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BIS/BAS Levels and Psychiatric Disorder: An Epidemiological Study

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Behavioral inhibition and behavioral activation levels have been theorized to relate to a broad range of psychopathologies. To date, however, studies have focused on a single diagnosis, and the measures used to assess different psychopathologies have varied greatly. This study assessed how levels of behavioral inhibition and behavioral activation relate to lifetime diagnoses of depression, anxiety, drug abuse and dependence, alcohol abuse and dependence, attention deficit hyperactivity disorder, and conduct disorder. A representative community sample of 1,803 individuals between the ages of 19 and 21 in the Miami area was surveyed with the Composite International Diagnostic Interview and the Behavioral Inhibition and Behavioral Activation Scales (BIS/BAS; C. S. Carver & T. White, 1994). Results supported the role of BIS as a vulnerability factor for depression and anxiety and of BAS Fun Seeking for drug abuse and noncomorbid alcohol diagnoses. Other models were not supported. Goals in understanding BIS and BAS are described, including the need for prospective studies with a broader array of behavioral indices.

KEY WORDS: personality; epidemiology; motivation; psychopathology.

Substantial evidence has begun to accrue for long-term individual differences in vulnerability to disorders, in part bolstered by the strong genetic concordance for many disorders. Theorists have emphasized the behavioral function of neurobiological systems as a way of conceptualizing links between biological systems, personality traits, and different forms of psychopathology. Although several systems have been proposed (cf. Eysenck, 1981; Zuckerman, 1999), an array of articles have focused on two broadband motivational systems that are hypothesized to regulate approach and withdrawal behavior in response to environmental cues (see Carver & Scheier, 1998; Depue & Collins, 1999; Gray, 1982): the behavioral inhibition system (BIS) and the behavioral activation system (BAS).[5]

BAS is hypothesized to facilitate goal-motivated behavior in the face of cues of incentive. There is evidence that dopamine-secreting neurons of the ventral tegmental area potentiate motivation in the face of cues of incentive (see Depue & Collins, 1999; Depue & Zald, 1993; Winters, Scott, & Beevers, 2000). Once activated, BAS is expected to generate increased approach behavior, motor activity, and feelings of elation, desire, and hope (Depue & Zald, 1993). In the face of conditioned signals of punishment and novelty (Gray, 1971, 1982), BIS is hypothesized to activate responses of inhibition and avoidance, as well as feelings of anxiety and arousal.

Constellations of BIS and BAS have been theorized to explain a broad range of psychopathologies (cf. Fowles, 1993). For example, it has been proposed that high BIS levels are related to anxiety (Gray, 1982), and low BIS levels to attention deficit hyperactivity disorder (Barkley, 1997; Quay, 1988a, 1988b) and psychopathy (Fowles, 1980). High BAS engagement has been theorized to help explain conduct disorder and antisocial personality disorder (Quay, 1993), and low BAS engagement has been seen as related to depression (Depue, Krauss, & Spoont, 1987). Variability in BAS activity has been proposed to explain

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[5]The latter system has been labeld as the behavioral approach system, the behavioral faciliatation system, and the behavioral activation system.

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bipolar disorder, with mania seen as the outcome of high BAS activity (Depue et al., 1987). Models emphasizing relative levels of BIS and BAS have also been suggested. For example, psychopathy, attention deficit hyperactivity disorder (ADHD), and undersocialized aggressive conduct disorder have been theorized to involve elevations of behavioral activation compared to behavioral inhibition (Milich, Hartung, Martin, & Haigler, 1994; Newman & Wallace, 1993; Newman, Wallace, Schmitt, & Arnett, 1997; Quay, 1993, 1997). Despite some inconsistencies, BIS and BAS have been hypothesized to relate to a wide range of disorders.

Considerable evidence supports expected links between BIS/BAS and specific psychopathologies, including ADHD (Matthys, van-Goozen, de-Vries, Cohen-Kettenis, & van-Engeland, 1998; Milich et al., 1994; Schacher, Tannock, & Logan, 1993), psychopathy (cf. Newman et al., 1997), anxiety (Turner, Beidel, & Wolff, 1996), depression (Henriques, Glowacki, & Davidson, 1994), and hypomania (Meyer, Johnson, & Carver, 1999). Although some inconsistent findings have emerged (cf. Iaboni, Douglas, & Baker, 1995), a growing body of evidence documents expected associations. In addition to cross-sectional studies during symptomatic periods, BIS/BAS levels have been found to predict the longitudinal course of some disorders: high BAS appears to predict more mania within bipolar disorder (Johnson et al., 2000; Meyer, Johnson, & Winters, 2001) and slower recovery within unipolar depression (Kasch, Rottenberg, Arnow, & Gotlib, in press). There is also tentative evidence that BIS/BAS models may help guide treatment development. For example, behavioral activation interventions have been found to account for much of the depression relief provided by cognitive–behavioral treatment (Gortner, Gollan, Dobson, & Jacobson, 1998; Jacobson et al., 1996). In sum, BIS and BAS appear important for understanding vulnerability to symptoms, and potentially important for guiding treatment development.

Despite the growing evidence, methodological issues have arisen. In particular, different assessment traditions have emerged within research on different types of psychopathology. For example, laboratory tasks that manipulate punishment and reinforcement contingencies have been used to support BIS/BAS models of ADHD (Casey, Castellanos, Giedd, & Marsh, 1997; Schachar et al., 1993; Matthys et al., 1998; Milich et al., 1994), psychopathy (cf. Newman et al., 1997; Newman, Patterson, & Kosson, 1987), and depression (Henriques et al., 1994). A self-report measure of BIS and BAS (Carver & White, 1994) has been used to study manic symptoms (Meyer et al., 1999, 2001).

Psychophysiological indices of BIS/BAS have been developed as well, including heart rate responses to reward and electrodermal phasic responses to threat (Fowles, 1980). Despite some inconsistencies (cf. Clements & Turpin, 1995), electrodermal responses to anticipated punishment have been used broadly (Arnett, 1997) and have been found to differentiate individuals with psychopathic traits from controls (Fowles & Furuseth, 1994). Other psychophysiological research has been spurred by findings that self-report BAS scores (adjusting for BIS levels) correlate with left-sided midfrontal resting cortical activity (Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997). EEG asymmetry has differentiated currently and formerly depressed individuals from normal controls (Allen, Iacono, Depue, & Arbisi, 1993; Gotlib, Ranganath, & Rosenfeld, 1998; Henriques & Davidson, 1990).

Because of these separate measurement traditions, BIS/BAS studies on anxiety, externalizing disorders, and mood disorders are difficult to compare. To date, little evidence is available to examine how BIS/BAS levels—measured using one approach—relate to a broad range of psychopathologies. Some authors have remained skeptical about how two dimensions could explain so many types of symptom profiles.

