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Interacting Mechanisms of Impulsivity in Bipolar Disorder and Antisocial Personality Disorder

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Abstract

Background—Bipolar disorder and antisocial personality disorder (ASPD) overlap in clinical characteristics and behavioral consequences. Impulsivity is prominent in both, but there is little information on how specific mechanisms of impulsivity differentiate, bridge, or underlie the disorders.

Methods—Subjects, all males, were controls (n=46), bipolar disorder without cluster B personality disorder (n=21), ASPD without bipolar disorder (n=50), and bipolar disorder with ASPD (n=16). Impulsivity measures were the Immediate Memory Task (IMT), a continuous performance test of response inhibition measuring ability to evaluate a stimulus before responding, and the Two Choice Impulsivity Paradigm (TCIP), a choice between smaller-sooner and larger-later reward. Data were analyzed using general linear models analysis.

Results—Subjects with bipolar disorder had fewer IMT correct detections and slower reaction times than controls. Reaction times were faster with combined diagnoses than in bipolar disorder alone. TCIP responding in either diagnosis alone resembled controls, but was more impulsive in combined disorders. These differences persisted after correction for age and education, which had significant independent effects. In combined ASPD and bipolar disorder, increased reaction speed, impulsive response bias, and reward-delay impulsivity occurred independent of substance-use disorder history.

Conclusions—Impulsivity was increased in the combined disorders over either disorder alone. Results were consistent with at least partially distinct mechanisms of impulsivity in ASPD and bipolar disorder. Compensatory mechanisms for impulsivity in uncomplicated ASPD or bipolar disorder appear to be compromised or lost when the disorders are combined.

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Keywords

bipolar disorder; antisocial personality disorder; personality disorders; substance-related disorders; impulsive behavior; continuous performance test; attention; reward; inhibition (psychology); recurrence

Introduction

Impulsivity, a pattern of action without reflection or regard to consequences, is related to the initiation of action and early responses to stimuli, and has a prominent role in clinical problems associated with psychiatric disorders (Moeller et al., 2001). Impulsivity can result from failures in regulation of attention, motivation, arousal, delay of reward, and/or behavioral monitoring (Barratt & Patton, 1983). In bipolar disorder, impulsivity is increased (Swann et al., 2009a); potentially related to increased suicidal (Swann et al., 2005), aggressive (Elbogen & Johnson, 2009), or criminal (Modestin et al., 1997) behavior. Impulsivity is associated with similar problems in Cluster B personality disorders, including antisocial personality disorder (ASPD) and borderline personality disorder, which can be difficult to distinguish from bipolar disorder in practice.

Mechanisms of impulsivity could be specific to psychiatric conditions or could cut across seemingly disparate disorders. The relationship between bipolar disorder and ASPD may provide evidence about specificity of impulsivity across psychiatric illnesses. In ASPD, impulsivity occurs without the strong relationship to mania that characterizes bipolar disorder, or the affective instability associated with bipolar disorder or borderline personality disorder. Further, while many patients with bipolar disorder also have ASPD (Fan & Hassell, 2008), the diagnosis of ASPD requires specific behaviors beginning early in life (First et al., 1997), facilitating the distinction between individuals with and without ASPD, whether or not bipolar disorder is present.

Bipolar disorder shares some of its most destructive clinical features with ASPD. Bipolar disorder is associated with increased prevalence of conviction for crimes and other behavior also associated with ASPD (Quanbeck et al., 2005). Bipolar disorder has more severe outcome if ASPD is present (Gillberg et al., 1993;Barzman et al., 2007). Arrest (Calabrese et al., 2003) and incarceration (Kemp et al., 2008) are more prevalent in bipolar disorder than in community controls; this may require comorbid substance-use or personality disorder (Elbogen & Johnson, 2009). There is a strong comorbidity of ASPD and bipolar disorder (Fan & Hassell, 2008). Onset of bipolar disorder is earlier in individuals with both disorders (Goldstein & Levitt, 2006). In one report, 55% of newly diagnosed adolescents with bipolar disorder already had histories of antisocial behavior (Barzman et al., 2007).

