distress	BIS	BAS RR	BAS Drive	BAS Fun	SP	SR
Anxious Arousal	.89	.33**	.75**	.15*	-.10	.02	-.05	.26**	.091
Anhedonic Depression		.91	.54**	.21**	-.31**	-.15*	-.17**	.43**	-.10
General Distress			.94	.31**	-.09	-.03	.01	.39**	.08
BIS				.76	.19**	.07	.13*	.43**	.09
BAS RR					.73	.39**	.50**	-.05	.33**
BAS Drive						.78	.42**	-.17**	.49**
BAS Fun							.74	-.12	.40**
SP								.85	.02
SR									.79

Note. * $p < .05$; ** $p < .01$. Cronbach's alpha presented on the diagonal.

To separate measurement error from latent constructs, a principal components analysis (PCA) was run with a Promax oblique rotation to allow factors to correlate. A PCA with the six

BIS and BAS subscales produced two factors with eigenvalues over one, together explaining 63% of the variance. The first factor (see Table 2) appeared to be a BAS factor upon which each of the relevant subscales loaded highly; the second factor appeared to be a BIS factor upon which the two relevant subscales loaded equally highly. Cross loadings were minimal and the two factors correlated .02, suggesting that these constructs as measured by the BIS/BAS Scales and the SPSRQ are independent.

Table 2

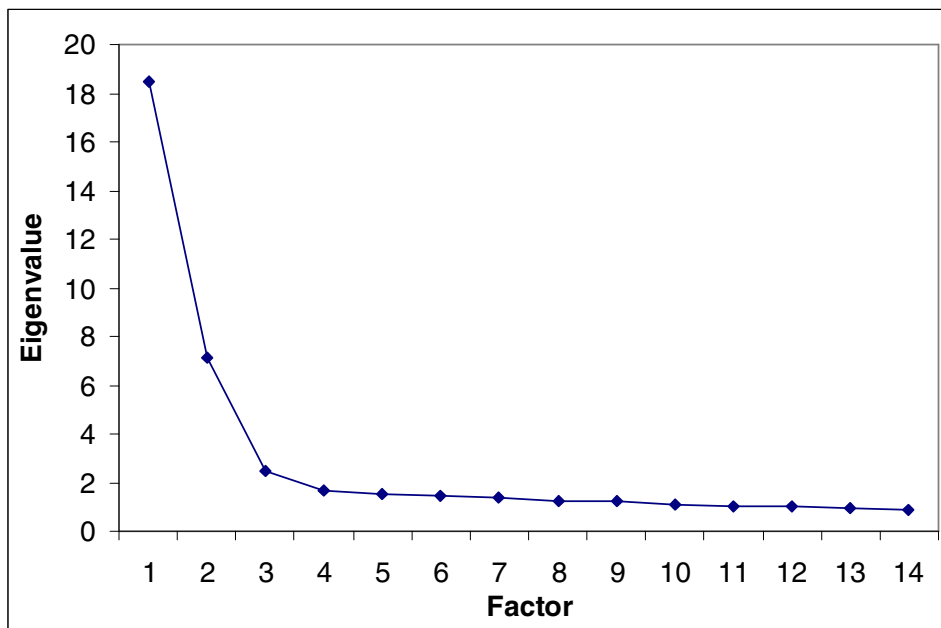
Structure Matrix of PCA with BIS/BAS and SPSRQ Subscales

	BAS Component	BIS Component
SP	-.15	.85
SR	.72	.07
BIS	.20	.83
BAS Reward Responsiveness	.74	.18
BAS Drive	.77	-.11
BAS Fun	.78	.00

Results from a PCA with the MASQ subscales indicated a one factor solution accounting for 75% of the variance. This was at odds with the three factor solution found by Watson and Clark (1995), so a second PCA was run with the 62 items rather than the subscales from this

measure. Eleven factors with eigenvalues over one resulted, but a scree plot (see Figure 1) suggested that two factors accounting for 43% of the variance fit