Beyond measurement, several design and sampling issues have been common in studies of BIS and BAS. For example, most studies have used small samples. Moreover, most research studies have examined clinical samples drawn from treatment or institutional settings. Because difficulties with personality may increase the likelihood of treatment-seeking, clinical samples may overrepresent the presence of maladaptive traits (Cohen & Cohen, 1984); epidemiological research provides a more conservative methodology to test the BIS/BAS model. To date, no epidemiological studies of BIS/BAS and multiple psychopathologies have been conducted. In addition, many studies have failed to attend to comorbidity.

This study provides the first epidemiological study of these systems and their relation to a broad range of psychiatric disorders. BIS/BAS levels were measured using a psychometrically sound self-report scale (Carver & White, 1994). Analyses focused on the associations of BIS and BAS levels with a broad range of diagnoses, while taking comorbidity into account. BIS and BAS levels were hypothesized to show a broad range of associations with lifetime diagnoses, including (1) lower BAS with depression, (2) higher BIS with anxiety, (3) lower BIS with externalizing disorders, and (4) higher BAS with alcohol and drug abuse.

METHODS

Participants

This study was built on a previous three-wave investigation based in the Miami-Dade public school system (Vega & Gil, 1998). All 48 of the county's public middle schools and all 25 public high schools, as well as alternative schools, participated in the prior study. Wave 1 data were obtained from students in sixth and seventh grade and wave 3 when these same students were in eighth and ninth grade. Informed consent forms were sent to parents of the total population of 9,763 male students scheduled to enter sixth and seventh grades, and of 669 female students from six schools selected to approximate the ethnic composition of all county middle schools. Of these 10,432 prospective participants, 7,386 completed questionnaires at wave 1, 6,646 at wave 2, and 5,924 at wave 3. Detailed analyses provided assurance that wave one participants were highly representative of the population from which they were drawn and that this was also true for the wave 3 participants (Vega & Gil, 1998).

All measures were gathered in a follow-up assessment after wave 3, which included 952 young men and 851 young women in the transition to adulthood (ages 19–21). The target sample for this study included all female participants who had participated in waves 1 and 3 of the earlier investigation and a random subsample of the male participants. The original female sample was supplemented by 909 young women randomly drawn from the class rosters of the original cohort of sixth- and seventh grade students. Comparisons of the characteristics of this supplementary sample with those of the original female sample and with those of their male counterparts revealed the single difference of lower parental socioeconomic status (SES). To correct for this bias, the sample of women was weighted to conform to the SES distribution observed among male participants.

As Table I indicates, the follow-up success rate was 76.4%—a high completion rate for a study in which 93% of respondents were between 19 and 21 years of age at the time of interview. However, substantial losses (41.8%) were experienced among the supplementary sample of women who had no previous involvement in the study, primarily because of the inability to locate them nearly 10 years after their sixth- or seventh-grade attendance. Combining these losses across samples yielded an overall success rate of 70.1%.

Comparisons of the characteristics of those sampled from the original study with those successfully interviewed as young adults are presented in Table II. These data, of course, are not available for the supplementary sample of new women. As shown in columns 1 and 2, those interviewed are similar to the original random sample on a wide array of early adolescent behaviors and family characteristics that may be relevant to mental health or substance abuse risk. Comparisons of those interviewed with those lost to interview (columns 2 and 3) also suggests rather close correspondence. In only 5 of the 29 comparisons is a statistically significant difference observed. Those lost to interview were less likely to live in a two-parent household at both wave 1 and wave 3, they were less likely to have reported using drugs other than marijuana in grade 6–7, they came from families with lower average income, and they were less likely to have fathers who had ever used alcohol. This latter observation may be an artifact of the greater likelihood of father absence among those lost to interview. Despite the relatively low success rate with the supplementary female sample, these results allow the conclusion that the follow-up sample is largely representative of the population from which it was drawn.

Diagnostic Assessment

Data on lifetime and 1-year prevalence of psychiatric and substance disorders were obtained through computer-assisted personal interviews utilizing a *DSM-IV* version of the Michigan revision of the Composite International Diagnostic Interview (CIDI). The

Table I. Percentage of Individuals From the Original Sample Successfully Followed

	Original sample						New sample		Original and new sample	
	Both genders		Men		Women		Women		Both genders	
	n	%	*n*	%	*n*	%	*n*	%	*n*	%
Interviewed	1286	76.4	956	75.1	330	80.5	517	58.2	1803	70.1
Refused	120	7.1	89	7.0	31	7.6	93	10.5	213	8.3
Not found	277	16.4	228	17.9	49	12.0	278	31.3	555	21.6
Total	1683	100.0	1273	100.0	410	100.0	888	100.0	2571	100.0

Table II. Comparisons of Original Sample, Current Sample, and Excluded Sample for Key Wave 1 and Wave 3 Variables

Variables	All ($N = 1{,}683$)	Interviewed ($N = 1{,}286$)	Refused and not found ($n = 397$)
Demographics Wave 1			
Mother education	3.73	3.77	3.60
Father education	3.78	3.80	3.72
Two-parent household	62.2%	64.3%	55.1%[a]
Demographics Wave 3			
Two-parent household	56.4%	59.3%	46.9%[a]
Wave 1			
Lifetime alcohol	38.1%	39.2%	34.5%
Lifetime marijuana	2.3%	2.4%	1.9%
Lifetime other drugs	3.8%	4.4%	2.5%[a]
Alcohol use problems	3.8%	3.9%	3.7%
Family alcohol use problems	15.9%	15.7%	16.2%
Family drug use problems	10.3%	10.1%	10.9%
Peer drug use	1.23	1.23	1.24
Depression symptoms	1.62	1.62	1.60
Wave 3			
Lifetime alcohol	63.0%	63.6%	61.0%
Lifetime marijuana	14.9%	14.1%	17.7%
Lifetime other drugs	8.9%	9.4%	7.7%
Alcohol use problems	10.1%	10.4%	8.9%
Drug use problems	4.7%	4.5%	5.3%
Family alcohol use problems	18.8%	18.0%	21.4%
Family drug use problems	12.5%	12.8%	11.3%
Sibling drug use approval	10.2%	10.1%	10.4%
Peer drug use	1.63	1.62	1.64
Depression symptoms	1.66	1.68	1.60
Parent reported variables			
Parents married	78.1%	78.3%	78.6%
Parents separated or divorced	21.9%	21.7%	21.4%
Household income	2.43	2.66	2.12[a]
Parent smoking	2.56	2.50	2.64
Mother lifetime alcohol use	56.4%	58.2%	51.9%
Father lifetime alcohol use	57.2%	58.5%	49.3%[a]
Father # days drink past month	6.57	6.44	6.93

[a] Interviewed differs significantly from Refused and not found.

CIDI is a structured interview based substantially on the Diagnostic Interview Schedule (DIS). It is designed to be administered by trained nonclinicians (Helzer & Robins, 1988). This version of CIDI was used in the National Comorbidity Study and is currently being used in the 10-year follow-up (Kessler, 1994).