Cluster B disorders including ASPD may be attenuated forms of a bipolar spectrum (Perugi & Akiskal, 2002). Alternatively, severe bipolar disorder may predispose to personality disorders or antisocial behaviors (Henry et al., 2001;Dunayevich et al., 2000;Swann et al., 2009b), with impulsivity-related complications like suicidal behavior (Garno et al., 2005) and substance-use (Kay et al., 2002).

Models of Impulsivity

Impulsivity can be measured as inability to fully appraise a stimulus before responding (rapid-response impulsivity), or inability to withhold response for a delayed larger reward (reward-delay impulsivity) (Barratt & Patton, 1983;Swann et al., 2002).

Rapid-response impulsivity can be measured by continuous performance tests (Dougherty et al., 2003a;Swann et al., 2002). Impulsive errors (errors of commission), are increased in bipolar disorder in the presence of mania (Swann et al., 2003;Fleck et al., 2005;Sax et al., 1998), a co-occurring substance-use disorder (Swann et al., 2004), or a recurrent course of illness (Swann et al., 2009b), but not in euthymic subjects without these complications (Swann et al., 2009b). Reaction times are slow in euthymic subjects with bipolar disorder (Fleck et al., 2001;Swann et al., 2009b), and response bias is conservative (Swann et al., 2009b). These characteristics may be counterintuitive for bipolar disorder but are consistent with a compensation mechanism that would reduce commission errors at the expense of reductions in response speed and correct detections (Carli & Samanin, 2000). Reaction times are faster with history of many episodes or substance-use disorder (Swann et al., 2009b), or of a medically severe suicide attempt (Swann et al., 2005). Subjects with ASPD have more impulsive response bias than controls; commission error rates and response bias correlate with severity of ASPD, even though self-reported impulsivity does not (Swann et al., 2009c).

Reward-delay impulsivity, inability to delay response for reward, is measured as choice between a smaller-sooner and larger-later reward (Dougherty et al., 2003a;Cherek et al., 1997). A study in which no group had ASPD alone found that reward-delay impulsivity was increased in addictive disorders combined with ASPD compared to addictive disorders alone (Petry, 2002). Reward-delay impulsivity is increased in cocaine dependence only with a history of aggressive behavior (Moeller et al., 2002). Reward-delay impulsivity may be increased in bipolar disorder (Swann et al., 2009b), but roles of comorbidities (Rogers et al., 2010) and affective state (Strakowski et al., 2009) are not established.

Rationale and hypotheses

We measured response inhibition and reward delay in men with ASPD and/or bipolar disorder, compared to healthy controls. Based on the existing literature, our hypotheses were 1) rapid-response impulsivity would be increased in either disorder; 2) reward-delay impulsivity would be increased in either disorder, and 3) both types of impulsivity would be increased in combined disorders over either alone. Because of potential interactions between substance-use disorders and bipolar disorder (Swann et al., 2004) or ASPD (Petry, 2002), we investigated the potential role of substance-use disorder in terms of each hypothesis.

Methods

Subjects

The study was approved by the Committee for the Protection of Human Subjects, IRB for the University of Texas Health Science Center at Houston and was conducted in accordance with the latest version of the Declaration of Helsinki. Potential subjects, responding to advertisements or fliers, were informed of study procedures and risks, and gave written informed consent, before any study-specific procedures. Subjects with bipolar disorder who were not in treatment were given referral information or, if needing immediate treatment, referred to an appropriate facility. Healthy controls had never met criteria for any axis I or Axis II disorder according to SCID-I or SCID-II (First et al., 1996;First et al., 1997). Negative breath alcohol and urine screens for drugs of abuse were required on study days; subjects with positive screens were rescheduled. Because of the strong tendency for ASPD to occur in men, we limited this study to men. Subjects were 46 controls, 50 with ASPD without bipolar disorder, 21 with bipolar disorder without Axis II disorder, and 16 with bipolar disorder also meeting DSM-IV criteria for ASPD, of whom 6 also met criteria for borderline personality disorder.

