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Trait impulsivity and increased pre-attentional sensitivity to intense stimuli in bipolar disorder and controls

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ABSTRACT

Impulsivity and sensation seeking are stimulus-oriented traits. Because they differ in degree of intention and planning, they may have distinct neurophysiological mechanisms. Impulsivity is prominent in bipolar disorder, and may be related to pre-attentional information filtering and stimulus-orientation. We investigated specificity of relationships between impulsivity and sensitivity to stimulus intensity in bipolar disorder and controls, using intensity–sensitivity of auditory evoked potentials. Seventy-six subjects (37 healthy controls, 39 with bipolar disorder) were administered an intensity–sensitivity paradigm. Additional measures included Barratt Impulsiveness Scale (BIS-11) and Eysenck Impulsivity and Venturesomeness scores. State-dependent rapid-response impulsivity was measured using the Immediate Memory Task. Intensity-sensitivities of the auditory evoked P1N1, N1P2, P1, N1, and P2 potentials were assessed as the slope of amplitude relative to loudness. Analyses used general linear models (GLM) with impulsivity-related measures as dependent variables and age, gender, education, and diagnosis as dependent variables. BIS-11 total, motor, and attentional impulsivity scores correlated positively with pre-attentional N1 and P1N1 intensity–sensitivity slopes in bipolar disorder, but not in controls. BIS-11 nonplanning and Eysenck Venturesomeness scores did not correlate with intensity–sensitivity. Intensity–sensitivity slopes did not correlate with rapid-response impulsivity. Correlations between N1 or P1N1 slopes and BIS-11 scores in bipolar disorder were not affected by age, education, WAIS, treatment, symptoms, or gender. Trait impulsivity in bipolar disorder may be related to poorly modulated stimulus-driven late pre-attentional responses to stimuli, potentially resulting in exaggerated responses to intense stimuli even before conscious awareness. Components of trait impulsivity are physiologically heterogenous relative to intensity–sensitivity.

1. Introduction

The initiation of action is an important aspect of normal and abnormal brain function. Impulsivity and sensation-seeking, related but potentially independent traits linking stimulus and action (Barratt et al., 2004; Patton et al., 1995), cross psychiatric diagnoses (Zuckerman and Kuhlman, 2000; Moeller et al., 2001; Fossati et al., 2007; Barratt et al., 2004). Impulsivity is increased in bipolar disorder, regardless of clinical state and pharmacological treatment, and is related to severe course of illness, including recurrent course, substance-use or personality disorder (Swann et al., 2004, 2009b, 2010), crime (Swann et al., 2011), or suicidal behavior (Swann et al., 2005). Neurophysiological bases of this increased impulsivity are not well understood. An important characteristic of bipolar disorder is that it is generally recurrent and can be progressive, and it is important to distinguish characteristics underlying or predisposing to the illness from those resulting from progression of the illness or state-dependent changes.

Impulsivity can be measured as a trait-like predisposition to impulsive behavior, or as behavioral responses sensitive to physiological state, environmental conditions, or pharmacological manipulations (Swann et al., 2002). The latter includes rapid-response impulsivity, representing inability to withhold a response to a stimulus that has not been fully appraised and measurable by suitably designed continuous performance or stop-signal tasks

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(Swann et al., 2002; Dougherty et al., 2003). Trait and state aspects of impulsivity may differ physiologically and have different roles in bipolar disorder.

In bipolar disorder, trait-like impulsivity is increased even in euthymic subjects, is not affected significantly by most treatments (Swann et al., 2009a), and is increased in unaffected first-degree relatives (Sanches et al., 2014). Therefore, increased trait-like impulsivity may represent a marker of susceptibility, or an endophenotype, for bipolar disorder. Neurophysiological correlates of trait-like impulsivity in bipolar disorder have not been identified.

On the other hand, rapid-response impulsivity is normal in euthymic patients with uncomplicated bipolar disorder but is increased with a recurrent course of illness (Swann et al., 2009b) or during mania (Swann et al., 2003; Strakowski et al., 2010), and is associated with complications of bipolar disorder including addiction (Swann et al., 2004), crime (Swann et al., 2011), and severe suicidal behavior (Swann et al., 2005), suggesting a relationship with illness progression.

Trait impulsivity may reflect poorly modulated stimulus-orienting (Zuckerman, 1979, 1991; Barratt, 1985; Dickman, 2000). Stimulus-orienting can be studied as change in amplitude of auditory evoked potentials relative to change in loudness of the stimulus (intensity–sensitivity). N1 and P2 event-related potentials form a complex that peaks between 60 and 250 ms after an auditory stimulus. Both increase in amplitude with increased loudness, expressed as intensity–amplitude slopes reflecting loudness-dependent changes in N1 and P2 amplitude. The combined N1P2 is widely used to measure intensity–sensitivity.

Intensity–sensitivity of N1P2 may have trait-like characteristics (Norra et al., 2008), and underlying mechanisms are under debate. N1P2 slope has been shown not to be influenced by acute pharmacological manipulation of serotonergic function (Norra et al., 2008; O'Neill et al., 2008). Genetic studies have suggested relationships to serotonergic 5HT-1B receptors (Juckel et al., 2008a), catechol O-methyltransferase (COMT) (Juckel et al., 2008b), Brain-derived Neurotrophic Factor (BDNF) (Park et al., 2013), and nitric oxide synthase (Kawohl et al., 2008), but not serotonin transporter (Juckel et al., 2007) regulation. These findings suggest involvement of integrated systems across neurotransmitters, and appear to be related to a chronic predisposition rather than being state-dependent (Norra et al., 2008). If that is the case, trait-like impulsivity in bipolar disorder may correlate with intensity–sensitivity.