Figure 1. Scree plot of MASQ items.



the data best. The first factor was composed of items from the Anxious Arousal and General Distress subscales as well as eight items from the Anhedonic Depression subscale. Items included statements such as “Was unable to relax,” “Heart was racing or pounding,” and “Was disappointed in myself.” Thus, it appears that this component is somewhat heterogeneous, tapping both anxious symptoms and negative affect. The second component appeared to specifically tap anhedonic depression. Items from this factor included classic signs of depression such as “Looked forward to things with enjoyment” (reverse scored), “Felt hopeful about the future” (reverse scored), and “Seemed to move quickly and easily” (reverse scored). Some

General Distress items from the first component tended to cross load moderately with this factor which is consistent with Watson and Clark's intention in constructing the measure. Interestingly, pairs of items from the Anhedonic Depression subscale that appeared to tap the same symptom often loaded on different components. For example, "Felt like there wasn't anything interesting or fun to do" loaded on the anxious/distressed component whereas "Felt like I had a lot of interesting things to do" (reversed scored) loaded on the anhedonic depression component. Reverse scored items from the Anhedonic Depression subscale tended to load on the anhedonic depression scale, suggesting that this may tap lack of positive affect. However, as lack of positive affect is hypothesized by Watson and Clark (1995) to be a key factor in depression, this does not seriously compromise the interpretation of this factor as depressive symptoms. However, this interpretation is confounded by the lack of reverse scored items on the Anxious Arousal and General Distress subscales. Finally, many items from the Anhedonic Depression subscale loaded more highly on the anxious/distressed component, suggesting the need for further revision of this scale. Although Keogh and Reidy's (2000) factor analysis of the MASQ resulted in three components, the pattern of factor loadings found is generally consistent with that reported here.

To predict the anxious and distressed symptom cluster, a stepwise regression was run (see Table 3). No demographic variables were significant predictors. A main effect of the BIS factor was significant, $F(277) = 44.88, p < .01$, and predicted 14% of the variance in symptoms such that greater BIS was associated with greater anxious arousal and general distress. The total score for negative life events predicted 4% of the variance over and above BIS, $F(276) = 4.11, p < .01$. Of the five categories of life events assessed by the Recent Life Change Questionnaire, only

Table 3

Stepwise Regression Predicting Anxious and Distressed Symptoms

	Predictors	Beta	R ² Change	p-value
Step 1	Age, Gender, Ethnicity	-.05 -.02 .03	.01	.19 .88 .71
Step 2	BIS factor	.35	.14	.00
Step 3	BAS factor	-.03	.00	.74
Step 4	Life Events	.20	.04	.00
Step 5	BIS*BAS	.14	.01	.04
Step 6	Events*BAS	-.04	.00	.99
Step 7	Events*BIS	.03	.00	.63
Step 8	BIS*BAS*Events	-.13	.02	.03

events related to home and family significantly predicted outcome, $F(276) = 2.276, p < .01$.

Although BAS by itself was not a predictor, the two-way interaction of BIS and BAS was significant over and above the main effects although it accounted for only 1% of the variance, $F(275) = 4.07, p < .05$. Post-hoc analyses indicated that higher BIS in conjunction with higher BAS predicted these symptoms. Finally, the three way interaction of BIS, BAS, and life events was significant, $F(272) = 5.52, p < .05$ and accounted for 2% of the variance after main effects and two-way interactions were entered into the regression. Further analyses of this interaction indicated that high BIS, low BAS, and a high score on the life events measure predicted anxious and distressed symptoms

Next, a stepwise regression predicting the anhedonic depression factor was run (see Table 4). No demographic variables were significant predictors. Results showed main effects for BIS,

Table 4

Stepwise Regression Predicting Anhedonic Depression Symptoms

	Predictors	Beta	R ² Change	p-value
Step 1	Age, Gender, Ethnicity	.05 -.08 .05	.00	.51 .58 .72
Step 2	BAS factor	-.26	.06	.00
Step 3	BIS factor	.35	.12	.00
Step 4	Life Events	.09	.01	.08
Step 5	BIS*BAS	.02	.00	.47
Step 6	Events*BAS	.04	.00	.63
Step 7	Events*BIS	-.01	.00	.78
Step 8	BIS*BAS*Events	.13	.01	.03