There is extensive comorbidity between bipolar disorder and cluster B personality disorders (Fan & Hassell, 2008;George et al., 2003). Subjects meeting criteria for any personality disorder other than ASPD but without ASPD were excluded. In combined bipolar disorder and ASPD, presence of borderline personality disorder had no relationship to age, education, affective symptoms, or impulsivity measures. Two subjects with bipolar disorder and ASPD also met criteria for narcissistic personality disorder; one of these also met criteria for borderline personality disorder.

Diagnosis and clinical state

Diagnosis used the Structured Clinical Interview for DSM-IV (SCID) (First et al., 1996) and the SCID-II (First et al., 1997). Symptoms were rated using the Change version of the Schedule for Affective Disorders and Schizophrenia (SADS-C), which is designed to measure depressive, manic, anxiety, and psychotic symptoms concomitantly (Spitzer & Endicott, 1978b). We used the augmented SADS-C (Bowden et al., 1994) with all ten mania rating scale items from the full SADS (Spitzer & Endicott, 1978a;Endicott & Spitzer, 1978), rather than the subset of five items in the conventional SADS-C (Spitzer & Endicott, 1978b). Raters were trained using standard rating materials. Diagnoses were confirmed in consensus meetings including co-authors A.C.S, F.G.M., and/or J.L.S.

Pharmacological treatment of subjects with bipolar disorder—Pharmacological treatment, independent of the study, was required to be stable for at least two weeks before study procedures. Treatments included lithium (three subjects, no monotherapy); anticonvulsant (23, 8 monotherapy); antipsychotic (15, 3 monotherapy); and antidepressant (12, 1 monotherapy). Eight were taking no medicines, 12 one class, 10 two, 4 three, and 2 four or more. Additional ASPD diagnosis had no effect on medicines prescribed (number of classes, X^2 (3 df)=1.9, p=0.6; individual medicine classes, Fisher Exact Test (FET) p>0.2). There were no significant relationships between impulsivity task performance and stable prescription of lithium, antidepressants, anticonvulsants, or antipsychotics ($t < 1$), or between task performance and number of drugs prescribed ($F(3,27) < 1$) (Swann et al., 2009b). The authors were not involved in treatment of the subjects.

Clinical characteristics—Subjects with bipolar disorder had SADS-C depression scores from 0–36 (mean±SD 10.3±7.7), mania 0–29 (9.5±7.1), anxiety 0–20 (6.6±4.5) and psychosis 0–8 (1.7±1.9). Affective state (SCID) was euthymic in 18, hypomanic/manic in 11, depressed in 10, depressed plus hypomanic/manic in 10; state was not related to age ($F(3,45)=0.6$), education ($F(3,45)=0.7$), or comorbid ASPD ($X^2(3df)=3.8$, p=0.29). Controls had depression scores of 0–6, mania 0–2, anxiety 0–3, and psychosis 0–1; for ASPD without bipolar disorder, depression was 0–20, mania 0–9, anxiety 0–13, and psychosis 0–1.

Substance-use disorders—All subjects with substance-use disorders (DSMIV criteria for abuse or dependence) had stimulant and/or alcohol-use disorders and were in partial or full remission. Subjects meeting criteria for an active substance-use disorder were excluded, and subjects testing positive for any illicit drug, stimulant, or alcohol were rescheduled for testing when their screens were negative and, with three positive screens, removed from the study. Thirty-seven subjects with ASPD alone met lifetime criteria for a substance- or alcohol-use disorder (3 alcohol only, 8 stimulant only, 26 both) and 13 did not; 20 with bipolar disorder alone met lifetime criteria for a substance-use disorder (4 alcohol only, 3 stimulant only, 13 both) and 6 did not; 12 with combined ASPD and bipolar disorder met lifetime criteria for a substance-use disorder (2 alcohol only, 2 stimulant only, 8 both) and 4 did not (for alcohol and/or stimulant use disorder across groups, X^2 (2df)=0.5, p=0.8).