The *DSM-IV* diagnoses assessed were major depression, dysthymia, generalized anxiety disorder (GAD), social phobia, panic disorder, posttraumatic stress disorder (PTSD), attention deficit (AD) disorder, hyperactivity disorder (HD), combined ADHD, childhood conduct disorder, antisocial personality (ASP) disorder, alcohol abuse and dependence, and drug abuse and dependence. In field trials by the World Health Organization, good interrater reliability (Cottlere et al., 1991; Wittchen et al., 1991), test-retest reliability (Wacker, Battegay, Mullejans, & Schlosseer, 1990), and validity (Farmer, Katz, McGuffin, & Bebbington, 1987; Janca, Robins, & Cottler, 1992; Wittchen, Burke, Semler, & Pfiester, 1989) have been documented for each of these modules of CIDI.

Several changes from the original CIDI were made. Consistent with the Michigan revision employed in the National Comorbidity Study (Kessler, 1994), ASP and PTSD modules from DIS were incorporated. Assessments of AD and HD from the revised DIS, and of childhood conduct disorder were added as well. The National Comorbidity Study strategy of preliminary screening was adopted and extended. Initial screening covered a central criterion for each of several diagnoses (major depression, dysthymia, GAD, panic disorder, AD, and hyperactivity disorder), and

on the lifetime use of individual licit and illicit drugs. The goal of this screening process was to reduce fall-off in reporting that might occur if the participant learned during the interview that positive, but not negative, responses tend to be followed by additional questions. Although computer administration, which automated complex skip patterns, greatly reduced the complexity of the interviewing task, each interviewer completed 9 days of training, most of which was devoted to the diagnostic portion of the questionnaire.

Results are presented in terms of the following summary categories of diagnoses: depressive disorders (major depression or dysthymia), anxiety disorders (GAD, panic disorder, social anxiety, and PTSD), drug abuse or dependence, alcohol abuse or dependence, and ADHD and conduct disorder (ADHD, and conduct disorder with or without ASP disorder). As this paper focuses on personality traits that are expected to relate to lifetime vulnerability, lifetime diagnoses are used in analyses. As expected, high rates of comorbidity of diagnoses were documented (see Table III). Comorbidity was more common than was the presence of only one diagnosis, and comorbidity was common across the entire range of diagnostic categories. Of the 1,060 individuals with at least one diagnosis, only 494 individuals met criteria for one and only one diagnostic category.

Table III. Frequencies and Percentages of Sample by Diagnostic Categories

	n	%
No diagnosis	708	40.0
Single diagnosis		
Depression	98	5.5
Anxiety	67	3.8
ADHD/conduct	125	7.1
Alcohol	110	6.2
Drug	94	5.3
Two diagnoses		
Depression & anxiety	31	1.8
Depression & ADHD/conduct	22	1.2
Depression & alcohol	15	0.8
Depression & drug	12	0.7
Anxiety & ADHD/conduct	27	1.5
Anxiety & alcohol	8	0.5
Anxiety & drug	10	0.6
ADHD/conduct & alcohol	35	2.0
ADHD/conduct & drug	53	3.0
Alcohol & drug	86	4.9
More than two diagnoses		
Three diagnoses	179	10.1
Four diagnoses	74	4.2
Five diagnoses	15	0.8
Total sample	1,768	100

Behavioral Inhibition/Behavioral Activation System Scales (BIS/BAS; Carver & White, 1994)

This is a 24-item self-report measure. The BAS scale assesses the tendency to experience strong positive affect or behavioral approach when cues of incentive are present. The BIS scale assesses the tendency to experience negative affect or behavioral inhibition when cues of threat are present. The BIS/BAS scales have good internal consistency. These scales have been more predictive of affective and behavioral responses after incentives and threat than related personality scales (Carver & White, 1994). BAS scores correlate with learning in reinforcement tasks (Zinbarg & Mohlman, 1998). BAS scores also predict more positive affect in response to positive stimuli (Germans & Kring, 2000), in anticipation of rewards in laboratory settings (Carver & White, 1994), and following daily life events involving success (Gable, Reis, & Elliot, 2000). Similar links for BIS and cues of punishment have been found (Carver & White, 1994; Gable et al., 2000). Normative data for a large community sample are also available (Jorm et al., 1999). As described above, these scales have predicted manic symptoms (Meyer et al.,1999, 2001), and relative left anterior brain activity (Sutton & Davidson, 1997).

BAS total scale includes three subscales: Fun Seeking, Drive, and Reward Responsiveness. Possible scores range from 4 to 16 for Drive and Fun Seeking, 9 to 20 for Reward Responsiveness, and 7 to 28 for BIS. The BAS Reward Responsiveness scale measures the tendency to respond with heightened energy and positive affect in the context of desired events or cues of potential future reward. This scale correlates moderately with extraversion, trait measures of positive affect, and positive affect after cues for exposure to pleasant stimuli (Carver & White, 1994; Germans & Kring, 2000; Heubeck, Wilkinson, & Cologon, 1998), and is negatively correlated with physical anhedonia (Germans & Kring, 2000). The Fun Seeking scale emphasizes the impulsive pursuit of pleasure. Unlike the Reward Responsiveness and Drive scales, the Fun Seeking scale correlates highly with novelty seeking (Cloninger, 1987) and disinhibition-constraint (Watson & Clark, 1993). The Drive scale emphasizes motivation to pursue goals, regardless of whether these goals are inherently pleasurable. As differing aspects of BAS have been emphasized across models of psychopathology, analyses examine each specific BAS subscale.

Analysis Plan

The primary objective was to evaluate the significance of BIS and BAS levels as risk factors for the

occurrence of one or more psychiatric disorders. The analytic strategy was to examine mean differences in BIS/BAS scores by the presence versus absence of each lifetime disorder while taking comorbidity into account. This was done by conducting a multivariate analysis of variance (MANOVA). The dependent variables were BIS, BAS Reward, BAS Drive, and BAS Fun Seeking. The five independent variables captured the presence or absence of lifetime diagnoses of (a) depression, (b) anxiety, (c) alcohol abuse or dependence, (d) drug abuse or dependence, and (e) ADHD or conduct disorder with or without ASP disorder. Interaction terms, then, were employed to estimate the significance of BIS and BAS for specific forms of comorbidity. For example, an interaction between depression and anxiety would suggest that BIS/BAS scales have unique implications for understanding the comorbidity of depression and anxiety compared to depression or anxiety alone. Wilk's Lambda was used to estimate the multivariate F. To examine significant multivariate effects, univariate ANOVAs were computed for each dependent variable: BIS, BAS Reward Responsiveness, BAS Drive, and BAS Fun Seeking. To provide another index of the magnitude of significant effects, odds ratios were calculated for each significant effect. The MANOVA allowed for an integrated examination of links of a broad array of diagnoses with BIS and BAS levels, but some more specific models have been described within the field. To address these, three MANOVAs were conducted to examine the role of (1) current depression, (2) subtypes of externalizing disorders, and (3) subtypes of ADHD. All analyses were completed using SPSS for Windows, Release 9.0. An alpha level of .05 was used for all tests.