$F(277) = 37.96, p < .01$, accounting for 12% of the variance, and BAS, $F(278) = 20.98, p < .01$, accounting for 6% of the variance over and above BIS. To determine whether BAS would remain significant once variance due to BIS was partialled out, another regression was run with BIS entered at step 2 and BAS at step 3. Results indicated that both predictors remained significant and accounted for similar proportions of variance as in the previous regression. Life events was not a significant predictor, $F(276) = 3.15, p < .10$. Whereas no two-way interactions

were significant, the three way interaction of BIS*BAS*Life events was significant, $F(272) = 4.88, p < .05$, although it accounted for only 1% of the variance over and above the other predictors. Further analyses of this interaction indicated that low BIS, low BAS, and a high score on the life events measure predicted greater anhedonic depression.

CHAPTER VI

DISCUSSION

Hypothesis 1 stated that higher BIS scores will predict greater anxious arousal symptoms. However, because the anxious arousal and general distress resulted as one factor on the principal components analysis, this study cannot examine anxious arousal separately from general distress. However, higher BIS scores did predict greater anxious arousal and greater general distress, which corroborates a wealth of prior research indicating that higher BIS may be a risk factor for the development of anxiety disorders. Surprisingly, the interaction of high BIS and high BAS predicted the anxious arousal/general distress factor over and above BIS alone. High BAS may predispose an individual to a more activated arousal when combined with high BIS.

Consistent with Hypothesis 2, lower BAS predicted greater anhedonic depression symptoms. This suggests that individuals lower on reward-seeking behaviors may have a greater risk for depression, perhaps due to experiencing fewer rewarding events in everyday life. In addition, higher BIS predicted greater anhedonic depression which may mean that an anxious and overvigilant personality style can lead to depression, perhaps by route of the BIS's attentional bias towards threatening information. Although BIS accounted for more variance (12%) than BAS (6%) this may be due to the relative psychometric strength of the BIS measures. Alternatively, it may be because high scorers on BIS endorse a pathologically anxious style of interacting with the world whereas low BAS scorers endorse the absence of a pathologically

impulsive style. Jackson and Francis (2004) suggest that “low BAS is related to obtaining rewards passively as a result of, for example, prayer as opposed to actively going out to get

rewards.” Thus, the low end of the BAS scale may not discriminate those at risk for symptoms. Finally, because the anhedonic depression component that resulted from the principal components analysis contained only 8 items that tapped these symptoms, predictive power may be increased with a longer measure of this construct.

Hypothesis 3 could not be fully tested because general distress or negative affect did not appear to be a separate factor on the item-level principal components analysis of the MASQ. Correlational data did suggest that in this sample BIS but not BAS was associated with general distress, although the General Distress subscale used in these analyses correlated highly (.75) with the Anxious Arousal subscale and thus it was likely that results for General Distress would mirror those for the anxious/distressed factor.

Hypothesis 4 stated that stressful life events interact with BIS and BAS to predict all symptoms. Life events predicted anxious/distressed symptoms but not anhedonic depression symptoms although the shortness of the depression scale may have limited the likelihood of significant results. These results suggest that life events are particularly important in conjunction with specific personality styles. Examination of the data indicated that there were no differences among types of life events reported by participants with anxious/distressed symptoms and anhedonic depression symptoms. Overall, participants most often tended to report life events involving moving off to college, changes in friends and romantic relationships, changes in work responsibilities and alterations in their family relationships.