Laboratory measures of impulsivity

Rapid-response impulsivity: the Immediate Memory Task (IMT) is a continuous performance test (Dougherty et al., 2000). 5-digit numbers are shown for 0.5 seconds, separated by 0.5-second blank intervals; subjects are instructed to respond if a number matches the previous number. Responses include: correct detections of matching numbers (33% of total stimuli); commission errors, when 4 of the 5 digits match (33%); and random errors, where no digits match (34%). Commission errors are taken as impulsive responses, e.g., inhibitory failures (Dougherty et al., 2003a;Dougherty et al., 2003b;Swann et al., 2002). Nonparametric signal detection methods yield discriminability (A'; ability to distinguish target from non-target stimuli) and response bias (beta; positive numbers represent conservative, negative numbers indicate liberal or impulsive bias) (Donaldson, 1992;Green & Swets, 1966).

Delayed reward: the Two-Choice Impulsivity Paradigm (TCIP) is a choice between a small reward after a 5 -sec delay or a larger reward after a 15-sec delay (Dougherty et al., 2003a). Short-delay responses are taken as impulsive (Cherek et al., 1997;Cherek & Lane, 1999;Dougherty et al., 2003a). This procedure has been widely used in studies of impulsive populations (Cherek et al., 1997;Cherek & Lane, 1999;Dougherty et al., 2003a). Results are displayed immediately on the computer monitor.

Statistics

For normally distributed variables, we used general linear model analysis of variance (GLM ANOVA) or linear regression analyses. TCIP data was not normally distributed (right-skewed); normal distributions were obtained by square-root transformation (Table 3). Post-hoc comparisons, when appropriate ANOVA was significant, used the Tukey Honestly Significant Difference Test (HSD) with correction for unequal n.

Results

Subject Characteristics

Table 1 shows that subjects with ASPD without bipolar disorder (Tukey HSD p=0.0002) had significantly fewer years of education than controls. Subjects with bipolar disorder were older than controls. Age and education were therefore included in all analyses.

Rapid-response impulsivity: IMT

Table 2 summarizes relationships between IMT performance and diagnosis. IMT measures were the dependent variables, ASPD and bipolar disorder were dichotomous predictor variables, and age and education were continuous independent variables. Subjects with bipolar disorder had fewer correct detections, slower reaction times, and more conservative response bias than controls. There were significant interactions between bipolar disorder and ASPD in reaction times, where ASPD alone had no significant effect, but combined ASPD and bipolar disorder was associated with faster reaction times than bipolar disorder alone.

Increased age correlated with slower reaction times; increased education correlated with lower commission error rates and better discriminability, independent of diagnosis (Table 2).

Rapid-response impulsivity and substance-use disorder

We investigated the role of substance-use disorder history and course of illness using 1) GLM limited to subjects with bipolar disorder, to investigate the role of substance-use disorder in effects of comorbid ASPD, and 2) GLM for ASPD and bipolar disorder in

subjects without a substance-use disorder, to see whether interactions between bipolar disorder and ASPD occurred without substance-use disorder.

We have reported faster IMT reaction times in bipolar disorder combined with substance-use disorders than in bipolar disorder alone (Swann et al., 2009b). Analysis limited to bipolar disorder, with IMT measures as dependent variables and substance-use disorder, ASPD, age, and education as independent variables, found a main effect of ASPD ($F(1,76)=4$, $p=0.05$) but no main or interaction effects for substance-use disorder ($F(1,76)<0.6$), on reaction time. Similarly, ASPD ($F(1,76)=4.6$, $p = 0.04$), but not substance-use ($F(1,76)<0.1$), had a significant main effect on response bias in subjects with bipolar disorder.

Among subjects without a substance-use disorder, there was a significant main effect of bipolar disorder on reaction time ($F(1,67)=4.6$, $p=0.04$): subjects with bipolar disorder had slower reaction times than controls (Tukey HSD $p=0.02$) but subjects with bipolar disorder plus ASPD did not differ from controls. Correct detections and response bias were reduced in bipolar disorder without a substance-use disorder, as in Table 2. Bipolar disorder also had a significant main effect on response bias ($F(1,67) = 4.8$, $p = 0.04$), with conservative response bias in bipolar disorder compared to ASPD or controls. Therefore, the apparent reversal by concurrent ASPD of effects of bipolar disorder on reaction time or bias did not require a substance-use disorder. By contrast, neither ASPD nor bipolar disorder had significant effects on commission errors per correct detection in subjects without a substance-use disorder.

