



Research report

Associations of age with reward delay discounting and response inhibition in adolescents with bipolar disorders

Snežana Urošević^{a,*}, Eric A. Youngstrom^b, Paul Collins^a, Jonathan B. Jensen^c,
Monica Luciana^a^a Department of Psychology, University of Minnesota, Twin Cities, USA^b Department of Psychology, University of North Carolina, Chapel Hill, USA^c Department of Psychiatry, University of Minnesota, Twin Cities, USA

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Objectives: Bipolar disorders' (BD) onset before age 18 is a potential marker for a more severe illness course. Adolescence is also a period of significant normative maturation of inhibitory control and reward-relevant decision-making processes, such as decreased delay discounting (i.e., decreased preference for smaller, immediate versus larger, delayed rewards). Adults with BD exhibit elevated delay discounting rates. Very little is known about developmental changes in delay discounting in adolescents with BD, or about associations between inhibitory control and delay discounting in BD. The present study addresses these questions.

Methods: The sample included 78 participants, ages 13 to 23, with BD or without history of mental illness. Group differences and group by age interaction effects on delay discounting (32 BD, 32 controls with valid responses), probability discounting (34 BD, 37 controls) and inhibitory control indices (34 BD, 38 controls) were assessed.

Results: Among healthy controls, less discounting of delayed rewards was associated with older age, whereas adolescents with BD did not show age-related associations. There were no group differences in probability discounting or inhibitory control.

Limitations: The cross-sectional nature of the study cannot fully rule out the less likely interpretation of group differences in cohort effects.

Conclusions: The lack of age-related improvement in delay tolerance in BD suggests disrupted development of executive control processes within reward contexts, which in turn may contribute to understanding more severe course of pediatric onset BD. Longitudinal studies are needed to examine delay discounting in relation to maturation of neural reward systems among adolescents with BD.

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1. Introduction

Two separate constructs are proposed to explain “excessive involvement in pleasurable activities that have a high potential for painful consequences” (p. 362, DSM-IV-TR, 2000), which often accompany manic/hypomanic periods in bipolar disorders (BD). Hypersensitivity of the behavioral approach system (BAS), a system implicated in facilitating approach to rewards in the

environment (Depue and Collins, 1999), could explain manic/hypomanic reward-related risk-taking and other bipolar symptoms (Depue and Iacono, 1989; Johnson et al., 2012; Urošević et al., 2008). Alternatively, manic/hypomanic risk-taking may reflect a general failure to inhibit behavioral responses regardless of the reward context (Swann, 2010). Supporting the latter hypothesis, high levels of trait impulsivity predict prospective first-time onset of mania (Alloy et al., 2012a).

Normative adolescence is also marked by high rates of engaging in reward-related risks, like substance use, risky sexual practices, and reckless driving (Eaton et al., 2006), which are attributed to both normative changes in BAS sensitivity and inhibitory control failures (e.g., Luciana et al., 2012). Empirical studies support normative increases in BAS sensitivity (Ernst et al., 2006; Urošević et al., 2012; Wahlstrom et al., 2010). High levels of BAS activity during typical adolescence may interact with inefficiencies in prefrontal control (Luciana et al., 2012; Steinberg, 2010) to promote

Abbreviations: BAS, behavioral approach system; BD, bipolar disorders; DD, delay discounting; DLPFC, dorsolateral prefrontal cortex; OFC, orbitofrontal cortex; PD, probability discounting; PFC, prefrontal cortex; VLPFC, ventrolateral prefrontal cortex

* Correspondence to: Department of Psychology, University of Minnesota, N218 Elliott Hall, 75 East River Parkway, Minneapolis, MN 55455, USA.

Fax: +612 626 2079.

E-mail address: snezana.urosevic@gmail.com (S. Urošević).

decisions based on immediate versus future goal-attainment. Consistently, there is evidence for greater intolerance for delayed rewards (i.e., delay discounting [DD]) in healthy adolescents and increased tolerance for these delays with maturation (Olson et al., 2007; Steinberg et al., 2009). Moreover, with maturation, healthy adolescents exhibit an increased ability to inhibit prepotent responses outside of reward contexts (Hooper et al., 2004). Compared to healthy adults, healthy adolescents exhibit weaker connectivity between frontal cortical regions involved in cognitive control (e.g., right inferior frontal gyrus) and other brain regions during an inhibitory control task (Hwang et al., 2010).

DD and inhibitory control are related but separate processes. Among healthy adolescents, the general ability to inhibit prepotent responses appears to be unrelated to disadvantageous decision-making within reward contexts (Hooper et al., 2004). Still, DD in particular is associated with inhibitory control in other studies of healthy adolescents (Olson et al., 2007). A recent meta-analysis of functional neuroimaging studies indicates distinct neural networks, but with some overlap in structures, implicated in DD versus response inhibition (Wesley and Bickel, 2014). There are no studies examining whether adolescents with BD are similar to healthy adolescents in their DD behaviors, inhibitory control, or in the age-related maturation of these skills. The present study addresses these questions.