RESULTS

Analyses were conducted to verify statistical assumptions. Histograms and descriptive printouts were examined for each dependent variable to determine the normality of distributions. As shown in Table IV, means and standard deviations for this sample were highly comparable to the original BIS/BAS validation sample (Carver & White, 1994). As expected, BAS subscales were moderately correlated with each other (see Table IV). As with the original validation sample, BIS was moderately correlated with BAS Reward Responsiveness, but relatively independent of the two other BAS subscales.

Results of a principal components factor analysis of the BIS/BAS items were highly congruent with the factor analysis results within the validation sample; 18 of the 20 items loaded on the same scales (eigenvalues >1.0) as the original results. The two reverse-scored items from BIS scale loaded on a separate factor within current factor analysis (*I rarely experience fear or nervousness* and *I have few fears compared to my friends*). In addition, the factor congruence of the current solution was highly comparable with that obtained in a large scale Australian study; for each of the original four scales, factor congruence scores were above .80. Given the similarity of structure, BIS/BAS scales were scored using previously published algorithms (Carver & White, 1994). The distributions of BIS/BAS scores across gender were also examined. Comparable to the original validation sample, women reported higher BIS and BAS Reward Responsiveness scores than did men, but did not differ from men on BAS Drive or Fun Seeking.

BIS and BAS Mean Scores by Lifetime Diagnosis

As described above, a MANOVA with BIS, BAS Reward, BAS Drive, and BAS Fun Seeking as dependent variables was conducted. The five independent variables included the lifetime presence or absence of (1) depression, (2) anxiety, (3) alcohol abuse or dependence, (4) drug abuse or dependence, and (5) ADHD or conduct disorders. Interaction effects are presented first, followed by main effects. Following this, odds ratios are provided as another index of the magnitude of significant effects.

Table IV. Correlations Among Subscales, Means, and Standard Deviations for the BIS and BAS Subscales

	Correlations			Entire ($N = 1{,}781$)		Men ($n = 939$)		Women ($n = 842$)	
	BIS	Reward	Drive	*M*	*SD*	*M*	*SD*	*M*	*SD*
BIS				19.67	3.37	18.90	3.20	20.52	3.34
Reward	.28***			18.11	1.84	17.98	1.84	18.27	1.82
Drive	−.00	.44***		13.18	2.16	13.26	2.09	13.09	2.24
Fun Seeking	−.02	.37***	.51***	12.55	2.30	12.62	2.27	12.46	2.33

*** $p < .001$.

Table V. Multivariate Analysis of Variance for BIS and BAS Subscales by the Lifetime Diagnoses of Depression, Anxiety, Alcohol Abuse/Dependence, Drug Abuse/Dependence, and ADHD/Conduct Disorder

	Multivariate[a]		Univariate effects[b]			
Effect	F	η^2	BIS	Reward	Fun Seeking	Drive
Depress × Anxiety × ADHD/Conduct × Alcohol × Drug	0.29	.001				
Anxiety × ADHD/Conduct × Alcohol × Drug	1.73	.004				
Depress × ADHD/Conduct × Alcohol × Drug	1.30	.003				
Depress × Anxiety × Alcohol × Drug	1.09	.002				
Depress × Anxiety × ADHD/Conduct × Drug	2.19	.005				
Depress × Anxiety × ADHD/Conduct × Alcohol	1.53	.004				
ADHD/Conduct × Alcohol × Drug	0.78	.002				
Anxiety × Alcohol × Drug	0.43	.001				
Anxiety × ADHD/Conduct × Drug	1.86	.004				
Anxiety × ADHD/Conduct × Alcohol	1.72	.004				
Depress × Alcohol × Drug	1.06	.002				
Depress × ADHD/Conduct × Drug	1.43	.003				
Depress × ADHD/Conduct × Alcohol	0.65	.001				
Depress × Anxiety × Drug	0.57	.001				
Depress × Anxiety × Alcohol	0.50	.001				
Depress × Anxiety × ADHD/Conduct	2.23	.005				
Alcohol × Drug	1.37	.003				
ADHD/Conduct × Drug	2.21	.005				
ADHD/Conduct × Alcohol	0.57	.001				
Anxiety × Drug	0.79	.002				
Anxiety × Alcohol	3.21**	.007	3.41	5.83*	3.84*	1.73
Anxiety × ADHD/Conduct	0.53	.001				
Depress × Drug	0.83	.002				
Depress × Alcohol	1.95	.004				
Depress × ADHD/Conduct	0.56	.001				
Depress × Anxiety	0.95	.002				
Drug	2.34*	.005	0.11	0.16	8.00***	0.37
Alcohol	0.44	.001				
ADHD/Conduct	2.01	.005				
Anxiety	4.72***	.009	10.30***	2.38	0.94	0.13
Depress	4.05**	.011	15.48***	0.03	0.00	0.06

Note. Depress = lifetime depression diagnosis; Anxiety = lifetime anxiety diagnosis; ADHD/Conduct = lifetime diagnosis of attention deficit or hyperactivity disorder or conduct disorder; Drug = lifetime diagnosis of substance abuse or substance dependence; Alcohol = lifetime diagnosis of alcohol abuse or alcohol dependence.

[a] $df = 4, 1731$.

[b] $df = 1, 1734$.

* $p < .05$. ** $p < .01$. *** $p < .001$.

Interaction Effects

Examination of the interaction terms suggested that most comorbid combinations did not have particular associations with BIS and BAS levels (see Table V). Only one significant interaction term emerged: Anxiety × Alcohol. Univariate ANOVAs were conducted to examine which of the BIS/BAS scales differed for Anxiety and Alcohol diagnoses. Those for Reward Responsiveness and BAS Fun Seeking were significant. The Anxiety × Alcohol interaction effects for BAS Drive and BIS were not significant.

Mean levels of Reward Responsiveness and Fun Seeking are presented in Table VI. *t* tests were used to examine whether an alcohol diagnosis was associated with differing BAS levels for individuals with and without an anxiety diagnosis. Then, *t* tests were used to examine whether an anxiety diagnosis was associated with differing BAS levels for individuals with and without an alcohol diagnosis. As four contrasts were conducted for each of the two significant dependent variables, alpha was set to .0125 (.05/4) for these analyses.