Reward delay: Two-Choice Impulsivity Paradigm

Table 3 shows that TCIP responding did not differ between controls and subjects with either ASPD or bipolar disorder. However, there was a significant interaction between ASPD and bipolar disorder, where subjects with combined diagnoses had more short-delay responses and fewer consecutive long-delay responses, indicating greater difficulty in delaying reward.

Reward-delay impulsivity and substance-use disorder

We assessed the role of substance-use disorder in the interaction between bipolar disorder and ASPD on TCIP with analyses analogous to those described above for the IMT, using square root-transformed data. GLM restricted to bipolar disorder revealed a significant main effect of ASPD on consecutive delayed responses (reduced; $F(1,16)=10.4$, $p<0.005$). Main effects or interactions with substance use disorder were not significant ($P>0.3$). Despite the relatively small number of subjects, these results suggest strongly that the interaction between bipolar disorder and ASPD in TCIP did not require history of a substance-use disorder.

In men without substance use disorder, GLM taking age and education into account revealed significant main effects of bipolar disorder for short-delay (increased; $F(1,39)=4.46$, $p=0.04$) and consecutive long-delay responses (reduced; $F(1,51)=6.3$, $p=0.02$). Main effects of ASPD, and interactions between ASPD and bipolar disorder, were not significant ($F(1,51) < 1$). These data suggest that history of a substance use disorder is associated with reduced impulsive responding in ASPD unless bipolar disorder is present, and that, in the absence of substance-use disorder, subjects with bipolar disorder have increased reward-delay impulsivity.

Discussion

Findings relative to hypotheses

In this paper we tested three general hypotheses on relationships between mechanisms of impulsivity in bipolar disorder and ASPD. The first, that subjects with bipolar disorder or ASPD would have higher rapid-response impulsivity than controls, was only partially confirmed. Table 2 shows that bipolar disorder had significant main effects on reaction times (slower) and response bias (conservative) that do not appear consistent with increased impulsivity but may be an adaptation to it.

The second hypothesis, that bipolar disorder or ASPD would increase reward-delay impulsivity compared to controls was partially supported, as TCIP responding did not differ between controls and subjects with bipolar disorder or ASPD alone across the entire group (Table 3), but was more impulsive than controls in subjects with bipolar disorder who did not have a substance-use disorder.

The third hypothesis was confirmed, since the combination of ASPD and bipolar disorder was associated with more impulsive TCIP performance than controls. Commission errors per correct detection appeared to be increased in ASPD (with or without bipolar disorder) but this effect was accounted for by education. Bipolar disorder combined with ASPD was associated with faster reaction times and more impulsive response bias than in bipolar disorder alone. Substance abuse history was not required for these effects, or for effects on TCIP.

Rapid-response impulsivity: IMT

Subjects with bipolar disorder had fewer correct detections, slower reaction times, and more conservative response bias than controls. These findings suggest an adaptation that could reduce commission errors at the cost of fewer correct detections and slower response speed (Swann et al., 2009b). Subjects with ASPD had a more impulsive response bias than those with bipolar disorder. Response bias in subjects with combined bipolar disorder and ASPD was similar to that in controls (Table 2).

Reaction times were slower in bipolar disorder than in controls, unless ASPD was also present (Table 2). We have reported that these subjects were more likely than other subjects with bipolar disorder to have many previous episodes of illness (Swann et al., 2010), and that subjects with many previous episodes had faster reaction times than other subjects with bipolar disorder (Swann et al., 2009b).

Delay to reward: Two-Choice Impulsivity Paradigm

Increased reward-delay impulsivity has been reported in aggressive subjects, most of whom had ASPD, though these results could have been confounded by comorbid disorders (Cherek & Lane, 1999;Dougherty et al., 2003a;Cherek et al., 1997). TCIP response rates in subjects with ASPD alone were similar to controls, and did not correlate with the number of ASPD symptoms (Swann et al., 2009c). This agrees with Table 3, where neither ASPD nor bipolar disorder alone had significant effects on TCIP. In contrast, subjects with combined bipolar disorder and ASPD had significantly more short-delay responses and fewer consecutive long-delay responses than controls, consistent with increased reward-delay impulsivity.