However, type of life events reported appeared to differentially predict symptom type. An examination of types of life events involved indicated that events involving home and family were particularly important for anxious/distressed individuals whereas events involving work,

home/family, and personal/social were particularly important for anhedonic depression. This suggests that stressful events involving the home and family have the greatest impact on mental health, perhaps due to decreases in social support. Financial events were not reported as commonly as other events, likely because many students are at least partially supported by their parents. Alternatively, financial worries may not be as stressful in this age group because college students do not expect themselves to be financially secure. Similarly, health events did not have much of an impact on symptoms in this sample whereas such stressors might be more important in an older or less healthy sample.

These results are consistent with a diathesis-stress model in which Gray's dimensions put an individual at risk for symptoms when negative life events occur. When exposed to stressful circumstances persons low in approach and high in avoidance are particularly vulnerable to internalizing psychopathology. Gray's model represents an advance from descriptive personality theories in that it provides a direct link from physiology and genetics to personality and symptomatology.

Overall, the relationships of personality and life stress to anxiety and depression are very similar. The effects of BIS, BAS, and negative life events are seen in each symptom class, and such similar personality and life event risk factors may explain why depression and anxiety frequently co-occur. An individual extremely high in avoidance or BIS and experiencing many life events would be at risk for both depressive and anxiety symptoms. However, among the few differences is that high BIS appears to play a role in both depression and anxiety, whereas low BAS appears to play a key role in depression only. High BAS interacts with high BIS to produce anxiety symptoms. As high BIS and life events predispose an individual to both depression and

anxiety, individuals in clinical settings are likely to present with comorbid or mixed anxious and depressive symptoms. This suggests the diagnosis of Mixed Anxiety-Depressive Disorder (MAD) may be helpful in capturing the true nature of an individual's problems and tailoring treatment to their particular deficits. It may be that individuals high in avoidance and low in approach would necessitate different therapeutic treatment than individuals with only a single personality diathesis.

Finally, the pattern of interactions of BIS and BAS may be best explained by the Joint Subsystems Hypothesis (Corr, 2002), which suggests that neither BIS nor BAS can be examined separately because each system inhibits the action of the other. Rather, it is the relative strength of each system compared to the other that determines the organism's decision to approach or avoid an ambiguous stimulus. Thus, low approach would exacerbate the effect of high avoidance, leading to an interaction. Corr (2002) suggests that although the systems may be functionally independent, the behavioral outcome is a joint product of the actions of the two systems. Consistent with the Joint Subsystems Hypothesis, these results indicate that BIS and BAS each influence anxious arousal and anhedonic depression. In the case of anxious arousal, main effects of BIS and BAS are apparent as well as the effect of a three way interaction with negative life events, BIS and BAS. In the case of anhedonic depression, the statistical interaction of BIS and BAS and the three way interaction with negative life events are significant. This indicates that both BAS and BIS in conjunction best predict mood and anxiety symptoms.

There are several limitations of this study. First, it examines a college student sample and the results may not be generalizable to other populations. Second, the measure of internalizing psychopathology did not result in three clear factors as expected. Future work could include

more measures of anxiety, depression, and distress, or revise the MASQ to test hypotheses about general distress separately from anxious arousal. In addition, the measures of BIS and BAS, although the best currently published, have several flaws including lower than ideal internal consistency, awkward wording of several questions, and few reverse coded items. Future studies revising measures of Gray's constructs are necessary. Finally, this study does not speak to the mechanism connecting extreme levels of BIS and BAS activation to psychopathology. One possible explanation is suggested by Jackson and Francis (2004), who propose that cognitions and attitudes mediate the relationship between RST and behavior. In the current study, high BIS and lowBAS may predict pessimistic cognitions which signal to the individual that rewards are not likely to be obtained or that punishments are extremely likely. This, in turn, could lead to avoidance and decreased reward seeking behaviors. Future research must incorporate the wealth of research on cognitive biases in depression and anxiety.

Despite its limitations, this study has several strengths. First, it is among the few studies examining the interaction of RST and environment and finding support for a diathesis stress model. The non-clinical sample allows testing the relationship of personality and life stress to symptoms across the continuum of low risk to high risk. Finally, the large sample size allowed for testing of smaller effects, such as the interactions between RST variables and life stress.

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