Among individuals with no anxiety diagnosis, a lifetime diagnosis of alcohol problems compared to no alcohol problems was associated with higher levels of Fun Seeking, but not Reward Responsiveness, $t_{\text{Reward Responsiveness}}\ (1{,}492) = -0.92$, *ns*; $t_{\text{Fun Seeking}}\ (726.47) = -5.93$, $p \leq .0005$. Among individuals with

Table VI. Means (and *SD*s) for Reward Responsiveness and Fun Seeking by Alcohol and Anxiety Diagnoses ($N = 1{,}766$)

	No alcohol diagnosis		Alcohol diagnosis	
	No anxiety diagnosis	Anxiety diagnosis	No anxiety diagnosis	Anxiety diagnosis
Dependent variable	$n = 1120$	$n = 176$	$n = 376$	$n = 94$
Reward	18.12 (1.84)	18.10 (2.03)	18.22 (1.68)	17.78 (1.93)
Fun Seeking	12.34 (2.35)	12.64 (2.89)	13.10 (2.06)	12.66 (2.35)

an anxiety diagnosis, a lifetime diagnosis of alcohol problems compared to no alcohol problems was not related to BAS Reward or Fun Seeking, $t_{\text{Reward Responsiveness}}$ (268) = 1.28, $p = .20$; $t_{\text{Fun Seeking}}$ (268) = −0.08, $p = .94$.

Among individuals with an alcohol diagnosis, neither BAS Reward Responsiveness, $t_{\text{Reward Responsiveness}}$ (468) = 2.24, $p < .03$, nor Fun Seeking differed by anxiety diagnosis, $t_{\text{Fun Seeking}}$ (468) = 1.78, $p = .08$. Among individuals with no alcohol diagnosis, having an anxiety diagnosis was not associated with different levels of BAS Reward Responsiveness or Fun Seeking , $t_{\text{Reward Responsiveness}}$ (222.64) = 0.12, $p = .90$; $t_{\text{Fun Seeking}}$ (1,294) = −1.56, $p = .12$.

Main Effects

Main effects were significant for depression, anxiety, and drug abuse/dependence, but not for ADHD/conduct disorder or alcohol use. As illustrated by the η^2 estimates (see Table V), the magnitude of effects was quite small. Totaling the η^2 estimates for each separate effect, less than 10% of the variance in BIS/BAS scores was accounted for. For each of the significant main effects, univariate ANOVAs were examined for each specific subscale (see Table V). A lifetime diagnosis of depression was associated with significantly higher BIS scores, $M_{\text{depression}} = 21.07$, $SD = 3.29$, $n = 314$, $M_{\text{no depression}} = 19.38$, $SD = 3.30$, $n = 1{,}455$, as was a lifetime diagnosis of anxiety, $M_{\text{anxiety}} = 20.63$, $SD = 3.34$, $n = 270$, $M_{\text{no anxiety}} = 19.51$, $SD = 3.34$, $n = 1{,}499$. None of the BAS subscales related significantly to a lifetime diagnosis of anxiety or depression.

Lifetime drug abuse/dependence was associated with significantly higher levels of BAS Fun Seeking, $M_{\text{drug use}} = 12.98$, $SD = 2.28$, $n = 485$, $M_{\text{no drug use}} = 12.39$, $SD = 2.29$, $n = 1{,}284$. BIS, BAS Reward, and BAS Drive were not significantly associated with a lifetime diagnosis of drug abuse/dependence.

Odds Ratios

To provide a descriptive index of the magnitude of significant effects in the above MANOVA analysis, odds ratios were computed using logistic regression. To avoid inflating type II error, these analyses are included only for the main effects that were significant within MANOVAs. Participants scoring in the upper 25% and lower 25% of the BAS Fun Seeking and BIS dimensions were compared.[6] Odds ratios were calculated taking comorbidity into account; that is, the odds of a lifetime episode of depression controlled for other diagnoses. The odds of meeting criteria for major depression among high compared to low BIS scorers was 3.28 for major depression (95% CI = 2.27–4.65) and 2.01 (95% CI = 1.37–2.93) for anxiety disorder. Among individuals with high Fun Seeking scores compared to those with low scores, the odds ratio of a drug use disorder was 1.44 (95% CI = 1.06–1.96).

Analyses Within Diagnostic Categories

The assumption that informs most work in this field is that BIS and BAS define variations in vulnerability to psychiatric disorders. However, there is some evidence that self-reported BAS scores decrease with the onset of depressive symptoms (Meyer et al., 2001). In light of this finding, analyses were conducted to examine whether BIS and BAS scores varied with current depression among only those individuals with a lifetime diagnosis of depression ($n = 314$). A Hotelling's t test on BIS, BAS Reward Responsiveness, BAS Fun Seeking, and BAS Drive suggested significant differences between individuals with and without a current diagnosis of depression, defined as meeting criteria over the preceding month, Hotelling's $t = .04$, $F_{4,\,309} = 2.88$, $p < .05$. Contrary to evidence from prior studies, however, none of the BAS scores was associated with current depression (all ps $> .35$). Rather, univariate ANOVAs indicated that individuals with current depression reported significantly higher BIS levels than those without current depression ($F_{1,\,312} = 6.48$, $p < .01$). Analyses that excluded currently depressed participants continued to reveal significantly higher BIS scores among those with a history

[6]Because of discrete score distribution, both high and low score subgroups included slightly more than 25% of the participants (Maximum 36.1% for low Drive).

of depressive disorder compared to nondisturbed controls ($M_{\text{controls}} = 19.34, SD = 3.38, n = 707, t(934) = -5.64$, $p < .0005$). Thus, the BIS–depression association does not appear to be an artifact of state-dependent effects.

MANOVA analyses were also conducted to determine whether specific externalizing diagnoses were more uniquely tied to patterns of BIS and BAS. That is, among individuals with a lifetime diagnosis of ADHD or conduct disorder, a MANOVA was conducted with the independent variables of ADHD (yes or no), conduct disorder (yes or no), and an interaction between ADHD and conduct disorder. Results did not support theory, that is, no interaction emerged between ADHD and conduct disorder, nor was the main effect for conduct disorder significant. A main effect emerged for ADHD. Contrary to hypothesized differences in BIS, univariate ANOVAs were not significant for BIS, nor for BAS Reward or Fun Seeking. Individuals with a lifetime diagnosis of ADHD reported higher levels of BAS Drive than did individuals without a lifetime diagnosis of ADHD ($M_{\text{ADHD}} = 13.75$, $SD = 2.12$, $n = 220$; $M_{\text{no ADHD}} = 13.26$, $SD = 2.04$, $n = 347$).

Analyses were also conducted to examine whether subtypes of ADHD displayed different profiles on BIS/BAS scales. A MANOVA among individuals with a diagnosis of ADHD was conducted, examining main effects for AD and for hyperactivity. Neither main effect was significant, Hotelling's $t_{\text{attention deficit}} = .01$, exact $F(4, 214) = 0.55$, $p > .05$, Hotelling's $t_{\text{hyperactivity}} = .02$, exact $F(4, 214) = 1.14$, $p > .05$.

DISCUSSION

In this epidemiological study, evidence is provided that BIS and BAS are associated with a broad range of psychopathologies. Specifically, current results suggest that BIS and BAS levels may be related to drug abuse, anxiety disorders, and noncomorbid alcohol-related diagnoses. Support was not obtained for models of ADHD and conduct disorder, or for a BAS model of depression. Overall, diagnostic status accounted for only a modest 10% of the variance in BIS/BAS scores. The absence of support for these models may relate to important methodological issues.