Substance-use history had no effect on TCIP in bipolar disorder or ASPD alone, consistent with our previous reports (Swann et al., 2009c;Swann et al., 2009b). ASPD and substance-use disorders were reported to increase reward-delay impulsivity in a study where no subjects had ASPD only (Petry, 2002). In cluster B personality disorder (ASPD or

borderline) and/or alcohol use disorder, reward-delay impulsivity was increased as a marker for alcohol-use disorder, whereas rapid-response impulsivity was increased in subjects with combined alcohol-use and Cluster B disorder (Rubio et al., 2007). Our results suggest that factors other than substance-use disorder, such as other comorbid conditions, age, or education, may have contributed to differences found in those studies.

Interactions between bipolar disorder and ASPD in impulsivity: Compensatory mechanisms

Impulsivity may represent imbalance between behavioral activation and inhibition (Fineberg et al., 2010). Uncomplicated bipolar disorder has high impulsivity as measured by questionnaires (Swann et al., 2009a) even when behavior and affective state are relatively stable (Swann et al., 2001). Non-manic patients with bipolar disorder appear to have normal baseline NE function (Swann et al., 1987;Greenspan et al., 1970) but have exaggerated behavioral reactions to NE (Price et al., 1984). The data in Table 2 are consistent with a compensatory response in bipolar disorder where potential impulsivity is counteracted by slowing of responses and a more conservative response bias. This apparent compensation appears to be reversed when ASPD is also present (Tables 2 and 3).

Relationship between ASPD and bipolar disorder

Bipolar disorder and ASPD had distinct patterns of IMT responses, and interacted in a non-additive manner on the IMT and TCIP (Tables 2 and 3). Because the prevalence of combined bipolar disorder and ASPD is substantially larger than the product of their individual prevalence rates (Fan & Hassell, 2008), the disorders are likely either to be mechanistically related, or to have confounding similarities that lead to the deceptive appearance that they are commonly combined. There is one report of a familial association of ASPD and bipolar disorder (Faraone et al., 1998). The data summarized here and the results in this paper suggest that 1) the two disorders have at least partially different mechanisms governing impulsivity, 2) susceptibility to impulsive behavior may have the same basic mechanism across the two disorders, with different compensating mechanisms in bipolar disorder and ASPD that are lost when the disorders are combined, and/or 3) a severe form of bipolar disorder is characterized by early antisocial behavior and increased impulsivity.

Limitations

Factors complicating the interpretation of these results include 1) bipolar disorder plus ASPD differed from those with ASPD alone, being more likely to have borderline personality disorder; 2) restriction to subjects without recent changes in treatment may have biased bipolar disorder toward more stable illness; 3) psychotropic treatments did not affect response-inhibition in bipolar disorder (Swann et al., 2009b), but the fact remains that most subjects with bipolar disorder (regardless of ASPD) were receiving a range of pharmacological treatments while subjects with ASPD alone were not; 4) psychiatric symptoms did not account for response inhibition in bipolar disorder (Swann et al., 2009b) or ASPD (Swann et al., 2009c), but subjects with bipolar disorder (with or without ASPD) had a wider range of psychiatric symptoms than ASPD alone; and 5) the study did not address psychopathy, a characteristic of ASPD potentially related to response inhibition and externalizing behavior disturbances (Blair & Mitchell, 2009).

Conclusions

Bipolar disorder and ASPD are associated with increased impulsivity, especially when combined. The results suggest distinct, potentially interacting mechanisms of impulsivity in bipolar disorder and ASPD. Subjects with uncomplicated bipolar disorder had reductions in

response speed and decreases in correct detections consistent with a compensatory effect buffering against impulsive responses. The results suggest that loss of this compensatory mechanism may lead to more severe impulsivity in the combined disorders.