The present study's findings may be clinically significant for several reasons. Onset of BD in adolescence/childhood predicts poor prognosis—greater rates of comorbidity, suicidality, episode recurrence, and shorter euthymic periods (Lewinsohn et al., 1995; Perlis et al., 2004; Schurhoff et al., 2000; Strober et al., 1995). Regardless of onset age, adolescent age predicts poorer functioning compared to younger ages in pediatric BD (Goldstein et al., 2009). Childhood/adolescent onset of BD is also more common than previously believed (Perlis et al., 2004; Van Meter et al., 2011). A recent longitudinal community study of 3,021 individuals found the first onset of mania to peak in the teens. The first onset of hypomania has two peaks—in early childhood and adolescence, and the first onset of major depression is between ages 12 to 25 (Beesdo et al., 2009). Deviations from typical patterns of reward responding and/or behavioral inhibition in adolescence may represent early indicators of BD vulnerability and/or BD course severity.

Little work has examined reward functioning in adolescents with BD, despite growing empirical support for the BAS/reward hypersensitivity model (Alloy et al., 2012a; Alloy et al., 2012b; Alloy et al., 2008; Johnson et al., 2000; Meyer et al., 2001; Nusslock et al., 2007; Salavert et al., 2007) and overall abnormalities in reward processing (Johnson et al., 2005; Murphy et al., 2001; Pizzagalli et al., 2008) in adult BD. There are deficits in learning reward contingencies in reversal learning tasks in studies combining children and adolescents with BD (Dickstein et al., 2004; Dickstein et al., 2010; Gorrindo et al., 2005). However, two studies failed to find deficits when examining decision-making with explicit reward contingencies (Rau et al., 2008), or with variable probabilities of monetary reward (Ernst et al., 2004) in pediatric BD. Additional data are needed to fully understand reward processing abnormalities in adolescents with BD.

DD tasks provide an opportunity to examine a specific aspect of reward processing—the tendency to prefer immediate rewards that stems from an inability/unwillingness to tolerate delayed reward delivery. The preferences for immediate rewards are sub-optimal when immediacy comes at the cost of smaller rewards. In healthy adolescents, maturation-related changes in DD appear to be specific, since probability discounting (PD; preference for smaller but certain versus larger but uncertain, probabilistic rewards) does not show maturational changes (Olson et al., 2007; Scheres et al., 2006). PD predicts concurrent rates of externalizing behaviors in normative adolescence, whereas DD is related to

executive functioning indices (Olson et al., 2007). Adult studies support psychological distinctions between these two discounting types (Green and Myerson, 2004). The assessment of both PD and DD may yield important insights regarding processes that contribute to BD.

Few studies have assessed DD rates in adults vulnerable to or with BD. In a study of young adults at risk for BD, hypomania-proneness was related to a greater discounting of delayed rewards in a two-choice task and greater N100 event-related amplitudes in response to immediate versus delayed rewards (Mason et al., 2012). In another study, adults with BD exhibited greater DD in a paradigm with hypothetical rewards, with discounting rates related to measures of trait impulsivity but not to current bipolar symptoms, psychotropic medications, or executive functioning (Ahn et al., 2011). In another study, adults at high-risk for BD exhibited delay aversion in a gambling task, but no motor response inhibition deficits, compared to controls (Wessa et al., 2015). Still, there are no examinations of whether DD in particular is related to deficits in response inhibition outside of reward contexts in BD.

Based on this past research on adult BD and normative adolescence, (1) we hypothesize that adolescents with BD will exhibit greater DD rates than healthy adolescents and a non-normative lack of age-related declines in DD. (2) In order to determine the specificity of DD abnormalities, we examined group differences and age associations in discounting of probabilistic rewards. Based on data from healthy adolescents (Olson et al., 2007), it is hypothesized that adolescents with BD will exhibit greater PD than healthy adolescents, but neither group will exhibit age-related decreases. (3) In order to determine whether differences in DD and PD are due to general inhibitory failures, we examined group differences and age-related associations with behavioral inhibition using a Go/NoGo task. We also examined relationships between DD, PD, and behavioral inhibition.

2. Methods

2.1. Participants

The present sample includes 78 participants, whose age range reflects the full range of BAS-relevant neurodevelopment (Sowell et al., 1999; Urošević et al., 2012), recruited into two groups—participants with BD and participants without DSM-IV Axis I disorders. Table 1 provides the sample's demographic and clinical information. Participants were recruited from the local community (flyers; volunteer participants database) and university-affiliated clinics (referrals, mailings to eligible families). Exclusion criteria were: history of neurological disorders; current major physical conditions; birth complications; history of extended loss of consciousness/severe head injury; IQ < 70; learning disabilities/severe developmental problems; uncorrected vision/hearing problems; learning English after age 5; and left-handedness (Oldfield, 1971), due to issues related to psychophysiological measures.

DSM-IV (American Psychiatric Association, 2000) Axis I disorders were assessed with the Kiddie-SADS-Present and Lifetime Version (K-SADS-PL; Axelson et al., 2009; Kaufman et al., 1996) semi-structured interview. For minors, one interviewer interviewed a parent and another interviewed the minor. Adult participants (age ≥ 18) provided all information themselves. A clinical psychologist (SU) conducted one interview for each participant and supervised consensus meetings where symptom ratings were derived based on all available information. A pediatric BD assessment expert (EAY) reviewed a subset of BD interviews (40%) for reliability purposes. Interrater reliability for K-SADS-PL symptom assessments was excellent (weighted kappa = .87).

In the BD group, participants with Bipolar Disorder NOS

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