Studies on relationships between impulsivity and intensity–sensitivity have focused mostly on the N1P2. Results have been inconsistent, with positive correlations (Barratt et al., 1987; Carrillo-de-la-Pena and Barratt, 1993; Brocke et al., 1999; Juckel et al., 1995; Herrmann et al., 2002; Norra et al., 2003) and insignificant or negative correlations (Wang et al., 1999a; Brocke et al., 1999; Tuchtenhagen et al., 2000; Carrillo-de-la-Pena and Barratt, 1993; Schwerdtfeger and Baltissen, 2002; Wang et al., 1999b). The amplitude–intensity slope of N1P2 may be lower in bipolar disorder than in controls (Park et al., 2010), whereas in healthy controls N1P2 slope correlated with hypomanic or hyperthymic traits (Hensch et al., 2007). These inconsistencies might be related to the fact that N1 and P2, which combine to form the N1P2 complex, have different functional significances.

The N1, generated in primary auditory cortex and anterior cingulate cortex (Enge et al., 2011) reflects involuntary, stimulus-driven triggering of attention (Naatanen and Picton, 1987; Rinne et al., 2006; Naatanen, 1992, pp. 113–135); P2 reflects early allocation of attention with initial conscious awareness of the stimulus (Naatanen, 1992, p. 222). This may be relevant to potential relationships between the N1 or P2 and trait impulsivity or associated characteristics.

Impulsivity and sensation-seeking are related but potentially independent traits linking stimulus and action (Barratt et al., 2004; Patton et al., 1995) across psychiatric diagnoses (Zuckerman and Kuhlman, 2000; Moeller et al., 2001; Fossati et al., 2007; Barratt et al., 2004). Trait impulsivity and sensation seeking may relate differently to neurophysiological correlates of stimulus-intensity assessed by N1 and P2. We know of only three reports investigating relationships between impulsivity or sensation-seeking and intensity–sensitivity of N1 and P2 independently. In a study combining controls and prisoners, trait impulsivity correlated positively with the visual N1 slope (Carrillo-de-la-Pena and Barratt, 1993). In studies of healthy controls that did not measure impulsivity, sensation-seeking correlated more strongly with the slope for P2 than for N1 (Brocke et al., 1999, 2000).

Therefore, trait impulsivity may reflect increased late pre-attentional orientation to intense stimuli, with increased N1 amplitude–intensity slopes with higher trait impulsivity, suggesting augmenting of the N1 in people with high trait impulsivity compared to those with lower impulsivity. Sensation-seeking may reflect early attention allocation to and initial conscious awareness of intense stimuli, with increased amplitude-sensitivity of P2 with higher venturesomeness, suggesting augmenting of the P2 in people with high sensation-seeking or venturesomeness. To address the specificity of relationships between impulsivity and pre-attentive stimulus orientation in bipolar disorder, this study focuses on N1 and P2 rather than the N1P2 complex. In addition to comparing intensity–amplitude slopes in bipolar disorder to healthy controls, we addressed the relationship between those slopes and impulsivity.

We used the Barratt Impulsiveness Scale (BIS-11) (Patton et al., 1995) and I_7 Impulsiveness scale (Eysenck et al., 1985) to assess trait impulsivity. Both measures are designed to address impulsivity independent from sensation seeking, assessed with the I_7 Venturesomeness scale (Eysenck et al., 1985; Miller et al., 2004; Luengo et al., 1991; Eysenck, 1993).

We hypothesized that (1) impulsivity is more strongly related to the N1 than to the P2 slope, whereas venturesomeness is more strongly related to the P2 slope, (2) these characteristics are stronger in bipolar disorder than in controls, reflecting the relative importance of impulsivity in the pathology of bipolar disorder, (3) relationships between intensity–amplitude slopes and impulsivity involve trait-like impulsivity rather than state-dependent impulsivity as measured by performance tests.

2. Methods

2.1. Participants

The study complied with the Declaration of Helsinki and was approved by the Committee for the Protection of Human Subjects, IRB for the University of Texas Health Science Center at Houston (protocol HSC-MS-05-0036). Subjects, recruited through print advertisements, received thorough descriptions of the study, with full opportunity for questions, and signed informed consent before research-related procedures. Inclusion criteria included: age 18–55, good hearing by self-report (corrected-to-) normal vision, and no history of traumatic brain injury or epilepsy. Structured Clinical Interviews for DSM-IV axis-I and axis-II disorders (SCID-I and SCID-II) (First et al., 1996, 1997) were administered by trained staff to establish diagnosis, confirmed by consensus of co-investigators (ACS, JLS, FGM).

Subjects were 37 healthy controls without history or known family history of Axis I or Axis II psychiatric disorder, and 39 with bipolar disorder. Among those with bipolar disorder, 36 had bipolar I disorder, 3 had bipolar II disorder, and none had bipolar disorder