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Table 1

Demographic characteristics

Group	**Bipolar Disorder**	**ASPD**	**N**	**Age Mean ± SD**	**Education**
1	No	No	46	31.2 ± 9.7	14.5 ± 2.5
2	No	Yes	50	34.8 ± 9.7	12.3 ± 1.7
3	Yes	No	21	36.9 ± 12.0	13.6 ± 2.5
4	Yes	Yes	16	38.2 ± 9.1	13.5 ± 2.9
2-way ANOVA, F (1,129) (p in parentheses)			BP	**5.5 (0.02)**	0.1
			ASPD	1.5	**7.0 (0.009)**
			BP X ASPD	0.4	**5.6 (0.02)**
Post-hoc, Tukey Honest Significant Difference corrected for unequal sample size, $p < 0.05$				None	**1 versus 2 (0.0002)**

ASPD=Antisocial personality disorder; BP=bipolar disorder; N = number of subjects.

Table 2

IMT Performance in Bipolar Disorder and ASPD

G	BP	ASPD	N	CD, %	CE, %	CE/CD	CD reaction time, msec	CE reaction time, msec	A'	Bias (β)
1	No	No	46	83.2 ± 10.2	25.4 ± 15.5	0.302 ± 0.160	455 ± 66	447 ± 74	0.869 ± 0.055	−0.178 ± 0.521
2	No	Yes	50	82.9 ± 10.3	32.1 ± 14.0	0.390 ± 0.165	492 ± 73	494 ± 75	0.839 ± 0.068	−0.342 ± 0.431
3	Yes	No	21	76.8 ± 15.0	23.8 ± 14.9	0.322 ± 0.196	559± 87	541 ± 101	0.841 ± 0.089	−0.004 ± 0.499
4	Yes	Yes	16	78.7 ± 11.0	29.6 ± 13.8	0.381 ± 0.168	497 ± 74	492 ± 91	0.826 ± 0.072	−0.198 ± 0.410
GLM F (1,123)		Bipolar		**6.6****	0.6	0.1	**9.7** ***	**7.5****	2.4	**4.3***
		ASPD,		0.1	2.0	1.97	0.2	0.2	1.2	1.9
		Bipolar × ASPD		0.6	0.4	0.1	**15.4*****	**12.6*****	0.1	0.4
		Age		0.1	0.5	0.6	**6.2***	**6.3***	0.4	0.6
		Education		0.1	**8.5*****	**8.7*****	1.5	0.3	**5***	3.6
Post hoc, Tukey HSD (p < 0.05)				None	None	None (1 vs 2, 0.06)	**3 vs 1, 2, or 4**	**3 vs 1, 2, or 4**	None	None (2 vs 3, 0.06)

Significance of F:

* p < 0.05,

** p < 0.01,

*** p < 0.005

G=Group; ASPD=Antisocial personality disorder; BP=bipolar disorder; CD=Correct Detections; CE=Commission errors; A'=discriminability (0.5-1, higher is better); for β (−1 to 1), lower is impulsive bias, higher is conservative bias.

Table 3

Effect of ASPD and/or Bipolar Disorder on Two-Choice Impulsivity Paradigm Performance

Group (n)	ASPD	Bipolar Disorder	Median; 25th-75th percentiles	
			Short-delay responses, %	Number of consecutive long-delay responses
1 (36)	No	No	22; 2–50	12; 5–44
2 (7)	No	Yes	13; 4–38	29; 4–45
3 (45)	Yes	No	18; 8–45	24; 9–39
4 (15)	Yes	Yes	48; 12–58	5; 3–16
F (1,97) Bipolar disorder (p)			1.74 (0.19)	0.75
F (1,97) ASPD (p)			**4.65 (0.035)**	2.32
F (1,97) Bipolar disorder X ASPD (p)			**3.82 (0.05)**	**5.85 (0.017)**
F (1,97) Age (p)			0.09	0.05
F (1,97) Education (p)			0.01	0.01
Post hoc (Tukey HSD corrected for unequal sample size) (p)			**1 v 4 (0.035)**	**2 or 3 v 4 (0.05)**

GLM was conducted using square-root transformed data; original data are shown