This paper provides some of the first information on the significance of BIS and BAS levels for comorbid outcomes. Comorbidity did not appear to be central to understanding associations between BIS/BAS and disorder, with one important exception. For individuals with a lifetime history of alcohol problems, high levels of Fun Seeking were documented only among those without comorbid anxiety. These findings are congruent with increasing attention to subtypes in alcohol research, such as individuals with and without mood regulation difficulties (cf. Kushner, Abrams, Thuras, & Hanson, 2000; Zucker, 1994). For example, prior research has reported that sensation seeking, a construct that is correlated with Fun Seeking, was tied specifically to alcohol abuse in the absence of comorbid anxiety (Conrod, Pihl, Stewart, & Dongier, 2000).

For drug-related diagnoses, only one subscale of BAS appeared important: Fun Seeking, parallel with alcohol-related diagnoses without comorbid anxiety. This finding is congruent with previous findings regarding sensation seeking and impulsivity as risk factors for substance abuse problems (Conrod et al., 2000). These results suggest that specific aspects of behavioral activation are important to consider—only one component of behavioral activation was associated with substance abuse.

The finding that BIS is linked with anxiety diagnoses is highly congruent with theory: BIS models were developed in the context of anxiolytic research (Gray, 1982). This finding is also congruent with previous results from human research measuring laboratory behavior in novel social situations (Gest, 1997; Kagan, 1988), as well as self-report measures of harm avoidance and neuroticism (Starcevic, Uhlenhuth, Fallon, & Pathak, 1996).

BIS/BAS levels in this study did not appear linked with ADHD or conduct disorder, whether examined as a group of diagnoses or as individual diagnoses. In contrast to the current reliance on the BIS/BAS self-report measure, previous studies have used card sorting tasks and psychophysiological measures. The reliance on self-report in the current study may be a problem in the context of disorders characterized by reporting bias and poorer insight. Behavioral measures also have the capacity to elucidate specific elements such as response perseveration and attention problems. As highlighted by a recent review, behavioral inhibition can be conceptualized as including motivational, executive, and automatic inhibitory processes, each of which could correspond to separate cognitive and personality assessments (Nigg, 2000). It is important for the field to begin assessing more specific behavioral and cognitive aspects of these personality systems.

As with ADHD and conduct disorder, the findings regarding depression are not concordant with theory. Previous theory has suggested that depression would be associated with lower BAS levels. In contrast, individuals with a lifetime history of depression manifested higher BIS levels, but not lower BAS levels compared to normal controls. Although two previous studies have found high BIS levels associated with depression symptoms (Meyer et al., 1999, 2001), the current study is the first to control for comorbid anxiety as an explanation for the higher self-reported BIS levels among individuals with a history of depression. These BIS-relevant findings are somewhat

congruent with research indicating that neuroticism robustly predicts depressive symptoms (Jorm et al., 2000), and that depression and neuroticism may be linked genetically (Kendler, Neale, Kessler, Heath, & Eaves, 1993).

A similar absence of support for BAS as a predictor of depression emerged in a recent study of bipolar disorder (Meyer et al., 2001). Many of the studies that have found an association between low BAS and depression history have used a substantially different measure—lower resting left frontal cortical EEG activity (cf. Henriques & Davidson, 1990). In some of these studies, however, EEG asymmetry has been correlated with the *ratio* of BAS to BIS scores (Sutton & Davidson, 1997); as such, these findings are limited in their ability to clarify the relative importance of BAS versus BIS in vulnerability to depression. Nonetheless, given evidence for decreased sensitivity to reinforcement during episodes of depression (Henriques et al., 1994) and evidence that both positive and negative affectivity are important to consider in vulnerability to depression (Clark, Watson, & Mineka, 1994), further research is warranted. As with ADHD and conduct disorder, a study including a broad battery of BIS/BAS measures is needed. Moreover, certain subgroups of depressed individuals, such as those with prominent anhedonia, recurrent episodes of depression, or episodes in the absence of major life events, may be more likely to demonstrate BAS deficits.

In placing these findings regarding ADHD, conduct disorder, and depression in context, sampling issues are important to consider. The absence of previously reported relationships with these diagnoses may arise from the general and representative nature of the present sample. Previous support for the BIS/BAS model in these instances has been obtained largely within clinical or institutional samples, and as mentioned previously, such sample procedures may artificially overestimate maladaptive personality traits.

On a theoretical level, a further goal is to evaluate how maladaptive personality traits may interact with environmental conditions. For example, high BIS levels may predict greater reactivity to life events involving danger, such that more robust associations between personality and disorder may emerge in the context of life events. Moreover, the cumulative effects of major life experiences, as well as changes in underlying biological systems, may influence vulnerability. Both BIS and BAS levels appear to decrease over time (Jorm et al., 1999). The relatively young age of the current sample may limit the accuracy of lifetime risk estimates, as many of the undiagnosed individuals in this sample may develop psychiatric symptoms as they face other challenges in life. Moreover, it remains possible that Fun Seeking is more critical for understanding alcohol and drug problems during young adulthood than during later developmental stages. In sum, the current study fails to address a broad range of questions regarding the influence of BIS and BAS on the course of psychopathology over time and within different contexts.

Current findings suggest that BIS and BAS are associated with anxiety, drug abuse, and nonanxious alcohol abuse. Results of this study did not support predicted links of self-reported BIS and BAS with ADHD, conduct disorder, or depression. These results highlight several issues that merit further consideration. First, BAS does not appear to be a unified construct in relation to psychopathology, and it is important to attend to the components of BAS carefully. Second, it is unclear whether behavioral, physiological, and self-report measures of BIS and BAS have the same relations with psychopathology, even though these measures appear to correlate with each other in normative samples. Finally, studies that are designed to rule out third variable explanations for the current associations are needed.

REFERENCES

Allen, J. J., Iacono, W. G., Depue, R. A., & Arbisi, P. (1993). Regional electroencephalographic asymmetries in bipolar seasonal affective disorder before and after exposure to bright light. *Biological Psychiatry, 33,* 642–646.

Arnett, P. A. (1997). Autonomic responsivity in psychopaths: A critical review and theoretical proposal. *Clinical Psychology Review, 17,* 903–936.

Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin, 121,* 65–94.

Carver, C. S., & Scheier, M. F. (1998). *On the self-regulation of behavior*. New York: Cambridge University Press.

Carver, C. S., & White, T. (1994). Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: The BIS/BAS scales. *Journal of Personality and Social Psychology, 67,* 319–333.

Casey, B. J., Castellanos, F. X., Giedd, J. N., & Marsh, W. L. (1997). Implication of right frontostriatal circuitry in response inhibition and attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 36,* 374–383.

