Interacting mechanisms of impulsivity in bipolar disorder and antisocial personality disorder

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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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1. 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 and Patton, 1983). In bipolar disorder, impulsivity is increased (Swann et al., 2009a); potentially related to increased suicidal (Swann et al., 2005), aggressive (Elbogen and 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 and 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 and Johnson, 2009). There is a strong comorbidity of ASPD and bipolar disorder (Fan and Hassell, 2008). Onset of bipolar disorder is earlier in individuals with both disorders (Goldstein and 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 and 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).

1.1. 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 and 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 (Carl and 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, can be 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.

1.2. 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.

2. Methods

2.1. 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 immediate 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, 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 and 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.

2.2. 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 and Endicott, 1978b). We used the augmented SADS-C (Bowden et al., 1994) with all ten mania rating scale items from the full SADS (Spitzer and Endicott, 1978a; Endicott and Spitzer, 1978), rather than the subset of five items in the conventional SADS-C (Spitzer and 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.

2.2.1. 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); anticonvululant (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 ($r < 1$), 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.
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