Clark, L. A., Watson, D., & Mineka, S. (1994). Temperament, personality, and the mood and anxiety disorders. *Journal of Abnormal Psychology, 103,* 103–116.

Clements, K., & Turpin, G. (1995). Effects of feedback and task difficulty on electrodermal activity and heart rate: An examination of Fowles' three arousal model. *Journal of Psychophysiology, 9,* 231–242.

Cohen, P., & Cohen, J. (1984). The clinician's illusion. *Archives of General Psychiatry, 41,* 1178–1182.

Conrod, P. J., Pihl, R. O., Stewart, S. H., & Dongier, M. (2000). Validation of a system of classifying female substance abusers on the basis of personality and motivational risk factors for substance abuse. *Psychology of Addictive Behaviors, 14,* 243–256.

Cottlere, L. B., Robins, L. N., Grant, B. F., Blaine, J., Towle, L. H., Wittchen, H., & Sartorius, N. (1991). Participants in the WHO/ADAMHA Field Trials. The CIDI-Core substance abuse and dependence questions: Cross-cultural and nosological issues. *British Journal of Psychiatry, 159,* 653–658.

Depue, R. A., & Collins, P. F. (1999). Neurobiology of the structure of personality: Dopamine, facilitation of incentive motivation, and extraversion. *Behavioral and Brain Sciences, 22,* 491–569.

Depue, R. A., Krauss, S. P., & Spoont, M. R. (1987). A two-dimensional threshold model of seasonal bipolar affective disorder. In D. Magnuson & A. Ohman (Eds.), *Psychopathology: An interactional perspective* (pp. 95–123). San Diego, CA: Academic Press.

Depue, R. A., & Zald, D. H. (1993). Biological and environmental processes in nonpyschotic psychopathology: A neurobiological perspective. In C .G. Costello (Ed.), *Basic issues in psychopathology* (pp. 127–237). New York: Guilford Press.

Eysenck, H. J. (1981). General features of the model. In H. J. Eysenck (Ed.), *A model for personality* (pp. 1–37). New York: Springer.

Farmer, A. E., Katz, R., McGuffin, P., & Bebbington, P. A. (1987). A comparison between the Present State Examination and The Composite International Diagnostic Interview. *Archives of General Psychiatry, 44,* 1064–1068.

Fowles, D. C. (1980). The three arousal model: Implications of Gray's two-factor learning theory for heart rate, electrodermal activity, and psychopathy. *Psychophysiology, 17,* 87–104.

Fowles, D. C. (1993). Biological variables in psychopathology: A psychobiological perspective. In P. B. Sutker & H. E. Adams (Eds.), *Comprehensive handbook of psychopathology* (2nd ed., pp. 57–82). New York: Plenum Press.

Fowles, D. C., & Furuseth, A.M. (1994). Electrodermal hyporeactivity and antisocial behavior. In D. K. Routh (Ed.), *Disruptive behavior disorders in childhood*. New York: Plenum Press.

Gable, S. L., Reis, H. T., & Elliot, A. J. (2000). Behavioral activation and inhibition in everyday life. *Journal of Personality and Social Psychology, 78,* 1135–1149.

Germans, M. K., & Kring, A. (2000). Hedonic deficit in anhedonia: Support for the role of approach motivation. *Personality and Individual Differences, 28,* 659–672.

Gest, S. D. (1997). Behavioral inhibition: Stability and associations with adaptation from childhood to early adulthood. *Journal of Personality and Social Psychology, 72,* 467–475.

Gortner, E. T., Gollan, J. K., Dobson, K. S., & Jacobson, N. S. (1998). Cognitive–behavioral treatment for depression: Relapse prevention. *Journal of Consulting and Clinical Psychology, 66,* 377–384.

Gotlib, I. H., Ranganath, C., & Rosenfeld, J. P. (1998). Frontal EEG alpha asymmetry, depression, and cognitive functioning. *Cognition and Emotion, 12,* 449–478.

Gray, J. A. (1971). *The psychology of fear and stress* . London: Weidenfeld & Nicolson.

Gray, J. A. (1982). *The neuropsychology of anxiety* . New York: Oxford University Press.

Harmon-Jones, E., & Allen, J. J. B. (1997). Behavioral activation sensitivity and resting frontal EEG asymmetry: Covariation of putative indicators related to risk for mood disorders.*Journal of Abnormal Psychology, 106,* 159–163.

Helzer, J. E., & Robins, L. N. (1988). The Diagnostic Interview Schedule: Its development, evolution, and use. *Social Psychiatry and Psychiatric Epidemiology, 23,* 6–16.

Henriques, J. B., & Davidson, R. J. (1990). Asymmetrical brain electrical activity discriminates between previously depressed subjects and healthy controls. *Journal of Abnormal Psychology, 99,* 22–31.

Henriques, J. B., Glowacki, J. M., & Davidson, R. J. (1994). Reward fails to alter response bias in depression. *Journal of Abnormal Psychology, 103,* 460–466.

Heubeck, B. G., Wilkinson, R. B., & Cologon, J. (1998). A second look at Carver and White's (1994) BIS/BAS scales. *Personality and Individual Differences, 25,* 785–800.

Iaboni, F., Douglas, V., & Baker, A. (1995). Effects of reward and response costs on inhibition in ADHD children. *Journal of Abnormal Psychology, 104,* 232–240.

Jacobson, N. S., Dobson, K S., Truax, P. A., Addis, M. E., Koerner, K., Gollan, J. K., et al. (1996). A component analysis of cognitive–behavioral treatment for depression. *Journal of Consulting and Clinical Psychology, 64,* 295–304.

Janca, A., Robins, L. N., & Cottler, L. B. (1992). Clinical observation of CIDI assessments: An analysis of the CIDI fields trials-wave II at the St. Louis site. *British Journal of Psychiatry, 160,* 815–818.

Johnson, S. L., Sandrow, D., Meyer, B., Winters, R., Miller, I., Keitner, G., et al. (2000). Increases in manic symptoms following life events involving goal-attainment. *Journal of Abnormal Psychology, 109,* 721–727.

Jorm, A.F., Christensen, H., Henderson, A. S., Jacomb, P. A., Korten, A. E., & Rodgers, B. (1999). Using the BIS/BAS scales to measure behavioural inhibition and behavioural activation: Factor structure, validity and norms in a large community sample. *Personality and Individual Differences, 26,* 49–58.

Jorm, A. F., Christensen, H., Henderson, A. S., Jacomb, P. A., Korten, A. E., & Rodgers, B. (2000). Predicting anxiety and depression from personality: Is there a synergistic effect of neuroticism and extraversion? *Journal of Abnormal Psychology, 109,* 145–149.

Kagan, J. (1988). The concept of behavioral inhibition to the unfamiliar. In J. S. Reznick (Ed.), *Perspectives on behavioral inhibition* (pp. 1–23). Chicago: University of Chicago Press.

Kasch, K. L., Rottenberg, J., Arnow, B. A., & Gotlib, I. H. (2002). Behavioral activation and inhibition systems and the severity and course of depression. *Journal of Abnormal Psychology, 111,* 589–597.

Kendler, K. S., Neale, M. C., Kessler, R. C., Heath, A. C., & Eaves, L. J. (1993). A longitudinal twin study of personality and major depression in women. *Archives of General Psychiatry, 50,* 853–862.

Kessler, R. C. (1994). The National Comorbidity Survey of the United States. *International Review of Psychiatry, 6,* 365–376.

Kushner, M.G., Abrams, K., Thuras, P., & Hanson, K. L. (2000). Individual differences predictive of drinking to manage anxiety among non-problem drinkers with panic disorder. *Alcoholism: Clinical and Experimental Research, 24,* 448–458.

Matthys, W., van-Goozen, S., de-Vries, H., Cohen-Kettenis, P.T., & van-Engeland, H. (1998). The dominance of behavioral activation over behavioral inhibition in conduct disordered boys with or without attention deficit hyperactivity disorder. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 39,* 643–651.

Meyer, B., Johnson, S. L., & Carver, C. S. (1999). Exploring behavioral activation and inhibition sensitivities among college students at risk for mood disorders. *Journal of Psychopathology and Behavioral Assessment, 21,* 275–292.

Meyer, B., Johnson, S. L., & Winters, R. (2001). Responsiveness to threat and incentive in bipolar disorder: Relations of the BIS/BAS scales with symptoms. *Journal of Psychopathology and Behavioral Assessment, 23,* 133–143.

Milich, R., Hartung, C. M., Martin, C. A., & Haigler, E. D. (1994). Behavioral disinhibition and underlying processes in adolescents with disruptive behavior disorders. In D. K. Routh (Ed.), *Disruptive behavior disorders in childhood* (pp. 109–138). New York: Plenum Press.

Newman, J. P., Patterson, C. M., & Kosson, D. S. (1987). Response perseveration in psychopaths. *Journal of Abnormal Psychology, 96,* 145–148.

Newman, J. P., & Wallace, J. F. (1993). Psychopathy and cognition. In P.C. Kendall and K.S. Dobson (Eds.), *Psychopathology and cognition* (pp. 294–349). New York: Academic Press.

Newman, J. P., Wallace, J. F., Schmitt, W.A., & Arnett, P.A. (1997). Behavioral inhibition system functioning in anxious, impulsive and psychopathic individuals. *Personality and Individual Differences, 23,* 583–592.

Nigg, J. T. (2000). On inhibition/disinhibition in developmental psychopathology: Views from cognitive and personality psychology and a working inhibition taxonomy. *Psychological Bulletin, 126,* 220–246.

Quay, H.C. (1988a). Atttention deficit disorder and the behavioral inhibition system: The relevance of the neuropsychological theory of Jeffrey A. Gray. In L.M. Bloomingdale & J. Sergeant (Eds.), *Attention deficit disorder: Criteria, cognition, and intervention* (pp. 117–126), New York, Pergamon.

Quay, H. C. (1988b). The behavioral reward and inhibition systems in childhood behavior disorder. In L. Bloomingdale (Ed.), *Attention deficit disorder: III. New research in treatment, psychopharmacology, and attention* (pp. 176–186). New York: Pergamon.

Quay, H. C. (1993). The psychobiology of undersocialized aggressive conduct disorder: A theoretical perspective. *Development and Psychopathology*, *5*, 165–180.

Quay, H. C. (1997). Inhibition and attention deficit hyperactivity disorder. *Journal of Abnormal Child Psychology*, *25*, 7–13.

Schacher, R. J., Tannock, R., & Logan, G. (1993). Inhibitory control, impulsiveness, and attention deficity hyperactivity disorder. *Clinical Psychology Review*, *13*, 721–739.

Starcevic, V., Uhlenhuth, E. H., Fallon, S., & Pathak, D. (1996). Personality dimensions in panic disorder and generalized anxiety disorder. *Journal of Affective Disorders*, *37*, 75–79.

Sutton, S. K., & Davidson, R. J. (1997). Prefrontal brain asymmetry: A biological substrate of the behavioral approach and inhibition systems. *Psychological Science*, *8*, 204–210.

Turner, S. M., Beidel, D. C., & Wolff, P. L. (1996). Is behavioral inhibition related to the anxiety disorders? *Clinical Psychology Review*, *16*, 157–172.

Vega, W. A., & Gil, A. G. (1998). *Drug use and ethnicity in early adolescence* . New York: Plenum Press.

Wacker, H. R., Battegay, R., Mullejans, R., & Schlosseer, C. (1990). Using the CIDI-C in the general population. In C. N. Stefanis, A. D. Rabavilas, & C. R. Soldatos (Eds.), *Psychiatry: A world perspective* (pp. 138–143). Amsterdam, The Netherlands: Elsevier Science.

Watson, D., & Clark, L. A. (1993). Behavioral disinhibition versus constraint: A dispositional perspective. In D. M. Wegner & J. W. Pennebaker (Eds.), *Handbook of mental control Century psychology series* (pp. 506–527). Upper Saddle River, NJ: Prentice-Hall.

Winters, R. W., Scott, W. D., & Beevers, C. G. (2000). Affective distress as a central and organizing symptom in depression: Neurobiological mechanisms. In S. L. Johnson, A. M. Hayes, T. Field, N. Schneiderman, & P. McCabe (Eds.), *Stress, coping and depression: Proceedings of the fifteenth annual stress and coping conference* (pp. 177–222). Mahweh, NJ: Erlbaum.

Wittchen, H. U., Burke, J. D., Semler, G., & Pfiester, H. (1989). Recall and dating of psychiatric symptoms: Test–retest reliability of time-related symptoms in a standardized psychiatric interview. *Archives of General Psychiatry*, *46*, 437–443.

Wittchen, H., Robins, L.N., Cottler, L. B., Sartorius, N., Burke, J. D., & Regier, D. A. (1991). Participants in the Multicentre WHO/ADAMHA Field Trials: Cross-cultural feasibility, reliability and sources of variance in the Composite International Diagnostic Interview (CIDI). *British Journal of Psychiatry*, *159*, 645–653.

Zinbarg, R. E., & Mohlman, J. (1998). Individual differences in the acquisition of affectively valenced associations. *Journal of Personality and Social Psychology*, *74*, 1024–1040.

Zucker, R. A. (1994). Pathways to alcohol problems and alcoholism: A developmental account of the evidence for multiple alcoholisms and for contextual contributions of risk. In R. A. Zucker, G. M. Boyd, & J. Howard (Eds.), *The development of alcohol problems: Exploring the biopsychosocial matrix of risk* (NIAAA Research Monograph No. 26, NIH Publication No. 94–3495, pp. 255–282).

Zuckerman, M. (1999). *Vulnerability to psychopathology: A biosocial model* . Washington, D.C.: American Psychological Association.